IPCS

INTERNATIONAL PROGRAMME ON CHEMICAL SAFETY

Environmental Health Criteria 169

Linear Alkylbenzene Sulfonates and Related Compounds

nder the joint sponsorship of the United Nations Environment Programme, ternational Labour Organisation, and the World Health Organization

WORLD HEALTH ORGANIZATION

THE ENVIRONMENTAL HEALTH CRITERIA SERIES

Acetaldehyde (No. 167, 1995)
Acetonitrile (No. 154, 1993)
Acrolein (No. 127, 1991)
Acrylamide (No. 49, 1985)
Acrylonitrile (No. 28, 1983)
Aged population, principles for evaluating the effects of chemicals (No. 144, 1992)
Aldicarb (No. 121, 1991)
Aldrin and dieldrin (No. 91, 1989)
Allethrins (No. 87, 1989)
Amitrole (No. 158, 1994)
Ammonia (No. 54, 1986)
Anticoagulant rodenticides (No. 175, 1995)
Arsenic (No. 18, 1981)
Asbestos and other natural mineral fibres (No. 53, 1986)
Barium (No. 107, 1990)
Benomyl (No. 148, 1993)
Benzene (No. 150, 1993)
Beryllium (No. 106, 1990)
Biomarkers and risk assessment: concepts and principles (No. 155, 1993)
Biotoxins, aquatic (marine and freshwater) (No. 37, 1984)
Brominated diphenylethers (No. 162, 1994)
Butanols - four isomers (No. 65, 1987)
Cadmium (No. 134, 1992)
Cadmium - environmental aspects (No. 135, 1992)
Camphechlor (No. 45, 1984)
Carbamate pesticides: a general introduction (No. 64, 1986)
Carbaryl (No. 153, 1994)
Carbendazim (No. 149, 1993)
Carbon disulfide (No. 10, 1979)
Carbon monoxide (No. 13, 1979)
Carcinogens, summary report on the evaluation of short-term *in vitro* tests (No. 47, 1985)
Carcinogens, summary report on the evaluation of short-term *in vivo* tests (No. 109, 1990)
Chlordane (No. 34, 1984)
Chlordecone (No. 43, 1984)
Chlorine and hydrogen chloride (No. 21, 1982)
Chlorobenzenes other than hexachlorobenzene (No. 128, 1991)
Chlorofluorocarbons, fully halogenated (No. 113, 1990)
Chlorofluorocarbons, partially halogenated (ethane derivatives) (No. 139, 1992) (methane derivatives) (No. 126, 1991)
Chloroform (No. 163, 1994)
Chlorophenols (No. 93, 1989)
Chromium (No. 61, 1988)
Cresols (No. 168, 1995)
Cyhalothrin (No. 99, 1990)
Cypermethrin (No. 82, 1989)
Cypermethrin, alpha- (No. 142, 1992)
DDT and its derivatives (No. 9, 1979)
DDT and its derivatives - environmental aspects (No. 83, 1989)
Deltamethrin (No. 97, 1990)
Diaminotoluenes (No. 74, 1987)
1,2 Dichloroethane (No. 62, 1987, 1st edition) (No. 176, 1995, 2nd edition)
2,4-Dichlorophenoxyacetic acid (2,4-D) (No. 29, 1984)
2,4-Dichlorophenoxyacetic acid - environmental aspects (No. 84, 1989)
1,3-Dichloropropene, 1,2-dichloropropane and mixtures (No. 146, 1993)
Dichlorvos (No. 79, 1988)
Diethylhexyl phthalate (No. 131, 1992)
Dimethoate (No. 90, 1989)
Dimethylformamide (No. 114, 1991)
Dimethyl sulfate (No. 48, 1985)
Diseases of suspected chemical etiology and their prevention, principles of studies on (No. 72, 1987)
Dithiocarbamate pesticides, ethylenethiourea, and propylenethiourea: a general introduction (No. 78, 1988)
Electromagnetic fields (No. 137, 1992)
Endosulfan (No. 40, 1984)
Endrin (No. 130, 1992)
Environmental epidemiology, guidelines on studies in (No. 27, 1983)
Epichlorohydrin (No. 33, 1984)
Ethylene oxide (No. 55, 1985)
Extremely low frequency (ELF) fields (No. 35, 1984)
Fenitrothion (No. 133, 1992)
Fenvalerate (No. 95, 1990)
Fluorine and fluorides (No. 36, 1984)

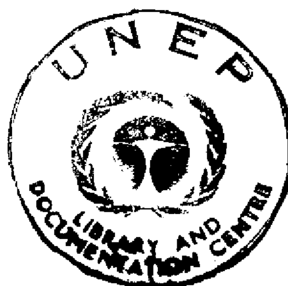
continued at end of book

This report contains the collective views of an international group of experts and does not necessarily represent the decisions or the stated policy of the United Nations Environment Programme, the International Labour Organisation, or the World Health Organization.

Environmental Health Criteria 169

LINEAR ALKYLBENZENE SULFONATES AND RELATED COMPOUNDS

First draft prepared at the National Institute of Health Sciences, Tokyo, Japan, and the Institute of Terrestrial Ecology, Monks Wood, United Kingdom



Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization

World Health Organization
Geneva, 1996

The **International Programme on Chemical Safety (IPCS)** is a joint venture of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. The main objective of the IPCS is to carry out and disseminate evaluations of the effects of chemicals on human health and the quality of the environment. Supporting activities include the development of epidemiological, experimental laboratory, and risk-assessment methods that could produce internationally comparable results, and the development of manpower in the field of toxicology. Other activities carried out by the IPCS include the development of know-how for coping with chemical accidents, coordination of laboratory testing and epidemiological studies, and promotion of research on the mechanisms of the biological action of chemicals.

WHO Library Cataloguing in Publication Data

Linear Alkylbenzene Sulfonates and Related Compounds.

(Environmental health criteria ; 169)
1.Alkane sulfonates - adverse effects 2.Environmental exposure
3.Guidelines
I.Series

ISBN 92 4 157169 1 (NLM Classification: QU 98)
ISSN 0250-863X

The World Health Organization welcomes requests for permission to reproduce or translate its publications, in part or in full. Applications and enquiries should be addressed to the Office of Publications, World Health Organization, Geneva, Switzerland, which will be glad to provide the latest information on any changes made to the text, plans for new editions, and reprints and translations already available.

©World Health Organization 1996

Publications of the World Health Organization enjoy copyright protection in accordance with the provisions of Protocol 2 of the Universal Copyright Convention. All rights reserved.

The designations employed and the presentation of the material in this publication do not imply the expression of any opinion whatsoever on the part of the Secretariat of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries.

The mention of specific companies or of certain manufacturers' products does not imply that they are endorsed or recommended by the World Health Organization in preference to others of a similar nature that are not mentioned. Errors and omissions excepted, the names of proprietary products are distinguished by initial capital letters.

PRINTED IN FINLAND
95/10757 VAMMALA 5500

CONTENTS

WHO TASK GROUP ON ENVIRONMENTAL HEALTH CRITERIA FOR LINEAR ALKYLBENZENE SULFONATES AND RELATED COMPOUNDS ... 1

NOTE TO READERS OF THE CRITERIA MONOGRAPHS ... 3

ENVIRONMENTAL HEALTH CRITERIA FOR LINEAR ALKYLBENZENE SULFONATES AND RELATED COMPOUNDS ... 4

1. OVERALL SUMMARY, EVALUATION, AND RECOMMENDATIONS ... 7
   1.1 Identity and analytical methods ... 7
   1.2 Sources of human and environmental exposure ... 8
   1.3 Environmental concentrations ... 8
      1.3.1 Linear alklylbenzene sulfonates ... 8
      1.3.2 α-Olefin sulfonates and alkyl sulfates ... 9
   1.4 Environmental transport, distribution, and transformation . 9
      1.4.1 Linear alklylbenzene sulfonates ... 9
      1.4.2 α-Olefin sulfonates ... 10
      1.4.3 Alkyl sulfates ... 10
   1.5 Kinetics ... 11
   1.6 Effects on experimental animals and *in vitro* test systems .. 11
   1.7 Effects on humans ... 12
   1.8 Environmental effects ... 13
      1.8.1 Linear alklylbenzene sulfonates ... 13
         1.8.1.1 Aquatic environment ... 13
         1.8.1.2 Terrestrial environment ... 14
         1.8.1.3 Birds ... 15
      1.8.2 α-Olefin sulfonates ... 15
         1.8.2.1 Aquatic environment ... 15
         1.8.2.2 Terrestrial environment ... 15
      1.8.3 Alkyl sulfates ... 15
         1.8.3.1 Aquatic environment ... 15
         1.8.3.2 Terrestrial environment ... 16
   1.9 Human health risk evaluation ... 16
   1.10 Evaluation of effects on the environment ... 18
   1.11 Recommendations for protection of human health and the environment ... 19
   1.12 Recommendations for further research ... 19

A. Linear alkylbenzene sulfonates and their salts. 22
A1 SUMMARY 22

A2. IDENTITY, PHYSICAL AND CHEMICAL PROPERTIES, AND ANALYTICAL METHODS 22
A2.1 Identity (sodium salt) 22
A2.2 Physical and chemical properties 23
A2.3 Analysis 27
A2.3.1 Isolation 27
A2.3.2 Analytical methods 28
A2.3.2.1 Nonspecific methods 28
A2.3.2.2 Specific methods 29

A3. SOURCES OF HUMAN AND ENVIRONMENTAL EXPOSURE ... 36
A3.1 Natural occurrence 36
A3.2 Anthropogenic sources 36
A3.2.1 Production levels and processes 36
A3.2.2 Uses 38

A4. ENVIRONMENTAL TRANSPORT, DISTRIBUTION, AND TRANSFORMATION 39
Section summary 39
A4.1 Transport and distribution between media 40
A4.1.1 Wastewater treatment 40
A4.1.2 Surface waters, sediments, and soils 41
A4.1.3 Fate models 45
A4.2 Environmental transformation 46
A4.2.1 Biodegradation 46
A4.2.1.1 Aerobic degradation 46
A4.2.1.2 Anaerobic degradation 58
A4.2.2 Abiotic degradation 59
A4.2.2.1 Photodegradation 59
A4.2.2.2 Cobalt-60 irradiation 59
A4.2.3 Bioaccumulation and biomagnification 59
A4.2.3.1 Aquatic organisms 60
A4.2.3.2 Terrestrial plants 63

A5. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE 64
Section summary 64
A5.1 Environmental levels 65
A5.1.1 Wastewater, sewage effluent, and sludge 65
A5.1.2 Sediment 70

A5.1.3 Surface water 70
A5.1.4 Soil and groundwater 85
A5.1.5 Drinking-water 85
A5.1.6 Biota 86
A5.2 Environmental processes that influence concentrations of linear alkylbenzene sulfonates 86
A5.2.1 Changes in chain length distribution during environmental removal of linear alkylbenzene sulfonates 86
A5.2.2 Specification of linear alkylbenzene sulfonates in surface waters 87
A5.3 Estimation of human intake 88

A6. KINETICS 89
Section summary 89
A6.1 Absorption, distribution, and excretion 89
A6.2 Biotransformation 92

A7. EFFECTS ON LABORATORY MAMMALS AND *IN VITRO* TEST SYSTEMS 94
Section summary 94
A7.1 Single exposures 94
A7.2 Short-term exposure 97
A7.2.1 Mouse 97
A7.2.2 Rat 97
A7.2.2.1 Administration in the diet 97
A7.2.2.2 Administration by gavage 99
A7.2.2.3 Dermal application 100
A7.2.2.4 Subcutaneous injection 101
A7.2.3 Guinea-pig 101
A7.2.4 Monkey 101
A7.3 Long-term exposure; carcinogenicity 102
A7.3.1 Mouse 102
A7.3.1.1 Administration in the diet 102
A7.3.1.2 Administration in the drinking-water ... 102
A7.3.2 Rat 107
A7.3.2.1 Administration in the diet 107
A7.3.2.2 Administration in the drinking-water ... 109
A7.3.2.3 Administration by gavage 110
A7.3.2.4 Dermal application 110
A7.4 Skin and eye irritation; sensitization 111
A7.4.1 Studies of skin 111
A7.4.2 Studies of the eye 116

A7.5 Reproductive toxicity, embryotoxicity, and teratogenicity 116
A7.6 Mutagenicity and related end-points 117
A7.6.1 Studies *in vitro* 117
A7.6.2 Studies *in vivo* 117
A7.7 Special studies 127
A7.7.1 Studies *in vitro* 127
A7.7.2 Biochemical effects 128

A8. EFFECTS ON HUMANS 129
Section summary 129
A8.1 Exposure of the general population 129
A8.2 Clinical studies 129
A8.2.1 Skin irritation and sensitization 129
A8.2.2 Effects on the epidermis 130
A8.2.3 Hand eczema 132
A8.2.4 Occupational exposure 133
A8.2.5 Accidental or suicidal ingestion 133

A9. EFFECTS ON OTHER ORGANISMS IN THE LABORATORY AND THE FIELD 135
Section summary 135
A9.1 Effect of chain length on the toxicity of linear alkylbenzene sulfonates 137
A9.2 Microorganisms 138
A9.3 Aquatic organisms 139
A9.3.1 Aquatic plants 139
A9.3.1.1 Freshwater algae and cyanobacteria 139
A9.3.1.2 Marine algae 141
A9.3.1.3 Macrophytes 141
A9.3.2 Aquatic invertebrates 141
A9.3.2.1 Acute toxicity 141
A9.3.2.2 Short-term and long-term toxicity 149
A9.3.2.3 Biochemical and physiological effects 152
A9.3.3 Fish 152
A9.3.3.1 Acute toxicity 152
A9.3.3.2 Chronic toxicity 154
A9.3.3.3 Biochemical and physiological effects 155
A9.3.3.4 Behavioural effects 166
A9.3.3.5 Interactive effects with other chemicals 166
A9.3.4 Amphibia 168
A9.3.5 Studies of the mesocosm and communities 169
A9.3.6 Field studies 172

A9.3.7 Toxicity of biodegradation intermediates and impurities of linear alkylbenzene sulfonates 173
A9.3.7.1 Individual compounds 173
A9.3.7.2 Effluents 174
A9.4 Terrestrial organisms 174
A9.4.1 Terrestrial plants 174
A9.4.2 Terrestrial invertebrates 176
A9.4.3 Birds 176

B. α-Olefin sulfonates 177
B1 SUMMARY 177

B2. IDENTITY, PHYSICAL AND CHEMICAL PROPERTIES, AND ANALYTICAL METHODS 177
B2.1 Identity 177
B2.2 Physical and chemical properties 178
B2.3 Analytical methods 178

B3. SOURCES OF HUMAN AND ENVIRONMENTAL EXPOSURE 180
B3.1 Natural occurrence 180
B3.2 Anthropogenic sources 180
B3.2.1 Production levels and processes 180
B3.2.2 Uses 181

B4. ENVIRONMENTAL TRANSPORT, DISTRIBUTION, AND TRANSFORMATION 182
Section summary 182
B4.1 Transport and distribution between media 182
B4.2 Biotransformation 182
B4.2.1 Biodegradation 192
B4.2.1.1 Aerobic biodegradation 182
B4.2.1.2 Anaerobic degradation 184
B4.2.2 Abiotic degradation 185
B4.2.3 Bioaccumulation and biomagnification 185
B4.3 Interaction with other physical, chemical, and biological factors 185

B5. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE 186

B6. KINETICS 187
Section summary 187
B6.1 Absorption, distribution, and excretion 187
B6.2 Biotransformation 188

B7. EFFECTS ON LABORATORY MAMMALS AND *IN VITRO* TEST SYSTEMS 189
Section summary 189
B7.1 Single exposures 189
B7.2 Short-term exposure 189
B7.3 Long-term exposure; carcinogenicity 189
B7.3.1 Mouse 189
B7.3.2 Rat 190
B7.4 Skin and eye irritation; sensitization 191
B7.5 Reproductive toxicity, embryotoxicity, and teratogenicity 193
B7.6 Mutagenicity and related end-points 194
B7.7 Special studies 194

B8. EFFECTS ON HUMANS 196
Section summary 196
B8.1 Exposure of the general population 196
B8.2 Clinical studies 196
B8.2.1 Skin irritation and sensitization 196
B8.2.2 Effect on the epidermis 197
B8.2.3 Hand eczema 198
B8.2.4 Accidental or suicidal ingestion 198

B9. EFFECTS ON OTHER ORGANISMS IN THE LABORATORY AND THE FIELD 199
Section summary 199
B9.1 Microorganisms 199
B9.2 Aquatic organisms 199
B9.2.1 Aquatic plants 199
B9.2.2 Aquatic invertebrates 199
B9.2.3 Fish 199
B9.3 Terrestrial organisms 200
B9.3.1 Terrestrial plants 200
B9.3.2 Terrestrial invertebrates 200
B9.3.3 Birds 200

C. Alkyl sulfates 205
C1. SUMMARY 205

C2. IDENTITY, PHYSICAL AND CHEMICAL PROPERTIES, AND ANALYTICAL METHODS 205
C2.1 Identity 205
C2.2 Physical and chemical properties 206

C2.3 Analysis ... 207
C2.3.1 Isolation ... 207
C2.3.2 Analytical methods ... 207

C3. SOURCES OF HUMAN AND ENVIRONMENTAL EXPOSURE 208
Section summary ... 208
C3.1 Natural occurrence ... 208
C3.2 Anthropogenic sources ... 208
C3.2.1 Production levels and processes ... 208
C3.2.2 Uses ... 209

C4. ENVIRONMENTAL TRANSPORT, DISTRIBUTION, AND TRANSFORMATION ... 210
Section summary ... 210
C4.1 Transport and distribution between media ... 210
C4.2 Biotransformation ... 211
C4.2.1 Biodegradation ... 211
C4.2.1.1 Biodegradation pathway; mechanism ... 211
C4.2.1.2 Biodegradation in the environment ... 211
C4.2.1.3 Anaerobic degradation ... 214
C4.2.2 Abiotic degradation ... 214
C4.2.3 Bioaccumulation and biomagnification ... 214
C4.3 Interaction with other physical, chemical, and biological factors ... 215
C4.4 Ultimate fate following use ... 216

C5. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE ... 217
Section summary ... 217
Environmental levels ... 217

C6. KINETICS ... 218
Section summary ... 218
C6.1 Absorption, distribution, and excretion ... 218
C6.2 Biotransformation ... 220

C7. EFFECTS ON LABORATORY MAMMALS AND *IN VITRO* TEST SYSTEMS ... 224
Section summary ... 224
C7.1 Single exposures ... 224
C7.2 Short-term exposure ... 226
C7.2.1 Rat ... 226
C7.2.1.1 Administration in the diet ... 226
C7.2.1.2 Administration in the drinking-water ... 230
C7.2.1.3 Dermal application ... 230

C7.2.2 Rabbit ... 231
C7.3 Long-term exposure; carcinogenicity ... 231
C7.3.1 Mouse ... 231
C7.3.2 Rat ... 232
C7.3.2.1 Administration in the diet ... 232
C7.3.2.2 Administration in the drinking-water ... 232
C7.4 Skin and eye irritation; sensitization ... 234
C7.4.1 Local irritation ... 234
C7.4.1.1 Skin ... 234
C7.4.1.2 Eye ... 235
C7.4.2 Skin sensitization ... 236
C7.5 Reproductive toxicity, embryotoxicity, and teratogenicity 236
C7.6 Mutagenicity and related end-points ... 238
C7.7 Special studies ... 238

C8. EFFECTS ON HUMANS ... 240
Section summary ... 240
C8.1 Exposure of the general population ... 240
C8.2 Clinical studies ... 240
C8.2.1 Skin irritation and sensitization ... 240
C8.2.2 Effects on the epidermis ... 242
C8.2.3 Hand eczema ... 243
C8.2.4 Accidental or suicidal ingestion ... 243

C9. EFFECTS ON OTHER ORGANISMS IN THE LABORATORY AND THE FIELD ... 244
Section summary ... 244
C9.1 Microorganisms ... 244
C9.2 Aquatic organisms ... 245
C9.2.1 Aquatic plants ... 245
C9.2.1.1 Freshwater algae ... 245
C9.2.1.2 Macrophytes ... 246
C9.2.2 Aquatic invertebrates ... 246
C9.2.3 Fish ... 247
C9.2.4 Tests in biocenoses ... 251
C9.3 Terrestrial organisms ... 251

APPENDIX I ... 253

REFERENCES ... 255

RESUME ... 295

RESUMEN ... 313

WHO TASK GROUP ON ENVIRONMENTAL HEALTH CRITERIA FOR LINEAR ALKYLBENZENE SULFONATES AND RELATED COMPOUNDS

Members

Dr R.S. Chhabra, National Institutes of Health, Institute of Environmental Health Sciences, Research Triangle Park, North Carolina, USA

Dr A. Granmo, University of Göteborg, Marine Research Station at Kristineberg, Fiskebackskil, Sweden

Ms K. Hughes, Priority Substances Section, Health and Welfare Canada, Ottawa, Ontario, Canada

Mr H. Malcolm, Institute of Terrestrial Ecology, Huntingdon, United Kingdom

Dr E. van der Plassche, Toxicology Advisory Centre, National Institute of Public Health and Environmental Protection, Bilthoven, Netherlands

Dr J. Sekizawa, Division of Information on Chemical Safety, National Institute of Hygienic Sciences, Tokyo, Japan

Ms R. Takei, Research Planning and Administration Department, Lion Corporation, Tokyo, Japan

Dr D.G. Van Ormer, Health Effects Division, Office of Pesticides Programs, Environmental Protection Agency, Washington DC, USA

Professor P.N. Viswanathan, Industrial Toxicology Research Centre, Lucknow, India

Representatives/Observers

IUTOX

Dr P. Montuschi, Department of Pharmacology, Catholic University of the Sacred Heart, Rome, Italy

CEFIC

Dr J.L. Berna, Petresa, Madrid, Spain (20–21 October)

Dr L. Cavalli, Enichem Augusta Industriale Srl, Milan, Italy (18–19 October)

IASD

Dr G. Holland, UNILEVER Ltd, Environmental Safety Laboratory, Sharnbrook, United Kingdom

Dr M. Stalmans, Procter & Gamble ETC, 100 Temselaan, Strombeek-Bever, Belgium

Secretariat

Dr H.-J. Poremski, Umweltbundesamt, Berlin, Germany (21 October)

Dr E. Smith, International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland (*Secretary*)

Dr B. Wittann, Umweltbundesamt, Berlin, Germany (18–20 October)

NOTE TO READERS OF THE CRITERIA MONOGRAPHS

Every effort has been made to present information in the criteria monographs as accurately as possible without unduly delaying their publication. In the interest of all users of the environmental health criteria monographs, readers are requested to communicate any errors that may have occurred to the Director of the International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda, which will appear in subsequent volumes.

* * *

A detailed data profile and a legal file can be obtained from the International Register of Potentially Toxic Chemicals, Case Postale 356, 1219 Châtelaine, Geneva, Switzerland (Telephone no. 979 9111).

* * *

This publication was made possible by financial support from the US Environmental Protection Agency, USA, and from the European Commission.

ENVIRONMENTAL HEALTH CRITERIA FOR LINEAR ALKYLBENZENE SULFONATES AND RELATED COMPOUNDS

A WHO Task Group on Environmental Health Criteria for Linear Alkylbenzene Sulfonates and Related Compounds met at the World Health Organization, Geneva, on 18-22 October 1993. Dr E. Smith, IPCS, welcomed the participants on behalf of Dr M. Mercier, Director of IPCS, and of the three IPCS cooperating organizations (UNEP, ILO, and WHO). The Group reviewed and revised a draft document and evaluated the risks for human health and the environment of exposure to linear alkylbenzene sulfonates, α-olefin sulfonates, and alkyl sulfonates.

The sections of the first draft on toxicology and human health were prepared at the National Institute of Health Sciences (NIHS), Tokyo, Japan, and the sections on the environment at the Institute of Terrestrial Ecology (ITE), Monks Wood, United Kingdom.

Dr E. Smith of the IPCS Central Unit was responsible for the scientific content of the monograph and Mrs E. Heseltine, St Léon-sur-Vézère, France, for the editing.

The authors who contributed to the first draft were:
Dr S. Dobson, ITE, Monks Wood, United Kingdom
Dr R. Hasegawa, NIHS, Tokyo, Japan
Dr Y. Hayashi, NIHS, Tokyo, Japan
Dr K. Hiraga, Public Health Research Laboratory, Tokyo, Japan
Dr P. Howe, ITE, Monks Wood, United Kingdom
Dr Y. Ikeda, NIHS, Tokyo, Japan
Dr Y. Kurokawa, NIHS, Tokyo, Japan
Dr H. Malcolm, ITE, Monks Wood, United Kingdom
Dr A. Matsuoka, NIHS, Tokyo, Japan
Dr K. Morimoto, NIHS, Tokyo, Japan
Dr M. Nakadate, NIHS, Tokyo, Japan
Dr K. Oba, Lion Chemical Corporation, Tokyo, Japan
Dr J. Sekizawa, NIHS, Tokyo, Japan
Dr T. Sohuni, NIHS, Tokyo, Japan
Dr M. Takahashi, NIHS, Tokyo, Japan
Dr R. Takei, Lion Chemical Corporation, Tokyo, Japan
Dr S. Tanaka, NIHS, Tokyo, Japan

Dr S. Tomiyama, Lion Chemical Corporation, Tokyo, Japan
Dr T. Yamaha, NIHS, Tokyo, Japan
Dr S. Yoshikawa, Environmental Research Institute, Kawasaki, Japan
Dr M. Wakabayashi, Water Quality Management Centre, Tokyo, Japan
Dr Y. Watanabe, Central Railway Hospital, Tokyo, Japan

Dr P. Howe, Dr H. Malcolm, and Dr J Sekizawa also contributed to the second draft.

The efforts of all who helped in the preparation and finalization of the monograph are gratefully acknowledged.

1. OVERALL SUMMARY, EVALUATION, AND RECOMMENDATIONS

1.1 Identity and analytical methods

Linear alkylbenzene sulfonates (LAS), α-olefin sulfonates (AOS), and alkyl sulfates (AS) are anionic surfactants with molecules characterized by a hydrophobic and a hydrophilic (polar) group. Commercial mixtures consist of isomers and homologues of related compounds, which differ in physicochemical properties, resulting in formulations for various applications.

LAS, AOS, and AS can be analysed by nonspecific methods. The assay usually used is one for substances that react with methylene blue, which responds to any compound containing an anionic and hydrophobic group. It thus suffers from analytical interference if used for environmental samples; furthermore, the sensitivity of this method is about 0.02 mg/litre. Although nonspecific alternatives to this method have been developed, they are not commonly used. Specific methods for environmental analysis are available only for LAS and AS. An improved method based on methylene blue reactivity and high-performance liquid chromatography (HPLC) is available for analysis of AOS.

LAS are nonvolatile compounds produced by sulfonation of linear alkylbenzene. Commercial products are always mixtures of homologues of different alkyl chain lengths (C_{10}–C_{13} or C_{14}) and isomers differing in the phenyl ring positions (2 to 5 phenyl). All of the homologues and isomers of LAS can be determined in environmental samples and other matrices by specific analytical methods such as HPLC, gas chromatography, and gas chromatography–mass spectrometry.

AOS are nonvolatile compounds produced by sulfonation of α-olefins. They are mixtures of two compounds, sodium alkene sulfonate and hydroxyalkane sulfonate, with alkyl chain lengths of C_{14}–C_{18}.

AS are nonvolatile compounds produced by sulfation of oleochemical or petrochemical alcohols. They are mixtures of homologues with alkyl chain lengths of C_{10}–C_{18}. Specific analytical methods are being developed for environmental monitoring.

1.2 Sources of human and environmental exposure

LAS, AOS, and AS are used as active ingredients in household and personal care products and in specialized applications. After use, such detergent compounds are discharged into the environment in wastewater.

There is occupational exposure to these compounds. The exposure of the general human population and of environmental organisms depends on the application of LAS, AOS, and AS (and other surfactants), on local sewage treatment practices, and on the characteristics of the receiving environment.

In 1990, worldwide consumption figures were about 2 million tonnes of LAS, 86 000 tonnes of AOS, and 289 000 tonnes of AS.

1.3 Environmental concentrations

1.3.1 *Linear alkylbenzene sulfonates*

Concentrations of LAS have been quantified by means of a specific, sensitive analytical method in almost every environmental compartment in which they might be present. The concentrations decrease progressively in the order wastewater > treated effluent > surface waters > the sea.

In areas where LAS are the predominant surfactants used, the concentrations are usually 1–10 mg/litre in wastewater, 0.05–0.1 mg/litre in effluents treated biologically, 0.05–0.6 mg/litre in effluents treated with a percolating filter, 0.005–0.05 mg/litre in surface waters below sewage outfalls (with concentrations decreasing rapidly to 0.01 mg/litre downstream of the outfall), < 1–10 mg/kg in river sediments (≤ 100 mg/kg in highly polluted sediments near discharge zones), 1–10 g/kg in sewage sludge, and < 1–5 mg/kg in sludge-amended soils (initially 5–10 mg/kg; ≤ 50 mg/kg have been reported after atypically high applications of sludge). The concentrations of LAS in estuarine waters are 0.001–0.01 mg/litre, although higher levels occur where wastewater is discharged directly. The concentrations in offshore marine waters are < 0.001–0.002 mg/litre.

It should be noted that the environmental concentrations of LAS vary widely. This variation is due to differences in analytical methods, in the characteristics of sampling sites (ranging from highly polluted

areas with inadequate sewage treatment to areas where sewage undergoes extensive treatment), in season (which can account for a difference of twofold), and in consumption of LAS.

Environmental monitoring shows that there has been no accumulation of LAS in environmental compartments over time. The concentrations in soil do not increase with time but decrease owing to mineralization. As LAS do not degrade under strictly anaerobic conditions (to generate methane), it cannot be concluded that they are mineralized in anaerobic sediments. With current use, the rate of assimilation of LAS in all receiving environmental compartments is equal to the rate of input, implying a steady state.

1.3.2 *α-Olefin sulfonates and alkyl sulfates*

Limited data are available on the concentrations of AOS in the environment owing to the difficulty of analysing them in environmental samples. Nonspecific colorimetric methods (such as that based on methylene blue) allow detection of anionic surfactants in general, but they suffer from analytical interferences and are not suitable for determining specific concentrations of AOS. A specific method is being developed for measuring AS in environmental samples.

Studies conducted in the laboratory indicate that AOS and AS are mineralized rapidly in all environmental compartments and are virtually entirely removed from sewage during treatment. The concentrations in surface water, sediments, soil, estuarine water, and the marine environment are probably low. The levels of AOS in river water have been found to be low.

1.4 Environmental transport, distribution, and transformation

At temperatures below 5–10 °C, the biodegradation kinetics of LAS, AOS, and AS is reduced because of a reduction in microbial activity.

1.4.1 *Linear alkylbenzene sulfonates*

The routes by which LAS enter the environment vary among countries, but the main route is via discharge from sewage treatment works. When wastewater treatment facilities are absent or inadequate, sewage may be discharged directly into rivers, lakes, and the sea.

Another route of entry of LAS to the environment is by the spreading of sewage sludge on agricultural land.

Throughout their passage into the environment, LAS are removed by a combination of adsorption and primary and ultimate biodegradation. LAS are adsorbed onto colloidal surfaces and onto suspended particles, with measured adsorption coefficients of 40–5200 litres/kg depending on the media and the structure of the LAS. They biodegrade in surface water (half-life, 1–2 days), aerobic sediments (1–3 days), and marine and estuarine systems (5–10 days).

During primary sewage treatment, about 25% of LAS (range, 10–40%) are adsorbed onto and removed with waste sludge. They are not removed during anaerobic sludge digestion but are removed during aerobic treatment of sludge, with a half-life of about 10 days. After application of sludge to soil, 90% of LAS are generally degraded within three months, with a half-life of 5–30 days.

The whole-body concentration factors for LAS range from 100 to 300, for the sum of ^{14}C-LAS and ^{14}C metabolites. Uptake by fish occurs mainly through the gills, with subsequent distribution to the liver and gall-bladder after biotransformation. LAS are excreted rapidly, and there is therefore no evidence that they undergo biomagnification.

1.4.2 *α-Olefin sulfonates*

Fewer data are available on the environmental transport, distribution, and transformation of AOS than for LAS. It can be inferred that AOS are transported into the environment in a manner similar to that established for LAS, AS and other detergent surfactants, and the environmental fate of AOS is similar to that of LAS and AS. It is readily biodegraded under aerobic conditions, and primary biodegradation is complete within 2–10 days, depending on the temperature. Limited data are available on the bioaccumulation of AOS; no bioaccumulation was observed in fish. There are no data on abiotic degradation.

1.4.3 *Alkyl sulfates*

AS are transported into the environment by mechanisms similar to those that operate for LAS and AOS. They are readily biodegradable under aerobic and anaerobic conditions in the laboratory and under environmental conditions; primary biodegradation is complete within 2–5 days. The whole-body bioconcentration factor ranges from 2 to 73

and varies with the chain length of alkyl sulfate homologues. AS are taken up, distributed, biotransformed, and excreted by fish in the same way as LAS and are not bioconcentrated in aquatic organisms.

1.5 Kinetics

LAS, AOS, and AS are readily absorbed by the gastrointestinal tract, widely distributed throughout the body, and extensively metabolized. LAS undergo ω- and β-oxidation. The parent compounds and metabolites are excreted mainly through the kidney, although a proportion of an absorbed dose may be excreted as metabolites in the faeces by biliary excretion. Only minimal amounts of LAS, AOS, and AS appear to be absorbed through intact skin, although prolonged contact may compromise the integrity of the epidermal barrier, thereby permitting greater absorption; high concentrations may reduce the time required for penetration.

1.6 Effects on experimental animals and *in vitro* test systems

The oral LD_{50} values for sodium salts of LAS were 404–1470 mg/kg body weight in rats and 1259–2300 mg/kg body weight in mice, suggesting that rats are more sensitive than mice to the toxicity of LAS. An oral LD_{50} of 3000 mg/kg body weight was measured for a sodium salt of AOS in mice. The oral LD_{50} values of AS in rats were 1000–4120 mg/kg body weight. LAS, AOS, and AS irritate the skin and eye.

Minimal effects, including biochemical alterations and histopathological changes in the liver, have been reported in subchronic studies in which rats were administered LAS in the diet or drinking-water at concentrations equivalent to doses greater than 120 mg/kg body weight per day. Although ultrastructural changes were observed in liver cells at lower doses in one study, the changes appeared to be reversible. Effects were not seen at similar doses in other studies, but the organs may have been examined more closely in the initial study. Reproductive effects, including decreased pregnancy rate and litter loss, have been reported in animals administered doses > 300 mg/kg per day. Histopathological and biochemical changes were observed after long-term dermal application to rats of solutions of > 5% LAS, and after 30 days' application to the skin of guinea-pigs of 60 mg/kg body weight. Repeated dermal application of ≥0.3% solutions of LAS induced fetotoxic and reproductive effects, but also induced maternal toxicity. Few data are available from studies in experimental animals that allow

evaluation of the potential effects of AOS in humans. No effects were observed in rats administered oral doses of 250 mg/kg body weight per day chronically, but fetotoxicity was seen in rabbits administered a maternally toxic dose of 300 mg/kg body weight per day. Application of AOS to the skin and eyes of experimental animals induced local effects.

Although the effects of short- and long-term exposure of animals to AS have been investigated in several studies, most suffered from inadequate histopathological examination or small group sizes; furthermore, the highest doses used in the long-term studies did not produce any toxic effects, so that an NOAEL could not be established. Effects have, however, been reported consistently in rats administered AS in the diet or drinking-water at concentrations equivalent to 200 mg/kg body weight per day or more. Local effects have been observed on the skin and eyes after topical application of concentrations of about 0.5% AS or more. Maternally toxic and fetotoxic effects have been observed at higher concentrations.

Most of the long-term studies are inadequate to evaluate the carcinogenic potential of LAS, AOS, and AS in experimental animals, owing to factors such as small numbers of animals, limited numbers of doses, absence of a maximal tolerated dose, and limited histopathological examination in the majority of studies. In those studies in which the pathological findings were adequately reported, maximal tolerated doses were not used, and the doses did not produce toxic effects. Subject to these limitations, however, the studies in which animals were administered LAS, AOS, or AS orally gave no evidence of carcinogenicity; long-term studies in which AOS was applied by skin painting studies also showed no effect.

On the basis of limited data, these compounds do not appear to be genotoxic *in vivo* or *in vitro*.

1.7 Effects on humans

The results of patch tests show that human skin can tolerate contact with solutions containing up to 1% LAS, AOS, or AS for 24 h with only mild irritation reactions. These surfactants caused delipidation of the skin surface, elution of natural moisturizing factor, denaturation of the proteins of the outer epidermal layer, and increased permeability and swelling of the outer layer. Neither LAS, AOS, nor AS induced skin sensitization in volunteers, and there is no conclusive evidence that

they induce eczema. No serious injuries or fatalities have been reported following accidental ingestion of these surfactant by humans.

1.8 Environmental effects

1.8.1 *Linear alkylbenzene sulfonates*

1.8.1.1 *Aquatic environment*

LAS have been studied extensively both in the laboratory (short- and long-term studies) and under more realistic conditions (micro- and mesocosm and field studies). In general, a decrease in alkyl chain length or greater internalization of the phenyl group is accompanied by a decrease in toxicity. Observations in fish and *Daphnia* indicate that a decrease in chain length of one unit (e.g. C_{12} to C_{11}) results in an approximately twofold decrease in toxicity.

The results of laboratory tests are as follows:

—*Microorganisms*: The results are highly variable owing to the use of a variety of test systems (e.g. inhibition of activated sludge; mixed cultures and individual species). The EC_{50} values range from 0.5 mg/litre (single species) to > 1000 mg/litre. For microorganisms, there is no linear relationship between chain length and toxicity.

—*Aquatic plants*: The results are highly species dependent. For freshwater organisms, the EC_{50} values are 10–235 mg/litre (C_{10}–C_{14}) in green algae, 5–56 mg/litre ($C_{11.1}$–C_{13}) in blue algae, 1.4–50 mg/litre ($C_{11.6}$–C_{13}) in diatoms, and 2.7–4.9 mg/litre ($C_{11.8}$) in macrophytes; marine algae appear to be even more sensitive. In algae, there is probably no linear relationship between chain length and toxicity.

—*Invertebrates*: The acute $L(E)C_{50}$ values for at least 22 freshwater species are 4.6–200 mg/litre (chain length not specified; C_{13}) for molluscs; 0.12–27 mg/litre (not specified; $C_{11.2}$–C_{18}) for crustaceans; 1.7–16 mg/litre (not specified; $C_{11.8}$) for worms, and 1.4–270 mg/litre (C_{10}–C_{15}) for insects. The chronic $L(E)C_{50}$ values are 2.2 mg/litre ($C_{11.8}$) for insects and 1.1–2.3 mg/litre ($C_{11.8}$–C_{13}) for crustaceans. The chronic no-observed-effect concentration (NOEC; based on lethality or reproductive effects) is 0.2–10 mg/litre (not specified; $C_{11.8}$) for crustaceans. Marine invertebrates appear to be more sensitive, with LC_{50} values of 1 to >100 mg/litre (almost all C_{12}) for 13 species, and NOECs of 0.025–0.4 mg/litre (not specified for all tests) for seven species tested.

—*Fish*: The acute LC_{50} values are 0.1–125 mg/litre (C_8–C_{15}) for 21 freshwater species; the chronic $L(E)C_{50}$ values are 2.4 and 11 mg/litre

(not specified; $C_{11.7}$) for two species; and the NOECs are 0.11–8.4 to 1.8 mg/litre (not specified; $C_{11.2}$–C_{13}) for two species. Again, marine fish appear to be more sensitive, with acute LC_{50} values of 0.05–7 mg/litre (not specified; $C_{11.7}$) for six species and chronic LC_{50} values of 0.01–1 mg/litre (not specified) for two species. In most of the reports, the chain length was not reported. An NOEC of < 0.02 mg/litre (C_{12}) was reported for marine species.

The average chain length of products commonly used commercially is C_{12}. Compounds of many different chain lengths have been tested in *Daphnia magna* and fish, but the length tested in other freshwater organisms has usually been $C_{11.8}$. The typical acute $L(E)C_{50}$ values for C_{12} LAS are 3–6 mg/litre in *Daphnia magna* and 2–15 mg/litre in freshwater fish, and the typical chronic NOECs are 1.2–3.2 mg/litre for *Daphnia* and 0.48–0.9 mg/litre for freshwater fish. The typical acute LC_{50} values for LAS of this chain length in marine fish are < 1–6.7 mg/litre.

Saltwater organisms, especially invertebrates, appear to be more sensitive to LAS than freshwater organisms. In invertebrates, the sequestering action of LAS on calcium may affect the availability of this ion for morphogenesis. LAS have a general effect on ion transport. Biodegradation products and by-products of LAS are 10–100 times less toxic than the parent compounds.

The results obtained under more realistic conditions are as follows: LAS have been tested in all freshwater tests at several trophic levels, including enclosures in lakes (lower organisms), model ecosystems (sediment and water systems), rivers below and above the outfall of wastewater treatment plants, and in experimental streams. C_{12} LAS were used in almost all cases. Algae appear to be more sensitive in summer than in winter, as the 3-h EC_{50} values were 0.2–8.1 mg/litre after photosynthesis, whereas in model ecosystems no effects were seen on the relative abundance of algal communities at 0.35 mg/litre. The no-effect levels in these studies were 0.24–5 mg/litre, depending on the organism and parameter tested. These results agree fairly well with those of laboratory tests.

1.8.1.2 *Terrestrial environment*

Information is available for plants and earthworms. The NOECs for seven plant species tested in nutrient solutions are < 10–20 mg/litre; that for three species tested in soils, based on growth, was 100 mg/kg (C_{10}–C_{13}). The 14-day LC_{50} for earthworms was > 1000 mg/kg.

1.8.1.3 Birds

One study of chickens treated in the diet resulted in an NOEC (based on egg quality) of > 200 mg/kg.

1.8.2 α-Olefin sulfonates

There are limited data on the effects of AOS on aquatic and terrestrial organisms.

1.8.2.1 Aquatic environment

Only the results of laboratory tests are available:

— *Algae*: EC_{50} values of > 20–65 mg/litre (C_{16}–C_{18}) have been reported for green algae.

— *Invertebrates*: LC_{50} values of 19 and 26 mg/litre (C_{16}–C_{18}) have been reported for *Daphnia*.

— *Fish*: The acute LC_{50} values are 0.3–6.8 mg/litre (C_{12}–C_{18}) for nine species of fish. On the basis of short-term studies in brown trout (*Salmo trutta*), golden orfe (*Idus melanotus*), and harlequin fish (*Rasbora heteromorpha*), it can be concluded that the toxicity of C_{14}–C_{16} compounds is about five times lower than that of C_{16}–C_{18}, with LC_{50} values (all measured concentrations) of 0.5–3.1 (C_{16}–C_{18}) and 2.5–5.0 mg/litre (C_{14}–C_{16}). Two long-term studies in rainbow trout showed that growth is the most sensitive parameter, resulting in an EC_{50} of 0.35 mg/litre. In a marine fish, the grey mullet (*Mugal cephalus*), the 96-h LC_{50} value was 0.70 mg/litre.

1.8.2.2 Terrestrial environment

One study of plants in nutrient solutions showed NOECs of 32–56 mg/litre. In a study of chickens treated in the diet, an NOEC (based on egg quality) of > 200 mg/kg was reported.

1.8.3 Alkyl sulfates

1.8.3.1 Aquatic environment

AS have been studied in short- and long-term studies and in one study under more realistic conditions. Their toxicity is again dependent on the alkyl chain length; no information was available on any difference in toxicity between linear and branched AS.

The results of the laboratory tests are as follows:

— *Microorganisms*: The EC_{50} values in a marine community were 2.1–4.1 mg/litre (C_{12}). The NOECs in *Pseudomonas putida* were 35–550 mg/litre (C_{16}–C_{18}).

— *Aquatic plants*: The EC_{50} values were > 20–65 mg/litre (C_{12}–C_{13}) in green algae and 18–43 mg/litre (C_{12}) in macrophytes. The NOECs were 14–26 mg/litre (C_{12}–C_{16}/C_{18}) in green algae.

— *Invertebrates*: The LC_{50} and EC_{50} values were 4–140 mg/litre (C_{12}/C_{15}–C_{16}/C_{18}) in freshwater species and 1.7–56 mg/litre (all C_{12}) in marine species. The chronic NOEC in *Daphnia magna* was 16.5 mg/litre (C_{16}/C_{18}) and those in marine species were 0.29–0.73 mg/litre (chain length not specified).

— *Fish*: The LC_{50} values were 0.5–5.1 mg/litre (not specified; C_{12}–C_{16}) in freshwater species and 6.4–16 mg/litre (all C_{12}) in marine species. No long-term studies were available.

It should be noted that many of these studies were carried out under static conditions. As AS are readily biodegradable, their toxicity may have been underestimated. In a 48-h study with *Oryzias latipes*, the LC_{50} values were 46, 2.5 and 0.61 mg/litre (measured concentrations) for C_{12}, C_{14}, and C_{16} compounds, respectively. This and other studies indicate that toxicity differs by a factor of five for two units of chain length. In a flow-through biocenosis study with compounds of C_{16}–C_{18}, an NOEC of 0.55 mg/litre was observed.

1.8.3.2 *Terrestrial environment*

NOEC values of > 1000 mg/kg (C_{16}–C_{18}) were reported for earthworms and turnips.

1.9 Human health risk evaluation

LAS are the most widely used surfactants in detergents and cleaning products; AOS and AS are also used in detergents and personal care products. The primary route of human exposure is, therefore, through dermal contact. Minor amounts of LAS, AOS, and AS may be ingested in drinking-water and as a result of residues on utensils and food. Although limited information is available, the daily intake of LAS via these media can be estimated to be about 5 mg/person. Occupational exposure to LAS, AOS, and AS may occur during the formulation of

various products, but no data are available on the effects in humans of chronic exposure to these compounds.

LAS, AOS, and AS can irritate the skin after repeated or prolonged dermal contact with concentrations similar to those found in undiluted products. In guinea-pigs, AOS can induce skin sensitization when the level of γ-unsaturated sultone exceeds about 10 ppm.

The available long-term studies in experimental animals are inadequate to evaluate the carcinogenic potential of LAS, AOS, and AS, owing to factors such as study design, use of small numbers of animals, testing of insufficient doses, and limited histopathological examination. In the limited studies available in which animals were administered LAS, AOS, or AS orally, there was no evidence of carcinogenicity; the results of long-term studies in which AOS were administered by skin painting were also negative. These compounds do not appear to be genotoxic *in vivo* or *in vitro*, although few studies have been reported.

Minimal effects, including biochemical alterations and histopathological changes in the liver, have been reported in subchronic studies of rats administered LAS in the diet or drinking-water at concentrations equivalent to a dose of about 120 mg/kg body weight per day, although no effects were observed in studies in which animals were exposed to higher doses for longer periods. Dermal application of LAS caused both systemic toxicity and local effects.

The average daily intake of LAS by the general population, on the basis of limited estimates of exposure via drinking-water, utensils, and food, is probably much lower (about three orders of magnitude) than the levels shown to induce minor effects in experimental animals.

The effects of AOS in humans observed in the few studies available are similar to those reported in animals exposed to LAS. As insufficient data are available to estimate the average daily intake of AOS by the general population and on the levels that induce effects in humans and animals, it is not possible to evaluate with confidence whether exposure to AOS in the environment presents a risk to human health. The levels of AOS in media to which humans may be exposed are likely to be lower than those of LAS, however, as AOS are used less.

Effects have been reported consistently in a few, limited studies in rats administered AS in the diet or drinking-water at concentrations equivalent to doses of 200 mg/kg body weight per day or more. Local

effects on the skin and eyes have been observed after repeated or prolonged topical application. The available data are insufficient to estimate the average daily intake of AS by the general population. Since AS surfactants are not used as extensively as those containing LAS, however, intake of AS is likely to be at least three orders of magnitude lower than the doses shown to induce effects in animals.

1.10 Evaluation of effects on the environment

LAS, AS, and AOS are used in large quantities and are released into the environment via wastewater. Risk assessment requires comparison of exposure concentrations with concentrations that cause no adverse effects, and this can be done for several environmental compartments. For anionic surfactants in general, the most important compartments are sewage water treatment plants, surface waters, sediment- and sludge-amended soils, and estuarine and marine environments. Both biodegradation (primary and ultimate) and adsorption occur, resulting in decreased environmental concentrations and bioavailability. Reduction in chain length and loss of the parent structure both result in compounds that are less toxic than the parent compound. It is important that these considerations be taken into account when the results of laboratory tests are compared with potential effects on the environment. Furthermore, in assessing the risk associated with environmental exposure to these three anionic compounds, comparisons should be made with the results of tests for toxicity of compounds of the same chain length.

The effects of LAS on aquatic organisms have been tested extensively. In laboratory tests in freshwater, fish appeared to be the most sensitive species; the NOEC for fathead minnow was about 0.5 mg/litre (C_{12}), and these results were confirmed in tests under more realistic conditions. Differences have been observed among phytoplankton: in acute 3-h assays on phytoplankton, the EC_{50} values were 0.2–0.1 mg/litre (C_{12}–C_{13}), whereas no effects on relative abundance were found in other tests at 0.24 mg/litre ($C_{11.8}$). Marine species appeared to be slightly more sensitive than most other taxonomic groups.

A broad range of concentrations of all three anionic compounds occurs in the environment, as shown by extensive measurements of LAS. Owing to this broad range, no generally applicable environmental risk assessment can be made for these compounds. A risk assessment must involve appropriate understanding of the exposure and effect concentrations in the ecosystem of interest.

Accurate data on exposure to AS and AOS are needed before an environmental risk assessment can be made. Models are therefore being used to assess exposure concentrations in the receiving environmental compartments. Data on the toxicity of AS and AOS to aquatic organisms, especially after chronic exposure to stable concentrations, are relatively scarce. The available data show that the toxicity of AOS and AS is similar to that of other anionic surfactants.

Saltwater organisms appear to be more sensitive than freshwater organisms to these compounds; however, their concentrations are lower in seawater, except near wastewater outlets. The fate and effects of these compounds in sewage in seawater have not been investigated in detail.

For an evaluation of the environmental safety of surfactants such as LAS, AOS, and AS, actual environmental concentrations must be compared with no-effect concentrations. Research requirements are determined not only by the intrinsic properties of a chemical but also by its pattern or trend of consumption. As these can vary considerably among geographic areas, assessment and evaluation must be carried out regionally.

1.11 Recommendations for protection of human health and the environment

1. As exposure to dusts may occur in the workplace (during processing and formulation), standard occupational hygiene practices should be used to ensure protection of workers' health.

2. The composition of formulations for consumer and industrial use should be designed to avoid hazard, particularly for formulations that are used for cleaning or laundering by hand.

3. Environmental exposure and effects should be appropriately monitored to provide early indications of any overloading of relevant environmental compartments.

1.12 Recommendations for further research

Human health

1. Since the skin is the primary route of human exposure to LAS, AOS, and AS and since no adequate long-term studies of dermal toxicity or

carcinogenicity in experimental animals are available, it is recommended that suitably designed long-term studies in which these compounds are applied dermally be conducted.

2. In view of the lack of definitive data on the genotoxicity of AOS and AS, additional studies should be performed *in vivo* and *in vitro*.

3. In view of the inadequacies of the available studies on reproductive and developmental toxicity, definitive studies should be carried out in laboratory animals to obtain data on the effects and on the effect and no-effect levels of LAS, AOS, and AS.

4. As exposure to LAS, AOS, and AS is not adequately defined, the exposure of the general population should be monitored, particularly when these surfactants are used for cleaning and laundering by hand.

5. Since LAS, AOS, and AS may enhance the transport of other chemicals in environmental media and modulate their bioavailability and toxicity in surface waters, river sediments, and soils to which humans may be exposed, interactions with other environmental chemicals and the consequences for humans should be investigated.

Environmental safety

6. Additional studies should be carried out on the mechanisms of adsorption and desorption of AOS and AS. Studies should also be done on the partitioning of LAS, AOS, and AS between dissolved and suspended colloidal particles in water. Mathematical models of sorption coefficients should be developed and validated on the basis of physical-chemical parameters.

7. Studies of the biodegradation of AOS and AS in sludge-amended soils and river sediments should be carried out when exposure occurs. Studies in river sediments (aerobic and anaerobic zones) should be performed downstream of treated and untreated wastewater and sewage outfalls.

8. Environmental concentrations of LAS, AOS, and AS should be monitored regionally and nationally in order to obtain information on exposure. Analytical methods should be developed for detecting low levels of AOS and AS in relevant environmental compartments.

9. National databases should be developed on the concentrations of LAS, AOS, and AS in wastewater and rivers and on the types, efficiency, and

location of wastewater treatment plants, in order to facilitate an assessment of the impact of discharges of these surfactants to the environment.

10. Long-term studies of the toxicity of AOS and AS to fish (freshwater and marine) and aquatic invertebrates should be conducted in order to establish the relative sensitivity of these species.

A. Linear alkylbenzene sulfonates and their salts

A1. SUMMARY

See Overall Summary, Evaluation, and Recommendations (pp. 7–21).

A2. IDENTITY, PHYSICAL AND CHEMICAL PROPERTIES, AND ANALYTICAL METHODS

A2.1 Identity (sodium salt)

Chemical formula: $C_nH_{2n-1}O_3S$ Na (*n*: 16–20) (for current commercial products)

Chemical structure:

$$CH_3(CH_2)_jCH(CH_2)_kCH_3$$
$$|$$
$$O$$
$$|$$
$$SO_3Na$$

j,*k*: integers (*j* + *k* = 7–11)

Common name: Sodium linear alkylbenzenesulfonate

Common synonyms: LAS, LAS sodium salt, linear alkylbenzenesulfonic acid sodium salt, linear dodecylbenzenesulfonic acid sodium salt, sodium straight chain alkylbenzenesulfonate

CAS Registry number: 68411-30-3 (LAS sodium salt, C10–13 alkyl)

Common trade names: Ablusol DBC, Agrilan WP, Alkasurf CA, Arylan, Atlas G-3300B, Atlox, Biosoft, Berol, Calsoft, Demelan CB-30, Elecut S-507, Elfan, Emulphor ECB, Emulsogen Brands, Gardilene, Hexaryl, Idet, Kllen, Lutopon SN, Manro, Marlopon, Marlon A, Nacconol 90 F, Nansa HS 80, Nansa Lutersit, Neopelex, Sandozin

AM, Sipex, Sulfamin, Sulframin, Surfax 495, Teepol, Tersapol, Tersaryl, Ufaryl DL 80P, Witconate (McCutcheon, 1993)

Abbreviations: LAS, LAS-Na

Specification: LAS are anionic surfactants which were introduced in the 1960s as more biodegradable replacements for highly branched alkylbenzene sulfonates. LAS are produced by sulfonation of linear alkylbenzene (LAB) with sulfur trioxide (SO_3), usually on a falling film reactor or with oleum in batch reactors. The corresponding sulfonic acid is subsequently neutralized with an alkali such as caustic soda. The hydrocarbon intermediate, LAB, is currently produced mainly by alkylation of benzene with *n*-olefins or *n*-chloroparaffins using hydrogen fluoride (HF) or aluminium chloride ($AlCl_3$) as a catalyst, and the LAS derivatives are thus generally referred to in that context (Cavalli et al., 1993a). Currently, 74% of world production of LAB is via HF and 26% via $AlCl_3$ (Berna et al., 1993a).

LAS are a mixture of homologues and phenyl positional isomers, each containing an aromatic ring sulfonated at the *para* position and attached to a linear alkyl chain of C_{10}–C_{14} (in Europe, predominantly C_{10}–C_{13}) at any position except the terminal one. The product is generally used in detergents in the form of the sodium salt.

Some of the typical characteristics of LAS, including the distribution of alkyl chain lengths and the positions of the phenyl rings in the two types of LAS used in laundry detergents, are shown in the box below. The United States Toxic Substances Control Act inventory lists LAS homologues with chain lengths up to C_{18} (Tables 1 and 2), but these products are not currently used for commercial purposes.

A2.2 Physical and chemical properties

The properties of LAS differ greatly depending on the alkyl chain length. Table 3 shows the Krafft points (temperature at which 1 g of LAS dissolve in 100 ml of water) and the relative critical micelle concentrations of the single homologues.

Typical characteristics of linear alkylbenzene sulfonates used in laundry detergents:

Appearance (commercial product):	White paste (containing water)
Average length of alkyl carbon chain:	11.8
Average relative molecular mass:	342
Unsulfonated matter:	1–2%
Alkyl chain distribution:	
C_{10}	10–15%
C_{11}	25–35%
C_{12}	25–35%
C_{13}	15–30%
C_{14}	0–5%

Phenyl ring position	LAS (LAB-HF[a])	LAS (LAB-$AlCl_3$[b])
2-phenyl	18	28
3-phenyl	16	19
4-phenyl	17	17
5-phenyl	24	18
6-phenyl	25	18

From Cavalli et al. (1993a)
[a] Hydrofluoric acid-catalysed process
[b] Aluminium chloride-catalysed process

Table 1. Mixtures of linear alkylbenzene sulfonates and their salts found in the United States Toxic Substances Control Act inventory

Generic benzene-sulfonic acid groups	CAS number	
	Acid	Salts
(C_{10-13})Alkyl-[a]		68411-30-3 (sodium salt)
(C_{10-16})Alkyl-	68584-22-5	68584-23-6 (calcium salt)
		68584-26-9 (magnesium salt)
		68584-27-0 (potassium salt)
Mono (C_{6-12})alkyl-		68608-87-7 (sodium salt)
Mono(C_{7-17})alkyl-		68953-91-3 (calcium salt)
		68953-94-6 (potassium salt)
Mono(C_{9-12})alkyl-		68953-95-7 (sodium salt)
Mono(C_{10-16})alkyl-		68910-31-6 (ammonium salt)
		68081-81-2 (sodium salt)
Mono(C_{12-18})alkyl-		68648-97-5 (potassium salt)

[a] There may be more than one alkyl substituent per benzene ring (United States Environmental Protection Agency, 1981).

Table 2. Individual linear alkylbenzene sulfonates (LAS) found in the United States Toxic Substances Control Act inventory

Parent sulfonic acid (abbreviation)	Empirical formula	CAS Registry number		
		Acids	Sodium salts	Other salts
Dodecylbenzene (C_{10} LAS)	$C_{16}H_{26}O_3S$	1322-98-1 (140-60-3)[a]	1322-98-1 (2627-06-7)[a]	
Undecylbenzene (C_{11} LAS)	$C_{17}H_{28}O_3S$	50854-94-9	27636-75-5	NH_4 salt, 61931-75-7
Dodecylbenzene (C_{12} LAS)	$C_{18}H_{30}O_3S$	27176-87-0	25155-30-0 (2211-98-5)[a] (68628-60-4)[b] (18777-54-3)[c]	Al salt, 29756-98-7; NH4 salt, 1331-61-9; Ca salt, 26264-06-2; K salt, 27177-77-1; also numerous salts with alkyl amines
Tridecylbenzene (C_{13} LAS)	$C_{19}H_{32}O_3S$	25496-01-9	26248-24-8	Also salts with alkyl amines
Tetradecylbenzene (C_{14} LAS)	$C_{20}H_{34}O_3S$	30776-59-1 (47377-10-2)[a]	28348-61-0 (1797-33-7)[a]	
Pentadecylbenzene (C_{15} LAS)	$C_{21}H_{36}O_3S$	61215-89-2		K salt, 64716-02-5
Hexadecylbenzene (C_{16} LAS)	$C_{22}H_{38}O_3S$	(16722-32-0)[a]		K salt, 64716-00-3
Heptadecylbenzene (C_{17} LAS)	$C_{23}H_{40}O_3S$	39735-13-2		

From United States Environmental Protection Agency (1981)

[a] Specifies *para* substitution

[b] Specifies *para* substitution at second position on alkyl chain

Table 3. Relationship between alkyl chain length, Krafft point, and critical micelle concentration (CMC) of linear alkylbenzene sulfonates

Alkyl chain length	Krafft point (°C)	CMC x 10^{-3}(25 °C)
10	–1	5.8
12	3	1.1
14	8	0.24
15	–	0.11
16	13	–

From Ohki & Tokiwa (1970)

The solubility of surfactants in water, defined as the concentration of dissolved molecules in equilibrium with a crystalline surfactant phase, increases with rising temperature. For surfactants, a distinct, sharp bend (break point) is observed in the solubility/temperature curve. The steep rise in solubility above the sharp bend is caused by micelle formation. The point of intersection of the solubility and critical micelle curves plotted as a function of temperature is referred to as the Krafft point, which is a triple point at which surfactant molecules coexist as monomers, micelles, and hydrated solids. The temperature corresponding to the Krafft point is called the Krafft temperature. Above the Krafft temperature and critical micelle concentration, a micellar solution is formed and higher than aqueous solubility may be obtained.

As commercial LAS are a mixture of homologues and phenyl-positional isomers, their properties may differ. Even some products with the same alkyl chain distribution (same average carbon number) have different properties, depending on the 2-phenyl isomer content. The solubility in water of commercial LAS used for detergents (average alkyl carbon length, 11.8), for example, which is important for liquid formulations, is typically about 25% at 25 °C for LAS (LAB via HF) and about 38% at 25 °C for LAS (LAB via $AlCl_3$) (Cavalli et al., 1993a).

As LAS are anionic surfactants, they lower the surface tension of water so that it can wet and penetrate fabrics more easily to loosen and remove soils and stains. Micelles, which are formed at low concentrations, solubilize oil and stains effectively (Ohki & Tokiwa, 1969). Other important properties of LAS are detergency, foaming, sensitivity to Ca and Mg ions, wetting, and surface tension, which reach their optimal values generally when the alkyl chain length is about C_{12} (Yamane et al., 1970).

A physico-chemical property often used in environmental modelling is the octanol–water partition coefficient (K_{ow}). Although it is impossible to measure the K_{ow} for surface-active compounds like LAS, it can be calculated. Roberts (1989) modified the fragment method of Leo & Hansch (1979) in order to take the branching of position into account. He thus defined a function, log (CP + 1), where CP is found by pairing off carbon atoms along the two branches up to the terminus of the shorter branch. (In the case of LAS, CP is the carbon number of the shorter of the integers j and k noted in section 2.1.) This gave the formula:

$$\log K_{ow} = ALK\text{-}1.44 \log (CP + 1),$$

where ALK is log K_{ow} calculated without a branch factor.

In order to calculate log K_{ow} for multicomponent materials like LAS, the calculated K_{ow} for each component is multiplied by the mole fraction of the corresponding component, the products are summed, and the logarithm is calculated to give log WAK (WA, weighted average).

A2.3 Analysis

A2.3.1 Isolation

A number of analytical methods are available for the determination of LAS in water, but the primary method is assay as methylene blue-active substances (MBAS). The methylene blue reaction responds to any compound containing an anionic centre and a hydrophobic centre, because such compounds tend to form an extractable ion pair when they combine with cationic dyes such as methylene blue; as only the oxidized form is blue, many positive interferences may occur. Negative interference in MBAS analysis is seen in the presence of cationic substances such as proteins and amines (Swisher, 1970, 1987). Therefore, isolation of LAS from a sample is one of the most important aspects of their analysis. Most analytical methods include appropriate procedures for isolation.

A2.3.2 *Analytical methods*

The analytical methods available for determining LAS in water include nonspecific methods, involving colorimetric, fluorimetric, and atomic adsorption techniques, and specific methods involving techniques such as high-performance liquid chromatography (HPLC), gas chromatography (GC) and GC–mass spectrometry (MS).

A2.3.2.1 *Nonspecific methods*

The simplest procedure for the determination of LAS in aqueous solution is a two-phase titration method. LAS are titrated in a mixed aqueous chloroform medium with a standard solution of a cationic reagent, such as benzethonium chloride (Hyamine 1622), and a small amount of indicator, such as a mixture of dimidium bromide and acid blue. The end-point is determined by a change in the colour of the organic solvent (ISO 2271, 1972).

The main nonspecific analytical method used is assay for MBAS, described above. Colorimetric techniques are routinely used to determine low concentrations of anionic surfactants, including LAS, in aqueous samples and have been used extensively in testing and environmental monitoring of these materials. The colorimetric methods have the same common analytical basis, that is, formation of solvent extractable compounds between the anionic surfactant and an intensely coloured cationic species. The most commonly used cationic reagent for this purpose is methylene blue (Swisher, 1970, 1987). The same principle has been used as the basis of many other procedures for the determination of anionic surfactants.

It has been shown or predicted that organic sulfates, sulfonates, carboxylates, phenols, and even simple inorganic anions such as cyanide, nitrate, thiocyanate, and sulfate can be methylene blue-reactive (Swisher, 1970, 1987). The negative interferences that can occur as a result of direct competition of other 'cationic' materials are generally considered to be less important than positive interferences, and the entities detected by the analysis are correctly referred to as MBAS.

The procedure developed by Longwell & Maniece (1955) and the improved version of Abbott (1962) are considered to be the best methods for the determination of MBAS in aqueous samples. The sensitivity of these procedures is such that levels of 0.01–0.02 mg/litre MBAS can be determined.

The MBAS response can be used as an acceptable overestimate of the synthetic anionics present in domestic wastewaters, but these materials may comprise only a small proportion of the total MBAS in surface waters (Waters & Garrigan, 1983; Matthijs & De Henau, 1987). Berna et al. (1991) found that LAS contributed 75% of the MBAS in integrated sewage and 50% in treated water. Direct methylene blue analysis of extracts derived from sludge, sediment, and soil invariably leads to highly inflated estimates of LAS (Matthijs & De Henau, 1987). Numerous attempts have been made to improve the specificity of methylene blue analysis, by using a variety of separation steps before the usual colorimetric estimation. Such indirect procedures are usually lengthy, difficult, and still susceptible to interference. A number of analytical methods for the determination of LAS involving extraction and methylene blue are summarized in Table 4.

Many other cationic dyes and metal chelates have been used as colorimetric (and fluorimetric) reagents for the determination of anionic surfactants, including LAS. Use of the cationic metal chelates has also led to the development of sensitive atomic absorption methods for indirect determination of anionic surfactants in fresh, estuarine, and marine waters. Although these alternative systems may offer some advantages over the methylene blue cation method, they cannot match the wide experience gained with methylene blue analysis. Some examples of analytical methods based on the use of alternative cationic reagents are shown in Table 5.

A2.3.2.2 Specific methods

Good progress has been made towards developing methods for the specific determination of the many homologues and phenyl-positional isomers of LAS in almost all laboratory and environmental matrices (liquid and solid) at concentrations down to micrograms per litre. High-resolution GC techniques have allowed determination of all the major components of LAS (homologues and phenyl-positional isomers) in environmental samples. Waters & Garrigan (1983) and Osburn (1986) reported improved microdesulfonation–GC procedures for the determination of LAS in both liquid and solid matrices.

Derivatization techniques offer an alternative approach to desulfonation for increasing the volatility of LAS for GC (or GC–MS) analysis (Hon-nami & Hanya, 1980a; McEvoy & Giger, 1986; Trehy et al., 1990. The GC–MS technique was also applied, after ion-pair, supercritical fluid extraction and derivatization, to five sewage sludges, and the LAS

Table 4. Analytical methods for anionic surfactants in environmental water using methylene blue and extraction

Method	Isolation method/ procedure	Limit of detection (mg/litre)	Interference	Reference
Absorption photometry	Extract LAS in water into chloroform as ion-pair with MB; measure absorption of chloroform solution at 650 nm	50–300	Urea, thiocyanate, chloride	Jones (1945)
	Extract from alkaline solution, wash with acidic MB	10–100	As above	Longwell & Maniece (1955)
	Remove impurities from MBreagent by chloroformextraction	0.1–1	As above	Abbot (1962)
	Remove MBAS by TLC	0.1–1		Oba & Yoshida (1965)
	Remove MBAS on polymer bead column			Takeshita & Yoshida (1975)
	Remove MBAS on ion exchange column	0.02		Yasuda (1980)
UV absorption photometry	Re-extract LAS into water; measure UV absorption at 222 nm	1		Uchiyama (1977)
Infra-red spectometry	Use to reduce interference from MBAS	1000		Ambe & Hanya (1972)
Gas chromatography	Convert into fluorine derivative; measure by ECD	0.02		Tsukioka & Murakami (1983)

Table 4 (contd)

Method	Isolation method/ procedure	Limit of detection (mg/litre)	Inter- ference	Reference
HPLC	Remove MB by cation exchange, HPLC	0.1		Hashimoto et al. (1976)
	Remove MB by anion exchange, HPLC	0.02		Saito et al. (1982)

LAS, linear alkylbenzene sulfonates; MB, methylene blue; MBAS, methylene blue-active substances; TLC, thin-layer chromatography; UV, ultraviolet radiation; ECD, electron capture detection; HPLC, high-performance liquid chromatography

Table 5. Analytical methods involving reagents other than methylene blue

Method	Isolation method/ procedure	Limit of detection (mg/litre)	Inter- ference	Reference
Absorption photometry	1-Methyl-4-(4-diethyl-aminophenylazo)pyrid-inium iodide; measure chloroform solution at 564 nm	0.04	Fe[III]	Higuchi et al. (1982)
	Bis[2-(5-chloro-2-pyridylazo)-5-diethyl-aminophenolato]Co [III] chloride; measure benzene solution at 560 nm	0.06		Taguchi et al. (1981); Kobayashi et al. (1986)
	Ethylviolet; measure benzene or toluene solution at 540 nm	0.01		Motomizu et al. (1982); Yamamoto & Motomizu (1987)
Atomic absorption spectrometry	Bis[2-(5-chloro-2-pyridylazo)-5-diethylaminophenolato] Co [III] chloride; measure Co by atomic absorption spectrometry	1×10^{3}	Hydro-chlorite ion	Adachi & Kobayashi (1982)

Table 5 (contd)

Method	Isolation method/ procedure	Limit of detection (mg/litre)	Interference	Reference
	Potassium dibenzo-18-crown-6; measure K	0.05	Alkali, alkaline earth metals	Nakamura et al. (1983)
	Cu[II] ethylenediamine derivatives; measure Cu	0.03×10^{-3}		Gagnon (1979); Sawada et al. (1983)
Absorption photometry	Bis(ethylenediamine)Cu; determine Cu after addition of 1-(2-pyridylazo)-2-naphthol at 560 nm	5×10^{-3}		Rama Bhat et al. (1980)
GC–MS	Extract solid phase on C_8 column; derivatize LAS with sulfonyl chloride for GC–MS	1×10^{-3}		Trehy et al. (1990)

LAS, linear alkylbenzene sulfonates; GC–MS, gas chromatography-mass spectrometry

were found to occur at 3.83–7.51 g/kg on a daily basis (Field et al., 1992). These GC procedures, however, involve extensive sample pre-treatment and depend on conversion of the isolated LAS into a suitably volatile form for GC determination; they are therefore time-consuming.

HPLC offers a more convenient means for determining homologues of LAS in all types of environmental matrices routinely. Several researchers have reported HPLC procedures for LAS which involve trace enrichment of the surfactant as the first step (Kikuchi et al., 1986; Matthijs & De Henau, 1987; Castles et al., 1989; Di Corcia et al., 1991). Takita & Oba (1985) developed a modified analytical method based on MBAS–HPLC measurement. Further HPLC methods, some requiring no sample preparation, are listed in Table 6.

Table 6. Analytical methods for linear alkylbenzene sulfonates (LAS) by specific analysees

Extraction method	Analytical conditions	Limit of detection (mg/litre)	Reference
Recover LAS on column chromatograph packed with polymer beads	Column, silica gel, mobile phase hexane:ethanol containing sulfuric acid; UV at 225 nm	0.02–0.03	Takano et al. (1975)
Extract LAS with methyl-isobutyl ketone	Column, ODS; mobile phase, ethanol:water; UV at 225 nm	0.05	Matsueda et al. (1982)
Recover LAS by ion-exchange column chromatography	Column, cyanopropyl-modified silica; mobile phase, ethanol:water; UV at 225 nm	0.04	Saito et al. (1982)
Direct analysis	Column, ODS; mobile phase, methanol: water with sodium perchlorate; fluorescence detector capable of determining alkyl homologue distribution	0.1	Nakae et al. (1980)
Extract LAS using mini-column	Column, ODS; mobile phase, acetonitrile:water with sodium perchlorate; fluorescence detector	0.1×10^{-3}	Kikuchi et al. (1986)
Concentrate LAS using mini-cartridge column connected in sequence with HPLC system	Column, ODS; mobile phase, acetonitrile:water with sodium perchlorate; fluorescence detector	3×10^{-3}	Takami et al. (1987)

Table 6 (contd)

Extraction method	Analytical conditions	Limit of detection (mg/litre)	Reference
Extract LAS with methyl-isobutyl ketone	Column, ODS; mobile phase, aceto-nitrile:water (gradient elution to sharpen peak) with sodium perchlorate; UV at 222 nm	NR	Inaba & Amano (1988)
Extract from solids with methanol on Soxhlet	Column, octyl-modified silica; mobile phase, 2-propanol:water: acetonitrile (gradient elution) with sodium perchlorate; fluorescence detector	0.8 (injected weight)	Marcomini & Giger (1987)
Two-step solid phase extraction with C2 and SAX cartridges	Column, C1 Sphesorb; mobile phase, THF:water with sodium perchlorate; fluorescence detector	7×10^{-3}	Castles et al. (1989)
Extract LAS using Carbopack B (graphitized carbon black) cartridge	Column, C8-DB (Supelco); mobile phase, methanol:water with sodium perchlorate; fluorescence detector	0.8×10^{-3}	Di Corcia et al. (1991)
Concentrate LAS on anion-exchange pre-column connected to HPLC system	Column, Wakosil 5C4; mobile phase, acetonitrile:water with sodium perchlorate; UV at 220 nm	10×10^{-3}	Yokoyama & Sato (1991)

Table 6 (contd)

Extraction method	Analytical conditions	Limit of detection (mg/litre)	Reference
Ion-pair extraction under SFE conditions using	Column, capillary gas chromatography, 20 m; mass spectrometry with	NR	Field et al. (1992)
tetralhyl-ammonium ion pair reagents, coupled with ion-pair derivatization	electron impact ionization operating in selected ion mode		
Solid-phase extraction for purification and concentration	HPLC column, Bandapat C_{18} gradient elution water:acetonitrile and 0.15 mol/litre $NaClO_n$	10×10^{-3} (water phase) 0.1 (solid phase)	Matthijs & De Henau (1987)

UV, ultraviolet spectrometry; ODS, octadecyl silica; HPLC, high-performance liquid chromatography; NR, not reported; SFE, supercritical fluid extraction

A3. SOURCES OF HUMAN AND ENVIRONMENTAL EXPOSURE

A3.1 Natural occurrence

LAS do not occur naturally.

A3.2 Anthropogenic sources

LAS are synthetic surfactants that were introduced as prime components of almost all types of household surfactant products in the early 1960s to replace alkylbenzene sulfonates (ABS), which were then in widespread use. The change-over from ABS to LAS took place gradually, starting in the United Kingdom (1960) and then spreading to Germany (1961), the United States of America (1963), Japan (1965) and to other European countries (Brenner, 1968; Husmann, 1968; Waldmeyer, 1968; Tomiyama, 1972).

After use, LAS are discharged into wastewater. As the surfactant components of the detergent products are soluble, they eventually reach raw sewage at concentrations of 1–7 mg/litre (Rapaport et al., 1987). Unlike ABS, which has a branched alkyl chain structure, LAS with a linear, straight alkyl chain structure are readily biodegradable. Their use has alleviated significant environmental hazards such as foaming and residual surfactant in water.

A3.2.1 Production levels and processes

Annual world production of surfactants, excluding soap, in 1990 was estimated to be about 7 million tonnes (Colin A. Houston & Associates, Inc., 1990; Richtler & Knaut, 1991). World consumption of LAS in 1989 was about 2.43 million tonnes, 50% of which was used in North America, western Europe, and Japan (Hewin International Inc., 1992). Worldwide consumption of LAS in 1990 was about 2 million tonnes, with the following geographical distribution: western Europe, 23%; North America, 19%, eastern Asia, 16%, South-east Asia, 12%; eastern Europe, 11%; western Asia, 7%; South America, 7%; and Africa, 5% (CEFIC, 1992). Berna et al. (1993a) reported that, in 1990, 380 000 tonnes were used in western Europe, 180 000 tonnes in eastern Europe, 110 000 in Africa, 100 000 tonnes in western Asia, 305 000

in eastern Asia, 180 000 in South-east Asia, 295 000 in North America, and 140 000 in Latin America. An additional demand of 650 000 tonnes is expected by the year 2000. The estimates for 1990 show an increase over 1987, when LAS production in the United States, Japan, and western Europe was about 1.4 million tonnes, on the basis of global demand for linear alkylbenzene (Painter & Zabel, 1988), and consumption of LAS was about 307 500 tonnes in the United States, 485 000 tonnes in western Europe, and 145 000 tonnes in Japan (Richtler & Knaut, 1988).

LAS are complex mixtures of isomers and homologues in proportions dictated by the starting materials and reaction conditions. LAS are manufactured by reacting the parent alkylbenzenes with sulfuric acid or sulfur trioxide to give the corresponding sulfonic acid, which is then neutralized to the desired salt. This is usually the sodium salt but ammonium, calcium, potassium, and triethanolamine salts are also made. The reactions are smooth and the yields nearly quantitative. Commercial LAS contain linear alkyl chains 10–14 carbons in length, with phenyl groups placed at various internal positions on the alkyl chain, with the exception of 1-phenyl (Painter & Zabel, 1988).

LAS are manufactured in an enclosed process; under normal conditions, therefore, exposure can occur only at the stage of detergent formulation, by inhalation or dermally. Dermal exposure is generally short and accidental, whereas exposure by inhalation can occur continually.

The concentration of surfactants in water from washing machines is 0.2–0.6%. LAS are estimated to represent 5–25% of the total surfactant mixture.

In Germany in 1988, when annual consumption of LAS in the western states was about 85 000 tonnes, daily consumption was 3.8 g per inhabitant per day. As consumption of drinking-water was 190 litres per inhabitant per day, the average LAS concentration in sewage was 20 mg/litre. Consumption of LAS per capita in other countries is shown in Table 7 (Huber, 1989).

Table 7. Specific consumption of linear alkylbenzene sulfonates (LAS) in various countries

Country	Water usage (litres per capita per day)	LAS usage (g per capita per day)	Reference
Germany	–	3.8	Huber (1989)
	185	2.2	Wagner (1978)
United States	560	2.6[a], 2.1[b]	Rapaport et al. (1987)
United Kingdom	208	3.5[c], 2.7[c]	Standing Technical Committee on Synthetic Detergents (1978, 1989)
Spain	–	5.6[a], 2.6[b]	Berna et al. (1989)
Japan	493	2.7	Ministry of Health and Welfare (1992); Hewin International Inc. (1992)

[a] Calculated from sales
[b] Calculated by analysis
[c] Methylene blue-active substances

A3.2.2 Uses

LAS are the most widely used surfactants in detergent and cleaning products, in both liquid and powder preparations and for household and industrial use. The amount of LAS in a product depends on several factors, including the type of application (washing-up products, light- and heavy-duty powders and liquids) and the formulation, but is usually 5–25%. Small amounts of LAS are used in non-detergent applications, but these represent less than 5% of total world consumption.

A4. ENVIRONMENTAL TRANSPORT, DISTRIBUTION, AND TRANSFORMATION

Section summary

The way in which LAS enter the environment varies between countries, but the major route is via discharge from sewage treatment works. Direct discharge of sewage to rivers, lakes, and the sea occurs when wastewater treatment facilities are absent or inadequate. Another route of entry of LAS into the environment is via disposal of sewage sludge on agricultural land.

Throughout their journey into the environment, LAS are removed by a combination of adsorption and primary or ultimate biodegradation. LAS adsorb onto colloidal surfaces and suspended particles, with measured adsorption coefficients of 40–5200 litres/kg, depending on the medium and structure of the LAS. LAS undergo primary biodegradation in all environmentally relevant compartments, such as raw sewage, sewage treatment water, surface waters, sediments, and soils. They are readily and ultimately mineralized under aerobic conditions in the laboratory and the field. They tend not to be biodegraded under methanogenic conditions or if the initial LAS concentration is so high that microbial degradation is inhibited (> 20–30 mg/litre). Typical half-lives for aerobic biodegradation of LAS are 1–8 days in river water, 1–2 days in sediments, and 5-10 days in marine systems. The rate of biodegradation depends on temperature: biodegradation is rapid between 10 and 25 °C; at lower temperatures, biodegradation kinetics are reduced, in close association with microbial activity. During primary sewage treatment, LAS are partially adsorbed onto and removed with waste sludge to an extent of about 25% (range, 10–40%). LAS are not removed during anaerobic sludge digestion but are removed during aerobic treatment with a half-life of about 10 days. Application of the sludge to soil generally results in 90% degradation within three months, with a half-life of 5–30 days.

LAS are not bioconcentrated or biomagnified in aquatic organisms. They are readily absorbed through the gills and body surface of fish and are distributed via the blood to the systemic organs. Most LAS-related compounds (parent compound and metabolites) have been detected in the gall-bladder and hepatopancreas of fish. They are usually cleared rapidly, with a half-life of two to three days.

A4.1 Transport and distribution between media

Detergent chemicals such as LAS are normally discharged after use into sewers in communal wastewater. The proportion of wastewater that is subjected to sewage treatment varies widely between countries. In most advanced countries, 50 to > 90% may be treated, whereas in less developed countries the proportion may be as little as 5–30% (Eurostat, 1991). In countries where there is no or inadequate sewage treatment, LAS are removed from the environment via adsorption and mineralization in the receiving surface waters.

Anionic surfactants such as LAS can adsorb onto the solid substrates associated with sewage, sludge, sediment, and soil; the extent of adsorption is dependent on the composition and physical nature of the solid matrix. Measured values of the adsorption constant (K_d) for LAS on a range of solid substrates were compiled by Painter & Zabel (1989), who reported K_d values of 590–1400 litres/kg for primary sludge, 660–5200 litres/kg for activated sludge, and 40–360 litres/kg for river water sediment.

A4.1.1 Wastewater treatment

Under certain conditions, up to 50% of the LAS present can be biodegraded in sewers before entering sewage treatment (Moreno et al., 1990). In large-volume batch biodegradation tests with acclimatized sludge, the MBAS levels decreased to 10% of the initial concentration within 15 days. During biodegradation, the toxicity of the test solution decreased in parallel with the reduction in MBAS. A relative enrichment of the shorter chain homologues was observed by GC analysis concurrently with the decrease in MBAS levels, indicating preferential removal of the higher homologues (Dolan & Hendricks, 1976).

The distribution and fate of LAS have been established in the course of mass balance studies at sewage treatment plants in Spain (Berna et al., 1989), Italy (Cavalli et al., 1991), Switzerland (Giger et al., 1989), Germany (De Henau et al., 1989), and the United States (Rapaport & Eckhoff, 1990; McAvoy et al., 1993). Efficient, well-operated activated sludge plants generally remove most of the LAS during aerobic treatment, and the overall removal of LAS in primary settlement and secondary aerobic treatment stages can be ≥ 98% (Berna et al., 1991). Smaller amounts of LAS were removed (77 ± 15%) in less efficient, trickling filter plants (McAvoy et al., 1993).

The main mechanism for removal of LAS during sewage treatment is biodegradation (Berna et al., 1991), but a significant fraction (on average, 20–30%) of the LAS entering sewage treatment plants may be removed on primary sewage solids and do not undergo aerobic sewage treatment (Giger et al., 1989). Instead, the sludge is digested under anaerobic conditions, and in some countries a high proportion may then be applied raw or digested to agricultural land as a source of plant nutrients (Berna et al., 1991). In Germany and the United Kingdom, 40–45% of sewage sludge is disposed of in this way (Waters et al., 1989). Since LAS do not undergo significant anaerobic biodegradation under methanogenic conditions, concentrations of 3–12 g/kg can be found on dried solids in sludge (see Section 5, Table 10). Any LAS in sludge applied to agricultural soil should then be rapidly biodegraded, since the receiving soil environment is aerobic. In Germany and the United Kingdom a typical application of digested sludge was estimated to add LAS at a rate of 7–16 mg/kg soil (Waters et al., 1989).

Adsorption can account for 15–40% of the removal of LAS from raw sewage during the primary settlement stage of treatment (Berna et al., 1989; Giger et al., 1989). Berna et al. (1989) reported that precipitation and adsorption were particularly important in removing LAS from wastewater containing high concentrations of calcium and magnesium ions.

The percentage adsorption of C_{10}, C_{11}, C_{12}, and C_{13} LAS onto activated sludge, Amazon clay, and various bacteria and algae was directly related to the chain length and phenyl position: longer homologues and more terminal phenyl isomers were adsorbed much more readily than other forms. Adsorption of LAS at a concentration of 23 mg/litre was found to be pH-dependent, with adsorption increasing as the pH decreased from 7 to 3 (Yoshimura et al., 1984a).

A4.1.2 *Surface waters, sediments, and soils*

The half-life for the removal of LAS by combined sorption and settling < 12 km below a sewage outfall in Rapid Creek, South Dakota, United States, was 0.25 days. The biodegradation half-life was 1.5 days (Rapaport & Eckhoff, 1990). The partition coefficient of LAS between natural water and sediment was reported to increase with increasing chain length and with the position of the phenyl nearer to the end of the chain. Adsorption was increased when either the concentration of suspended solids or fractional organic carbon was increased (Amano & Fukushima, 1993).

Freshwater pearl oysters are cultivated in Lake Nishinoko, Japan, which has an area of 2.8 km^2, an average depth of 1.5 m, and a residence period of 27 days. The water of the lake was found to contain total concentrations of MBAS of 0.01–0.02 mg/litre and LAS concentrations of 0.005–0.018 mg/litre. The partition coefficients of LAS (K_d) were 70 litres/kg for bottom sediment:water and 11 litres/kg for oyster:water (Sueishi et al., 1988). The authors concluded that when a river flows into a semi-enclosed lagoon, the fate of the surfactants is dominated by mass transfer between media and transformation due to degradation rather than spatial transportation.

LAS were present in Swiss soils that had been treated with sludge for 10 years; however, the application rates were six times higher than normal. The reported half-lives were 5–80 days. The authors noted that it is not entirely correct to use half-life to describe the loss of LAS from soils, because there is competition between biodegradation and sorption on and into soil particulates, and LAS may persist at very low 'threshold' levels. During the 330-day study, the levels of LAS decreased from 45 mg/kg dry soil to a residual level of 5 mg/kg (Giger et al., 1989).

A comparison of the measured concentration of LAS with detailed records on the amount of sludge applied on 51 fields in England indicated that loss of LAS exceeded 98% in fields that had not recently been sprayed with sludge; losses from fields that had been sprayed recently were calculated to be 70–99% of the estimated cumulative load. The calculated half-lives for removal of LAS from soil sprayed with sludge were 7–22 days. Examination of the distribution of homologues suggested that loss of LAS is the result of biodegradation and not leaching (Holt et al., 1989). In a study of the disappearance of LAS from sludge-amended soils at two locations, the average half-lives were 26 days when sludge was applied at a rate of 1.6 kg dry sludge per m^2 (giving a concentration of LAS of 16.4 mg/kg soil) and 33 days when sludge was applied at 5 kg wet sludge per m^2 (concentration, 52.5 mg/kg soil) (Berna et al., 1989). In another study, the half-life for LAS in soil was more than three months; there was no evidence that they accumulate in soil over time (Rapaport & Eckhoff, 1990).

When C_{13} LAS were applied to various soil (surface) types at a concentration of 0.05 mg/kg under laboratory test conditions, the half-lives were 1–5 days, with an average of two days. There was no significant variation with regard to soil type. In a second experiment, the average half-life of C_{12} LAS applied to subsurface soil was 20 days (Larson et al., 1989).

After grass, radishes, and garden beans were grown for 76 days in soil treated with ^{14}C-LAS at a rate of approximately 1.2 g/m^2, 98% of radioactive residues were recovered, with 63.6% released to the atmosphere, 26.8% found in the soil, 6.6% in plant biomass, and 0.9% leached out in percolated water. When potatoes were grown on the soil for 106 days, 97.9% of the radioactivity was recovered, and 72.3% was released to the atmosphere, 18.3% to the soil, 5.9% in plant biomass, and 1.4% leached into percolated water (Figge & Schoberl, 1989).

LAS in a plume of contaminated groundwater on Cape Cod, United States, were degraded rapidly and was found only within 0.6 km of the sewage disposal bed (Thurman et al., 1986).

Effects on the biodegradation of LAS applied at 50 mg/litre of aqueous dispersion were studied in three Japanese soil types inoculated with sewage. The rate of sludge application used in this study was not typical of that found in the environment. Primary degradation, as measured by the presence of MBAS, reached 70% within 16 days. Addition of andosol (allophane) and weathered granite (kaolin and illite) both reduced primary degradation, and 40–50% of the LAS was still present after 30 days, indicating that the rate of microbial degradation of LAS adsorbed onto soils containing large amounts of allophane and/or sesquioxides was reduced. A montmorillonite soil did not affect the rate of degradation (Inoue et al., 1978).

The behaviour of C_{10}–C_{13} LAS and C_{12} LAS at concentrations of 50 and 100 mg/litre was studied by HPLC in perfusion tests on two types of soil, a clay loam and a sandy loam. The sandy loam, with a lower content of humus and clay, adsorbed less LAS with a longer lag. During the first three days of perfusion, only adsorption occurred, 50% being adsorbed; after nine days, decomposition was observed and only 16.6% of the LAS remained; after 15 days, the LAS had almost completely disappeared (Abe & Seno, 1987).

LAS were applied at a rate of 5 g/m^2 to three soil types: loamy orthic luvisol under agricultural land, sandy acidic dystric combisol under a pine forest, and combisol irrigated with wastewater. The half-life in loamy soil was five days; 80% was degraded after 12 days, and none was detectable after 28 days. With 45 mm of precipitation, about 8% of the LAS percolated to a depth of 10–30 cm. The LAS moved significantly more slowly than radioactively labelled water. LAS were less mobile in the sandy soil, with a maximal percolation depth of 5 cm after two weeks, whereas water percolated 15 cm. The half-life in the sandy soil

was 10 days, with 80% degradation after 19 days and total degradation after 28 days. The LAS were bound to organic material in the humic litter, which probably slowed degradation and reduced mobility. In combisol irrigated with wastewater, the LAS were bound mainly in the upper 5 cm, with some percolation to 10-30 cm after an application of 180 mm of wastewater. The half-life was 12 days, and 80% was degraded within 21 days; however, there was no further degradation after 28 days, and the remaining LAS were tightly bound. Increasing the application rate to 50 g/m^2 had no effect on percolation; however, the half-life was doubled. Samples collected during the winter showed much slower degradation, with half-lives of 68–117 days. Percolation was also much deeper; the authors suggest this was due to a higher rate of precipitation and extensive evaporation (Litz et al., 1987).

In a study of the adsorption of LAS in aqueous solution onto clay grumusol and sandy regosol soils, a linear adsorption isotherm was obtained. The release of the surfactant was proportional to the initial adsorption and the soil type, suggesting ready desorption. More LAS was adsorbed by the clay soil than by the sandy soil (Acher & Yaron, 1977).

Hydroxy aluminium and iron adsorbed LAS more readily and with a much larger capacity than other soil constituents, such as organic matter, silica gel, layer silicates, and calcium carbonate (Volk & Jackson, 1968).

In a study of the adsorption of LAS applied at a concentration of 2 mg/litre to a variety of Wisconsin (United States) soils, a highly significant correlation was found between adsorption and organic matter content (including the iron and aluminium components), phosphate fixing capacity, and aluminium content. The removal of sesquioxides reduced the adsorption of LAS to zero; however treatment of montmorillonitic soils with H_2O_2 and $Na_2S_2O_4$ increased adsorption by oxidizing and removing the organic matter, indicating that montmorillonite can adsorb LAS. Treatment of soils with H_2O_2 increased adsorption because iron and aluminium were released from organic chelates (Krishna Murti et al., 1966).

Adsorption of LAS to microorganisms was found on the basis of calculated adsorption isotherms to be more important than adsorption to humic substances (Urano et al., 1984; Urano & Saito, 1985).

A4.1.3 Fate models

One model of the fate of LAS predicted the sorption coefficient to within one order of magnitude. The sorption distribution coefficient was consistently underpredicted, so that when the concentrations of LAS in interstitial and overlying water were predicted from concentrations in sediment they were overestimated. The model thus provided conservative estimates for assessing safety in aquatic media (Hand et al., 1990).

The reported concentrations of LAS in Rapid Creek, South Dakota, United States, were compared with expected concentrations generated by the quantitative water-air-sediment interaction fugacity model, which is based on physical, chemical, reactive, and transport properties and emission rates into rivers. In general, close agreement was reached: in both cases, LAS had a residence time of about two days. The authors pointed out that the results might differ if the model were applied in situations that differed hydrodynamically (Holysh et al., 1986).

A model to predict surface water concentrations of LAS in German and American rivers included the following parameters: river flow and velocity, sewage treatment plant location and type, discharge volume, and connected population. The values obtained were in general agreement with those measured. The authors also investigated a septic tank discharge at a Canadian site by applying a groundwater model, which was based on hydrogeological, biodegradation, and sorption data. The predicted and measured concentrations were in good agreement (Hennes & Rapaport, 1989).

A mathematical model was derived to explain a downstream decrease in the concentration of LAS in the Lake Teganuma estuary, Japan. The model included the adsorption coefficient, the biodegradation rate constant, and the rate of transport (diffusive and settling) flux of LAS between water and sediment. The model predictions and laboratory findings were used to confirm that biodegradation is the predominant mechanism for removal of LAS from the estuary (Amano et al., 1991). A model based on data from the Lake Biwa basin was devised to predict the fate of LAS in Japanese rivers, assuming that complete mixing occurs in any given cross-section of a river. The parameters included the cross-sectional mean concentration of LAS, time elapsed, flow velocity, longitudinal dispersion coefficient, decay due to biodegradation and sedimentation, water depth, and river width (Sueishi et al., 1988).

The measured concentrations of LAS in United States river water under critical flow conditions were mirrored by the predictions of a simple dilution model, which predicts chemical concentrations below the mixing zone of wastewater treatment plants. The model is based on three large databases, which link river flow, treatment type and wastewater discharge volume; the output of the model is a frequency distribution of concentrations just below the mixing zone of treatment plant outfalls. The model predicted that 95% of the river waters below that point would have concentrations of LAS of < 0.35 mg/litre during critical low-flow periods. The sampling sites selected for this study were reported to have a low dilution factor for mixing effluent with surface water, however. The predictions therefore represent a 'worst-case scenario', since the 95 percentile value represents critical low-flow periods, in which the lowest ever recorded flow is used for a consecutive period of seven days within 10 years (McAvoy et al., 1993).

A4.2 Environmental transformation

A4.2.1 Biodegradation

A4.2.1.1 Aerobic degradation

Studies on aerobic biodegradation of LAS can be divided into those of primary degradation and those of ultimate degradation. Primary degradation of LAS occurs during the initial reactions in the metabolic pathway, and the products are often shorter-chain homologues. The ultimate degradation of LAS is that of the entire molecule to its biodegradation end-products, CO_2, H_2O, and NH_4. These products are used in cell synthesis or, in the case of CO_2, excreted. The ultimate degradation of LAS normally requires the action of several species of bacteria.

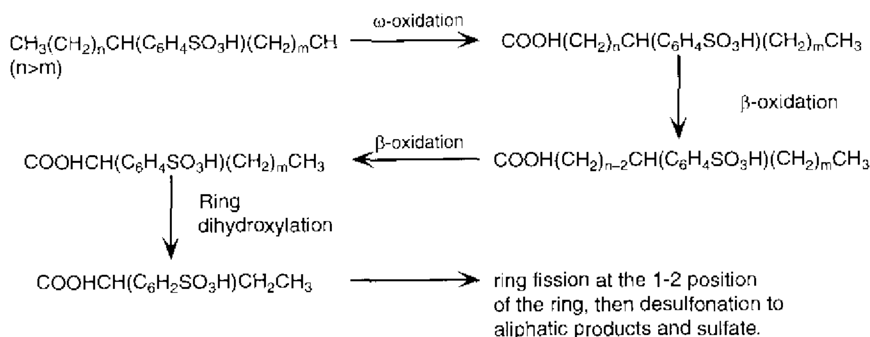
The degradation pathway of LAS has been described (Huddleston & Allred, 1963; Swisher, 1963). The steps, shown in Figure 1, are: ω-oxidation of the end of the alkyl chain, rapid β-oxidation of the chain, and oxidation of the ring.

Swisher (1981) pointed out that ultimate biodegradation (at least 80%) is achievable under the correct conditions, which include:

(i) the presence of mixed bacterial species,

(ii) free access to new bacteria during the test,

Figure 1. Postulated metabolic pathway of linear alkylbenzene sulfonates



From Painter (1992)

(iii) acclimatization,
(iv) enough growth factors and food, and
(v) limitation of the LAS concentration to that found in the environment.

Biodegradation of LAS begins at the terminus of the alkyl chain with an ω-oxidation and is followed by successive cleavage of C_2 fragments (β-oxidation) (Huddleston & Allred, 1963; Swisher, 1963). The resulting sulfocarboxylic acids have a chain length of four to five carbon atoms (Schöberl, 1989). These intermediates are further biodegraded by oxidative scission of the aromatic ring and cleavage of the sulfonate group (Setzkorn & Huddleston, 1965; Swisher, 1967). Catabolites of further oxidation steps are fed into the central metabolic pathways, i.e. the Krebs cycle and glyoxylate cycle (Schöberl, 1989).

LAS degradation begins at the longest end of the linear alkyl chain, with ω- and β-oxidation, and proceeds up to the sulfophenylmono-carboxylic acids (one to two CH_2 groups) (Divo & Cardini, 1980). Under mild conditions, as in river water, intermediates such as sulfo-phenylcarboxylic acids are often not degraded, as the greater distance between sulfophenyl groups and the far end of the hydrophobic group increases the speed of primary biodegradation (Swisher, 1976). Once the terminal methyl group has been attacked, primary biodegradation is rapid (Swisher, 1970; Gledhill, 1975). Short-chain sulfophenylmonocarboxylic acids were not degraded by *Pseudomonas* but were degraded by mixed cultures of microorganisms (Leidner et al.,

1976). The initial attack that opens the aromatic ring is the rate limiting step for ultimate biodegradation: once the ring is opened, degradation is rapid.

Enzymological methods were used to show that the same sequence of steps occurs when ring degradation proceeds via the catechol derivative. A variety of microorganisms isolated from soil, sewage, and river water showed at least five distinct metabolic routes for the degradation of LAS: ω- and β-oxidation of the side-chain; oxidation and desulfonation followed by cleavage of the aromatic nucleus; reductive desulfonation of the ring; and metabolic α-oxidation of the side-chain, followed by β-oxidation and desulfonation. Metabolism of alkyl chains shorter than four carbons was initiated through the aromatic nucleus by hydrolytic or reductive desulfonation of the ring (Cain et al., 1971). LAS may also be cleaved by biochemical mechanisms (Schöberl, 1989).

Primary degradation

(i) Low levels of biomass

Measurement of MBAS was compared with measurement of total organic carbon for detecting biodegradation in shake cultures. With the MBAS method, LAS had lost 98% of their activity within five days, whereas 34% of the total carbon had disappeared by that time, and 70% was lost by the end of the 31-day test (Sekiguchi et al., 1975a).

In a modification of the screening test of the Organisation for Economic Co-operation and Development (OECD), accepted by the European Commission, the percentage of dissolved organic carbon was found to have decreased by more than 80% within four weeks. The authors cautioned that the decrease in LAS may not have been due solely to biological degradation, since 40–50% of organic carbon was also removed from abiotic controls, suggesting that adsorption may account for part of the removal of LAS (Canton & Slooff, 1982). When aerobic biodegradation of 10 mg/litre LAS was followed during a 10-day incubation period at 27 °C, primary degradation, measured by the MBAS method, was complete within 8–10 days, and the theoretical CO_2 production reached 20–25% within 10 days. At a concentration of 20 mg/litre, no degradation was observed, but this elevated concentration may have inhibited the microbial inoculum (Itoh et al., 1979).

The rate and degree of biodegradation of LAS are dependent on temperature. In an unacclimatized microbial population, no more than

25% biodegradation was achieved at 5 °C during a 28-day test, whereas at 15, 25, and 35 °C about 90% degradation was achieved within 7–14 days. At 45 °C, the microbial population degraded 75% of the LAS within 14 days, but this rate of degradation was not maintained, probably because of loss of the acclimatized seed due to the high temperature. A clearer effect of temperature was observed when the microorganisms were acclimatized to LAS before the test. Under these conditions, the rate of biodegradation increased steadily with increasing temperature from 15 to 35 °C (Hollis et al., 1976).

(ii) Wastewater treatment

In the OECD screening test, there was 95% loss of LAS, measured by the MBAS method, and similar losses were measured in OECD confirmatory test No. 1 with 20 mg/litre LAS. In the closed-bottle test with a concentration of LAS of 2 mg/litre, there was 90–95% analytical loss (by the MBAS method) and 60–65% loss of biochemical oxygen demand. Coupled-unit tests with 10 mg/litre LAS and a mean hydraulic retention time of 6 h showed 94% removal of chemical oxygen demand (values > 73% indicate benzene ring opening) (Fischer & Gerike, 1975). In activated sludge, 80–90% of dissolved organic carbon and benzene rings disappeared within 6 h (Swisher, 1972). A bacterium similar to *Klebsiella pneumoniae*, isolated from sewage, degraded 93% of a concentration of LAS reported as 1% (10 g/litre), as measured by the MBAS method (Hong et al., 1984). A direct correlation was found between the rate of primary degradation of 1.5 mg/litre $C_{11.7}$ LAS and the initial bacterial population size (Yediler et al., 1989).

The biodegradation of C_9–C_{13} LAS at concentrations of 25, 50, and 65 mg/litre was monitored in activated sludge at 100 mg/litre over a period of 12 days. Four methods were used: MBAS, chemical oxygen demand, dissolved organic carbon, and ultra-violet spectrophotometry. The results obtained with the MBAS method showed a percentage loss of 94–97% for the three concentrations of LAS, whereas the other methods showed losses of ~ 50% at 25 mg/litre LAS and ~ 70% at 50 and 65 mg/litre. The specific rate of biodegradation was calculated to be 3.6 mg/g per h, on the basis of loss of chemical oxygen demand (Pitter & Fuka, 1979).

The degradation ratio (biochemical oxygen demand:total oxygen demand) for LAS by a synthetic sewage solution after five days was 0.81 for a concentration of 3 mg/litre and 0.14 for 10 mg/litre. Concentrations of 30 and 100 mg/litre LAS were not degraded during the 14-day test.

Even after acclimatization to a concentration of 5 mg/litre LAS for one month, the two higher concentrations were not degraded, probably because these levels inhibited the microbial inoculum (Urano & Saito, 1985).

The percentage removal of biochemical oxygen demand and of LAS were found to be significantly correlated in activated sludge and in a trickling filter system under laboratory and field conditions, implying that a well-functioning sewage treatment plant effectively removes LAS (Tang, 1974).

LAS at a concentration of 150 mg/litre were inoculated into sewage water collected from French water treatment plants. In six out of eight experiments, primary degradation was almost complete (90%) within seven days, but in the other two experiments only 45–55% degradation was achieved. The authors concluded that rapid biodegradation of LAS requires the presence of a community of several bacterial species, including *Flavobacterium*, *Pseudomonas*, and *Acinetobacter* (Gard-Terech & Palla, 1986).

In an extended aeration activated sludge plant, 95–99% of LAS was removed. Degradation of LAS and reduction of biochemical oxygen demand were strongly correlated, in a 1:1 ratio (Knopp et al., 1965). In long-term laboratory tests, 95–97% of LAS was removed by activated sludge (Janicke & Hilge, 1979).

In a wastewater treatment plant where the input water had an MBAS concentration of 6.2–9.4 mg/litre, at least 99% of the LAS present was removed during treatment, biodegradation accounting for 85%. The relative composition of long-chain (C_{12}–C_{13}) homologues adsorbed on the suspended solids was increased in comparison with the relative incidence of short-chain (C_{10}–C_{11}) homologues detected in the aqueous phases. Sulfophenylcarboxylates were identified as intermediates of the biodegradation of LAS but were detected only in the aqueous and not in the adsorbed phases (Cavalli et al., 1993b).

Biodegradation of LAS in field trials with trickling filter sewage was 86–95%, and average biochemical oxygen demand removal was 93.8%. Thus, the LAS appeared to be removed almost as rapidly as the naturally occurring organic material. The linear correlation between degradation and temperature (7.5–17.5 °C) was highly significant. Further degradation (94–99%) took place after additional aeration (Mann & Reid, 1971).

MBAS degradation did not correspond to biodegradation of LAS (20–200 mg/litre) in laboratory sludge units, because of the presence of intermediates not accounted for by analysis of MBAS (Janicke, 1971).

(iii) Surface waters

Primary degradation, measured by HPLC, of 5 mg/litre C_{11} LAS in a static lake microcosm was complete within 18 days. The sulfophenylcarboxylic acid intermediates produced were completely degraded within 22 days (Eggert et al., 1979).

Aerobic degradation of 5 mg/litre LAS in river water, measured by MBAS levels, was 100% after seven days at 25 °C. Under microaerophilic conditions at 25 and 35 °C), no degradation took place within 10 days (Maurer et al., 1971; Cordon et al., 1972).

In die-away tests with water from various sites on the Tama River, Japan, primary biodegradation (measured by the MBAS method) was complete within 7–15 days, but total organic carbon was completely removed within an incubation time of 45 days. In a study of LAS in seawater collected from the mouth of the Tama River, degradation was only 50% complete within 60 days, as measured by total organic carbon (Sekiguchi et al., 1975b). In a study to monitor detergent-degrading bacteria from the Han River, Republic of Korea, the lowest density was found in January and the highest in July; the dominant group throughout the year was *Pseudomonas* (Bae et al., 1982). Mixed and pure isomers of LAS were metabolized readily (97.5%) by bacteria collected during the summer from a sewage lagoon, but bacteria collected from under the ice during the winter were not able to metabolize LAS (Halvorson & Ishaque, 1969).

Primary biodegradation of C_{10}–C_{13} LAS was dependent on incubation temperature in die-away tests with water from the Tama River, Japan: primary biodegradation was complete within two days at 27 °C, within six days at 15 °C, and within three days at 21 °C; at a water temperature of 10 °C, however, only 20% of the LAS had been degraded within the nine-day test (Kikuchi, 1985). The optimal temperature for the biodegradation of LAS in a river water die-away test was found to be 25 °C (Yoshimura et al., 1984b).

Degradation of 10 mg/litre LAS in a simulated river model was found to be almost complete within 20 days, on the basis of MBAS levels in water and sludge; however, ultra-violet spectrophotometry showed

that 40% of the LAS remained in the water and 25% in the sludge. LAS with an alkyl chain length of C_{10} were degraded more slowly than those with a chain length of C_{14}, and LAS compounds with sulfylphenyl groups near the terminal part of the alkyl chains were degraded more easily than those with such groups further from the end (Fujiwara et al., 1975).

In a study of the biodegradation of LAS (10 mg/litre) and a 1:1 LAS:ABS (10 mg/litre) mixture in canal water with an unaerated or aerated system, LAS were rapidly degraded in the unaerated system, by 14.9% within two days and 40.7% within seven days. Biodegradation was more rapid in the aerated tanks, with 40.4% degraded within two days and 74.5% after seven days. Addition of sewage to the test system further increased the rate of degradation in the aerated system: addition of 0.5 ml/litre sewage resulted in degradation of 78.2% after two days and 89.4% after seven days, and addition of 1.0 ml/litre sewage resulted in degradation of 89.7% after two days and 99.8% within three days. No results were reported for the unaerated system. The LAS–ABS mixture was degraded more slowly than pure LAS: after two days, 12.3% was degraded without aeration, 36.4% with aeration, 60.1% with addition of 0.5 ml/litre sewage, and 78.3% after addition of 1.0 ml/litre sewage. The corresponding degradations calculated after seven days were 32.5, 66.0, 80.7, and 87.3%. The authors concluded that degradation of these detergents was increased by aerating the tank and by increasing the number of microflora by adding sewage (Abdel-Shafy et al., 1988).

In the Lake Teganuma estuary (Japan), an average of 66% of LAS is removed, with seasonal variability, ranging from 28% in winter to 100% in summer. Laboratory studies (based on HPLC methods) of estuarine water showed that LAS degraded with a half-life of eight days at 5 °C and 0.2 days at 25 °C. Model calculations and field monitoring showed that biodegradation is 10 times more important in the removal of LAS from the estuary during summer than is the settling of solids or adsorption to bottom sediments. At lower temperatures, biodegradation and the other removal mechanisms are of equal importance (Amano et al., 1991).

In well water, biodegradation of all LAS homologues (C_{10}–C_{13}) and isomers (maximal concentration, 2.5 mg/litre) after an acclimatization period of one day was reported to follow zero-order kinetics (Yakabe et al., 1992).

In seawater, primary biodegradation of 20 mg/litre LAS was 70% after 10 days; the half-life was six to nine days (Vives-Rego et al., 1987).

(iv) Soil

In soil degradation tests, levels of 2.5 mg/kg MBAS were reached within 15 days of the addition of 20 mg/kg LAS (Cordon et al., 1972). The biodegradation of LAS in soil was studied by measuring the amounts of ferroin reagent-active substance and total organic carbon. At 50 mg/litre LAS, total organic carbon disappeared within 50 days, whereas total ferroin reagent-active substance was completely lost after only 10 days. Both chemical and physical properties of the soils affected the loss of LAS: more LAS was adsorbed onto clay loam than sandy loam, and biodegradation occurred more readily in the clay loam (Abe, 1984). In a further study (initial concentration not given), loss of C_{10}–C_{13} and C_{12} LAS was complete within 15 days when measured as ferroin reagent-active substances; however, when measured as total organic carbon, residues remained until day 50 in the clay loam and beyond day 60 in the sandy loam (Abe & Seno, 1987).

Ultimate degradation

A number of studies have been conducted of the biodegradation of phenyl-radiolabelled LAS, in which $^{14}CO_2$ production was measured.

(i) Screening tests

In a simple shake-flask system with LAS, CO_2 evolution reached 60% or more of the theoretical value (Gledhill, 1975).

Four gram-negative bacteria synergistically mineralized 10 mg/litre ^{14}C-LAS. After 13 days of incubation, 29% of the ^{14}C-LAS was mineralized to $^{14}CO_2$. Pure cultures were unable to mineralize the LAS, although three of them carried out primary biodegradation, measured by the MBAS method (Jimenez et al., 1991). *Pseudomonas, Alcaligenes, Necromonas,* and *Moraxella* spp. isolated from activated sludge and river water degraded the alkyl chains of C_{12} LAS, while a group of unidentified Gram-negative bacteria cleaved the benzene ring. A mixture of the two groups degraded LAS completely (Yoshimura et al., 1984b).

(ii) Wastewater treatment

Mixed cultures of microorganisms found under natural conditions or in sewage treatment plants can readily degrade LAS, to 95% of MBAS and > 80% of dissolved organic carbon (Schöberl, 1989).

During a 19-day OECD screening test for the biodegradation of ^{14}C-LAS, there was a high degree of ring mineralization, as seen by the evolution of 55% as $^{14}CO_2$. In a continuous system, 80% of the LAS was evolved as CO_2, with a mean retention time of 3 h; 2–3% remained as unaltered surfactant and 15–25% as the sulfophenylcarboxylic acid intermediates (Steber, 1979).

Loss of MBAS (primary biodegradation) and ring cleavage were found to be nearly complete (> 98%) during simulated waste treatment of ^{14}C-LAS. During simulated secondary waste treatment, 62% of alkyl and ring carbon was converted to CO_2, 28–30% was assimilated into biomass, and 8–10% remained as soluble residue. In die-away tests, 85–100% of the substrates of LAS were converted to CO_2 within 91 days (Nielsen et al., 1980; Nielsen & Huddleston, 1981).

Continuous-flow experiments were conducted with mixed bacterial cultures isolated from a detergent plant wastewater containing five species of *Achromobacter* and two species of *Acinetobacter*. All were more efficient at primary degradation than ultimate degradation of LAS at concentrations of 20 and 50 mg/litre. One species of each genus could effect primary degradation even after isolation (Hrsak et al., 1982).

In a semi-continuous activated sludge method, 95% of the phenyl ring of radiolabelled LAS was cleaved, indicating near complete biodegradation of the whole molecule. Complete primary degradation (MBAS method) of C_{10}, C_{12}, and C_{14} LAS was followed by 99–100% ultimate degradation (HPLC and ultra-violet fluorescence). In die-away tests with 10 mg/litre of C_{10}, C_{12}, and C_{14} LAS, primary degradation was rapid and complete; 100% of C_{12} LAS was removed within four days. Almost complete ultimate degradation was observed within the 80-day test, with 90% ring cleavage of C_{10} LAS and C_{11} LAS within 10 days and 70% ring cleavage of C_{14} LAS within 30 days; however, no HPLC analysis was carried out on C_{14} LAS after day 30 (Huddleston & Nielsen, 1979).

The biodegradation of LAS (C_9–C_{14}) by a mixed bacterial culture was studied in river water, forest soil, and wastewater from a detergent

plant. The bacteria were acclimatized to 10 mg/litre LAS. Under continuous-flow conditions, LAS at a concentration of 20.8 mg/litre were 96% degraded, and a concentration of 46 mg/litre was 64% degraded. Only 8–10% of the breakdown products were completely mineralized; however, under the flow-through conditions of this test, water-soluble compounds were usually removed via the aqueous effluent and were not present long enough to allow mineralization. Acclimatization considerably increased the kinetics of mineralization (Hrsak et al., 1981).

(iii) Surface water and sediment

Detritus is a significant site of surfactant removal, and LAS were found to be the most sorptive of the surfactants tested. In wastewater from a pond containing submerged oak leaves, degradation followed first-order kinetics, with a half-life of 12.6 days. LAS were mineralized more slowly by leaves from a control pond, and an S-shaped pattern of degradation was seen (Federle & Ventullo, 1990).

In river water in which the biomass levels were 10–100 times higher below than above a sewage outfall, primary degradation of added $C_{11.6}$ LAS (11 mg/litre) and background LAS (0.37 mg/litre) was rapid in water taken from below the outfall, with a half-life of 0.23 days (based on measurements of MBAS). Mineralization of the benzene ring was rapid in water from below the outfall containing sediment (500 mg/litre), with a half-life of 0.7 days. Water taken above the sewage outfall also underwent ring mineralization, but the rate of degradation was about 25% of that seen for water from below the outfall, with a half-life of 2.7 days. When samples were incubated in the absence of sediment, ring degradation was much slower, with half-lives of 1.4 days in water taken from below the outfall and ~14 days in water taken above it. In all cases, degradation was immediate in water taken below the outfall, but occurred after a three-day lag in water taken above (Larson & Payne, 1981).

Degradation of C_{10}–C_{14} homologues of LAS at concentrations of 10 or 100 µg/litre followed first-order kinetics in both river water and river water plus sediment; the half-time for mineralization of the benzene ring was 15–33 h. The length of the alkyl chain and the phenyl position had no significant effect, and there was no effect of suspended sediment or competing homologues (Larson, 1990).

LAS were degraded in leaf litter, creek water, periphyton, and sediment at temperatures as low as 4 °C, with half-lives of 6–11 days. Temperature changes altered the dependence of the biodegradation of LAS: the half-lives increased by less than a factor of two over an 18 °C temperature range. Under realistic conditions, temperature had less effect than was predicted on the basis of classical thermodynamic studies in the laboratory (Palmisano et al., 1991). The dependence of the biodegradation of LAS follows a classical Arrhenius relationship down to about 12 °C, with a tenfold increase in reaction kinetics for every 2 °C drop in temperature (Larson, 1990).

Mineralization of LAS in saturated subsurface sediment from a wastewater pond and in a pristine pond was monitored by amending the sediment with ^{14}C-LAS and measuring the evolution of $^{14}CO_2$. Mineralization in both sediments exhibited first-order kinetics. LAS were mineralized without a lag in wastewater sediment, with half-lives of 3.2–16.5 days. In the control pond, LAS were mineralized much more slowly, with half-lives of 5.2–1540 days, and only after a lag of 2–40 days; the lag tended to increase with increasing depth. These findings confirm the assumption that acclimatization considerably increases the kinetics of LAS mineralization (Federle & Pastwa, 1988).

A study was conducted of the biodegradation of LAS by microorganisms associated with the roots of two aquatic plants, duckweed (*Lemna minor*) and cattail (*Typha latifola*). Microorganisms from the roots of cattail mineralized ^{14}C-LAS without a lag, attaining 17% evolution of $^{14}CO_2$ within the 35-day experiment. Microbiota associated with duckweed roots did not mineralize LAS. The fact that the plants came from a pristine pond or from a wastewater pond had no effect on the ability of the microorganisms to mineralize LAS (Federle & Schwab, 1989).

More than 70% of parent LAS (20 mg/litre) in natural seawater at 22 °C was biodegraded within 10 days, with an estimated half-life of 6–9 days (Vives-Rego et al., 1987). In an investigation of the primary biodegradation kinetics of LAS (10 mg/litre) in natural seawater in the presence of sediments (250 g/litre), 60% remained after 20 days at 15 °C and almost 100% of LAS at 5 °C; however, at 20 and 25 °C, only a small percentage of the original concentration remained (Sales et al., 1987). In another study in seawater, 97% of parent LAS (10 mg/litre) was biodegraded within two weeks (von Bock & Mann, 1971).

More than 85% of LAS ($C_{11.8}$) in estuarine water underwent primary biodegradation, measured as MBAS removal, after 11 days (Arthur D.

Little Inc., 1991). In water from Chesapeake Bay, United States, 75% of MBAS were removed within three days (Cook & Goldman, 1974). In a study of effluent-exposed estuarine waters, with phenyl-radiolabelled C_{13} LAS, production of $^{14}CO_2$ represented 42% of the added label. Addition of sediment from the site (1 g/litre) increased the $^{14}CO_2$ yield to 60%. In both tests, the half-life for mineralization of LAS was about seven days. Up to 54% of a radiolabelled control chemical, glucose, was mineralized. Thus, mineralization of LAS occurs rapidly in pre-exposed estuarine systems, with half-lives shorter than the typical hydraulic residence times of such estuaries (Shimp, 1989).

(iv) Soils and groundwater

A simple shake-flask system was used to determine CO_2 evolution in a test to assess the ultimate biodegradability of LAS by microorganisms in soil and sewage. At 30 mg/litre, high relative-molecular-mass LAS were biodegraded more slowly than those with a low relative molecular mass. Ultimate biodegradation could not be assessed precisely within the 28-day test period, but CO_2 removal was 37–77% and dissolved organic carbon removal was 59–84%. Ultimate biodegradation of the entire molecule (total CO_2) occurred concomitantly with biodegradation of the benzene ring ($^{14}CO_2$). Ring desulfonation, measured as ^{35}S-LAS, was rapid and occurred mainly after primary biodegradation (MBAS method) (Gledhill, 1975).

The kinetics of the ultimate biodegradation of C_{10}–C_{14} LAS to CO_2 was studied in a sludge-amended soil at 0.1–10 times environmental concentrations. All four homologues underwent rapid degradation, with half-lives for the breakdown of the benzene ring of 18–26 days (Ward & Larson, 1989).

Microbial mineralization of 50 µg/kg ^{14}C-LAS was examined in soil types ranging from a loamy sand impacted with sewage effluent to a highly organic alpine soil, by monitoring the evolution of $^{14}CO_2$. LAS were mineralized without a lag in all soils; mineralization exhibited first-order kinetics in nine of the 11 soil types. Asymptotic yields of CO_2 ranged from 16 to 70%; the half-lives were 1.1–3.7 days. The degradation rates were not correlated with microbial activity, pH, total organic content, or previous exposure (Knaebel et al., 1990).

After ^{14}C calcium and sodium salts of LAS were applied to two silty loam soils, the distribution of ^{14}C was similar. After 60 days, 31–47% of

the applied ^{14}C had evolved as $^{14}CO_2$ and 31–40% was present as soil residue, possibly as a combination of parent and metabolized surfactant (Kawashima & Takeno, 1982).

A4.2.1.2 *Anaerobic degradation*

Degradation of LAS (measured as MBAS) was much slower under anaerobic conditions in activated sludge than under aerobic conditions. No degradation had taken place after one day; up to 20% had been degraded between days 3 and 21, and 36% after 28 days. When soil and wastewater were used, only 20% of the MBAS had disappeared within 28 days (Oba et al., 1967). No significant removal of LAS was reported in an anaerobic sludge digester at a Swiss sewage treatment plant (Giger at al., 1989).

In a review of the fate of LAS in anaerobic and aerobic sewage treatment plants, it was concluded that drying anaerobic sludge on open beds considerably reduces the LAS content. Anaerobic degradation of LAS is, however, limited, as the addition of LAS at 15 g/kg raw sewage (about 15 g/litre raw sewage) may inhibit anaerobic degradation. In the laboratory, digestion of LAS was impaired at concentrations of > 15–20 g/kg, and a concentration of 20 g/kg seriously inhibited gas production, especially when other potentially inhibitory compounds were present. The concentration of LAS normally found in sewage (5–10 g/kg) is, however, unlikely to inhibit anaerobic degradation (Painter & Zabel, 1989). About 15–35% of LAS in raw sewage is physically removed in primary settlers in sewage treatment plants, accounting for most of the LAS found in anaerobic sludge. Precipitation of LAS is correlated with water hardness, since the solubilities of the calcium and magnesium salts of LAS are very low; the solubility products ranged from 2.2×10^{-10} for C_{10} LAS to 6.2×10^{-13} for C_{13} LAS (Berna et al., 1989). The effect of water hardness was confirmed by mass balance analysis of Na^+, Ca^{2+}, and Mg^{2+} (Berna et al., 1993b). The content of total calcium and magnesium in anaerobically digested sludge was 43 times higher than that in water. High contents of LAS in the sludge (up to 30 g/kg) did not inhibit the anaerobic digestion process (Painter & Mobey, 1992), probably because LAS were present as calcium and magnesium salts and therefore had reduced bioavailability.

LAS were not degraded in an anaerobic sediment from a pond receiving wastewater from a laundromat. Despite an exposure period of 25 years, no anaerobic degradation was reported (Federle & Schwab, 1992).

Pre-aerobic treatment of LAS may cause changes in the molecule that permit subsequent degradation under anaerobic conditions (Ward, 1986).

A4.2.2 Abiotic degradation

The mechanisms of abiotic degradation of LAS reported below are not of environmental significance, since biodegradation and sorption are rapid, effective removal mechanisms.

A4.2.2.1 Photodegradation

In a study of the kinetics of the photodecomposition of C_{12} LAS, using a continuous-flow reactor, the initial concentrations were 60–182 mg/litre and the radiation wavelength was 200–450 nm. Conversion of LAS to intermediate products occurred within 1 min, yielding 7 mol CO_2 per mol LAS, and was complete within 20 min. The reaction rate was increased by two orders of magnitude by ferric perchlorate (Matsuura & Smith, 1970).

Rapid photodegradation of LAS (50 mg/litre) occurred within 1–2 h in an aqueous, aerated titanium dioxide suspension without noble metal catalysts. There was rapid decomposition of the aromatic ring and slower oxidation of the aliphatic ring. Photodegradation was dependent on the simultaneous presence of titanium dioxide, oxygen, and light (Hidaka et al., 1985).

A4.2.2.2 Cobalt-60 irradiation

The decomposition of LAS was studied in distilled water irradiated with cobalt-60 gamma rays, which react with water to produce oxygen, peroxide, hydrogen peroxide, and other strong oxidizing agents. A concentration of 10 mg/litre LAS was reduced to 7.8 mg/litre by absorption of 10 Gy and to 0.9 mg/litre by absorption of 100 Gy. The rate of irradiation was found to be less important than the total absorbed energy (Rohrer & Woodbridge, 1975).

A4.2.3 Bioaccumulation and biomagnification

Studies of the bioaccumulation potential of LAS have all been carried out with LAS labelled with ^{14}C or ^{35}S. It should be noted that as these techniques do not usually allow consideration of metabolic

transformation the actual bioaccumulation of the parent compound may be overestimated. Toxic concentrations of the breakdown products of LAS are discussed in section A9.3.7.

A4.2.3.1 Aquatic organisms

Bioaccumulation has been studied in daphnids and fish (Table 8). LAS are readily absorbed through the gills and body surface of fish and are subsequently distributed via the blood to the organs and tissues; most LAS accumulate in the gall-bladder and hepatopancreas. Clearance is usually rapid, with a half-life of two to three days. Short-chain LAS are accumulated to a lesser degree than long-chain LAS.

Only 1% of 0.5 mg/litre LAS added to water was retained in *Daphnia magna* within three or four days after transfer to 'clean' water. Almost all of the chemical was in the form of intact LAS. In fathead minnows (*Pimephales promelas*), metabolic transformation occurred. All tissues monitored showed some uptake, with concentration factors ranging from 79–372 in muscle to 21 000–70 000 in gall-bladder. Within four days of transfer to 'clean' water, 85% of the LAS had been lost, and almost 100% was lost within 10 days (Comotto et al., 1979).

In an experiment in which the aqueous concentrations of an initial concentration of 1.1 mg/litre LAS decreased by 20% during the test, the compounds were concentrated in the gills of carp (*Cyprinus carpio*) within 2 h of exposure, with a concentration factor of 40. Skin surface, muscle, brain, kidney, hepatopancreas, and gall-bladder showed more gradual uptake of LAS over the 24 h of exposure, with concentration factors ranging from 4.1 for skin surface to 1000 for gall-bladder. Blood, gonads, and spleen also took up LAS but were not monitored throughout the period of exposure. LAS was lost rapidly from all tissues except the gall-bladder during 48 h in 'clean' water (Kikuchi et al., 1978).

In the bluegill (*Lepomis macrochirus*), a steady state was reached within 120–168 h. The bioconcentration factor was calculated by a kinetic method to be 286 for a concentration of LAS of 0.8 mg/litre and 132 for 0.08 mg/litre. LAS were cleared rapidly after the fish were transferred to 'clean' water, with 99% eliminated within 336 h; the time for clearance was 29–30 h (Bishop & Maki, 1980). In another study, a steady state was reached within seven days; the bioconcentration factor in whole body using a kinetic method was reported to be 104; and the half-time for clearance was two to five days during a depuration period of 14 days. The authors postulated that fish excrete LAS in the urine and

Table 8. Bioconcentration factors for linear alkylbenzene sulfonates in aquatic invertebrates and fish

Organism	Static/flow	Exposure concentration (mg/litre)	Duration of test (days)	Chain length	Steady state	Bioconcentration factor	Tissue	Reference
Daphnia magna	Flow	0.07	3	C_{12}	?	490		Comotto et al. (1979)
		0.11				560		
		044				720		
		0.09	3	C_{13}	Yes	1250		
		0.11				1050		
		0.41				1325		
Cyprinus carpio	Static	61.1	1	C_{12}	Yes	4.1	Skin surface	Kikuchi et al. (1978)
						1000	Gall-bladder	
	Flow	0.5	4	C_{12}	Yes	20	Whole body	Wakabayashi et al. (1978)
						30	Hepatopancreas	
						9000	Gall-bladder	
		0.0091	5	C_{12}	Yes	16	Whole body	Wakabayashi et al. (1981)
		0.3				400		
		1.0				300		
Pimephales promelas	Flow	0.1	11	C_{12}	Yes	551	Whole body	Comotto et al. (1979)
				C_{13}		1223		
				C_{12}, C_{13}		269		

Table 8 (contd)

Organism	Static/flow	Exposure concentration (mg/litre)	Duration of test (days)	Chain length	Steady state	Biocon-centration factor	Tissue	Reference
Lepomis macrochinus	Flow	0.063 0.064	28	C_{12}	Yes	260 120	Whole body	Bishop & Maki (1980)
	Flow	0.5	35	$C_{11.7}$	Yes	107 5000	Whole body Gall-bladder	Kimerle et al. (1981)

Static, water unchanged for the duration of the test; flow, concentration in water maintained continuously

excrete shorter-chain carboxylates with the benzene ring intact across the gill membranes. Both forms may also be excreted in the faeces (Kimerle et al., 1981).

A4.2.3.2 Terrestrial plants

Foliar uptake of the calcium and sodium salts of ^{14}C-LAS (chain length not specified) by peanuts was studied seven and 30 days after application. No movement of LAS was detected: 70–80% remained within the same leaf to which the compound was applied, and no LAS were detected in other parts of the plant (Kawashima & Takeno, 1982).

Aqueous solutions of ^{14}C-LAS (chain length not specified) were applied to soil (orthic luvisol), and ryegrass (*Lolium perenne*) was grown under laboratory conditions for up to seven days. Uptake of LAS after three days was 80 mg/kg at an application rate of 1 mg/kg dry weight, 370 mg/kg at a rate of 5 mg/kg, and 18 900 mg/kg at 50 mg/kg. After seven days, levels of 600, 5000, and 19 300 mg/kg were measured at the three dose levels, respectively (Litz et al., 1987).

^{14}C-LAS (chain length not specified) were applied under field conditions to both loamy orthic luvisol and sandy dystric cambisol soils irrigated with wastewater at rates of 5 and 50 g/m^2. After 49 days, rye grass grown in the loamy soil contained residues of 130 and 1000 mg/kg dry weight at the two exposure rates, respectively. Plants grown in the sandy soil contained 230 and 470 mg/kg, respectively, after 54 days (Litz et al., 1987).

Two plant-soil microcosms were exposed to ^{14}C-LAS (chain length not specified), and LAS degradation and percolation were followed for up to 109 days. The initial soil concentrations were 16.2 µg/g dry soil in potato soil (sandy) and 27.2 µg/g in grass, bean, and radish soil (clay-like). The concentrations of radiolabelled compounds in the plants decreased rapidly: at the end of exposure, 39.1–65.8 µg LAS equivalents per g fresh weight of plant were found in potatoes (study duration, 76 days) and 62.1–213.3 µg/g in grass, radishes, and beans (study duration, 109 days) (Figge & Schoberl, 1989).

A5. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

Section summary

The concentrations of LAS have been quantified by means of a specific, sensitive analytical method in almost every environmental compartment in which they might be present. The concentrations decrease progressively from wastewater to treated effluent and surface waters, and low concentrations are found in the sea.

The environmental concentrations of LAS are directly dependent on use patterns, the type and efficiency of sewage treatment, and the characteristics of the receiving environment. In areas where LAS are the predominant surfactants used, typical concentrations are 1–10 mg/litre in wastewater, 0.05–0.1 mg/litre in effluents that have undergone biological treatment, 0.05–0.6 mg/litre in effluents passed through a percolating filter, 0.005–0.050 mg/litre in surface waters below sewage outfalls (with concentrations decreasing rapidly to 0.01 mg/litre downstream from the outfall), < 1–10 mg/kg in river sediments (up to 100 mg/kg in highly polluted sediments near discharge zones), 1–10 g/kg in sewage sludge, and < 1–5 mg/kg in sludge-amended soils. The initial concentration of LAS in sludge-amended soils is 5–10 mg/kg, but up to 50 mg/kg have been reported after atypically heavy appli-cations. The concentration of LAS in estuarine waters is 0.001–0.010 mg/litre but is higher where wastewater is discharged directly. The concentrations in offshore marine waters are < 0.001–0.002 mg/litre.

A wide range of environmental concentrations has been reported, owing to use of different analytical methods; differences in characteristics of sampling sites, which range from highly polluted areas with inadequate sewage treatment to areas where sewage undergoes extensive treatment; seasonal differences, which can account for a twofold variation; and differences in the use of LAS.

Monitoring has shown no accumulation of LAS in environmental compartments over time. The concentrations in soil do not increase with time but are diminished due to mineralization. As LAS are not degraded under strictly anaerobic condition, they are not mineralized in anaerobic sediments. With current use of LAS, the rates of their assimilation in all receiving environmental compartments is equal to their rate of input, implying a steady state.

A5.1 Environmental levels

LAS have been measured in most environmental compartments, including discharge (raw sewage), sewers, sewage treatment plants, sludge-amended soils and land fill, river water, river sediments, subsurface soils, groundwater, and estuaries (Berna et al., 1991).

A decline in the concentrations of anionic surfactants in the environment, as assessed by measurement of MBAS, was seen in Europe, Japan, and the United States after ABS was replaced by LAS (Sullivan & Evans, 1968; Sullivan & Swisher, 1969; Gerike et al., 1989). Similar declines have been observed more recently in countries such as Thailand, where the change to LAS detergents is also more recent (Berna et al., 1991).

A5.1.1 Wastewater, sewage effluent, and sludge

The concentrations of LAS in sewage influent and effluent at sewage treatment plants are shown in Table 9; those in sewage sludge are given in Table 10.

The efficiency of wastewater treatment plants in removing LAS is reported to exceed that of removal of biochemical oxygen demand. Activated sludge removed an average of 98% LAS, trickling filters removed 80%, and primary clarification, 27%. The average concentration in raw sewage was 3.5 mg/litre, and those in effluent were 2.1 mg/litre after primary treatment and 0.06 mg/litre in activated sludge. The average chain length of LAS was $C_{12.5}$ in sewage sludge and C_{12} in influent sewage (Rapaport & Eckhoff, 1990).

The amount of LAS removed in a sewage treatment plant was 93% on the basis of total organic carbon and 98.1% on the basis of a specific method. The contribution of LAS to the total organic carbon was estimated to be 0.93% in treated water and 3.0% in digested sludge; 75.9% of LAS present in the raw sewage was mineralized during treatment and 7% was in the form of sulfoxyphenylcarboxylates, a product of the biodegradation of LAS, suggesting that biodegradation of LAS had reached a steady state. These figures were obtained by analysis for sulfoxyphenylcarboxylates (Berna et al., 1993b).

In another study, 40% of LAS was removed in a wastewater treatment plant. The half-life for removal from the sewer pipe was calculated to be 11 h (Moreno et al., 1990).

Table 9. Concentrations of methylene blue-active substances (MBAS) and linear alkylbenzene sulfonates (LAS) in sewage influent and effluent

Location	Year	Material	Concentration (mg/litre)		Reference
			MBAS	LAS	
Switzerland (29 sites, 1 sampling)	1986	Raw sewage		0.95–3.9	Brunner et al. (1988)
		Effluent		0.007–0.33	
Germany (11 sites, 1 sampling)	1985	Influent (activated sludge)	5.1 (1–13.3)	4 (0.54–12.4)	Matthijs & De Henau (1987)
		Influent (trickling filter)	8.8 (8.1–9.9)	7.4 (6.8–8.4)	
		Effluent (activated sludge)	0.19 (0.09–0.28)	0.07 (0.05–0.11)	
		Effluent (trickling filter)	1.1 (0.84–1.5)	0.76 (0.61–0.94)	
United Kingdom (several samples)	1982	Effluent	0.69 (0.58–0.81)	0.31 (0.21–0.42)	Gilbert & Pettigrew (1984)
River Thames area (5 sites, several samples)	1987	Sludge		15.1–341	Holt et al. (1989)
Israel (4 sites)	1983	Influent	9.6–10.6[a]		Zoller (1985)
		Effluent	0.3–11.0[a]		
United States	1979	Effluent		0.078–0.303	Eganhouse et al. (1983)
(4 sites, 45 samples	1976–86	Influent		3.7 ± 1.1	Rapaport & Eckhoff (1990)
		Effluent (activated sludge)		0.05 ± 0.04	
		Effluent (trickling filter)		0.6 ± 0.3	
		Effluent (primary)		2.2 ± 0.4	

Table 9 (contd)

Location	Year	Material	Concentration (mg/litre)		Reference
			MBAS	LAS	
United States		Influent	5.9–6.5	5.7–6.5	Osburn (1986)
(1 sampling)		Influent	3.7–5.2	3.8–4.9	
		Effluent	0.39–1.02	0.14–0.60	
(2 sites, 9 samples)	1983	Raw influent	4.17	3.73	Sedlak & Booman (1986)
		Primary influent	3.18	2.97	
		Primary effluent	1.66–2.82	1.73–2.51	
		Final effluent	0.03–0.06	0.02–0.05	
Canada (4 sites, 45 samples yearly)	1976-86	Influent		2.0 ± 0.6	Rapaport & Eckhoff (1990)
		Effluent (activated sludge)	0.09 ± 0.05		
		Effluent (primary)		1.7–2.3	
Japan					
(5 sites, 60 samples)	1972-73	Influent	5.1–14.0		Oba et al. (1976)
		Effluent	0.3–4.7		
(6 sites, 1–2 samples)	1984	Influent (suspended particles)		0.236–1.504	Takada & Ishiwatari (1987)
		Effluent (suspended particles)		0.0001–0.001	

[a] Total anionic surfactants (mainly LAS)

Table 10. Concentrations of methylene blue-active substances (MBAS) and linear alkylbenzene sulfonates (LAS) in sewage sludge

Location	Year	Material	Concentration (mg/litre)		Reference
			MBAS	LAS	
Switzerland					
(8 and 12 sites, 1 sampling)		Digested sludge		2900–11 900	McEvoy & Giger (1985, 1986)
(29 sites, 1 sampling)	1986			50–13 800[a]	Brunner et al. (1988)
Spain					
(5 sites, several samplings)		Activated sludge (anaerobic digestion)		7000–30 200[a]	Berna et al. (1989)
		Aerated, settling system		400–700[a]	
Finland (12 sites, 1 sampling)		Digested sludge		3400–6300[a]	McEvoy & Giger (1986)
Belgium (11 sites, 1 sampling)	1985	Aerobic sludge	5399 (3042–8133)	281 (182–432)	Matthiijs & De Henau (1987)
		Digested sludge	9017 (3632–17 006)	4917 (1327–9927)	
Germany (4 sites, 45 samples yearly)	1981-86			4920 (1330–9930)	Rapaport & Eckhoff (1990)

Table 10 (contd)

Location	Year	Material	Concentration (mg/litre)		Reference
			MBAS	LAS	
United States					
(4 sites, 45 samples yearly)	1981–86			4660 ± 1540	Rapaport & Eckhoff (1990)
12 sites, NY, (1 sampling)		Digested sludge		6900[a]	McEvoy & Giger (1986)
(12 sites, CA, 1 sampling)		Digested sludge		5200[a]	
(1 sampling)		Primary sludge	110–126	107–127	Osburn (1986)
(2 sites, OK, 9 samples)	1983	Primary sludge	4610–6120	5340–6310	Sedlak & Booman (1986)
		Secondary sludge	520–990	410–860	
		Anaerobic digester	6860	6660	
		Aerobic digester	3820	4250	
		Drying bed (anaerobic)	170	160	
		Drying bed (aerobic)	230	150	
Southern California (marine)	1981	Effluent particulates		1342	Eganhouse et al. (1983)

[a] Dry weight

A5.1.2 Sediment

The concentrations of LAS in sediment are shown in Table 11, and those in sediment samples collected at various distances from sites of effluent outfall are shown in Table 12.

Concentrations of LAS > 10 mg/kg were measured in sediments from the upper estuaries near Tokyo Bay and < 1 mg/kg in the lower estuaries. The concentrations of LAS in sediments decreased offshore, falling below 0.01 mg/kg in sediments sampled 10 km from the mouths of the rivers. The authors suggested that loss of LAS was due to rapid degradation in the coastal zone (Takada et al., 1992a).

It was reported in one study that C_{13} was the most abundant homologue of LAS in river sediment (Yoshikawa et al., 1985); another group found that C_{12} was the most abundant of the LAS in estuarine sediments and that no C_{10} were present (Utsunomiya et al., 1980). C_{12} and C_{13} LAS predominated in sediment and C_{10} and C_{11} homologues were the most abundant in water (Hon-Nami & Hanya, 1980b). The average chain length of LAS in Japanese river sediments was $C_{11.8}$–$C_{12.2}$ (Hon-Nami & Hanya, 1980b; Yoshimura et al., 1984a).

In a study of marine sediments from an area adjacent to the point of discharge from a submarine sewer, LAS were detected only in the vicinity of the discharge, at a concentration of 0.1 mg/kg, and not in sediment sampled 50 m outside this area. The average chain length was $C_{11.7}$. In a comparison of the chain lengths of LAS detected in various environmental compartments and those used in detergent products, the LAS detected in sludge and sediment were relatively higher homologues and those in the water phase were lighter (Prats et al., 1993).

The average concentration of LAS in river sediments sampled upstream of an activated sludge treatment plant outfall was 1.1 mg/kg, and those in sediments downstream of the plant were 0.3–3.8 mg/kg (McAvoy et al., 1993).

A5.1.3 Surface water

The concentrations of LAS in water are shown in Table 13 and those in samples taken at various distances from sites of effluent outfall in Table 14.

Table 11. Concentrations of methylene blue-active substances (MBAS) and linear alkylbenzene sulfonates (LAS) in sediments in the United States and Japan

Location	Year	Concentration (mg/kg)		Reference
		MBAS	LAS	
United States				
Rivers (activated sludge)			0.3–3.8	McAvoy et al. (1993)
Rivers (trickling filter)			0.2–340	
Mississippi River	1991–92		< 0.01–5	Tabor et al. (1993)
Japan				
Tokyo Bay (1 sampling, few samples)	1969	35 (33–37)		Ambe (1973)
River (1 sampling, few samples)		61 (55–65)		
River Sagami estuary (16 sites, 1 sampling)		7.9–39[a]	ND–17	Utsunomiya et al. (1980)
Sagami Bay (16 sites, 1 sampling)		5.1–15	ND	
Rivers	1977		< 1–260	Environment Agency Japan (1978)
Lake Suwa (1 site, 3 samples)	1977		1.0-7.0	
Rivers (9 sites, 7 samples, 1 year); (1 site 52 samples)	1982–83		107 (ND–567)	Takada & Ishiwatari (1987); Takada et al. (1992b)
Estuaries (1 site, 52 samples)	1983–84		4.82 (0.12–36.6)	Takada et al. (1992b)
Tokyo Bay (9 sites, 7 samples, 1 year)	1980		71.0	Takada & Ishiwatari (1987)
Tokyo Bay	1984		0.02 (ND–0.06)	Takada et al. (1992a)
Sumida River (12 sites, 1 sampling)	1982		0.069	Kikuchi et al. (1986)
Tama River (3 sites, 8 samples)	1977		3.5–86.3	Hon-Nami & Hanya (1980b)
Tama River (10-12 sites)	1982		0.141	Kikuchi et al. (1986)
Tokyo Bay (10-12 sites)	1982		< 0.001–0.002	

Table 11 (contd)

Location	Year	Concentration (mg/kg)		Reference
		MBAS	LAS	
Japan (contd).				
Tsurumi River (7 sites, 12 samples)	1984		17–45[a]	Yoshikawa et al. (1985)
Tama River	1981		2.79–10.72	Yoshimura et al. (1984b)
Ports and coast	1977		< 1–2.9	Environment Agency Japan (1978)

ND, not determined
[a] Dry weight

Table 12. Concentrations of methylene blue-active substances (MBAS) and linear alkylbenzene sulfonates (LAS) in sediment of rivers in Germany and the United States at various distances from effluent outfalls

Location	Year	Sampling site (distance from effluent outfall)	Concentration (mg/litre)		Reference
			MBAS	LAS	
German rivers (14 sites, several samples)	1978–82	Below outfall		1.5–174[a]	De Henau et al. (1986)
United States					
Rivers (4 sites, 45 samples)	1978–82	Below outfall		190	Rapaport &
yearly		< 5 miles (8.0 km)		11.9	Eckhoff
		> 5 miles (8.0 km)		5.3	(1990)
(1 sampling)		0.5 miles (0.8 km)	118–317	100–322	Osburn (1986)
		4.4 miles (7.1 km)	4.1–19	2.0–5.1	
		7.4 miles (11.9 km)	7.5–10.6	1.3–4.4	
Rapid Creek, South Dakota	1979–80	0.8 km		44.6–275	Games (1983)
		7 km		3.2–9.1	
		11.7 km		2.1–8.4	
		25.3 km		2.7–10.1	
		48 km		1.4	
		87.2 km		1.5	
Little Miami River, Ohio		Downstream from sewage		ND–1.2	Hand et al. (1990)
4 sites, 1 sampling)		treatment plants		24.7–290[b]	
Rivers (4 sites, 45 samples)	1978–82	Below outfall		190	Rapaport &
		Above outfall[c]		1.0–1.2	Eckhoff (1990);
		Below outfall (left)[c]		0.3–1.6	McAvoy et al.
		Below outfall (middle)[c]		0.6–3.8	(1993)

Table 12 (contd)

Location	Year	Sampling site (distance from effluent outfall)	Concentration (mg/litre)		Reference
			MBAS	LAS	
United States (contd)					
Rivers (4 sites, 45 samples) (contd).	1978–82	Below outfall (right)[c]		0.8–3.4	
		Above outfall[d]		0.2–0.9	
		Below outfall (left)[d]		0.2–130	
		Below outfall (middle)[d]		0.6–124	
		Below outfall (right)[d]		9–340	

[a] 13 of the 14 samples contained < 25 mg/kg and 10 contained < 10 mg/kg
[b] Suspended solids
[c] Activated sludge
[d] Trickling filter

Table 13. Concentrations of methylene blue-active substances (MBAS) and linear alkylbenzene sulfonates (LAS) in water

Location	Year	Water sample	Concentration (mg/litre)		Reference
			MBAS	LAS	
Freshwater					
United States					
Rivers (4 sites, 45 samples yearly)	1978–86			0.041–0.115	Rapaport & Eckhoff (1990)
Little Miami River, Ohio (4 sites, one sampling)				< 0.05	Hand et al. (1990)
		Interstitial		ND–0.08	
Illinois River (one sampling)[a]	1959–65		0.54		Sullivan & Swisher (1969)
	1965–66		0.22		
	1968		0.05–0.06		
Rapid Creek, South Dakota	1979–80			0.01–0.270	Games (1983)
Mississippi River (36 sites)	1991–92			< 0.01–0.3	McAvoy et al. (1993)
(350 samples)			< 0.01–0.046	< 0.005	Tabor et al. (1993)
Japan					
Rivers (23 sites, 51 samples)	1977			< 0.01-2.9	Environment Agency Japan (1978)
Rivers (1 sampling)				0.018-0.59	Tsukioka & Murakami (1983)
Oohori River (6 sites monthly)	1987–88			~0.5–1.6	Amano et al. (1991)
Lake Teganuma (6 sites monthly)	1987–88			ND–~0.7	
Tama River (3 sites, 8 samples)	1977–78		0.24–1.24	0.108–0.491	Hon-Nami & Hanya (1980a)

Table 13 (contd)

Location	Year	Water sample	Concentration (mg/litre)		Reference
			MBAS	LAS	
Japan (contd)					
Rivers, Hyogo Prefecture (70 sites)				0.004–2.5	Kobuke (1985)
Tama River (3 sites, 1 sampling)				0.035–0.219	Yoshikawa et al. (1984)
Tama River (10-12 sites)	1982			0.128	Kikuchi et al. (1986)
Sumida River (10-12 sites)	1982			0.005–0.01	Kikuchi et al. (1986)
Rivers (1 sampling)			0.06–0.12		Saito & Hagiwara (1982)
Rivers, Niigata Prefecture (6 sites, 1 sampling)			0.02-2.63	0.18 (max)	Motoyama & Mukai (1981)
Rivers, coastal area, Hiroshima Prefecture (20 sites)				0.019 (0.001–0.06)	Okamoto & Shirane (1982)
Inland Sea, Eastern Seto (4 sites, 1 sampling)	1975		0.016–0.077		Yoshida & Takeshita (1978)
(17 sites, 1 sampling)	1976		0.01–0.048		
Tsurumi River, Kanagawa (7 sites once)	1984–76	Surface	0–0.8	0.01–0.29	Yoshikawa et al. (1985)
Yodo River, Osaka (several sites)	1989	Surface		0.043–0.089	Nonaka et al. (1990)
Tama River, Tokyo (2 sites, 4 samples)	1981	Surface		0.2	Yoshimura et al. (1984b)
Sumidogawa River (2 samples)	1983	Suspended		0.0048–0.054	Takada & Ishiwatari
Tomogawa River (5 samples)		particles		0.0005–0.0025	(1987)
Teshiro River, Nagoya (4 sites, 4 samples)	1989	Surface		0.01–0.27	Kojima (1989)

Table 13 (contd)

Location	Year	Water sample	Concentration (mg/litre)		Reference
			MBAS	LAS	
Japan (contd)					
Lake Biwa, Shiga	1988	Surface		0.00	Shiga Prefecture (1988)
Teganuma, Chiba (1 site, 12 samples)	1988	Surface		ND–0.423	Amano et al. (1989)
River (several sites)	1988	Surface		0.019–1.4	Nonaka et al. (1989)
Nagoya Bay	1989	Surface		0.00	Kojima (1989)
Rivers, Fukuoka City				ND–1.6	Ohkuma (1981)
Europe					
River Rhine (several sites)	1971–72	0.08–0.24			Hellmann (1978)
Saar River (11 sites, 1 sampling)	1985		0.13 (0.03–0.25)	0.04 (0.01–0.09)	Matthijs & De Henau (1987)
German rivers (several sites)	1976–79		0.075–0.5		Fischer (1980)
Dutch river (Amsterdam drinking-water supply) (8 sites)			0.004–0.141	0.003–0.037	Waters (1976)
Florence, Italy (several samples)	1983	Aqueduct	0 .01–0.1		Mancini et al. (1984)
(several sites)	1982	Well water	0.00–0.01		
United Kigdom					
Rivers	1982		0.04–0.26	0.012–0.08	Gilbert & Pettigrew (1984)
Rivers (8 sites)			0.035–0.217	0.009–0.097	Waters (1976)
Rivers (4 sites)	1977–78		0.022–0.473	0.007–0.173	Waters & Garrigan (1983)

Table 13 (contd)

Location	Year	Water sample	Concentration (mg/litre)		Reference
			MBAS	LAS	
Groundwater	1992			< 0.01–0.02	Field et al. (1992)
Estuarine and marine water					
North Sea (19 sites)	1989			< 0.0005–0.0012	Stalmans et al. (1991)
Krka River estuary, Croatia	1990	Wastewater		0.42–0.78	Terzic & Ahel (1993)
(below 50 m > 50 m)		Estuarine water	0.003–0.007 0.001–0.002		
Tokyo Bay, Japan (8 samples)	1978		0.03–0.07	< 0.003–0.014	Hon-Nami & Hanya (1980a)
Tokyo Bay, Japan (10–12 samples)	1982			0.001–0.03	Kikuchi et al. (1986)
Osaka Bay, Japan (several sites)	1988	Surface		ND–0.0072	Nonaka et al. (1989)

ND, not detected
[a] 10–20% of MBAS were LAS

Table 14. Concentrations of methylene blue-active substances (MBAS) and linear alkylbenzene sulfonates (LAS) in water at various distances from effluent outfalls

Location	Year	Sampling site (distance from effluent outfall)	Concentration (mg/litre)		Reference
			MBAS	LAS	
United States					
Rivers (4 sites, 45 samples yearly)	1978–86	Below outfall		0.115	Rapaport & Eckhoff (1990)
		< 5 miles (8 km)		0.079	
		> 5 miles (8 km)		0.041	
(1 sampling)		0.5 miles (0.8 km)	0.400	0.270	Osburn (1986)
		4.4 miles (7.1 km)	0.300	0.150	
		7.4 miles (11.9 km)	0.250	0.120	
		15.8 miles (25.4 km)	0.240	0.100	
		30.0 miles (48.3 km)	0.130	0.040	
		55.0 miles (88.5 km)	0.100	0.010	
Rapid Creek, South Dakota	1979–80	0.8 km		0.270	Games (1983)
		7 km		0.150–0.190	
		11.7 km		0.120	
		25.3 km		0.080	
		48 km		0.040	
		87.2 km		0.010	
Rivers		Above outfall		< 0.01–0.9	McAvoy et al. (1993)
		Below outfall (left)		< 0.01–0.33	
		Below outfall (middle)		< 0.01–0.3	
		Below outfall (right)		< 0.01–0.3	

Table 14 (contd)

Location	Year	Sampling site (distance from effluent outfall)	Concentration (mg/litre)		Reference
			MBAS	LAS	
Canadian rivers (4 sites, 45 samples yearly)	1978–86	Below outfall		0.053	Rapaport & Eckhoff (1990)
Rio Grande, Brazil (1 sampling, 50 samples)	1979	90 m	0.05–4.5		Kantin et al. (1981)
German rivers (several sites)	1976–79	Unpolluted	0.075		Fischer (1980)
		Polluted	0.2–0.5		
(4 sites, 45 samples yearly)	1978–86	Below outfall		0.01–0.09	Rapaport & Eckhoff (1990)
United Kingdom					
Rivers (several samples)	1982	Above discharge	0.04 (0.02-0.07)	0.012 (0.008–0.019)	Gilbert & Pettigrew (1984)
		Close to discharge	0.26 (0.11–0.47)	0.08 (0.01–0.17)	
		5–16 km	0.16 (0.08–0.23)	0.04 (0.008–0.095)	
Avon River (4 sites)	1977–78	Head water	0.03–0.039	0.009–0.015	Waters & Garrigan (1983)
		0.5 km	0.21–0.371	0.056–0.173	
		6 km	0.095–0.22	0.011–0.095	
Tean River (4 sites)	1977–78	Head water	0.035–0.073	0.008–0.019	
		Directly below sewage treatment	0.208–0.473	0.067–0.144	
		5 km	0.145–0.234	0.019–0.07	

Table 14 (contd)

Location	Year	Sampling site (distance from effluent outfall)	Concentration (mg/litre)		Reference
			MBAS	LAS	
United Kingdom (contd)					
Trent River (4 sites)	1977–78	Head water	0.022–0.052	0.01–0.011	
		20–35 km below head water	0.08–0.227	0.007–0.072	
Nene River tributary (4 sites)	1978		0.104	0.011	
		In the vicinity of sewage effluent disharge	0.206–0.216	0.035–0.037	
		3.5 km	0.184	0.035	
		13.5 km	0.06	0.007	

After replacement of branched-chain ABS, which are only sparingly biodegradable, with the straight-chain LAS, the concentrations of MBAS decreased in many rivers. ABS were replaced by LAS in Japan in the late 1960s; the ratio of LAS to total ABS in river water rose from 20 to 70% in 1967–70 and had reached 90% by 1973 (Miura et al., 1968; Ihara et al., 1970; Oba et al., 1975). The levels of MBAS were monitored in the Illinois River, United States, from 1959 to 1966; those in 1965 and 1966 reflected the change in surfactant usage (Sullivan & Evans, 1968), and this trend continued in 1967 and 1968 (Sullivan & Swisher, 1969). In the River Rhine, the level of anionic detergents, measured as MBAS, fell steadily between 1971 and 1977 (Hellmann, 1978). In water samples from 140 sites on four German rivers, MBAS concentrations fell by 90% between 1964 and 1987 (Gerike et al., 1989).

The mean level of MBAS in rivers in the United Kingdom was 0.15 mg/litre. On average, only 26% was attributable to LAS (by microdesulfonation and gas–liquid chromatography), but the levels of LAS and their contribution to the total MBAS concentration varied according to the sampling site, with a higher proportion of LAS in samples from sites near sewage effluent discharge points (Waters & Garrigan, 1983). Similar findings were reported by Gilbert & Pettigrew (1984), who found that LAS represented 45% of total MBAS in actual sewage. Sites immediately below sewage outfalls were found to have higher MBAS:LAS ratios than sites further downstream (Osburn, 1986).

In Lake Biwa basin, Japan, during the summer months of 1983, LAS were found in a wide range of concentrations. The highest, measured as MBAS, were > 0.2 mg/litre at river mouths. The levels in rivers flowing from densely populated areas were 0.05–0.2 mg/litre MBAS and those flowing from less populated areas were < 0.05 mg/litre. The middle stream zone of the River Isasa, in a densely populated area, contained levels of 0.36–1.91 mg/litre, and surfactant levels in residential areas showed daily fluctuations related to discharge (Sueishi et al., 1988). Several observations apply to these studies. Firstly, the fact that daily fluctuations were observed indicates that the samples may have been taken from the actual discharge plume, so that the wastewater effluent may not have been completely mixed with the recipient surface water. Secondly, in several Japanese studies of heavy discharge zones, anionic surfactants could not be detected in surface waters, although the analytical detection limit of MBAS in the mid-1980s was 0.05–0.1 mg/litre. Thirdly, sewage treatment at several of the sites has improved considerably over the last decade.

Seasonal trends in the concentrations of LAS were observed in the Oohori River and Lake Teganuma, Japan, in 1987 and 1988, with low levels in summer and high levels in winter (Amano et al., 1991).

The concentrations of LAS were measured in the Tamagawa River, Japan, at two-week intervals for two years, by sampling water from the boundary between freshwater and brackish zones. The concentrations measured in winter were about five times higher than those measured in summer, when long-chain homologues tended to be depleted. The distribution of isomers also showed a clear seasonal trend, with a greater loss of external isomers in summer. The seasonal changes are thought to be the result of differences in water temperature and microbial activity. The flux of LAS in the river was estimated to be 320 tons/year (293 tonnes/year), which exceeds the total amount of LAS accumulated in the bay sediment, indicating that > 99.9% of LAS in the estuary and the bay was degraded (Takada et al., 1992b).

The concentrations of LAS in suspended particles from tributaries of Tokyo Bay, Japan, were 0.5–53.8 µg/litre. Those in suspended particles from a wastewater influent were 297–504 µg/litre and those in the effluent, 0.1–1.22 µg/litre (Takada & Ishiwatari, 1987).

The concentrations of LAS in the estuary of the Krka River, Croatia, were 420–780 µg/litre near municipal wastewater outlets; 50 m from the wastewater outlets, the concentrations were 7.2 µg/litre at a depth of 0.5 m and 3.2 µg/litre at a depth of 6 m. The concentrations in water sampled more than 50 m from the input area were 1–2 µg/litre. The Krka River estuary was reported to be highly stratified, with vertical transport of pollutants reduced by the freshwater–saline boundary. The concentrations of LAS were negatively correlated with salinity; the maximum concentration, 24 µg/litre, was detected in the surface monolayer. An increase in the relative abundance of lower homologues of LAS (C_{10} and C_{11}) was reported in comparison with the original distribution of homologues in the wastewater, indicating more rapid depletion of higher homologues, possibly by biodegradation and fast settling with particles from sewage (Terzic & Ahel, 1993).

In a comparison of the distribution of homologues of LAS in the Tama River, Japan, with those established for active substances used in commercial detergents, the levels of C_{12} and C_{13} LAS were found to decrease over time and those of C_{10} and C_{11} to increase (Hon-Nami &

Hanya, 1980a). C_{11} was the commonest LAS homologue in river water (Kobuke, 1985; Yoshikawa et al., 1985), and no C_{13} LAS were present (Utsunomiya et al., 1980). The average chain length of LAS in Japanese rivers was $C_{10.9}$–$C_{11.2}$ (Nakae et al., 1980; Yoshimura et al., 1984a; Kobuke, 1985).

Several research groups have confirmed that such changes in chain length occur during the environmental passage of LAS. In a study in which the concentration of homologues of LAS was measured quantitatively by HPLC during activated sludge treatment and lagoon treatment of wastewater in Spain, the average chain length decreased from $C_{11.7}$ in raw material, to $C_{11.3}$ in the dissolved phase of raw wastewater, and to $C_{10.3}$ in the dissolved phase of treated effluent. A slight increase in average chain length was reported for the solids compartment in each of these systems, adding to laboratory findings that the longest homologues adsorb most strongly to sediment. The reduction in average chain length in the water compartments was environmentally significant, since shorter homologues of LAS are less toxic to aquatic organisms. Thus, the LC_{50} values for daphnia were higher for shorter homologues (> 20 mg/litre for C_{11} and 10 mg/litre for $C_{11.7}$) (Prats et al., 1993).

The Japanese Soap & Detergent Association (1992) reported a decrease in LAS concentrations in the Tama River near Tokyo, Japan, from 2.3 mg/litre in 1967 to 0.2 mg/litre in 1991. The decrease was attributed to the development of sewage systems along the river: sewage coverage was 26% in 1974 and 89% in 1990. This information can be used to estimate concentrations of LAS in developing countries with inadequate sewage systems but where detergent use is increasing.

Low levels of LAS were reported in water from the Scheldt River estuary and in a series of samples from the North Sea (see Table 13). The concentrations in the estuary decreased rapidly from about 0.010–0.012 mg/litre to values below the limit of analytical detection (0.5 µg/litre) concurrently with an increase in salinity. The concentrations decreased more rapidly than on the basis of dilution alone, indicating that removal occurred rapidly. The authors did not report whether the removal of LAS was related to adsorption onto settling solids, to biodegradation, or to a combination of the two. The concentration of LAS in samples taken offshore was consistently below the limit of detection (Stalmans et al., 1991).

A5.1.4 Soil and groundwater

The levels of LAS in sludge-amended soil were 0.9–1.3 mg/kg in German soils used for agriculture. A level of 2.2 mg/kg was found in the United Kingdom in soil that was used only for the disposal of sludge (De Henau et al., 1986). MBAS were found at a level of 24.7 mg/kg (14.4–37.5 mg/kg) and LAS at 1.4 mg/kg (0.9–2.2 mg/kg) in German agricultural soils that had been amended with sludge (Matthijs & De Henau, 1987). The levels of LAS in soils near the River Thames, United Kingdom, in 1987 to which sludge had been applied previously were < 0.2–2.5 mg/kg. Soils that had received an application of sludge during 1987 had levels of LAS of < 0.2–19.8 mg/kg (Holt et al., 1989).

Levels of 13–47 mg/kg were found on the surface of sludge-amended soil in the United States in 1979; < 5 mg/kg were found at a depth of 15–90 cm (Rapaport & Eckhoff, 1990).

A concentration of 22.4 mg/kg LAS was measured in agricultural soil that had recently been amended with anaerobically digested sludge. The concentration was 3.1 mg/kg six months after application of the sludge and 0.7 mg/kg after 12 months (Prats et al., 1993). HPLC, fluorescence detection, and mass spectrometry were used to analyse samples of a groundwater plume which originated from an underground discharge of sewage. It was found that 96% of the LAS was removed from the aqueous phase during sewage treatment and an additional 3% during infiltration with groundwater. The concentrations in groundwater were below the detection limit of 0.01–0.02 mg/litre. The disappearance of LAS during groundwater infiltration was calculated to follow first-order kinetics. LAS were detected (by mass spectrometry) at only trace levels in groundwater sampled 20–500 m down the gradient from the infiltration zone (Field et al., 1992).

A5.1.5 Drinking-water

The concentration of LAS reported in Dutch tap-water was 0.003 mg/litre; MBAS levels were about three times higher. In tap-water in the United Kingdom, the concentration of LAS was 0.007 mg/litre; that of MBAS was again three times higher (Waters, 1976). The concentrations of LAS in Italian well-water were below the analytical limit of detection of 0.0084 mg/litre (Mancini et al., 1984). LAS were not detected in Japanese drinking-water in the 1970s at a limit of detection of 0.001 mg/litre (Yushi, 1978).

A5.1.6 Biota

The concentrations of LAS in biota are shown in Table 15.

Table 15. Total body concentrations of linear alkylbenzene sulfonates in biota in Japan

Organism	Year	Location	Concentration (mg/kg dry weight)	Reference
Algae	1980–81	River	< 1–368	Katsuno et al. (1983)
Pond snail (*Sinotaia quadratus histrica*)	1979	River	0.4–1.81	Tanaka & Nakanishi (1981)
Gizzard shad (*Konosirus punctatus*)	1982 1983	Bay	< 1 or < 2 < 0.1–0.3	Tokai et al. (1990)

A5.2 Environmental processes that influence concentrations of linear alkylbenzene sulfonates

A shift towards LAS of lower chain lengths has been reported in environmental samples in comparison with the distribution of chain lengths in raw materials. It has also been reported that about 50% of the total LAS in samples of water is associated with either suspended particles or dissolved organic matter. Reductions in both the chain length and the concentration of dissolved LAS will result in decreased aquatic toxicity (see also section 9).

A5.2.1 Changes in chain length distribution during environmental removal of linear alkylbenzene sulfonates

The concentrations of LAS and related compounds were measured in 350 samples of water and sediment from the Mississippi River, United States. Those in surface water were < 0.005 mg/litre. LAS in sediment had longer chains than those in the overlying water column (Tabor et al., 1993).

A gradual reduction in the average chain length of homologues was observed as they passed through a wastewater treatment plant: untreated wastewater, $C_{12.1}$; treated effluent, C_{12}; surface water below a sewage outfall, $C_{11.7}$ (Castles et al., 1989). Isomers of C_{13} LAS have partition coefficients that are typically one order of magnitude higher than those of the corresponding isomers of the C_{12} LAS homologues (Amano et al., 1991).

A5.2.2 *Specification of linear alkylbenzene sulfonates in surface waters*

In most programmes for monitoring LAS in the environment, the total sample of waste or surface water is analysed, and separate concentrations of LAS in the fractions of dissolved and suspended solids are not determined. In a study in which these concentrations were reported, the mean levels of dissolved LAS were 8.4 mg/litre in raw wastewater (range, 5.6–11.4 mg/litre) and 5.5 mg/litre in the suspended solid fraction. In the seven wastewaters studied, an average of about 65% was present in the filtered (filtration, < 1 µm) 'dissolved' fraction and 35% in the 'solids-associated' fraction. In treated effluent, 85% of LAS was in the dissolved fraction and 15% in the solids-associated fraction (Berna et al., 1993b). In wastewater treatment works, 49–63% of the LAS was in the dissolved phase and 37–51% in the solids-associated phase (Berna et al., 1989). In filtered (0.7 µm) wastewater containing LAS at 2.55–2.95 mg/litre, 25–30% LAS was dissolved, and the remaining 70–75% was associated with the solid phase (Cavalli et al., 1991).

The average chain length of homologues of LAS in raw wastewater was lower in the dissolved phase ($C_{11.2}$–$C_{11.4}$) than in the solids-associated phase ($C_{11.9}$–$C_{12.0}$). The authors reported that 39–43% of LAS was present in the dissolved phase and 57–61% in the solids phase (Prats et al., 1993).

Humic acids extracted from sediments and soils formed strong association complexes with LAS under environmental conditions, as observed with fluorescence quenching techniques. The bioavailabilty of LAS to aquatic organisms is reduced as a result of these complexes (McAvoy et al., 1993).

A5.3 Estimation of human intake

Human daily intake has been estimated on the assumption that LAS are taken up from drinking-water and from washing food, vegetables, dishes, and the skin. The estimates vary from 4.5 to 14.5 mg/day (Ikeda, 1965; Tokyo Metropolitan Government, 1974; Sterzel, 1992). The higher figure is based on dubious assumptions about the concen-trations of LAS on vegetables, and the lower value is probably a more realistic estimate.

The human intake of all anionic surfactants is estimated to be 0.044–0.944 mg/kg per day (Sterzel, 1992), and the maximum daily intake of ABS, 0.14 mg/kg per day (Ikeda, 1965).

A6. KINETICS

Section summary

LAS are readily absorbed by experimental animals in the gastrointestinal tract, are distributed throughout the body, and are extensively metabolized. The parent compound and metabolites are excreted primarily via the urine and faeces, although there are marked differences between the isomers in the route of excretion. The main urinary metabolites identified in rats are sulfophenylbutanoic acid and sulfophenylpentanoic acid, which are probably formed through ω-oxidation followed by β-oxidation of LAS, although the metabolic pathways in primates may differ. Although few data are available, it would appear that dermally applied LAS are not readily absorbed through the skin, although prolonged contact may compromise the epidermal barrier and permit more extensive absorption.

A6.1 Absorption, distribution, and excretion

After oral administration of 2 mg/animal of the calcium or sodium salt of ^{14}C-LAS (chain length, C_{12}) to Wistar rats, radiolabel was detected in plasma after 0.25 h, reaching maxima at 2 h (0.86 and 1.00 µg/g of the two salts, respectively), and then decreasing gradually with time; the mean biological half-lives were calculated to be 10.9 and 10.8 h, respectively. Four hours after oral administration of the calcium or sodium salt, the concentration of radiolabel was high in the digestive tract (especially in the stomach: 22.56 and 31.67 µg/g as the parent compound or metabolites; and large intestine: 43.24 and 27.26 µg/g) and in the urinary bladder (34.89 and 16.58 µg/g). The concentrations were also high in the liver (2.73 and 2.13 µg/g), kidney (1.19 and 1.35 µg/g), testis (0.08 and 0.11 µg/g), spleen (1.63 and 0.16 µg/g), and lung (0.49 and 0.44 µg/g). At 48 and 168 h, there was little further change. During the 168-h period after administration, 50% of the radiolabel on the calcium salt was excreted in urine and 51% in faeces, and 47% of that on the sodium salt was excreted in urine and 50% in the faeces (Sunakawa et al., 1979).

Doses of 1 mg per 200 g body weight of two radiolabelled LAS isomers (chain length, C_{12}) with the benzene sulfonate moieties at the 2 and 6 positions were administered orally and intravenously to rats; the same dose was also administered to anaesthetized rats with bile-duct cannulas by intravenous or intraduodenal injection. Forty-eight hours

after oral or intravenous administration, there were marked differences in the disposition of the isomers in the urine and faeces: most of the radiolabel associated with the 2 isomer (75.3%) was in the urine, whereas most of that on the 6 isomer (77.9%) was present in the faeces. After intravenous administration to bile duct-cannulated rats, 88.6% of the 2 isomer was recovered in the urine, whereas 83.1% of the 6 isomer was in the bile. Studies of absorption after intraduodenal administration showed that both isomers were extensively absorbed within 6 h (Rennison et al., 1987).

After a dose of 1.2 mg ^{35}S-LAS in aqueous solution was administered by gavage to bile duct-ligated rats, 89% was absorbed from the gastro-intestinal tract, as seen by the presence of radiolabel recovered in urine. Absorption probably occurred mainly via portal venous blood, since only 1.6% was recovered in the lymphatic system. When the same dose was administered to bile duct-cannulated rats, 46% of the radiolabel was recovered in urine, 29% in faeces, and 25% in bile after 90 h. Enterohepatic circulation was determined in a study in which the bile from one rat was transmitted to the intestine of another through a cannula; all of the radioactive LAS excreted in the bile was reabsorbed. In a separate study, 40–58% of single oral doses of ^{35}S-LAS ranging from 0.6 to 40.0 mg was excreted in the urine and 39–56% in the faeces within 72 h of administration (Michael, 1968).

The excretory pattern of ^{14}C-sodium dodecylbenzene sulfonate was examined in male rats administered a concentration of 1.4 mg/kg of diet daily for five weeks. The total intake was 1213 µg/rat, of which 81.8% was excreted during the dosing period, with 52.4% in the faeces and 29.4% in the urine. After a further week on a normal diet, however, only 7.8% of the estimated residual amount was found in excreta. Of a single intraperitoneal injection of 0.385 mg ^{14}C-sodium dodecylbenzene sulfonate/rat (2.26 mg/kg body weight), 84.7% was eliminated within the first 24 h and 94.5% within 10 days (Lay et al., 1983).

LAS were not detected in the uterus of pregnant ICR mice administered a single oral dose of 350 mg/kg body weight on day 3 of gestation (Koizumi et al., 1985).

^{14}C-LAS (chain length, C_{10}-C_{14}, predominantly C_{11}, C_{12}, and C_{13}) were applied at 250 µg/7.5 cm^2 in water to clipped dorsal skin of rats; the treated area was washed after 15 min, and the animals were restrained from grooming. Most of the radiolabel was rinsed off, but some of the ^{14}C-LAS (11 ± 4 µg/cm^2) were detected on the treated area; none were detected in urine or faeces 24 h after the application. In an

accompanying study *in vitro*, there was no measurable penetration of ^{14}C-LAS (chain length, C_{12}) through isolated human epidermis or rat skin 24 or 48 h after application (Howes, 1975).

A mixture of ^{35}S-LAS and white petrolatum (29 mg/0.3 ml) was applied to a 4-cm^2 area of the dorsal skin of guinea-pigs, and 24 h after the application about 0.1% of the applied dose was found in urine and about 0.01% in blood and the main organs. After dermal application of the same dose to rats and guinea-pigs, the concentration of ^{35}S in the liver was 9.7 μg/g equivalent of LAS in rats and about 0.4 μg/g in guinea-pigs (Hasegawa & Sato, 1978).

After a single oral administration of 150 mg/kg ^{14}C-LAS (mean relative molecular mass, 349) in aqueous solution to rhesus monkeys (*Macaca mulatta*), plasma concentrations of radiolabel reached a maximum equivalent to 41.2 μg/ml at 4 h and then declined over 6–24 h, with a biological half-life of about 6.5 h. The observed peak plasma concentration of radioactivity (33.6 μg/ml) and the biological half-life (about 5 h) after seven consecutive daily oral administrations of 30 mg/kg body weight were similar to those found after a single administration. The highest concentration of ^{14}C (238.6 μg/g) was found in the stomach 2 h after the last dose. Concentrations were also high in the intestinal tract (108 μg/g), kidney (135.6 μg/g), and liver (64.8 μg/g) and were moderately high in the lung (19.8 μg/g), pancreas (17.7 μg/g), adrenal glands (20.6 μg/g), and pituitary gland (17 μg/g). At 24 h, the concentrations were higher in the intestinal tract (255.4 μg/g) and liver (10.5 μg/g) than in plasma (2.4 μg/g), whereas those in most tissues were lower than those in plasma, indicating that there is no specific accumulation or localization of LAS and their metabolites in these tissues. After seven subcutaneous doses of 1 mg/kg per day of ^{14}C-LAS, most of the radiolabel remained in the skin; the concentration was generally highest at the injection site (113.96 μg/g). The levels of radiolabel were also high in the intestinal tract (2.41 μg/g), kidney (1.83 μg/g), lung (2.45 μg/g), spleen (2.43 μg/g), thyroid (1.24 μg/g), and pituitary (1.00 μg/g) at 2 h. The concentration in most tissues was generally lower at 4 h, except in the intestinal tract (3.50 μg/g), liver (1.74 μg/g), and kidney (1.92 μg/g). The high level of radiolabel in the intestinal tract probably indicates biliary excretion. The average rates of excretion of radiolabel in urine and faeces during 120 h after administration of single oral or subcutaneous doses of ^{14}C-LAS to male and female rhesus monkeys are shown in Table 16. In animals of each sex, radiolabel was excreted primarily in the urine after either route of administration (Cresswell et al., 1978).

Table 16. Excretion of ^{14}C-linear alkyl benzene sulfonates in rhesus monkeys

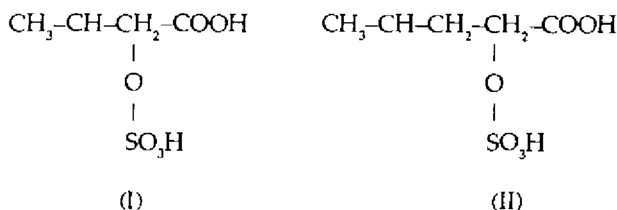
Route of administration	Sex	Concentration (%)	
		Urine	Faeces
Oral (30 mg/kg body weight)	Male	68.3	25.9
	Female	74.0	20.3
Subcutaneous (1 mg/kg)	Male	63.8	12.5
	Female	64.3	9.2

From Cresswell et al. (1978); values are average rates of excreted radioactivity during the 120-h period after a single dose.

When sodium ^{35}S-dodecylbenzenesulfonate (3.3 mmol/kg body weight) was administered in the diet to young pigs, at least 35% of the dose was absorbed through the intestinal tract. After 40 h, 30–40% of the dose had been excreted in urine and > 60% in faeces. The concentration of radiolabel after 200 h was relatively high in bristles and bones and low in liver, kidney, and spleen (quantitative data not presented). After 10 weeks, traceable amounts of ^{35}S (0.05% of the administered dose) were found in bristles, bones, skin, lung, and brain (Havermann & Menke, 1959).

A6.2 Biotransformation

The main metabolites isolated from the urine of rats administered ^{35}S-LAS orally were probably a mixture of sulfophenyl butanoic (I) and sulfophenyl pentanoic acids (II):



The material used in the experiment was a mixture of C_{10}–C_{14} LAS (mainly C_{11}, C_{12}, and C_{13}). The compounds in this mixture are probably degraded by ω-oxidation, followed by catabolism through a β-oxidation mechanism to form the above metabolites, with excretion of four or five carbons in the urine (Michael, 1968).

After oral administration of the calcium or sodium salt of ^{14}C-LAS to rats, two metabolites were detected in urine and four in faeces by thin-layer chromatography. The two urinary and two of the faecal metabolites were believed to be compounds similar to metabolites (I) and (II) previously identified by Michael (1968) (Sunakawa et al., 1979).

Thin-layer chromatography of urine extracts after oral or subcutaneous administration of ^{14}C-LAS to rhesus monkeys showed only trace amounts of the unchanged compound, and five metabolites more polar than LAS were detected. These metabolites have not been identified. Incubation of urine samples with β-glucuronidase or sulfatase did not affect the components, which were therefore probably not present as the corresponding conjugates (Cresswell et al., 1978).

A7. EFFECTS ON LABORATORY MAMMALS AND *IN VITRO* TEST SYSTEMS

Section summary

The oral LD_{50} values for sodium salts of LAS are 404–1470 mg/kg body weight in rats and 1259–2300 mg/kg body weight in mice. LAS irritate skin and eyes.

Minimal effects, including biochemical alterations and histopathological changes in the liver, were reported in subchronic studies in rats administered LAS in the diet or drinking-water at concentrations equivalent to a dose of about 120 mg/kg body weight per day. Although ultrastructural changes in liver cells were observed at lower doses in one study, these changes appeared to be reversible. Effects have not been seen at similar doses in other studies, but the organs may have been examined more closely in this study. Reproductive effects, including decreased pregnancy rate and litter loss, have been reported in animals administered doses > 300 mg/kg body weight per day. Histopathological and biochemical changes have been observed following long-term dermal application on rats of solutions of LAS at concentrations > 5% and after 30 days' dermal application on guinea-pigs of 60 mg/kg body weight. Repeated dermal application of solutions containing ≥ 0.3% LAS induced fetotoxic and reproductive effects, although these doses also induced maternal toxicity.

The available long-term studies are inadequate to evaluate the carcinogenic potential of LAS in experimental animals, owing to the small number of animals used, low or insufficient doses tested, the absence of a maximal tolerated dose, and limited histopathological examination. The limited studies available in which animals were administered LAS orally, however, provide no evidence of carcinogenicity.

Limited data also indicate that LAS are not genotoxic *in vivo* or *in vitro*.

A7.1 Single exposures

The LD_{50} values for the sodium and magnesium salts of LAS given orally, subcutaneously, or intravenously are summarized in Table 17. Rats appear to be more sensitive than mice to LAS, regardless of the

route of exposure. The LD_{50} values for LAS given orally were 1259–3400 mg/kg body weight in mice and 404–1900 mg/kg body weight in rats. Differences were seen according to the sex, strain, and age of the animals and the test material.

Table 17. Acute toxicity of linear alkylbenzene sulfonates

Species/ strain	Sex	Route	LD_{50}[a] (mg/kg body weight)	Test material[b]	Reference
Mouse					
NR	NR	Oral	2170	60% active ingredient	Yanagisawa et al. (1964)
DD	M	Oral	2300	34.55% solution	Tiba (1972)
ddY	M	Oral	1665	Purified	Kobayashi et
ICR-JCL	F	Oral	1950	Purified	al. (1972)
	M	Oral	1250	Commercial soln, 19.0%	Kuwano et al.
	F	Oral	1540	Commercial soln, 19.0%	(1976)
	M	Oral	1370	Commercial soln, 17.1%	
	F	Oral	1560	Commercial soln, 17.1%	
	M	Oral	2160	99.5% active ingredient of C_{10}–C_{13}	Ito et al. (1978)
	F	Oral	2250	99.5% active ingredient of C_{10}–C_{13}	
	M	Oral	2600	Magnesium salt of above	
	F	Oral	3400	Magnesium salt of above	
	M	s.c.	1250	99% active ingredient of C_{10}–C_{13}	Ito et al. (1978)
	F	s.c.	1400	99% active ingredient of C_{10}–C_{13}	
	M	s.c.	1529	Magnesium salt of above	
	F	s.c.	1550	Magnesium salt of above	
	M	i.v.	207	99% active ingredient of C_{10}–C_{13}	
	F	i.v.	298	99% active ingredient of C_{10}–C_{13}	
	M	i.v.	98	Magnesium salt of above	
	F	i.v.	151	Magnesium salt of above	
NR	NR	i.v.	120		Yanagisawa et al. (1964)
Rat					
FDRL	M,F	Oral	650	Nominal chain length, C_{12} (range C_9–C_{15})	Oser & Morgareidge (1965)

Table 17 (contd)

Species/ strain	Sex	Route	LD_{50}[a] (mg/kg body weight)	Test material[b]	Reference
Wistar					
6 w	M	Oral	873	Purified	Kobayashi et al. (1972)
6 w	F	Oral	760		
10 w	M	Oral	404		
10 w	F	Oral	409		
	M	Oral	1460	99.5% active ingredient of C_{10}–C_{13}	Ito et al. (1978)
	F	Oral	1470	99.5% active ingredient of C_{10}–C_{13}	
	M	Oral	1900	Magnesium salt of above	
	F	Oral	1840	Magnesium salt of above	
CRJ-SD	M	s.c.	840	99.5% active ingredient of C_{10}–C_{13}	
	F	s.c.	810	99.5% active ingredient of C_{10}–C_{13}	
	M	s.c.	710	Magnesium salt of above	
	F	s.c.	730	Magnesium salt of above	
	M	i.v.	119	99.5% active ingredient of C_{10}–C_{13}	
	F	i.v.	126	99.5% active ingredient of C_{10}–C_{13}	
	M	i.v.	27.2	Magnesium salt of above	
	F	i.v.	35.0	Magnesium salt of above	

NR, not reported; M, male; F, female; s.c., subcutaneous; i.v., intravenous; w, weeks

[a] As active ingredient

[b] Sodium salt, unless specifically indicated

The main clinical signs observed after oral administration of doses near or greater than the LD_{50} consisted of reduced voluntary activity, piloerection, diarrhoea, and weakness. Diarrhoea was more severe in rats than mice (Kobayashi et al., 1972). Convulsions, torsion, and paralysis of the hind limbs were also observed in some of mice (Kobayashi et al., 1972; Kuwano et al., 1976). Death usually occurred within 24 h of administration. Transient cardiac arrest, dyspnoea, cyanosis, respiratory collapse, and death occurred during intravenous injection (Ito et al., 1978).

At autopsy, hyperaemia and haemorrhage of the stomach and intestine, bloating of the intestine with thinning of its wall, and congestion of some internal organs were the main macroscopic findings; histological examination showed congestion and epithelial degeneration of the gastrointestinal mucosa (Kobayashi et al., 1972; Kuwano et al., 1976; Ito et al., 1978).

A7.2 Short-term exposure

A7.2.1 Mouse

In a study of the toxicity of a commercial preparation of LAS (17.1% active ingredient), 44 male and 16 female C57Bl TW mice were given subcutaneous injections according to the following schedule: 0.02 ml of 1% of the preparation for 10 consecutive days from the day of birth, 0.04 ml of the same solution for the following 10 days, 0.02 ml of a 10% solution five times over the next 10 days, and 0.04 ml of the same solution every other day for a further 30 or 60 days. Eight males and six females served as untreated controls. Epilation and dermatitis usually occurred in animals given continuous injections of the test material. Adhesions between some organs, most frequently between the spleen and kidney, were observed in those receiving injections from the day of birth. Neither the growth nor the survival of the animals was affected. Although the weights of the liver, kidney, and spleen were significantly increased in animals receiving treatment for 60 days, histopathological examination of the liver, kidney, adrenal glands, and thyroid by light and electron microscopy showed no evidence of toxicity (Kikuchi, 1978).

A7.2.2 Rat

A7.2.2.1 Administration in the diet

Groups of five male Wistar rats were fed diets containing LAS (60% active ingredient; chain length distribution: 10.6% C_{10}, 34.1% C_{11}, 27.7% C_{12}, 19.0% C_{13}, 8.7% C_{14}) at a concentration of 0, 0.6, 1.2, or 1.8% (equivalent to 180, 360, or 540 mg/kg body weight per day) for two and four weeks, and lipids in serum and liver were analysed. Body weight gain was suppressed in the group receiving 1.8% at four weeks, and the relative liver weight was increased at two weeks and thereafter in the groups receiving 1.2 and 1.8%. The levels of triglyceride and total lipids in the serum had decreased markedly at two weeks in all the experimental groups, and the levels of phospholipids and cholesterol in the serum had decreased significantly at two weeks in the groups given 1.2 and

1.8%. These changes were less apparent at four weeks, but triglyceride, phospholipid, and cholesterol levels in serum were significantly decreased in the group given 1.8%. Significant increases in triglyceride levels were seen in the liver after two weeks in the groups receiving 0.6 and 1.8%, and in cholesterol levels in the group given 0.6% (Yoneyama & Hiraga, 1977).

Technical-grade sodium LAS (87.9% active ingredient; chain length distribution: 1.8% C_{10}, 43.2% C_{11}, 32.2% C_{12}, 5.3% C_{14}, 1.5% C_{15}) were fed to five groups of 10 weanling Sprague-Dawley rats of each sex at a dietary level of 0, 0.02, 0.1, or 0.5% (equivalent to 8.8, 44, or 220 mg/kg body weight per day) for 90 days. No adverse effects were found on survival, growth, food conversion efficiency, haematological values, urinary analytical values, or absolute or relative organ weights. There were no gross or microscopic histological changes attributable to ingestion of the test material (Kay et al., 1965).

Technical-grade LAS (normal chain length, C_{12}; range, C_9–C_{15}; mean relative molecular mass, 346) were fed to three groups of weanling FDRL rats, each consisting of 15 males and 15 females, at a dose of 0, 0.05, or 0.25 g/kg body weight per day for 12 weeks. No adverse effects were noted on survival, behaviour, growth, food conversion efficiency, haematological measurements, blood chemistry, urine analytical values, organ weights, or gross or microscopic appearance, except for a slight increase in liver weight in females given 0.25 g/kg body weight per day (Oser & Morgareidge, 1965).

A diet containing LAS at a concentration of 1.5% (equivalent to 750 mg/kg body weight per day) or a control diet was given to groups of five male Wistar rats for 2, 4, or 12 weeks. LAS depressed body weight gain, and the relative liver weight was significantly increased after two weeks of treatment. The activities of alkaline phosphatase and glutamate-pyruvate transaminase in serum were significantly increased at each observation period, and cholesterol and protein levels were significantly decreased by four weeks. In the liver, the activities of glucose-6-phosphatase and glucose-6-phosphate dehydrogenase were decreased, and the activity of isocitrate dehydrogenase was increased at each observation point. Enzymatic examination of the renal cortex showed decreased activities of glucose-6-phosphatase and 5′-nucleotidase at each observation period, an increase in the activity of lactate dehydrogenase at 12 weeks, and increased activity of isocitrate dehydrogenase at 2 and 4 weeks. In the renal medulla, the activity of Na,K-ATPase was decreased, that of lactate dehydrogenase was

increased at 12 weeks, and that of isocitrate dehydrogenase was decreased at 2 weeks but increased at 12 weeks (Ikawa et al., 1978).

Groups of five male Wistar rats were given a diet or drinking-water containing LAS at a concentration of 0.4% (diet: 200 mg/kg body weight per day; drinking-water: 560 mg/kg per day) for two weeks in order to determine the effects of LAS on the synthesis of lipids in the liver. Lipids were thus measured in the liver, and uptake of acetate-1-^{14}C by the lipids was examined. Decreases in the levels of total lipids and triglyceride were seen in both groups, but there were no significant changes in phospholipid or cholesterol levels. Uptake of acetate-1-^{14}C by lipids in the liver was increased in both groups; uptake of phospholipids and triglycerides tended to increase, and that of phospholipids increased significantly in rats given LAS in the diet (Yoneyama et al., 1978).

A7.2.2.2 *Administration by gavage*

Groups of 12 male and 12 female Sprague-Dawley rats were given the magnesium salt of LAS by gavage at a dose of 0, 155, 310, or 620 mg/kg body weight for one month. Body weight gain was depressed in males and females at 620 mg/kg body weight; one male and two females at this dose also had diarrhoea and loss of appetite and subsequently died. Haematological examination revealed significant decreases in haemoglobin concentration and haematocrit in males at 620 mg/kg body weight. A significant increase in the activity of alkaline phosphatase and a significant decrease in calcium levels were seen in males at 310 or 620 mg/kg body weight; and a significant increase was seen in the activity of glutamate-oxalate transminase and a significant decrease in protein levels in females at those doses. Females at all doses had a significant decrease in calcium levels. At the highest dose, females had a significant increase in the activity of alkaline phosphatase, a significant decrease in cholesterol level, and increased weight of the liver, but the weight of the thymus decreased. The weight of the heart decreased in females at 310 and 620 mg/kg body weight. Histological examination of the liver revealed no abnormalities (Ito et al., 1978).

Groups of 12 male and 12 female Sprague-Dawley rats were given the sodium salt of LAS (chain length distribution: < 0.1% C_9, 10.1% C_{10}, 33.7% C_{11}, 31.0% C_{12}, 25.1% C_{13}) at a dose of 0, 125, 250, or 500 mg/kg body weight by gavage once a day. Diarrhoea was observed in the group receiving 500 mg/kg, and soft faeces were observed in the other two groups. Body weight gain was depressed in males of all groups and in females at 500 mg/kg. Haematological examination revealed no

abnormalities. Serum analysis revealed a significant increase in the activity of alkaline phosphatase in males at 500 mg/kg, a significant decrease in calcium levels in males of all groups, significant increases in the activity of gluatamate–oxalate transaminase and in blood–urea nitrogen in females at 500 mg/kg, a significant decrease in calcium level in females at 250 or 500 mg/kg, and significantly decreased protein and albumin levels in females of all groups. At 500 mg/kg, the weights of spleen and heart were significantly decreased in males; in females, liver weights were increased but the weights of the heart and thymus were decreased. No histological abnormalities were seen in the liver (Ito et al., 1978).

A7.2.2.3 *Dermal application*

Continued, repeated, or extremely high doses of LAS, like other detergents, compromise the integrity of the skin so that penetration occurs, causing a variety of anomalies. As the design of the following two studies was not adequate, the observations are not considered to be relevant to human risk assessment.

Application of 2 ml of a commercial preparation of LAS (23.4% active ingredient) to the thoracic skin of six male Wistar rats resulted in redness and wrinkling of the skin after 24 h. The redness then increased, the corium was lacerated, and bleeding occurred. These effects were most severe after five to seven days, but after a further 10 days the skin began to recover. Six rats died after 19 days, probably because of the extremely high dose used. The livers of three rats were examined by electron microscopy after three and 30 days and the findings compared with those in the control group. At three days, marked changes were seen in the components of the liver parenchymal cells, such as separation of the intracellular space, appearance of dark cells with high electron density, dysmorphia of mitochondria, extracellular prolapse of mitochondria, proliferation of rough-surfaced endoplasmic reticulum, lysosome proliferation, and a decrease in the prevalence of fatty droplets. At 30 days, many liver parenchymal cells were filled with abnormally divided and proliferated mitochondria, and an abnormal increase in smooth-surfaced endoplasmic reticula was noted. There were no granules of glycogen or fatty droplets. Structures resembling necrotic cells were also observed (Sakashita et al., 1974).

A commercial preparation of LAS (23.4% active ingredient) was applied dermally to male rats (number not given) at a dose of 5 mg/kg body weight active ingredient once a day for 30 days, and the liver was examined by electron microscopy. Degeneration was seen in part of the liver, in the form of atrophy and high density. Intra-mitochondrial

deposits and deformation of the Golgi apparatus were also noted (Sakashita, 1979).

A7.2.2.4 Subcutaneous injection

A commercial preparation of LAS (27% active ingredient) was given subcutaneously to groups of five male and five female Wistar rats at a dose of 2 ml/kg body weight per day of a 0, 0.02, 0.2, or 2% solution of the preparation for 25 or 50 days. Rats receiving the 2% solution had reduced body weight gain, increased weights of liver, kidney, and spleen, a low serum albumin:globulin ratio, low serum protein, and reduced ornithine aminotransferase activity in the liver (Hayashi, 1980).

A7.2.3 Guinea-pig

Twelve guinea-pigs were treated daily for 30 days with a solution of LAS in distilled water equivalent to 60 mg/kg body weight, which was applied to a 4-cm^2 area of clipped dorsal skin. Twelve controls received acetone at 0.5 ml. The animals were sacrificed after 30 days, and samples were taken from liver and kidney and homogenized for determination of enzymes, lipid peroxidation, glutathione, and protein. The activities of ß-glucuronidase, γ-glutamyl transpeptidase, 5-nucleotidase, and sorbitol dehydrogenase were increased in liver and kidney. Lipid peroxidation was increased in kidney but not in liver, and the glutathione content was unchanged in both organs. Extensive fatty changes were found in hepatic lobules, with dilation of sinusoids; tubular lesions were found in the kidney, predominantly in the proximal and distal portions (Mathur et al., 1992).

A7.2.4 Monkey

LAS (chain length, C_{10}–C_{13}) were given to four groups of three male and three female rhesus monkeys at a daily dose of 0, 30, 150, or 300 mg/kg body weight orally simultaneously with a dose of 0, 0.1, 0.5, or 1.0 mg/kg per day subcutaneously, for 28 days. Monkeys that received 300 mg/kg orally and 1.0 mg/kg subcutaneously vomited frequently, usually within 3 h of administration; these animals and those given 150 mg/kg orally and 0.5 mg/kg subcutaneously also had an increased frequency of loose or liquid faeces. Fibrosis at the injection sites was reported in all test animals, and the incidence and severity were related to dose. Treatment had no effect on ophthalmoscopic, haematological, or urinary parameters, on organ weight, or on histopathological appearance (Heywood et al., 1978).

The studies of short-term exposure to LAS are summarized in Table 18.

A7.3 Long-term exposure; carcinogenicity

A7.3.1 Mouse

A7.3.1.1 Administration in the diet

Groups of eight or nine ICR mice were given diets containing LAS at a concentration of 0.6 or 1.8% for nine months (corresponding to intakes of 500 and 1000 mg/kg body weight per day). There was no reduction in body weight gain at either dose, but the weight of the liver was increased in both males and females. Significant decreases were seen in the activities of hepatic lactate dehydrogenase and renal acid phosphatase in male mice (Yoneyama et al., 1976).

A7.3.1.2 Administration in the drinking-water

Drinking-water containing 100 ppm LAS (corresponding to 20 mg/kg body weight per day) was supplied to ddy mice (sex and number not stated) for six months, and they were then allowed to recover for two months. Mice were killed for electron microscopy of the liver at one, two, three, and six months and after the two-month recovery period. Hepatic damage was observed at one and six months, consisting of the disappearance of the nucleolus, atrophy of the Golgi apparatus, degranulation of rough-surfaced endoplasmic reticulum, degeneration of mitochondria, and increased numbers of primary and secondary lysosomes including autophagic vacuoles with a myelinated core. In mice examined after the two-month recovery period, some hepatic damage was seen, which was characterized by changes in mitochondrial structure and the presence of numerous fat droplets. Other cellular effects had reversed, indicating that the liver cells had recovered (Watari et al., 1977). Because an extremely high dose was used in this study, the observations have little relevance to human risk.

Groups of eight or nine ICR mice were given water containing LAS at a concentration of 0.07, 0.2, or 0.6% for nine months, corresponding to intakes of about 0.1, 0.25, or 0.6 g/kg body weight per day for males and 0.1, 0.25, or 0.9 g/kg body weight per day for females. Body weight gain was depressed in males and females at 0.6%, and there were dose-related increases in liver weight in females in all dose groups. In the group given 0.6% LAS, the activity of hepatic glutamate–oxalate

Table 18. Summary of studies of short-term exposure to linear alkylbenzene sulfonates (LAS)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Mouse, C57Bl TW 44 M, 16	LAS (a.i. 17.1%)	s.c.	63 or 76 mg/kg bw/day, 60–90 days	Abdominal adhesions, increased weights of liver, kidney, and spleen after 60-day treatment; no histopathological changes in liver, kidney, adrenal or thyroid glands	Kikuchi (1978)
Rat, Wistar 5 M	LAS, C_{10}–C_{14} (a.i. 60%)	Diet	0, 0.6, 1.2, 1.8%, 4 weeks	Decreased serum triglyceride, total lipids, phospholipids, and cholesterol; increased relative liver weight at 1.2 and 1.8%; suppression of body weight gain at 1.8%	Yoneyama & Hiraga (1977)
Rat, SD 10 M, 10 F	LAS, C_{10}–C_{15} (a.i. 8-9%)	Diet	0, 0.02, 0.1, 0.5%, 90 days	No adverse effects	Kay et al. (1965)
Rat, FDRL 15 M, 15 F	LAS, C_{9}–C_{15} (a.i. 39.5%)	Diet	0, 0.05, 0.25 g/kg bw per day, 12 weeks	Slight increase in liver weight in females at high dose	Oser & Morgareidge (1965)

Table 18 (contd)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Rat, Wistar 4 M	LAS (NS)	Diet	1.5%, 24 weeks	Increased activities of serum, hepatic, and renal enzymes; depressed body weight gain; increased relative liver weight	Ikawa et al. (1978)
Rat, CRJ-SD 12 M, 12 F	LAS, Na, C_{10}–C_{13} (a.i. 99.5%)	Gavage	125, 250, 500 mg/kg bw per day, 1 month	Altered serum enzyme activity and calcium levels at high doses; decreased serum protein and albumin levels in all treated females; decreased spleen and heart weights in males at highest dose; increased liver weight and decreased heart and thymus weights in females at highest dose; no histopathological abnormalities in liver	Ito et al. (1978)
Rat, CRJ-SD 12 M, 12 F	LAS Mg, C_{10}–C_{13} (a.i. 96.9%)	Gavage	155, 310, 620 mg/kg bw per day, 1 month	Altered haemoglobin, haematocrit, serum enzyme activities, calcium level at high doses; depressed body weight gain at highest dose; increased liver weight and decreased heart and thymus weights in females at highest dose; no histopathological abnormalities in liver	Ito et al. (1978)

Table 18 (contd)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Rat, Wistar 6 M	LAS detergent (a.i. 23.4%)	Dermal	2 ml/animal 3.5 x 4.5 cm, 30 days	Skin irritation; liver parenchymal changes with necrotic cells; no glycogen granules or fat droplets	Sakashita et al. (1974)
Rat, Wistar 6 M	LAS detergent (a.i. 23.4%)	Dermal	5 mg/kg bw, once/ day, 30 days	Degenerative changes in liver	Sakashita (1979)
Rat, Wistar 5 M, 5 F	LAS detergent (a.i. 27%)	s.c.	0, 0.02, 0.2, 2%, 2 ml/kg bw per day, 50 days	Depressed body weight gain; increased weights of liver, kidney, and spleen; and altered hepatic enzyme activities at highest dose	Hayashi (1980)
Rat, Wistar 8 M, 8 F	LAS (a.i. 60.2%)	Drinking-water	0.4%, 2 weeks	Decreased hepatic total lipids and triglycerides; increased uptake of acetate-1-^{14}C, phospholipids, and triglycerides	Yoneyama et al. (1978)
Guinea-pig 12 M, 12 F	LAS (NS)	Dermal	60 mg/kg bw, 30 days on 4 cm^2	Altered hepatic and renal enzyme activities; fatty degeneration in liver; renal tubular lesions	Mathur et al. (1992)

Table 18 (contd)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Rhesus monkey 3M, 3F	LAS C_{10}–C_{13} (a.i. 20.5%)	Gavage s.c.	0.30, 150, 300 mg/kg 0, 0.1, 0.5, 1.0 mg/kg bw per day, 28 days	Vomiting and diarrhoea; no ophthalmic, haematological or urinary changes; no effect on organ weights; no histopathological changes	Heywood et al. (1978)

M, male; F, female; a.i., active ingredient; s.c., subcutaneous

transaminase was significantly decreased in males and the activity of renal glucose-6-phosphatase was decreased in animals of each sex (Yoneyama et al., 1976).

A7.3.2 Rat

A7.3.2.1 Administration in the diet

LAS (98.1% active ingredient; chain length distribution, C_{10}–C_{14}) were fed to four groups of Charles River weanling rats, each consisting of 50 males and 50 females, at a dietary level of 0, 0.02, 0.1, or 0.5% (corresponding to 10, 50, or 250 mg/kg body weight per day) for two years. No adverse effects on growth or feed conversion efficiency were observed. Five males and females from each group were killed at 8 and 15 months, and all survivors at 24 months; all animals were necropsied, haematological values were determined, and tissues were taken for histological examination. No consistent change was seen that could be considered a toxic response. Animals that showed significant loss of weight, development of tumours, or other evidence of abnormalities were also sacrificed and their tissues preserved for study. The incidences of tumours and of common incidental diseases were similar in all dietary groups (Buehler et al., 1971).

Diets containing technical-grade LAS (chain length distribution: 10.6% C_{10}, 34.1% C_{11}, 27.7% C_{12}, 19.0% C_{13}, 8.7% C_{14}; mean relative molecular mass, 345.8) at a concentration of 0, 0.07, 0.2, 0.6, or 1.8% were given to groups of 10 Wistar rats of each sex for six months. The group given 1.8% had diarrhoea, markedly depressed growth, increased caecal weight, and marked degeneration of renal tubules. The group given 0.6% had slightly depressed growth, increased caecal weight, increased serum alkaline phosphatase activity, decreased serum protein, and degeneration of renal tubules. The group given 0.2% had increased caecal weight and slight degeneration of renal tubules. The group given 0.07%, corresponding to about 40 mg/kg body weight per day, showed no effects attributable to treatment (Yoneyama et al., 1972).

Groups of eight male and eight female Wistar rats were given diets containing LAS at a concentration of 0, 0.6, or 1.8% for nine months, corresponding to intakes of 230 or 750 mg/kg body weight per day for males and 290 or 1900 mg/kg body weight per day for females. In rats given 1.8% LAS, body weight gain was reduced in both males and females. Haematological examination revealed a significant decrease in leukocytes in males at 0.6% and significant decreases in mean corpuscular volume and mean corpuscular haemoglobin in females at

1.8%. The activity of glutamate–oxalate transferase and the levels of cholesterol and albumin in serum were significantly decreased and the activity of alkaline phosphatase and the levels of blood–urea nitrogen and cholinesterase were significant increased in males at 1.8%; females at that dose had a significant decrease in cholesterol level and a significant increase in alkaline phosphatase activity. At 0.6%, males had a significant decrease in glucose level, and females had a significant decrease in the activity of glutamate–pyruvate transaminase. The caecal weight of male rats and the liver and caecal weights of female rats at 1.8% were significantly increased. Enzymatic examination of the liver revealed dose-related decreases in the activities of glucose-6-phosphate dehydrogenase and lactate dehydrogenase in male rats. At 1.8%, males had significantly decreased activities of glucose-6-phosphatase, glutamate–pyruvate transaminase, and glutamate–oxalate transaminase and a dose-related decrease in the activity of glucose-6-phosphate dehydrogenase; females had significantly decreased activities of glucose-6-phosphatase and glutamate–oxalate transaminase. Enzymatic examination of the kidneys of females at 1.8% showed significantly decreased activities of glucose-6-phosphatase, Na,K-ATPase, and lactate dehydrogenase (Yoneyama et al., 1976).

Groups of 50 male and 50 female Wistar weanling rats were given diets containing LAS (10.6% C_{10}, 34.1% C_{11}, 27.7% C_{12}, 19.0% C_{13}, 8.7% C_{14}; mean relative molecular mass, 345.8) at a concentration of 0, 0.04, 0.16, or 0.6%. In each group, five rats of each sex were fed for one, three, six, or 12 months, and groups of 15 rats of each sex were fed for 24 months or more. The group fed 0.6% had slightly increased liver and caecal weights, and increased activity of glutamate–pyruvate transaminase and alkaline phosphatase in serum. The treatment had no adverse effect on the intake of food, body weight gain, general condition, mortality, or mean survival. On the basis of these results, it was concluded that a diet containing LAS at a concentration of 0.6% (300 mg/kg body weight per day) had no adverse effects on the rats (Yoneyama et al., 1977).

Groups of 50 male and 50 female Wistar rats were fed LAS (C_{10}–C_{14}) in the diet at a concentration of 0, 0.04, 0.16, or 0.6% and were then submitted to a detailed histopathological examination. After one month, proliferation of hepatic cells in the liver, slight swelling of the renal tubules, and narrowing of the tubular lumen were found in treated animals. Since these alterations later disappeared, they were considered to represent adaptation to the administration of LAS. No histological lesions were seen in the organs of rats that were fed for 24 months or

more that could be attributed to treatment. Various types of tumour were observed in both treated and control rats but did not appear to be due to LAS (Fujii et al., 1977).

A7.3.2.2 Administration in the drinking-water

Groups of eight to nine male and eight to nine female Wistar rats were given LAS at a concentration of 0, 0.07, 0.2, or 0.6% in drinking-water for nine months. Body weight gain was suppressed in males given 0.6%. Haematological examination revealed no significant change in any of the experimental groups, but a dose-related decrease in cholesterol level was seen in males. No change in organ weight was seen that was due to administration of LAS. Significant decreases in the activities of glutamate-oxalate transaminase and lactate dehydrogenase were seen in males at 0.2% and a dose-related increase in the activity of glutamate-oxalate transaminase in females. A significant decrease in renal Na,K-ATPase was seen in the group given 0.2%. The dose of 0.07% corresponded to intakes of LAS of 50 and 120 mg/kg body weight per day in males and females, and the dose of 0.2% to intakes of 120 and 170 mg/kg body weight per day, respectively (Yoneyama et al., 1976).

A commercial preparation of LAS (27% active ingredient) was given to groups of five male Wistar rats in drinking-water at a concentration of 0, 0.3, 3, 30, or 300 ppm (corresponding to 0.007, 0.07, 0.7, or 7 mg/kg body weight per day) for 60, 124, or 181 days. Although a reduction in body weight gain, changes in blood biochemistry, and increased ornithine aminotransferase activity in the liver were noted in some animals, they were not proportional to dose or feeding period (Hayashi, 1980).

Groups of 20 male Wistar rats were given water containing LAS (34.55% commercial solution) at a concentration of 0, 0.01, 0.05, or 0.1% for two years, the highest dose corresponding to an intake of about 200 mg/kg body weight per day. No changes attributable to the administration of LAS were seen in terms of growth, mortality, the weights of major organs, or histopathological appearance (Tiba, 1972).

A group consisting of 62 male and 62 female Wistar rats was given drinking-water containing LAS (mean relative molecular mass, 348; 38.74% active ingredient) at a concentration of 0.1% (corresponding to 140 mg/kg body weight per day), and a control group of 37 male and 37 females was given normal drinking-water. Five to 12 rats in the experimental group and three to 12 rats in the control group were killed at 3, 6, 12, and 18 months, and all surviving animals were killed at

24–26 months. Administration of LAS had no effect on the intake of water, mortality, body weight gain, or general condition. Histopathological examination revealed atrophy; fatty changes were found in hepatic cells in treated animals at six months, when there were also significant increases in the activities of glutamate–oxalate and glutamate–pyruvate transaminases and in the level of bilirubin. LAS had no effect on haematological parameters (Endo et al., 1980).

A group of 60 male and 60 female rats (strain not specified) received drinking-water containing 0.01% of a preparation containing 51% LAS for 100 weeks; a similar group was untreated. No detrimental effects on body weight and no pathological effects, including tumours, were reported (Bornmann et al., 1963).

A7.3.2.3 *Administration by gavage*

Groups of 20 male and 20 female Sprague-Dawley rats were given a solution of a magnesium salt of LAS at doses of 10, 75, 150, or 300 mg/kg body weight per day by gavage for six months. Body weight gain was suppressed, and slight decreases were observed in serum protein, albumin, and calcium ion level, but the changes were within the physiological range (Ito et al., 1978).

A7.3.2.4 *Dermal application*

A dose of 0.1 ml/kg body weight of a 0.5, 1.0, or 5.0% solution of magnesium LAS (in 3% polyethylene glycol) was applied to the backs of 20 male and 20 female Sprague-Dawley rats six times a week for six months. Slight redness at the application site was observed transiently in males and occasionally in females at 5%. Body weight was slightly suppressed in males at that dose, and one male in the control group and one at 5.0% died of unknown causes. Treatment had no definite effect in terms of food conversion efficiency, urinary, haematological, serum biochemistry, or histopathological findings, or organ weights (Ito et al., 1978). No systemic toxicity was reported in this study. Sakashita et al. (1974) and Sakashita (1979) (see section 7.2.2.3) may have obtained positive results because they used a shorter period of exposure, during which skin integrity may have been compromised, resulting in absorption of the preparation of LAS through the skin to produce systemic effects.

LAS (19.7% active ingredient) were applied to the dorsal skin of SLC-Wistar rats three times per week at a dose of 0.005, 0.025, or 0.125 ml/rat (equivalent to 1, 5, or 25 mg/rat) for 24 months. A dose of

0.025 ml of an LAS-based detergent containing 19.9% LAS (equivalent to 5 mg LAS per rat) and distilled water was given to controls. Each application was washed from the skin with warm water after 24 h. Treatment had no effect on organ weights or histopathological appearance, and there was no evidence of toxicity or carcinogenicity (Taniguchi et al., 1978).

Long-term studies of exposure to and the carcinogenicity of LAS are summarized in Table 19.

A7.4 Skin and eye irritation; sensitization

The potential of LAS to irritate the skin depends on the concentration applied. On the basis of the criteria of the European Commission and the OECD test guideline, LAS were classified as irritating to the skin at concentrations above 20% (European Committee of Organic Surfactants and Their Intermediates, 1990).

A7.4.1 Studies of skin

Solutions of LAS (chain length distribution, C_{10}–C_{13}; purity, 99.9%) were applied to the backs of groups of three male Wistar rats at a rate of 0.5 g of a 20 or 30% solution once a day for 15 days. On the sixteenth day of the experiment, the skin at the application site and the tissues of the tongue and oral mucosa (to examine the effects of licking) of the rats that received 30% were examined histologically. Body weight gain was reduced in the group exposed to 20%, and body weight was decreased in animals exposed to 30%. An infiltrating, yellow–red brown crust was observed after two to three days at 20% and after one to two days at 30%; at four to six days, the crust was abraded, and erosion was observed. Histological examination of the application site revealed severe necrosis of the region, from the epidermis cuticle to the upper layer of the dermis, severe infiltration of leukocytes in the necrotic site, diffuse inflammatory cell infiltration of all of the layers of the corium, and swelling of collagenous fibres in the dermis. Histological examination of the tongue showed no changes, but examination of the oral mucosa revealed atrophy and slight degeneration of the epithelium (Sadai & Mizuno, 1972).

Some batches of a paste of LAS (volume not stated) induced weak to moderate sensitization in guinea-pig skin at induction concentrations of 2–100% and challenge concentrations of 1–2%. A prototype liquid laundry detergent (10% LAS) induced sensitization at a challenge concentration of 1% (0.1% as LAS) (Nusair et al., 1988).

Table 19. Summary of studies of long-term exposure to linear alkylbenzene sulfonates (LAS)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Mouse, SLC-ICR 8-9 M, 8-9 F	LAS (a.i. 60%)	Diet	0, 0.6, 1.8%, 9 months	Increased liver weight; decreased hepatic and renal enzyme activities in males	Yoneyama et al. (1976)
Mouse, ddy (NR)	LAS (NS)	Drinking-water	20 mg/kg bw per day, 6 months end of treatment	Degenerative changes in liver, with partial recovery after	Watari et al. (1977)
Mouse, ICR 8-9 M, 8-9 F	LAS (a.i. 60%)	Drinking-water	0, 0.07, 0.2, 0.6, 1.8%, 9 months	Depressed body weight gain at high dose; dose-related increase in liver weight in all treated females; changes in hepatic enzyme activities at high dose	Yoneyama et al. (1976)
Rat, Wistar 10 M, 10 F	LAS, C_{10}–C_{14}	Diet	0, 0.07, 0.2, 0.6, 1.8%, 6 months	Dose-related depression of growth, caecal enlargement, and renal tubular degeneration at > 0.07%	Yoneyama et al. (1972)

Table 19 (contd)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Rat, Wistar 8 M, 8 F	LAS (a.i. 60%)	Diet	0, 0.6, 1.8%, 9 months	Depressed body weight gain at high dose; changes in haematological parameters, in serum and hepatic enzyme activities, and in cholesterol levels at both doses; changes in renal enzyme activities in females at high dose	Yoneyama et al. (1976)
Rat, Wistar 8–9 M, 8–9 F	LAS (a.i. 60%)	Drinking-water	0, 0.07, 0.2, 0.6%, 9 months	Depressed body weight gain in males at high dose; no changes in haematological parameters or organ weight; changes in serum and renal enzyme activities at 0.2%	Yoneyama et al. (1976)
Rat, Wistar 50 M, 50 F	LAS, C_{10}–C_{14} (a.i. 60%)	Diet	0, 0.04, 0.16, 0.6%, 24 months	Slight increase in liver and caecal weights and changes in serum enzym activities at high dose; no effect on body weight gain	Yoneyama et al. (1977)
Rat, Charles River 50 M, 50 F	LAS, C_{10}–C_{14} (a.i98.1%)	Diet	0, 0.02, 0.1, 0.5%, 2 years	No treatment-related effects	Buehler et al. (1971)

Table 19 (contd)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Rat, Wistar 50 M, 50 F	LAS, C_{10}–C_{14} (a.i. 60%)	Diet	0, 0.04, 0.16, 0.6%, 2 years	Transient changes in liver and kidney;no treatment-related histopathological abnormalities at end of study	Fujii et al. (1977)
Rat, SD 20 M, 20 F	LAS Mg, C_{10}–C_{13} (a.i. 96.9%)	Gavage	75, 150, 300 mg/kg bw per day, 6 months	Depressed body weight gain; no significant adverse effects	Ito et al. (1978)
Rat, Wistar, 5 M (a.i. 27%)	LAS detergent water	Drinking-	0, 0.3, 3, 30, 300 ppm, 181 days	Depressed body weight gain and changes in blood biochemistry and liver enzyme activity considered not to be related to treatment	Hayashi (1980)
Rat, Wistar, 20 M	LAS (a.i. 34.55%)	Drinking-water	0, 0.01, 0.05, 0.1%, 2 years	No adverse effects	Tiba (1972)
Rat, Wistar 62 M, 62 F	LAS (a.i. 38.74%)	Drinking-water	0, 0.1%, 26 months	Fatty changes and atrophy in liver; changes in hepatic enzyme activities; no effect on body weight gain	Endo et al. (1980)

Table 19 (contd)

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Rat 60 M, 60 F	LAS (Marlon BW 2043)	Drinking-water	0, 0.01%, 100 weeks	No adverse effects	Bornmann et al. (1963)
Rat, SD 20 M, 20 F	LAS Mg, C_{10}–C_{13} (a.i. 96.9%)	Dermal	0.5, 1.0, 5% in polyethylene glycol, 6 months	Slight reduction in body weight gain of males at high dose; no other adverse effects	Ito et al. (1978)
Rat, SLC-Wistar 25 M, 25 F	LAS (a.i. 19.7%)	Dermal	0, 6.7, 33.3, 167.0 mg/kg bw, 3 x per week, 2 years	No adverse effects	Taniguchi et al. (1978)
Rat, SLC-Wistar 25 M, 25 F	LAS detergent (a.i. 19.9%)	Dermal	0, 33.3 mg/kg bw 3 x per week, 2 years	No adverse effects	Taniguchi et al. (1978)

M, male; F, female; NS, not specified; a.i., active ingredient; SD, Sprague-Dawley

The biochemical and pathomorphological effects of LAS on the skin of four female albino CDRI guinea-pigs were investigated by shaving the abdominal skin and immersing the animals up to the neck in a 1% aqueous solution of neutralized LAS for 90 min daily for seven consecutive days. A control group was immersed in water according to the same schedule. After each immersion, the animals were washed and their skin dried. The animals were killed after seven days, and skin samples were taken. The skin of guinea-pigs exposed to the solution of LAS had increased activity of histidine decarboxylase, decreased sulfhydryl groups and histamine, and decreased activity of lactic dehydrogenase. It appeared to be shrunken, with thinner layers of dermis and epidermis than controls. There were also areas of scarring in the epidermis and ridging of epidermis and dermis (Misra et al., 1989a).

A7.4.2 *Studies of the eye*

A volume of 0.1 ml of a solution of LAS (relative molecular mass, 346.5) at five concentrations ranging from 0.01 to 1.0% was instilled into the eyes of rabbits (13 per group). The rabbits were observed for 24 h after application. The group receiving 0.01% had no abnormalities, but that given 0.05% had slight congestion. Concentrations of 0.5% and more induced marked reactions, such as severe congestion and oedema, increased secretion, opacity of the cornea, and disappearance of the corneal reflex (Oba et al., 1968a).

Solutions of LAS (chain length distribution, C_{10}–C_{14}; 80.9% C_{11}–C_{13}) at six concentrations ranging from 0.01 to 5.0% were instilled into the eyes of rabbits (three per group). The rabbits were observed for 168 h after application. The group given 0.01% had no reaction, but within 2 h those given 0.05% had slight congestion and those at 0.1% had considerable congestion or oedema, which had disappeared by 24 h. Animals given 0.5% or more had marked reactions, such as severe congestion and oedema, increased secretion, opacity of the cornea, and disappearance of the corneal reflex, for 24 h but then tended to recover; the signs had disappeared completely within 120 h (Iimori et al., 1972).

A7.5 Reproductive toxicity, embryotoxicity, and teratogenicity

The reproductive toxicity of LAS and formulations of LAS has been evaluated in studies by oral (gavage, diet, drinking-water), dermal (skin painting), and parenteral (subcutaneous) administration. Similar effects were seen, regardless of the route of application. The studies had a number of deficiencies, however, which are summarized below.

In some studies, widely separated dose levels were used (Palmer et al., 1975a; Takahashi et al., 1975; Tiba et al., 1976; Hamano et al., 1976), so that it is difficult to assess dose–response relationships and to interpret the results. Some of the studies included only one dose (Bornmann et al., 1963; Sato et al., 1972; Endo et al., 1980) and some two (Iimori et al., 1973; Nolen et al., 1975; Takahashi et al., 1975; Hamano et al., 1976; Tiba et al., 1976). The studies done on formulations are difficult to interpret, as the effects seen may have been due to another component. In some cases, the details of the formulation are not given, so that the dose of LAS is also unknown. Certain studies of dermal exposure (Sato et al., 1972; Masuda et al., 1973, 1974; Palmer et al., 1975a; Nishimura, 1976; Daly et al., 1980) involved levels that compromised the integrity of the skin and caused overt toxicity.

The teratogenic effects of some commercial formulations of LAS reported by Mikami and co-workers (1969), mainly in mice, were not reproduced in other studies. A number of studies indicated that LAS have some reproductive toxicity, but the effects were seen only at doses that caused maternal toxicity. No teratogenic effects were observed. These studies are summarized in Tables 20–22.

A7.6 Mutagenicity and related end-points

A7.6.1 *Studies in vitro*

Assays for mutagenicity were performed *in vitro* with two commercial products containing 17.1 and 19% LAS, either undiluted or diluted 10 and 100 times (Oda et al., 1977), 99.5% pure LAS (Fujita et al., 1977), 95.5% pure sodium salt, or 96.2% pure calcium salt (Inoue & Sunakawa, 1979), using *Bacillus subtilis* H17 (rec^+) and M45 (rec^-), *Salmonella typhimurium* TA98 and TA100 (including a metabolic activation system), and *Escherichia coli* WP2 *uvr*A. All of the assays gave negative results. LAS 99.5% pure (Fujita et al., 1977) were also tested in *S. typhimurium* TA1535 and TA1537, again with negative results. The sodium and calcium salts in the presence of various liver homogenates (Sunakawa et al., 1981) and a 22.2% solution of LAS (C_{10}–C_{14}, 10–200 µg/plate) (Inoue et al., 1980) were tested in *S. typhimurium* TA98 and TA100. No mutagenicity was seen.

A7.6.2 *Studies in vivo*

Groups of male ICR:JCL mice were given LAS at a dose of 200, 400, and 800 mg/kg body weight per day by gavage for five days and were killed 6 h after the final administration for examination of chromosomal

aberrations in bone-marrow cells. One commercial preparation containing 19.0% LAS was also given, at a dose of 800, 1600, or 3200 mg/kg body weight, and another containing 17.1% LAS at a dose of 1000, 2000, or 4000 mg/kg body weight once only by gavage. The highest doses were 50% of the respective LD_{50} values. Bone marrow was examined 6, 24 and 48 h after administration. There was no significant difference between any of the groups given LAS and the negative control group in the incidence of chromosomal aberrations. Mitomycin C, used as a positive control at 5 mg/kg body weight, induced severe chromosomal aberrations (Inoue et al., 1977).

Groups of five male Wistar rats, Sprague-Dawley rats, and ICR mice were given a diet containing 0.9% LAS for nine months. The equivalent doses were 450 mg/kg body weight per day in rats and 1170 mg/kg body weight per day in mice. There were no significant differences in the incidence of chromosomal aberrations between the experimental and control groups (Masubuchi et al., 1976).

After LAS (C_{10}–C_{15}) were fed to groups of six male and six female Colworth/Wistar rats in the diet at concentrations of 0.56 or 1.13%, equivalent to 280 or 565 mg/kg body weight per day, for 90 days, no alteratuons were seen in chromosomes in bone marrow (Hope, 1977).

In three male ddY mice given LAS at 100 mg/kg body weight by intraperitoneal injection, there was no differences between the treated animals and a control group in the incidence of polychromatic erythrocytes with micronuclei in bone-marrow cells (Kishi et al., 1984).

An assay to detect dominant lethal mutations was performed in seven male ICR:JCL mice given a diet containing 0.6% LAS at 300 mg/kg body weight per day for nine months. Each of the male mice was then mated with two female mice that had not been given LAS, and 11 of the 14 females became pregnant. The pregnant mice were laparotomized on day 13 of gestation to determine the numbers of luteal bodies, implantations, surviving fetuses, and dead fetuses. There were no significant differences in fertility, mortality of ova and embryos, the number of surviving fetuses, or the index of dominant lethal induction (Roehrborn) between the experimental and control groups (Masubuchi et al., 1976).

LAS were administered as a single oral dose of 2 mg to pregnant ICR mice on day 3 of gestation; on day 17 of gestation, each animal received a subcutaneous dose of 1, 2, or 10 mg/mouse and was killed

Table 20. Studies of the reproductive toxicity and teratogenicity of linear alkylbenzene sulfonates (LAS) and formulations of LAS, administered orally

Route	Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
LAS					
Diet	Charles River rats (20)	14, 70, 350 (0.02, 0.1, 0.5%)	84	Combined study of reproduction and teratogenicity (three generations); no effects attributable to LAS	Buehler et al. (1971)
Diet	SD rats (16)	78, 780 (0.1, 1.0%)	0–20	No abnormalities at either dose; few offspring at high dose	Tiba et al. (1976)
Gavage	ICR mice (NS)	300, 600	6,8,10	High incidence of cleft palate and exencephaly in fetuses at high dose	Mikami et al. (1969)
Gavage	ICR mice (14)	40, 400 (0.4, 4.0%)	0–6 7–13	No effects at low dose; reduced weight gain and pregnancy rate at high dose	Takahashi et al. (1975)
Gavage	ICR mice (25–33)	10, 100, 300	6–15	Reduced weight gain at all levels, particularly at highest dose; two dams died at highest dose; all fetuses of one dam died *in utero*; decreased body weight and delayed ossification in living fetuses but no increase in incidence of malformations	Shiobara & Imahori (1976)

Table 20 (contd)

Route	Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
LAS (contd).					
Gavage	ICR mice	14, 20, 350	1–3	No effect on implantation rate at any dose	Koizumi et al. (1985)
Gavage	CD rats (20) CD-1 mice (20) NZW rabbits (13)	0.2, 2.0, 300, 600	6–15, rats and mice 6–18, rabbits	No effects on any species at two lower doses Rats: reduced weight gain and one death at highest dose Mice: reduced weight gain, seven deaths, and four litter losses at 300 mg/kg bw per day; 18 deaths, one litter loss and one non-pregnancy at 600 mg/kg bw per day Rabbits: reduced weight gain, 11 deaths, two litter losses at 300 mg/kg bw per day; all animals died at highest dose	Palmer et al. (1975a)
Gavage	CD rats (30)	125, 500, 2000	6–15	Two-generation study of reproductive and developmental toxicity; delayed ossification significant at highest dose, slight at middle dose; no reproductive or developmental toxicity	Robinson & Schroeder (1992)

Table 20 (contd)

Route	Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
LAS (contd).					
Drinking-water	Charles River rats (10)	7 (0.01%)		Three-generation study of fertility; no teratogenic effects	Bornmann et al. (1963)
Drinking-water	Wistar rats (20)	70 (0.1%)		Four-generation study of reproductive toxicity; no effects attributable to LAS	Endo et al. (1980)
Drinking-water	Wistar rats (20) NZW rabbit (11)	383 mg/rat (0.1%) 3030 mg/rabbit (0.1%)	6–15 6–18	No effects in rats; rabbits had reduced weight gain and delayed ossification but no malformations	Endo et al. (1980)
17% LAS, 7% alcohol ethoxylate sulfate					
Gavage	CD rats (20) CD-1 mice (20)	0.8, 8, 1,200, 2400 1.064, 10.64, 1600, 320	6–15 6–15	No increase in major malformations or significant changes in anomalies	Palmer et al. (1975a)
	NZW rabbits (13)	0.8, 8, 1200, 2400	6–18		
45% LAS					
Diet	CD rats (25)	80, 400, 800 (0.1, 0.5, 1.0%)	6–15	No treatment-related effects on reproduction or embryonic development	Nolen et al. (1975)

Table 20 (contd)

Route	Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
1% LAS					
Gavage	ICR mice (18–23)	800, 1200, 1500, 3000	6–15	No increase in fetal malformations; decreased body weight and delayed ossification at 1200 mg/kg bw	Yamamoto et al. (1976)
19% LAS					
Gavage	IRC mice (9–13)	125, 4000	6	No effect on fetal viability or development	Hamano et al. (1976)

NS, not specified

Table 21. Studies of the reproductive toxicity and teratogenicity of linear alkylbenzene sulfonates (LAS) and formulations of LAS, administered dermally

Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
LAS				
CD rats (20)	0.6, 6.0, 60 (0.03, 0.3, 3.0%)	1–15	Slight reduction in body weight gain at highest dose; no effect on litter parameters at any dose; no evidence of malformations	Palmer et al. (1975a)
CD-1 mice (20)	5, 50 , 500 (0.03, 0.3, 3.0%)	2–13	Reduced body weight gain, fewer pregnancies, and total litter loss at highest dose; no malformations	
NZW rabbits (13)	0.9, 9, 90 (0.03, 0.3, 3.0%)	1–16	Marked reduction in body weight gain, fewer pregnancies, and two litter losses at highest dose; reduced body weight gain at 9 mg/kg bw per day; no malformations	
Wistar rats (20)	20, 100, 400 (1, 5, 20%)	0–20	Reduced body weight gain, decreased pregnancy rates and delayed ossification at highest dose; no effects at lower doses	Nishimura (1976)
Wistar rats (20)	20, 100, 400 (1, 5, 20%); rinse-off	0–20	Irritation at site and reduced body weight gain at two higher doses; no change in fetal parameters at any level	Daly et al. (1980)

Table 21 (contd)

Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
Wistar rats (contd)				
	0.1, 2, 10 (0.05, 0.1, 0.5%); leave on	0–20	No change in fetal parameters at any level	
ddy/s mice (16)	110 (2.22%)	0–13	No abnormalities in dams or fetuses	Sato et al. (1972)
ddy mice (4–10)	0.084, 0.84, 8.4 (0.017, 0.17, 1.7%)	2–14	No fetal or reproductive effects	Masuda et al. (1973, 1974)
ICR mice (25–30)	4.2, 8.4, 12.0, 16.5 (0.85, 1.7, 2.55, 3.4%)	1–13	Delayed ossification at two highest doses	
ICR mice (27–28)	15, 150, 1500 (0.03, 0.3, 3.0%)	6–15	Clear decrease in pregnancy rate and decrease in fetal weight at highest dose; no increase in malformations in fetus	Imahori et al. (1976)
17% LAS, 7% ethanol, 15% urea				
ICR mice (11–20)	2.5, 25, 75 (0.5, 5, 15%)	1–13	Decrease in pregnancy rate at highest dose; no other effects	Inoue & Masuda (1976)

Table 21 (contd)

Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
16.3% LAS				
ICR mice (17-50)	25, 50, 100 (5, 10, 20%)	0–13	Reduced pregnancy rate and some total litter losses at highest dose	Nakahara et al. (1976)
Unknown formulation				
ddy/s mice (21)	65 (15%)	0–13	Decreased body weight gain, decreased pregnancy rate, decreased fetal weight, and delayed ossification	Sato et al. (1972)
Unknown formulation				
IRC mice (27-39)	75, 100 (15, 20%)	0–12	Decreased pregnancy rates at both levels	Iimori et al. (1973)
Unknown formulation				
IRC mice (15–19)	30, 65, 85, 100, 125 (13.0, 17.0, 20.0, 25.0%)	0–13	Decreased pregnancy rates at all doses; decreased fetal body weight; delayed ossification at all doses except 65 mg/kg bw per day	Takahashi et al. (1975)

Table 22. Studies of the reproductive toxicity and teratogenicity of linear alkylbenzene sulfonates (LAS) and LAS formulations, administered subcutaneously

Species (no. of animals/group)	Dose (mg/kg bw per day)	Length of treatment (days)	Comments and results	Reference
LAS				
ICR mice (21–24)	0.4, 2.0, 10%	7–13	No significant effects on dams; high incidence of skeletal variations and delayed ossification, not dose-related; no abnormalities	Masuda & Inoue (1974)
ICR mice (12–19)	20, 200 (0.35, 1.00%)	0–3 8–11	Irritation at injection site and reduced pregnancy rate at highest dose; no malformations or anomalies	Takahashi et al. (1975)
17% LAS, 7% ethanol, 15% urea				
CR mice (16–17)	30, 150	7–13 0–13	No increase in major malformations or minor anomalies; increase in implantations at high dose given on days 0–13	Inoue & Masuda (1976)

24 h later. There was no difference among treated groups in the incidence of polychromatic erythrocytes with micronuclei in maternal bone marrow or fetal liver or blood. No mutagenic effect was found in any of the groups (Koizumi et al., 1985).

A7.7 Special studies

A7.7.1 Studies in vitro

The haemolytic action of LAS was investigated by mixing red blood cells from rabbits with solutions of LAS at concentrations of 1–1000 mg/litre at 38 °C for 30 min. Haemolysis occurred at concentrations ≥ 5 mg/litre (Yanagisawa et al., 1964). Red blood cells from rabbits were mixed with solutions of various concentrations of LAS (relative molecular mass, 346.5) at room temperature for 3 h. The 50% haemolytic concentration of LAS was 9 mg/litre (Oba et al., 1968a).

Purified LAS at various concentrations were added to 10 µl of normal plasma obtained from male rats, and prothrombin time was determined. Prothrombin time was prolonged; the 50% inhibitory concentration was about 0.6 mmol/litre. When LAS at various concentrations were added to a mixture of 1% fibrinogen and thrombin, the time of formation of a mass of fibrin was prolonged by inhibition of thrombin activity. The 50% inhibitory concentration was about 0.05 mmol/litre (Takahashi et al., 1974).

LAS influenced the thermal denaturation and decreased the fluorescence profile of bovine serum albumin *in vitro*, indicating protein–LAS interaction (Javed et al., 1988).

Eggs from female B6C3F1 mice were fertilized *in vitro* and incubated in culture medium containing LAS at concentrations between 0.015 and 0.03%; eggs grown in culture medium without LAS served as controls. Eggs exposed for 1 h, washed, and then cultured for five days developed normally to the blastocyst stage when the concentration of LAS was less than 0.025%; at concentrations higher than 0.03%, the eggs did not develop beyond the one-cell stage. With continuous exposure to LAS for five days, a concentration of 0.01% slightly impaired development to the blastocyst stage, and 0.025% prevented development to the one-cell stage (Samejima, 1991).

LAS with a chain length distribution of C_{10}–C_{14} did not induce transformation of cryopreserved primary cultures of Syrian golden hamster embryo cells *in vitro* (Inoue et al., 1979, 1980).

A7.7.2 Biochemical effects

The levels of amylase, alkaline phosphatase, glutamate–oxalate transaminase, and glutamate-pyruvate transaminase and of the electrolytes Ca, P, and Mg in serum were determined up to 24 h after a single oral administration of 2, 5, 50, or 100 mg/kg body weight of LAS (60% active ingredient) or dermal application of 5 ml of a 1, 5, 10, or 20% solution of LAS to rabbits (number not stated). The levels of total Ca, Ca^{2+}, Mg, and P were generally lower after either type of administration than before. Although there was no definite trend, the activities of the enzymes tended to decrease regardless of the route of the administration or the dose (Yanagisawa et al., 1964).

Groups of three male mice were given an intraperitoneal injection of 0.3 g/kg body weight of LAS (C_{14}) in order to study the effects on the formation of methaemoglobin, determined 0.5, 1, and 2 h afterinjection of LAS. The level of methaemoglobin in the experimental groups was not significantly greater than that in the control group at any time (Tamura & Ogura, 1969).

The effects of LAS (sodium dodecylbenzenesulfonate) on fasting blood glucose level and glucose tolerance curves were investigated in 40 male and 50 female albino rats pretreated with 0.25 g/kg body weight per day of LAS for three months. At the end of this period, the rats were divided into four groups and given distilled water, 6.1 g/kg body weight of glucose, 0.94 g/kg body weight of LAS, or 6.1 g/kg body weight of glucose plus 0.94 g/kg body weight of LAS by gavage. Blood glucose was then estimated at 30-min intervals. Administration of LAS in conjunction with glucose resulted in higher initial levels of blood glucose in male rats and persistently higher levels in females than did administration of glucose alone. Females in control and pretreated groups generally had higher blood glucose levels in response to administration of glucose or LAS plus glucose than did male rats (Antal, 1972).

A8. EFFECTS ON HUMANS

Section summary

Human skin can tolerate contact with solutions of up to 1% LAS for 24 h with only mild irritation. Like other surfactants, LAS can delipidate the skin surface, elute natural moisturizing factor, denature the proteins of the outer epidermal layer, and increase permeability and swelling of the outer layer. LAS do not induce skin sensitization in humans, and there is no conclusive evidence that they induce eczema. No serious injuries or fatalities have been reported following accidental ingestion of LAS-containing surfactant preparations.

A8.1 Exposure of the general population

Surface-active agents are used in shampoos, dish-washing products, household cleaners, laundry detergents, and other applications such as industrial cleaners. LAS are major components of such products. In general, the concentration of nonionic and ionic surfactants is 10–20%.

A8.2 Clinical studies

A8.2.1 Skin irritation and sensitization

LAS are mildly to moderately irritating to human skin, depending on the concentration. There is no evidence that they sensitize the skin in humans.

The relative intensity of skin roughness induced on the surface of the forearms of volunteers (a circulation method) due to contact with LAS of different alkyl chain lengths (C_8, C_{10}, C_{11}–C_{16}) was characterized mainly by gross visible changes. C_{12} LAS produced more skin roughening than LAS with longer or shorter alkyl chains. The degree of skin roughening *in vivo* correlated with the extent of protein denaturation measured *in vitro* (Imokawa et al., 1975a).

Primary skin irritation induced by an LAS formulation (average chain length, C_{12}; relative molecular mass, 346.5), by α-olefin sulfonates (AOS) (27% C_{15}, 25% C_{16}, 28% C_{17}, 8% C_{18}; relative molecular mass, 338.5), and by alkyl sulfates (AS) (C_{12}; relative molecular mass, 346.5) was compared in a 24-h closed-patch test on the forearms of seven male volunteers. A 1% aqueous solution (pH 6.8) of each substance was used,

and the relative intensity of skin irritation was scored by grading erythema, fissuring, and scales. The average score for LAS was similar to that for AOS but significantly lower than that for AS ($p < 0.05$) (Oba et al., 1968a).

In another comparison, the intensity of skin irritation induced by 1% aqueous solutions of LAS (C_{10}–C_{13}), AOS (C_{14}, C_{16}, C_{18}), and the sodium salt of AS (C_{12}–C_{15}) was studied in a 24-h closed-patch test on the forearm and in a test in which the substance was dripped onto the interdigital surface for 40 min once daily for two consecutive days at a rate of 1.2-1.5 ml/min. Skin reactions were scored by grading erythema in the patch test and by grading scaling in the drip test. In the patch test, the score for LAS was similar to that for AOS but significantly lower than that for AS. In the drip test, the score for LAS was similar to that for AS but higher than that for AOS (Sadai et al., 1979).

Repeated patch tests with LAS at aqueous concentrations of 0.05 and 0.2% produced mild to moderate primary irritation. In a study on the sensitization potential of LAS for human skin, a 0.1% aqueous preparation caused no sensitization in 86 subjects (Procter & Gamble Co., unpublished data).

No skin sensitization was seen in 2294 volunteers exposed to LAS or in 17 887 exposed to formulations of LAS (Nusair et al., 1988).

A8.2.2 *Effects on the epidermis*

The main effects of surface-active agents on the epidermal (stratum corneum) are:

- delipidation of the skin surface or outer layer;
- elution of natural moisturizing factor, which maintains the water content of the outer layer;
- denaturation of stratum corneum protein; and
- increased permeability, swelling of the outer layer, and inhibition of enzyme activities in the epidermis.

These effects and some others present a hazard to the skin; they are described below.

In an investigation of the relationship between the irritating potential of LAS *in vivo* and its ability to remove lipid from the stratum corneum *in vitro*, LAS removed detectable levels of lipids only at levels above the critical micelle concentration (0.04%). LAS removed only small amounts of cholesterol, free fatty acids, the esters of those materials, and possibly

squalene. At concentrations below that level, LAS can bind to and irritate the stratum corneum. The clinical irritation produced by LAS is therefore unlikely to be directly linked to extraction of lipid, and milder forms of irritation may involve binding of LAS to and denaturation of keratin as well as disruption of lipid (Froebe et al., 1990).

The results of the human arm immersion test with measurement of eluted amino acids and protein, the skin permeation test, freeing of sulfhydryl groups, and the patch test were compared for nine kinds of surfactant, including LAS, ABS, AS, alcohol ethoxylate sulfate, soap, nonionic surfactant, and amphoteric surfactant. LAS gave intermediate reactions in the patch test and the permeation test and showed a high level of sulfhydryl group freeing activity. The results of the tests for evaluating surfactants did not agree with those for the immersion test, which the author considered to provide the best simulation of actual use (Polano, 1968).

In a number of studies, denaturation of outer layer proteins was observed *in vitro* (Van Scott & Lyon, 1953; Harrold, 1959; Wood & Bettley, 1971; Imokawa et al., 1974; Okamoto, 1974; Imokawa et al., 1975b; Imokawa & Katsumi, 1976). Sodium dodecylbenzenesulfonate stimulated penetration of sodium ions through isolated human epidermis, partly because the detergent can denature proteins of the epidermal stratum corneum (Wood & Bettley, 1971). Sodium laurate and sodium lauryl sulfate were the most effective of several surfactants in inducing swelling of the horny layer (Putterman et al., 1977). The lysosome labilizing effects of surfactants, measured as the release of enzyme from lysosomes, were shown to diminish in the order cationic > anionic > nonionic surfactants (Imokawa & Mishima, 1979). When ovalbumin was used as a simulated epidermis protein, sodium lauryl sulfate was found to denature skin protein extensively by exposing concealed sulfhydryl groups in LAS of alkyl chain length C_8–C_{16} (Blohm, 1957).

In immersion tests of the hand and the forearm up to 5 cm above the wrist, falling off of skin scales diminished in the order: *sec*-alkane sulfonate > LAS > AOS, alcohol ethoxylate sulfate (Okamoto, 1974), but the distribution of carbon chain lengths among the samples was not described. In a comparison of skin roughening by a circulation method, the effects diminished in the order C_{12} AS > C_{12} AOS > C_{12} *sec*-alkane sulfonate > C_{12} LAS (Imokawa et al., 1974, 1975a,b). Skin roughening caused by several surfactants that are components of commercial products was studied by the method of Ito & Kakegawa (1972), in which

various concentrations are dripped onto the fingers. The effects diminished in the order C_{10}–C_{13} LAS = C_{12}–C_{15} AS > C_{11}, C_{13}, C_{15} alcohol ethoxylate sulfate (n = 0–3) > C_{14}, C_{16}, C_{18} AOS > C_{11}–C_{15} polyoxyethylene alkylether (Sadai et al., 1979).

A8.2.3 Hand eczema

The skin reaction to 0.04, 0.4, and 4.0% aqueous solutions of LAS (10.0% C_{10}, 34.3% C_{11}, 31.5% C_{12}, 24.7% C_{13}) was tested in a 24-h closed-patch test on the lower backs of 10 healthy volunteers and 11 patients with hand eczema (progressive keratosis palmaris). The incidence and intensity of skin reactions were greater in the group with hand eczema, but the difference was not statistically significant (Okamoto & Takase, 1976a,b).

In order to assess the possible etiological correlation between exposure to LAS and hand eczema, 0.04, 0.4, and 4% aqueous solutions of LAS were applied in 48-h closed-patch tests on the lower backs of 20 women with hand eczema and 42 with other skin diseases. The skin reaction was scored grossly from 0 to 5 on the basis of the occurrence or intensity of erythema, papules, and vesicles. The average score appeared to increase in parallel with the concentration of LAS but did not differ between the groups with hand eczema and other skin diseases (Sasagawa et al., 1978).

Nine proprietary household detergents were tested in 24-h closed-patch tests on the lower backs of 160 women with hand eczema. The surfactant concentrations in five of the products were: (i) 2% ABS-Na, 15% LAS-Na; (ii) 2% ABS-Na, 14% LAS-Na; (iii) 17% LAS-Na, 12% alcohol ethoxylate sulfate; (iv) 11% ABS-Na, 11% LAS-Na; (v) 19% LAS-Na. When the detergents were applied daily (for an unspecified period) at an aqueous concentration of 0.175–0.8%, positive responses were observed in 3.1% of the women, but they were considered not to be allergic because the redness of the skin disappeared completely within two days (Kawamura et al., 1970).

Three proprietary household detergents containing LAS were tested in 24-h closed-patch tests on the forearms of 13 women with 'housewives' dermatitis' and 13 with other skin diseases. The detergent was applied either undiluted or in a 0.2% aqueous solution. Undiluted solutions of all three detergents caused mild to moderate skin reactions, at incidences of 38.5, 48.1, and 73.1%, which did not differ between the groups with housewives' dermatitis and other skin diseases. The 0.2% aqueous solutions did not induce skin reactions (Ishihara & Kinebuchi, 1967).

Two series of field tests were conducted to estimate if exposure to a variety of synthetic detergent formulations was associated with causation or aggravation of hand eczema in women. In the first series, 162 female volunteers were divided into two groups and instructed to wear a rubber glove on either the left or the right hand while using the detergents. The test was conducted for one month, and the gross appearance of hands before and after the test period was compared. The relative intensity of noninflammatory keratosis of the hands was increased in individuals in both groups on hands that were covered and to a slightly greater extent on hands that were uncovered. In the second series of tests, 881 housewives were divided into three groups and instructed to use only one brand of household detergent, containing LAS, AOS, or ABS during the test period and to wear rubber gloves on both hands while using the detergent. The test was conducted for 1.5 months, and the gross appearance of hands before and after the test period was compared. Skin roughness was not worsened in any of the three groups (Watanabe et al., 1968).

A8.2.4 Occupational exposure

Sixty workers exposed at work to an atmosphere containing LAS at 8.64 mg/m^3 were tested for serum lipid and sugar content and for the activities of selected serum enzymes. The levels of total plasma lipids and plasma cholesterol were slightly lower in the exposed group than in controls, but no differences were noted for blood sugar, plasma phospholipid, plasma lipoprotein, α-amylase, leucine aminopeptidase, or pseudocholinesterase. The duration of exposure before testing was not indicated (Rosner et al., 1973).

In an investigation of the asthmagenic properties of sodium isononanoyl oxybenzene sulfonate, detergent industry workers were also tested with LAS. Three workers previously exposed to sodium isononanoyl oxybenzene sulfonate, three unexposed controls without asthma, and three controls with asthma were challenged with 0.01–100 µg of LAS. No changes were seen after inhalation of LAS in any of the subjects; but sodium isononanoyl oxybenzene sulfonate induced asthmatic symptoms in the previously exposed workers and not in the control groups (Stenton et al., 1990).

A8.2.5 Accidental or suicidal ingestion

No symptoms were seen in four cases of accidental ingestion of unknown amounts of a household synthetic detergent containing LAS as the main component (Hironaga, 1979).

A 32-year-old woman who had ingested 160 ml of a 21% aqueous solution of LAS with suicidal intent showed transient, slight mental confusion, vomiting, pharyngeal pain, hypotension, decreased plasma cholinesterase activity, and increased urinary urobilinogen, but all of these symptoms disappeared rapidly (Ichihara et al., 1967).

In a review of 1 581 540 cases of human exposure to a wide range of chemicals reported by the United States Poison Control Centers in 1989, 7983 people had been exposed to household automatic dishwasher preparations (alkali, anionic or nonionic, other or unknown) and 506 had required treatment in a health facility; 8950 had been exposed to household cleansers, with 894 requiring treatment; 12 876 had been exposed to laundry preparations, with 1542 treated; and 621 had been exposed to industrial detergents (anionic, cationic, nonionic), with 321 cases requiring treatment. There were no deaths, and only 12 of the treated cases were classified as 'major outcome'. Virtually all the reports involved accidental exposure. The compositions of the cleaning preparations, routes of exposure, and clinical descriptions were not provided (Litovitz et al., 1990).

A9. EFFECTS ON OTHER ORGANISMS IN THE LABORATORY AND THE FIELD

Section summary

LAS have been tested extensively, both in the laboratory and under field conditions, but the following aspects must be considered in interpreting test results. Comparison of the results of tests carried out on either mixtures of homologues of LAS or LAS of specified chain length is restricted, because the toxicity of LAS is influenced by the chain length, and homologues of lower chain length are less toxic than those with longer chains; furthermore, chain length was rarely specified in older studies. Studies of the effects of formulations of LAS on environmental biota are not included in this section.

Organisms are not exposed to a constant concentration of LAS in water, owing to the high adsorptivity and biodegradability of LAS. As LAS are adsorbed on suspended solids or food particles, they have reduced bioavailability. The adsorption kinetics of LAS also depend on the chain length of the homologues. Studies of aquatic toxicity involving flow-through or static renewal (at least daily) should therefore be given more prominence than studies based on static conditions, although flow-through and static renewal cannot be used in (semi-) chronic studies of lower organisms, such as daphnia. Studies in which the actual concentration was measured should likewise be given more consideration than those that rely on nominal concentrations.

The effects of LAS on the aquatic environment have been studied in short- and long-term studies in the laboratory and under more realistic conditions: micro- and mesocosm and field studies. In general, a decrease in alkyl chain length or a more internal position of the phenyl group is accompanied by a decrease in toxicity. Data on fish and daphnia indicate that a decrease in chain length of one unit (e.g. C_{12} to C_{11}) is accompanied by an approximately 50% decrease in toxicity, but there is no linear relationship between chain length and toxicity. In aquatic microorganisms, the effects are strongly related to variables such as the type of test system and use of mixed cultures as opposed to individual species. EC_{50} values range from 0.5 mg/litre (single species) to > 1000 mg/litre.

In freshwater fish, the acute LC_{50} values of C_8–C_{15} LAS are 0.1–125 mg/litre. The chronic $L(E)C_{50}$ values of LAS ($C_{11.7}$ and not

specified) in two species tested were 2.4 and 11 mg/litre, and NOECs ranging from 0.11 to 8.4 mg/litre have been reported for $C_{11.2}$–C_{13} (or not specified). Marine fish appear to be more sensitive, with acute LC_{50} values for $C_{11.7}$ (or not specified) in six species of 0.05–7 mg/litre, chronic LC_{50} values for LAS of unspecified chain length in two species of 0.01–1 mg/litre, and an NOEC for C_{12} in one species of < 0.02 mg/litre.

Results in aquatic plants are also species dependent. In freshwater plants, the EC_{50} values for LAS (with chain lengths shown in parentheses) were 10–235 mg/litre for green algae (C_{10}–C_{14}), 5–56 mg/litre for blue algae ($C_{11.1}$–C_{13}), 1.4-50 mg/litre for diatoms ($C_{11.6}$–C_{13}), and 2.7–4.9 mg/litre for macrophytes ($C_{11.8}$). Marine algae appear to be even more sensitive. There is probably no linear relationship between chain length and toxicity to algae.

The effects of LAS on freshwater algae have also been tested under realistic conditions in systems with various trophic levels, comprising enclosures in lakes (lower organisms), model ecosystems (sediment: water systems), a river below and above a wastewater treatment plant outfall, and experimental streams. In general, C_{12} LAS were used. Algae were more sensitive in summer, when the 3-h EC_{50} values with regard to photosynthesis were 0.2–8.1 mg/litre, whereas studies of model ecosystems showed no effects on the relative abundance of algal communities at 0.35 mg/litre. No effects were seen in these studies at 0.24–5 mg/litre, depending on the organism and parameter tested.

In aquatic invertebrates, the acute $L(E)C_{50}$ values were 4.6–200 mg/litre for molluscs (either C_{13} or not specified), 0.12–27 mg/litre for crustaceans ($C_{11.2}$–C_{18} or not specified), 1.7–16 mg/litre for worms ($C_{11.8}$ or not specified), and 1.4–270 mg/litre for insects (C_{10}–C_{15}). The chronic $L(E)C_{50}$ values were 2.2 mg/litre for insects ($C_{11.8}$) and 1.1–2.3 mg/litre for crustaceans ($C_{11.8}$–C_{13}). The chronic NOEC for crustaceans, on the basis of lethality or reproduction, was 0.2–10 mg/litre ($C_{11.8}$ or not specified). Marine invertebrates are more sensitive, with LC_{50} values of 1 to >100 mg/litre (almost all C_{12}) and NOEC values of 0.025–0.4 mg/litre (chain lengths not specified).

Biodegradation products and by-products of LAS are 10–100 times less toxic than the parent compound.

Fewer data are available on the effects of LAS in the terrestrial environment. For the plant species tested, the NOEC values were

< 10–20 mg/litre in nutrient solutions and 100 mg/kg (C_{10}–C_{13}) for growth of plants in soils. The 14-day LC_{50} for earthworms was > 1000 mg/kg.

One study in which chickens were treated in the diet resulted in an NOEC based on egg quality of > 200 mg/kg.

A9.1 Effect of chain length on the toxicity of linear alkylbenzene sulfonates

The ecotoxicity of homologues of LAS varies according to the length of the alkyl chain and the position of the benzene ring on this chain. In general, homologues with longer chains are more ecotoxic than shorter ones, and ecotoxicity increases with the proximity of the benzene ring to the end of the chain. The results of studies on the effect of LAS chain length on acute toxicity to fish are presented in Table 23.

Table 23. Effect of the chain length of linear alkylbenzene sulfonates (LAS) on their acute toxicity to freshwater fish

Homologue of LAS	Fathead minnow *Pimephales promelas* 48-h LC_{50} (mg/litre)[a]	Goldfish *Carassius auratus* 6-h LC_{50} (mg/litre)[b]	Guppy *Lebistes reticulatus* LC_{50} (mg/litre)[c]	Golden orfe *Idus idus melanotus* 96-h LC_{50} (mg/litre)[d]
C_{10}	43.0	61.0	50	16.6
C_{11}	16.0	22.5		6.5
C_{12}	4.7	8.5	5	2.6
C_{13}	0.4	3.3		0.57
C_{14}	0.4		1	0.26
C_{16}		0.087	1	0.68
C_{18}		0.38		15

[a] From Kimerle & Swisher (1977)
[b] From Gafa (1974)
[c] From Borstlap (1967)
[d] From Hirsch (1963)

The effect of chain length can also be seen on the basis of quantitative structure–activity relationships (Roberts, 1989, 1991) calculated from the octanol–water partition coefficients of homologues of LAS. The slope of the relationship varied from 0.64 to 0.78; therefore, using an average slope of 0.70, it was calculated that a decrease in chain length

from C_{12} to C_{11} reduced the aquatic toxicity of LAS by a factor of 2.4, with a corresponding decrease in the octanol–water partition coefficient of 0.54.

A9.2 Microorganisms

No adverse effects were seen on the performance of laboratory-scale activated sludge units after addition of ≤ 20 mg/litre LAS. At 50 mg/litre, nitrification was decreased in extended aeration units that were treating synthetic sewage (Janicke & Niemitz, 1973). A bacterium similar to *Klebsiella pneumoniae* isolated from sewage degraded LAS at a concentration of 10 ml/litre, but a concentration of 20 ml/litre inhibited the growth of the bacterium by 39% (Hong et al., 1984).

The toxicity of microorganisms in activated sludge increases with the length of the alkyl chain up to approximately C_{12} and then decreases (Table 24), presumably because of decreased bioavailability (e.g. greater sorption of these higher chain lengths) (Verge et al., 1993).

Table 24. Results of tests for the inhibition of activated sludge by the sodium salt of linear alkylbenzene sulfonates (LAS)

LAS	Chain length	3-h EC_{50} (mg/litre)
Pure homologues	C_{10}	1042-1200
	C_{11}	740-782
	C_{12}	500-723
	C_{13}	700-795
	C_{14}	900-1045
Commercial formulations	C_{11}	760
	$C_{11.5}$	550
	C_{13}	650

From Verge et al. (1993)

A mixed bacterial culture was acclimatized to 10 mg/litre LAS (C_9–C_{14}) and was then maintained in either river water, forest soil, or wastewater from a detergent plant, the concentration of LAS being increased every five days. At 20.8 and 46 mg/litre, no effect was reported on the specific growth rate of the bacteria; however, at 70 mg/litre, the growth rate was inhibited by 18%, and at 95 mg/litre growth was almost zero. Concentrations of 186 and 465 mg/litre LAS inhibited growth completely (Hrsak et al., 1981).

The acute toxicity of LAS (C_9–C_{14}) in naturally occurring bacteria was studied in freshwater and seawater samples by measuring ^{3}H-thymidine incorporation. The EC_{50} values were 0.5–1.66 mg/litre for all samples. Toxicity was found to increase with an increasing relative abundance of longer carbon chains (Martinez et al., 1989). For bacteria collected from the Rhone River plume (an estuarine area) and exposed to LAS, the EC_{50}, based on ^{3}H-thymidine incorporation, was 11.9 mg/litre (Martinez et al., 1991).

The 8-h EC_{50}, based on specific growth rate, of *Pseudomonas fluorescens* in solutions of $C_{11.1}$ LAS under static conditions was 3200–5600 mg/litre (Canton & Slooff, 1982).

The effect of $C_{11.6}$ LAS on the structure and function of microbial communities was studied in a flow-through model ecosystem containing several trophic levels at concentrations of 0.5 or 5 mg/litre. LAS had no effect on microbial structure at either dose level, but at 5 mg/litre it inhibited the degradation of both glucose and LAS. In an experiment in which LAS were supplied in sewage, neither microbial structure nor function was affected (Larson & Maki, 1982).

The effects of LAS on the microbial activity of soils were studied on the basis of Fe[III] reduction. The no-effect-level was found to be 250 mg/kg; the EC_{50} was about 500 mg/kg in a strongly adsorbing soil and 33–55 mg/kg in a poorly adsorbing soil (Welp & Brummer, 1985).

LAS at concentrations of 0.8–50 g/m^2 had no effect on respiration of loamy soil, sandy soil, or sandy soil irrigated with wastewater for one or 14 days (Litz et al., 1987).

A9.3 Aquatic organisms

A9.3.1 Aquatic plants

A9.3.1.1 Freshwater algae and cyanobacteria

The 96-h EC_{50} values for C_{13} LAS on population growth were 116 mg/litre for the green alga *Selenastrum capricornutum*, 5 mg/litre for the blue–green alga *Microcystis aeruginosa*, and 1.4 mg/litre for the diatom *Navicula pelliculosa*. The EC_{50} values for C_{12} LAS were 29 mg/litre for *Selenastrum* and 0.9 mg/litre for *Microcystis* (Lewis & Hamm, 1986). The EC_{50} for $C_{11.7}$ LAS on growth of *Selenastrum* was reported to be 83 mg/litre (Konno & Wakabayashi, 1987). The EC_{50} values for $C_{11.6}$ LAS were found to be 50–100 mg/litre for *Selenastrum*,

10–20 mg/litre for *Mycrocystis*, and 20–50 mg/litre for the diatom *Nitzschia fonticola* (Yamane et al., 1984). The seven-day EC_{50} for C_{12} LAS in the green alga *Chlorella pyrenoidosa*, based on growth, was 10 mg/litre (Kondo et al., 1983).

The 96-h EC_{50} values in algae grown in solutions of $C_{11.1}$ LAS under static conditions, measured as biomass, were 32–56 mg/litre for *Microcystis aeruginosa* and 18–32 mg/litre for *Chlorella vulgaris* (Canton & Slooff, 1982).

A study of the toxicity of various formulations of LAS to the algae *Scenedesmus subspicatus* and *Selenastrum capricornutum* (Table 25) indicated that commercial mixtures are as or slightly less toxic than homologues. This finding may be due to a difference in the sensitivity of the two algae, since those tested with the homologues were of a different origin than those tested with commercial LAS (Verge et al., 1993).

Table 25. Results of tests for the toxicity of the sodium salt of linear alkylbenzene sulfonates (LAS) in algae

LAS	Chain length	72-h EC_{50} (mg/litre)
Pure homologues	C_{10}	235
	C_{11}	118
	C_{12}	62
	C_{13}	33
	C_{14}	18
Commercial formulations	C_{11}	80
	$C_{11.6}$	80
	C_{13}	62

From Verge et al. (1993)

LAS (chain length not specified) significantly reduced the growth of the green alga *Selenastrum capricornutum* at a concentration of 40 mg/litre or more. A significant decrease in growth was also noted at 10 mg/litre, but no significant effect was observed at 20 or 30 mg/litre (Nyberg, 1988).

A9.3.1.2 *Marine algae*

Growth of *Gymnodinium breve* was reduced by 69% rafter nine days' exposure to C_{12} LAS (Kutt & Martin, 1977). These results were confirmed in a study in which C_{13} LAS were introduced at the bottom or surface of a water column: Exposure to LAS at concentrations > 0.025 mg/litre inhibited growth completely within two days (Hitchcock & Martin, 1977). These results suggest that *Gymnodinium breve* is more sensitive to the effects of LAS than other algae.

For $C_{11.7}$ LAS, the seven-day EC_{50} for growth and the two-day EC_{50} for ATP activity on the marine diatom *Thalassiosira pseudonana* were both 10 mg/litre (Kondo et al., 1983).

Exposure of the alga *Porhyra yezoensis*, a standard test species in Japan, to LAS (C_{10}-C_{14}) under semi-static conditions gave a 10-day E_{50} (based on growth) of 0.56 mg/litre (Takita, 1985).

A9.3.1.3 *Macrophytes*

The seven-day EC_{50} values for $C_{11.8}$ LAS on the duckweed *Lemna minor* under flow-through conditions were 2.7 mg/litre for frond count, 3.6 mg/litre for dry weight, and 4.9 mg/litre for root length. The time-independent EC_{50} for growth rate and doubling time was 4.8 mg/litre (Bishop & Perry, 1981).

A9.3.2 *Aquatic invertebrates*

A9.3.2.1 *Acute toxicity*

The acute toxicity of LAS to aquatic invertebrates is summarized in Tables 26 and 27. For marine invertebrates, the 96-h LC_{50} values for C_{12} LAS range from 3 mg/litre for barnacles to > 100 mg/litre for several other species (Table 26). Freshwater invertebrates show a range of 48-h LC_{50} values from 0.11 mg/litre (C_{16}) for a daphnid to 270 mg/litre ($C_{11.8}$) for an isopod (Table 27). Several marine invertebrate species are more sensitivite to LAS at the larval stage than as adults (Table 26).

Freshwater mussels (*Anodonta cygnea*) were more sensitive to LAS during the reproductive period than during the non-reproductive period, the 96-h LC_{50} being reduced from 200 to 50 mg/litre (Bressan et al., 1989).

Studies with *Daphnia magna* revealed a correlation between chain length and toxicity. The acute toxicity (24-h and 48-h LC_{50}) of LAS to

Table 26. Acute toxicity of linear alkylbenzene sulfonates (LAS) to estuarine and marine invertebrates

Organism	Size or age	Static or flow	Temp. (°C)	Salinity (‰)	LAS chain length	End-point	Concentration (mg/litre)[a]	Reference
Sea squirt (*Ciona intestinalis*)	Larva	Static	20		NS	6-h LC_{50}	1	Renzoni (1974)
Common mussel (*Mytilus edulis*)		Static	6–8	32–34	C_{12}	96-h LC_{50}	> 100	Swedmark et al. (1971)
		Static	15–17	32–34	C_{12}	96-h LC_{50}	50	
Mussel (*Mytilus galloprovincialis*)		Static[c]	18	35	NS	48-h LC_{50}	39.8	Bressan et al. (1989)
	Adult		18	35	NS	96-h LC_{50}	1.66	
Cockle (*Cardium edule*)		Static	6–8	32–34	C_{12}	96-h LC_{50}	5	Swedmark et al. (1971)
	Juvenile	Static	15–17	32–34	C_{12}	96-h LC_{50}	5	
Clam (*Mya arenaria*)		Static	6–8	32–34	C_{12}	96-h LC_{50}	70	
		Static	15–17	32–34	C_{12}	96-h LC_{50}	< 25	
Scallop (*Pecten maximus*)		Static	6-8	32–34	C_{12}	96-h LC_{50}	< 5	
Scallop		Static	15–17	32–34	C_{12}	96-h LC_{50}	< 5	
Decapod		Static	15–17	32–34	C_{12}	96-h LC_{50}	50	
(*Leander adspersus*)	Intermoult	Static	6–8	32–34	C_{12}	96-h LC_{50}	50	
	Postmoult	Static	6–8	32–34	C_{12}	96-h LC_{50}	25	
Hermit crab (*Eupagurus bernhardus*)		Static	6–8	32–34	C_{12}	96-h LC_{50}	> 100	

Table 26 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Salinity (‰)	LAS chain length	End-point	Concentration (mg/litre)[a]	Reference
Spider crab	Larva	Static	6–8	32–34	C_{12}	96-h LC_{50}	9	
(*Hyas araneus*)	Adult	Static	6–8	32–34	C_{12}	96-h LC_{50}	> 100	
Shore crab (*Carcinus maenus*)		Static	6–8	32–34	C_{12}	96-h LC_{50}	> 100	
Barnacle	Larva	Static	6–8	32–34	C_{12}	96-h LC_{50}	3	
(*Balanus balanoides*)	Adult	Static	6–8	32–34	C_{12}	96-h LC_{50}	50	
Brine shrimp (*Artemia salina*)		Static	25		C_{11}–C_{13}	24-h LC_{50}	33	Price et al. (1974)

Static: water unchanged for duration of test; NS, not specified; static′, static renewal: water changed every 12 h; flow, flow-through conditions: LAS concentration in water maintained continuously

[a] Based on nominal concentration

Table 27. Acute toxicity of linear alkylbenzene sulfonates (LAS) to freshwater invertebrates

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Bivalve mollusc (*Anodonta cygnea*)	11 cm	Static[f]	18		8.0	NS	96-h LC_{50}	200[b]	Bressan et al. (1989)
			18		8.0	NS	96-h LC_{50}	50[b,c]	
Bivalve mollusc (*Unio elongatulus*)	9 cm	Static[f]	18		8.0	NS	96-h LC_{50}	182.5[b]	
Snail (*Gonobasis* sp.)		Static	21	62	7.3	av. C_{13}	24-h LC_{50}	4.6[b]	Dolan & Hendricks (1976)
Snail (*Physa integra*)		Flow	15	41–47	7.5–7.7	NS	96-h LC_{50}	9[b]	Arthur (1970)
Amphipod (*Gammarus pseudolimnaeus*)		Flow	15	41–47	7.5–7.7	NS	96-h LC_{50}	7[b]	
Amphipod (*Gammarus* sp.)	4.3 mm	Static	22	165	7.9–8.4	$C_{11.8}$	48-h LC_{50}	3.3[b]	Lewis & Suprenant (1983)
Campeloma decisum		Flow	15	41–47	7.5–7.7	NS	96-h LC_{50}	27[b]	Arthur (1970)
Water flea (*Daphnia magna*)	< 24 h	Static	20	25		$C_{11.7}$	24-h LC_{50}	17	Wakabayashi et al. (1988)
	< 24 h	Static	21	120	7.4	C_{10}	48-h LC_{50}	9.55[d]	Maki & Bishop (1979)

Table 27 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Water flea (contd)	< 24 h	Static	21	120	7.4	C_{11}	48-h LC_{50}	1.15[d]	
(*Daphnia magna*)	< 24 h	Static	21	120	7.4	C_{12}	48-h LC_{50}	5.88–6.84[d]	
	< 24 h	Static	21	120	7.4	C_{13}	48-h LC_{50}	2.63[d]	
	< 24 h	Static	21	120	7.4	C_{14}	48-h LC_{50}	0.68–0.8[d]	
	< 24 h	Static	21	120	7.4	C_{16}	48-h LC_{50}	0.11–0.2[d]	
	< 24 h	Static	21	120	7.4	C_{18}	48-h LC_{50}	0.12[d]	
	< 18 h	Static				$C_{13.3}$	48-h LC_{50}	2.3[b]	Kimerle &
	< 18 h	Static				C_{10}	48-h LC_{50}	12.3[b]	Swisher (1977)
	< 18 h	Static				C_{11}	48-h LC_{50}	5.7[b]	
	< 18 h	Static				C_{12}	48-h LC_{50}	3.5[b]	
	< 18 h	Static				C_{13}	48-h LC_{50}	2.0[b]	
	< 18 h	Static				C_{14}	48-h LC_{50}	0.7[b]	
	< 24 h	Static	19			$C_{11.2}$	48-h LC_{50}	18-32[b]	Canton & Slooff (1982)
	< 24 h	Static	21	131	7.4–7.8	$C_{11.8}$	48-h LC_{50}	4.8[d]	Lewis (1983)
	< 24 h	Static	22	165	7.9–8.4	$C_{11.8}$	48-h LC_{50}	1.8–5.6[b]	Lewis & Suprenant (1983)
	< 24 h	Static	21	295–310	7.3–8.4	$C_{11.8}$	48-h LC_{50}	3.6–4.7[b]	Taylor (1985)
	< 48 h	Static	22	241	7.8	C_{11}	48-h EC_{50}	2.2[b,e]	Barera & Adams (1983)
		Flow				$C_{11.8}$	48-h LC_{50}	4.4[d]	Bishop & Perry (1981)
	< 12 h	Flow	21	120	7.4	$C_{11.8}$	96-h LC_{50}	23.94[d]	Maki (1979a)
	< 12 h	Flow	21	120	7.4	C_{13}	48-h LC_{50}	2.19[d]	

Table 27 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Water flea (*Daphnia pulex*)	< 24 h	Static	20	25		$C_{11.7}$	24-h LC_{50}	18	Wakabayashi et al. (1988)
	< 24 h	Static	21	120	7.4	C_{12}	48-h LC_{50}	8.62[d]	Maki & Bishop (1979)
	< 24 h	Static	21	120	7.4	C_{14}	48-h LC_{50}	0.59[d]	
	< 24 h	Static	21	120	7.4	C_{16}	48-h LC_{50}	0.15[d]	
Oligochaete (*Dero* sp.)	6.0 mm	Static	22	165	7.9–8.4	$C_{11.8}$	48-h LC_{50}	1.7[b]	Lewis & Suprenant (1983)
Roundworm (nematode) (*Rhabditis* sp.)	0.3 mm	Static	22	165	7.9–8.4	$C_{11.8}$	48-h LC_{50}	1.7[b]	
Flatworm (*Dugesia* sp.)	3.4 mm	Static	22	165	7.9–8.4	$C_{11.8}$	48-h LC_{50}	1.8[b]	
Branchiura sowerbyi		Static[f]	10	25	8.0	NS	96-h LC_{50}	10.8[b,f]	Bressan et al. (1989)
			10	25	8.0	NS	96-h LC_{50}	4.4[b]	
Worm (*Limnodrilus hoffmeisteri*)		Static[f]	10	25	8.0	NS	96-h LC_{50}	7.8[b,f]	
			10	25	8.0	NS	96-h LC_{50}	2.0[b]	
Isopod (*Asellus* sp.)	5.3 mm	Static	22	165	7.9–8.4	$C_{11.8}$	48-h LC_{50}	1.8[b]	Lewis & Suprenant (1983)

Table 27 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Midge (*Chironomus riparius*)	Larva	Flow	22	150	7.8–8.4	$C_{11.8}$	72-h LC_{50}	2.2[d]	Pittinger et al. (1989)
Midge (*Paratanytarsus parthenogenica*)	3.6 mm	Static	2147	165	7.9–8.4	$C_{11.8}$	48-h LC_{50}	1.8[d]	Lewis & Suprenant (1983)
Mosquito (*Aedes aegypti*)	Larva	Static				C_{10-13}	24-h LC_{50}	6[b]	Van Emden et al. (1974)
	Larva	Static				C_{10-15}	24-h LC_{50}	2[b]	
	3–4 d	Static	23			$C_{11.1}$	48-h LC_{50}	56-100[b]	Canton & Slooff (1982)
Mayfly (*Isonychia* sp.)	Larva	Static	10	53	7.5–7.8	$C_{11.6}$	24-h LC_{50}	13.6[b]	Dolan et al. (1974)
	Larva	Static	10	53	7.5–7.8	$C_{11.6}$	48-h LC_{50}	10.4[b]	
	Larva	Static	10	53	7.5–7.8	$C_{11.6}$	96-h LC_{50}	5.33[b]	
	Larva	Static	10	53	7.5–7.8	$C_{13.1}$	24-h LC_{50}	4.19[b]	
	Larva	Static	10	53	7.5–7.8	$C_{11.6}$	48-h LC_{50}	12.47[b]	
	Larva	Static	10	53	7.5–7.8	$C_{11.6}$	96-h LC_{50}	1.36[b]	

Static[r], static renewal: water changed every 12 h; NS, not specified; flow, flow-through conditions: LAS concentration in water maintained continuously; static: water unchanged for duration of test

[a] mg/litre $CaCO_3$

[b] Based on nominal concentration

[c] Test performed during the reproductive period

[d] Based on measured concentrations

[e] Based on immobilization

[f] Organism exposed in the presence of sediment

Daphnia magna increased with chain length between C_{10} and C_{14} (Kimerle & Swisher, 1977) and with chain lengths between C_{10} and C_{16} (Maki & Bishop, 1979), although similar values were obtained for C_{16} and C_{18} homologues. No significant difference in sensitivity was seen between *Daphnia magna* and *Daphnia pulex*. A similar result was obtained with homologue mixtures (Martinez et al., 1989): toxicity was correlated with the homologues in which long chains were the most abundant.

Partial biodegradation of LAS significantly reduces the specific toxicity (by unit weight) of the remaining LAS to *Daphnia magna*. For example, LAS with a high relative molecular mass and a 48-h LC_{50} of 2 mg/litre had an LC_{50} of 30–40 mg/litre after 80–85% degradation (Kimerle & Swisher, 1977); the longer homologues and more terminal isomers, which are the most toxic, are therefore also the more readily biodegraded. Shorter carboxylates formed during the degradation of LAS were three to four orders of magnitude less toxic than LAS (Swisher et al., 1978). Other workers also found a reduction in the acute toxicity of LAS to *Daphnia magna* during primary degradation (Gard-Terech & Palla, 1986).

Increasing hardness also increased the acute toxicity (48-h LC_{50}) of $C_{11.8}$ LAS from a nominal concentration of 7.1 mg/litre at 25 mg/litre $CaCO_3$ to 4.0 mg/litre at 350 mg/litre $CaCO_3$; however, significant additional physiological stress was induced if the hardness of the culture water was significantly different from that of the test water. Pre-exposure to 0.4 mg/litre LAS (one-tenth of the 48-h LC_{50}) for up to seven generations (14 weeks) had no significant effect on the susceptibility of daphnids to acute exposures (Maki & Bishop, 1979).

Loading density, ranging from 10 daphnids per 20 ml to 20 daphnids per 1000 ml, had no significant effect on the acute toxicity of $C_{11.8}$ LAS for *Daphnia magna* (Lewis, 1983). Daphnids fed a diet containing *Selenastrum* had a significant, twofold decrease in acute toxicity due to $C_{11.8}$ LAS in comparison with unfed daphnids (Taylor, 1985). The presence of sediment reduced the acute toxicity of LAS to the oligochaete worms *Branchiura sowerbyi* and *Limnodrilus hoffmeisteri*. The NOEC and LOEC for *B. sowerbyi* were 2.5 times higher in the presence of sediment, and those for *L. hoffmeisteri* were 4-4.5 times higher (Bressan et al., 1989; see also Table 27).

The 96-h EC_{50} values for duplicate studies of the effect of LAS on attachment of the podia of the sea urchin *Hemicentrotus pulcherrimus* were 3.7 and 3.8 mg/litre (Lee & Park, 1984).

The data from other studies (Lal et al., 1983, 1984a,b; Misra et al., 1984; Chattopadhyay & Konar, 1985; Misra et al., 1985; Devi & Devi, 1986; Misra et al., 1987, 1989a,b, 1991) could not be adequately interpreted because of deficiencies in the data or method, including inadequate characterization of the test material with regard to chain-length distribution and use of test material in an acidified form. The range of values for toxicity reported in these studies was 10-100 times greater than that in numerous studies of the same or similar species, and the high values have not been verified by these or other researchers. As the toxic effects reported are not considered to be representative of those of commercial LAS, the data were not used in evaluating the environmental effects of LAS.

A 72-h LC_{50} of 2.2 mg/litre was reported for $C_{11.8}$ LAS in newly hatched larvae of the midge (*Chironomus riparius*) (Pittinger et al., 1989).

A9.3.2.2 *Short-term and long-term toxicity*

The 21-day LC_{50} for the water flea (*Daphnia magna*) was 18 mg/litre, and the NOEC, based on survival, was 10 mg/litre under static renewal conditions. The 21-day EC_{50}, based on reproduction, was estimated to be > 10 mg/litre (Canton & Slooff, 1982). The 14-day EC_{50} for C_{12} LAS in *Daphnia carinata*, based on reproduction, was 16.8 mg/litre (Hattori et al., 1984).

Diet had a significant effect on the sensitivity of *Daphnia magna* to the chronic toxicity of $C_{11.8}$ LAS. The NOEC values showed a threefold variation of 1.2–3.2 mg/litre and the 21-day LC_{50} values a twofold variation of 2.2–4.7 mg/litre with diet. A threefold variation in toxicity in tests in *Daphnia* is not, however, unusual (Taylor, 1985).

Under continuous-flow conditions, a 21-day LC_{50} value of 1.67 mg/litre was found for daphnids (*Daphnia magna*) exposed to $C_{11.8}$ LAS and 1.17 mg/litre for those exposed to C_{13} LAS. The EC_{50} values for reproductive toxicity were 1.5 mg/litre for $C_{11.8}$ LAS and 11.1 mg/litre for C_{13} with respect to total young production, 2.3 mg/litre for $C_{11.8}$ and 1.4.1 mg/litre for C_{13} for average brood size, and 2.31 mg/litre for $C_{11.8}$ and 1.29 mg/litre for C_{13} for percentage of days on which reproduction occurred (Maki, 1979a).

Campeloma decisum, *Gammarus pseudolimnaeus*, and *Physa integra* were exposed to LAS at concentrations of 0.2–4.4 mg/litre for six weeks; amphipods were exposed for a further 15 weeks. Survival, growth,

reproduction, feeding, and mobility were studied. The maximum acceptable concentrations of LAS were found to be 0.2–0.4 mg/litre for *Gammarus* and 0.4–1.0 mg/litre for *Campeloma; P. integra* were not significantly affected (Arthur, 1970).

Fertilized eggs of sea urchins (*Paracentrotus lividus*) were treated with LAS at concentrations of 0–0.5 mg/litre for 40 days. The pattern of embryonic development was unaffected, but the mean length of the somatic rods of the echinoplutei were reduced successively with increasing LAS concentrations. A significant reduction in growth occurred at doses between 0.35 and 0.4 mg/litre; above 0.45 mg/litre, alterations in skeletal development were induced (Bressan et al., 1989).

Oligochaete worms (*B. sowerbyi*) were maintained in LAS at a concentration of 0.5, 2.5, or 5.0 mg/litre for up to 140 days in the presence of sediment. Exposed worms laid fewer cocoons and eggs, but the worms exposed to 5 mg/litre were the least affected. The percentage of degenerated cocoons, the percentage of worms hatching, the mean number of eggs per cocoon, and the mean embryonic development time were all unaffected by treatment. Worms exposed via the sediment only were not affected (Bressan et al., 1989).

Growth of mussels (*Mytilus galloprovincialis*) exposed to LAS at a concentration of 0.25 or 0.5 mg/litre for 220 days, expressed as mean length of the major axis of the shell, was significantly slowed ($p < 0.001$). The mean (± SE) increments in growth were: control, 3.11 ± 0.34; 0.25 mg/litre, 1.71 ± 0.15; 0.5 mg/litre, 1.48 ± 0.16 (Bressan et al., 1989).

Eggs of the common mussel, *M. edulis*, were exposed from the time of fertilization for 240 h. Fertility was decreased at the lowest concentration of 0.05 mg/litre and fertilization did not take place at concentrations in excess of 1 mg/litre. LAS at concentrations > 0.3 mg/litre inhibited the development of mussel larvae by delaying the transitory stages of larval development. Reduced growth rates were observed at concentrations > 0.1 mg/litre (Granmo, 1972).

Newly fertilized eggs of American oysters (*Crassostrea virginica*) were exposed to LAS (chain length not specified, but likely to be C_{13}) for 48 h. The percentage of eggs that developed normally was significantly reduced at concentrations greater than 0.025 mg/litre. The percentage survival of oyster larvae hatched in 'clean' water and exposed to LAS at a concentration of 1 mg/litre for 10 days was significantly decreased, and growth (mean length) was significantly reduced at 0.5 mg/litre (Calabrese & Davis, 1967).

Embryos of sea urchins (*P. lividus*) were exposed to LAS at concentrations of 0.25–0.5 mg/litre from the time of fertilization for 40 h. At concentrations > 0.45 mg/litre, skeletal development was totally inhibited; a significant decrease was observed at 0.3 mg/litre. The effect of LAS was found to be maximal at the end of gastrulation when calcium uptake is high (Bressan et al., 1991).

The effects of LAS were studied on the eggs and sperm of the sea squirt *Ciona intestinalis*. Fertility and hatchability were markedly reduced at 0.1 mg/litre when eggs and sperm were exposed for the entire developmental period; however, if they were exposed only before fertilization, fertility and hatchability were slightly reduced at 0.1 mg/litre but markedly at 1 mg/litre. Male gametes appeared to be particularly sensitive to the toxic effects of LAS (Renzoni, 1974).

Two marine benthic filter feeders, the sea squirts *Botryllus schlosseri* and *Botrylloides leachi* were exposed at different periods of development to LAS. When larvae were exposed from spawning for 6 h, the incidence of abnormal metamorphosis was significantly increased at 1 mg/litre LAS for *Botryllus* and 2 mg/litre for *Botrylloides*. The frequency of spontaneously settled larvae of both species also increased with exposure to LAS and seemed to be a selective effect of LAS. The frequency was significantly different from controls at 1 and 3 mg/litre for the two species, respectively. In a second experiment, young colonies were exposed to LAS for 15 days immediately after discharge by the parental colony. Growth rates were significantly decreased at 0.5 mg/litre for *Botryllus* and at 0.25 mg/litre for *Botrylloides*. When colonies were exposed from the end of metamorphosis, their growth rates were similarly affected, but the mortality rate was significantly lower. The effects of LAS thus appear to be exerted mainly on the pelagic phase of the life cycle (Marin et al., 1991).

No significant reduction in egg hatching of midges (*Chironomus riparius*) was seen at the highest concentration of $C_{11.8}$ LAS tested (18.9 mg/litre), but newly hatched larvae were more sensitive, with a 72-h LC_{50} of 2.2 mg/litre. In bioassays of part of the life cycle in a sediment and water system, the percentages of winged adults emerging were monitored after continuous exposure of larvae and pupae. The NOEC for sediment containing LAS was 319 mg/kg (dry weight). In the absence of sediment, the NOEC was 2.40 mg/litre. Both tests were conducted for about 20 days (Pittinger et al., 1989).

A9.3.2.3 Biochemical and physiological effects

Juvenile mussels (*M. galloprovincialis*) were exposed to LAS at a concentration of 0.25 or 0.5 mg/litre for 220 days. Oxygen uptake and the retention rate of neutral red (a measure of filtration rate) were significantly decreased, but no effect was detected on nitrogen excretion (measured as ammonia). When the experiment was repeated over a seven-day period at a concentration of LAS of 1 or 1.5 mg/litre, no significant effect was seen on nitrogen metabolism and the results for oxygen uptake were inconclusive. The filtration rate was again significantly reduced when compared with that in control mussels (Bressan et al., 1989).

The 48-h LC_{50} for lugworms (*Arenicola marina*) exposed to LAS was calculated to be 12.5 mg/litre (95% confidence interval, 8.6–18.2). When tissues from a lugworm exposed to a concentration close to that of the LC_{50} were examined for changes in morphology by both light and electron microscopy, serious damage was reported in the caudal epidermis, epidermic receptors, and gills; no effect was reported in the thoracic epidermis or the intestine. In the caudal epidermis, LAS destroyed the papillae, disrupting the internal structure, occasionally displacing the musculature below the papillae and thus giving it direct contact with seawater. Deciliation of the epidermic receptors was also reported. These effects were considered to indicate that the physiological response of damaged epidermic receptors was reduced or blocked by exposure to LAS. Changes in the morphology of the gills included destruction of the epithelium and blood vessels, causing complete solubilization of branch apexes, and development of holes at the base of the gills (Conti, 1987).

A9.3.3 Fish

A9.3.3.1 Acute toxicity

The acute toxicity of LAS to fish is summarized in Tables 28 and 29. Only a few studies were available on marine fish, providing two 96-h LC_{50} values, 1 and 1.5 mg/litre LAS. Tests in various species of freshwater fish gave a wide range of LC_{50} values: the 48-h values ranged from 0.2 mg/litre for brown trout (*Salmo trutta*) to 125 mg/litre for the golden orfe (*Idus idus memanotus*), and the 96-h values ranged from 0.1 mg/litre for brown trout to 23 mg/litre for white tilapia (*Tilapia melanopleura*).

The acute toxicity tended to increase with increasing carbon chain length. Thus, C_{14} LAS were more acutely toxic to bluegill (*Lepomis*

macrochirus) than C_{12} compounds (Swisher et al., 1964); the acute toxicity of LAS to the golden orfe increased with chain length from C_8 to C_{15} but decreased at C_{16} (Hirsch, 1963).; and a similar trend was found for fathead minnows (*Pimephalus promelas*) exposed to LAS with chain lengths of C_{10} to C_{14} (Kimerle & Swisher, 1977).

The 96-h LC_{50} values in bluegill (*Lepomis macrochirus*) were 0.64 mg/litre for C_{14} and 3 mg/litre for C_{12} LAS but 75 mg/litre for the intermediate degradation product, sulfophenylundecanoic acid disodium salt (Swisher et al., 1964). Biodegradation of LAS with a high relative molecular mass progressively shifted the homologue distribution in favour of shorter chain lengths and reduced the acute toxicity of the compound to bluegill (Dolan & Hendricks, 1976). Similar findings were reported for fathead minnow (Swisher et al., 1978), goldfish (*Carassius auratus*) (Divo & Cardini, 1980) and zebra fish (*Brachydanio rerio*) (Gard-Terech & Palla, 1986).

In rainbow trout (*Oncorhynchus mykissi*), addition of LAS (C_{10}–C_{15}) to activated sludge plant effluent increased the nominal 96-h LC_{50} from 0.36 to 29.5 mg/litre (Brown et al., 1978). No deaths were observed among bluegill exposed for 4–11 days to effluent from continuous-flow activated sludge units fed 100 mg/litre LAS (Swisher et al., 1964).

Water hardness was found to be the most significant environmental factor in the acute toxicity of LAS to bluegill, increasing with the level of hardness. At a water hardness of 15 mg/litre $CaCO_3$, the mean LC_{50} was 4.25 mg/litre; at 290 mg/litre $CaCO_3$, the LC_{50} was reduced to 2.85 mg/litre (Hokanson & Smith, 1971). Similarly, when water hardness was increased from 0 to 500 mg/litre $CaCO_3$, the LC_{50} for C_{18} LAS in goldfish was reduced from 15 to 5.7 mg/litre (Gafa, 1974). Exposure of the freshwater bleeker (*Puntius gonionotus*) to LAS gave 96-h LC_{50} values of 13.6 mg/litre at a water hardness of 50 mg/litre $CaCO_3$, 11.8 mg/litre at 110 mg/litre $CaCO_3$, and 11.4 mg/litre at 260 mg/litre $CaCO_3$ (Eyanoer et al., 1985).

The toxicity of $C_{11.7}$ LAS to the medaka (*Oryzias latipes*) increased with increasing salinity, but the effect was less pronounced than that of water hardness (Wakabayashi & Onizuka, 1986).

Temperature was reported to have no significant effect on the acute toxicity of LAS (Hokanson & Smith, 1971), but in another study increasing the water temperature from 28 to 35 °C marginally decreased the 96-h LC_{50} for the bleeker, from 11.8 to 11.5 mg/litre (Eyanoer et al., 1985).

A reduction in the dissolved oxygen concentration from 7.5 to 1.9 mg/litre reduced the 24-h LC_{50} in bluegill from 2.2 to 0.2 mg/litre. When the fish were first acclimatized to reduced oxygen levels, the effect was less pronounced (Hokanson & Smith, 1971).

No significant effect on the acute toxicity of LAS to bluegill was observed after a bentonite suspension was added to water at concentrations of 0, 50, or 200 mg/litre (Hokanson & Smith, 1971). Addition of gluten, however, reduced the 24-h and 48-h acute toxicity of LAS to both himedaka (*Oryzias latipes*) and cobalt suzume (*Chrysiptera hollisi*) (Iimori & Takita, 1979).

A9.3.3.2 *Chronic toxicity*

Exposure of the eggs of fathead minnows (*Pimephales promelas*) to LAS from laying until all surviving eggs had hatched under flow-through conditions gave a nine-day LC_{50} of 2.4 mg/litre, which would result in an LC_{50} of 3.4 mg/litre after 24 h of exposure (Pickering, 1966).

Eggs of cod (*Gadus morhua*) were exposed to LAS at a concentration of 0.005, 0.02, 0.05, or 0.1 mg/litre from fertilization until hatching under flow-through conditions. There were no significant effects at 0.005 mg/litre. At a concentration of 0.02 mg/litre, only 42% of the embryos completely developed into larvae, and there was an increased occurrence of tail malformations in comparison with controls. At 0.05 mg/litre, few eggs developed to embryos. No eggs developed to the blastula stage at a concentration of 0.1 mg/litre. In a repetition of the test at 0.05 mg/litre, fewer eggs and larvae died, but there was an increased frequency of abnormal embryos and inactive and crippled larvae (Swedmark & Granmo, 1981).

Eggs, larvae, and immature adult fathead minnows (*Pimephales promelas*) were exposed to LAS at a concentration of 0.34, 0.63, 1.2, or 2.7 mg/litre for up to four months. No significant effect was observed on the number of spawnings, the total number of eggs produced, the mean number of spawnings per female, the mean number of eggs per spawning, or the percentage hatchability; however, the two highest concentrations significantly reduced the survival of fry (Pickering & Thatcher, 1970).

The effects of $C_{11.8}$ and C_{13} LAS on the number of females, the number of spawnings, total number of eggs produced, and number of eggs per female were also studied in the fathead minnow over a period

of one year. As $C_{11.8}$ LAS had no significant effect on these parameters at a concentration of 1.09 mg/litre and a water hardness of 120 mg/litre $CaCO_3$, the NOEC was 0.9 mg/litre; C_{13} LAS were more toxic, and the NOEC was 0.15 mg/litre. At a lower water hardness (39 mg/litre), however, survival of larvae was impaired at 0.74 mg/litre (Maki, 1979a). NOECs in the fathead minnow in life-cycle and embryo-larval tests were dependent on mean alkyl chain length: 5.1–8.4 mg/litre for $C_{11.2}$, 0.48 mg/litre for $C_{11.7}$, and 0.11–0.25 mg/litre for $C_{13.3}$ (Holman & Macek, 1980).

The LC_{50} value of LAS in the eggs of carp (*Cyprinus carpio*) exposed from spawning to hatching was 11 mg/litre. In determinations of the sensitivity of eggs at different stages of development after spawning, the 24-h LC_{50} values were 15 mg/litre for eggs exposed between 2 and 26 h, 25 mg/litre for exposure between 26 and 50 h, and 32 mg/litre for exposure between 50 h and hatching (Kikuchi et al., 1976).

Bluegill (*Lepomis macrochirus*) were exposed to LAS from fertilization to yolk-sac resorption at a concentration of 1.8, 3.5, 4.6, or 5.5 mg/litre. The lowest concentrations did not affect hatchability or survival. Survival among those exposed to 3.5 mg/litre which hatched successfully was significantly reduced within two days of hatching, and 95% were dead by the end of the experiment. Eggs exposed to 4.6 or 5.5 mg/litre failed to hatch (Hokanson & Smith, 1971).

The NOEC of LAS in guppies (*Poecilia reticulata*), based on mortality, behaviour, and growth over 28 days, was 3.2 mg/litre (Canton & Slooff, 1982).

Studies of the short- and long-term toxicity of LAS to freshwater and marine fish are summarized in Tables 28 and 29.

A9.3.3.3 *Biochemical and physiological effects*

The main injury to the gills of catfish (*Heteropneustes fossilis*) exposed to LAS at 1 or 2.5 mg/litre was progressive separation of the lamellae from their vascular components. The activity of the enzymes of aerobic metabolism was decreased, and that of lactate dehydrogenase was strongly increased (Zaccone et al., 1985). Concentrations of 2.19 mg/litre $C_{11.8}$ LAS and 0.39 mg/litre C_{13} LAS significantly increased the 24-h mean ventilation rate (number of opercular closures per minute) of bluegill (*Lepomis macrochirus*) (Maki, 1979b).

Table 28. Toxicity of linear alkylbenzene sulfonates (LAS) to freshwater fish

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Brown trout (*Salmo trutta*)		Flow	15	26-30		NS	48-h LC_{50}	5.3	Reiff et al. (1979)
		Flow	15	26-30		NS	96-h LC_{50}	4.6	
		Flow	15	26-30		NS	48-h LC_{50}	2.3	
		Flow	15	26-30		NS	96-h LC_{50}	1.4	
		Flow	15	26-30		NS	48-h LC_{50}	0.4	
		Flow	15	26-30		NS	96-h LC_{50}	0.4	
		Flow	15	250		NS	48-h LC_{50}	2	
		Flow	15	250		NS	96-h LC_{50}	0.9	
		Flow	15	250		NS	48-h LC_{50}	0.7-0.9	
		Flow	15	250		C_{10}–C_{15}	48-h LC_{50}	0.2	
		Flow	15	250		C_{10}–C_{15}	96-h LC_{50}	0.1	
Masu trout (*Oncorhynchus masou*)	2 mo	Static[c]	8.5–9.6	30		$C_{11.7}$	96-h LC_{50}	4.4	Wakabayashi et al. (1984)
Rainbow trout (*Oncorhynchus mykiss*)		Flow	15	250		$C_{12.6}$	96-h LC_{50}	0.36[b]	Brown et al.(1978)
	40 d	Static[c]	8.8–10.9	25		$C_{11.7}$	96-h LC_{50}	4.7	Wakabayashi et al. (1984)
	4 d	Static[c]	10	25		$C_{11.7}$	96-h LC_{50}	2.1	Wakabayashi & Onizuka (1986)
	19 d	Static[c]	10	25		$C_{11.7}$	96-h LC_{50}	3.4	
Goldfish (*Carassius auratus*)		Static	20			C_{16}	6-h LC_{50}	61	Gafa (1974)
		Static	20			C_{17}	6-h LC_{50}	22.5	
		Static	20			C_{18}	6-h LC_{50}	8.5	

Table 28 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Goldfish (contd)		Static	20			C_{19}	6-h LC_{50}	3.3	
(*Carassius auratus*)		Static	20			C_{16}—C_{19}	6-h LC_{50}	7.6	
		Static	20			C_{16}–C_{19}	6-h LC_{50}	10	
		Static	20			C_{16}–C_{19}	6-h LC_{50}	12.2	
		Static	20	100		NS	6-h LC_{50}	8.2	Reiff et al. (1979)
		Static	20	100		NS	6-h LC_{50}	7	
		Static	20	100		NS	6-h LC_{50}	4.3	
	3.1–6.0	Flow	20–23	45–96	7.1–9.3		24-h LC_{50}	7.6	Tsai & McKee
	cm	Flow	20—23	45–96	7.1–9.3		48-h LC_{50}	7.5	(1978)
		Flow	20–23	45–96	7.1–9.3		72-h LC_{50}	7.0	
		Flow	20—23	45–96	7.1–9.3		96-h LC_{50}	6.2	
Bluegill sunfish	1.6 g	Static	23	76	7.5	av. C_{13}	48-h LC_{50}	0.72[b]	Dolan &
(*Lepomis macrochirus*)	1.6 g	Static	23	76	7.5	av. C_{13}	96-h LC_{50}	0.72[b]	Hendricks (1976)
		Flow	23	50	7.5	av. C_{13}	96-h LC_{50}	4[c]	Thatcher & Santner (1967)
	Finger	Static	25	15		$C_{11.2}$	48-h LC_{50}	4.0–4.5[b]	Hokanson &
	Finger	Static	25	290		$C_{11.2}$	48-h LC_{50}	2.8–2.9[b]	Smith (1971)
		Flow				$C_{11.8}$	96-h LC_{50}	1.7[c]	Bishop & Perry (1981)
Fathead minnow		Static				$C_{13.3}$	48-h LC_{50}	1.7[b]	Kimerle &
(*Pimephales promelas*)		Static				C_{10}	48-h LC_{50}	43[b]	Swisher (1977)
		Static				C_{11}	48-h LC_{50}	16[b]	

Table 28 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Fathead minnow (contd)		Static				C_{12}	48-h LC_{50}	4.7[b]	
		Static				C_{14}	48-h LC_{50}	0.4[b]	
	2–3 mo	Static		40		$C_{11.2}$	96-h LC_{50}	12.3[c]	Holman & Macek (1980)
	2–3 mo	Static		40		$C_{11.7}$	96-h LC_{50}	4.1[c]	
	2–3 mo	Static		40		$C_{13.3}$	96-h LC_{50}	0.86	
		Static	25				48-h LC_{50}	4.6	Pickering & Thatcher (1970)
		Static	25				96-h LC_{50}	5.0	
		Flow	15	43	7.2–7.9		96-h LC_{50}	3.4	McKim et al. (1975)
		Flow	23	50	7.5		96-h LC_{50}	4.2	Thatcher & Santner (1967)
		Flow	25				96-h LC_{50}	4.2-4.5	Pickering & Thatcher (1970)
	2.5 cm	Flow	18	116	7.9	C_{12}	96-h LC_{50}	3.5	Solon et al. (1969)
Harlequin fish (*Rasbora heteromorpha*)		Flow	20	20		NS	48-h LC_{50}	7.6	Reiff et al. (1979)
		Flow	20	20		NS	96-h LC_{50}	6.1	
		Flow	20	20		NS	48-h LC_{50}	5.1	
		Flow	20	20		NS	96-h LC_{50}	4.6	
		Flow	20	20		C_{10}–C_{15}	48-h LC_{50}	0.9	
		Flow	20	20		NS	96-h LC_{50}	0.7	
Carp (*Cyprinus carpio*)	4.4 mg	Static	22	25	7	$C_{11.7}$	12-h LC_{50}	5.6	Kikuchi et al. (1976)
		Static	22	25	7	$C_{11.7}$	48-h LC_{50}	5.6	

Table 28 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Carp (contd)	3.5–5.5	Static	21		7.5–7.8	NS	48-h LC_{50}	6.8	Lopez-Zavala et
	cm	Static	21		7.5–7.8	NS	96-h LC_{50}	5.0	al. (1975)
	7 d	Static[c]	22	25	7.0	$C_{11.7}$	48-h LC_{50}	5.6	Arima et al.
	6 mo	Static[c]	22	25	6.5–7.1	$C_{11.7}$	48-h LC_{50}	10	(1981)
	50 d	Static[c]	21	75		$C_{11.7}$	96-h LC_{50}	4.4	Wakabayashi et al. (1984)
	2 d	Static[c]	20	25		$C_{11.7}$	96-h LC_{50}	4.6	Wakabayashi &
	15 d	Static[c]	20	25		$C_{11.7}$	96-h LC_{50}	2.6	Onizuka (1986)
White tilapia	5–7 cm	Static	21		7.5–7.8	NS	48-h LC_{50}	26	Lopez-Zavala et
(*Tilapia melanopleura*)	5–7 cm	Static	21		7.5–7.8	NS	96-h LC_{50}	23	al. (1975)
Guppy (*Poecilia reticulata*)	3–4 wk	Static[c]	23			C_8–C_{14}	96-h LC_{50}	5.6–10	Canton & Slooff (1982)
Northern pike (*Esox lucius*)		Flow	15	43	7.2–7.9		96-h LC_{50}	3.7	McKim et al. (1975)
White sucker (*Catostomus commersoni*)		Flow	15	43	7.2–7.9		96-h LC_{50}	4	McKim et al. (1975)
Golden orfe		Static	18-20			C_8	48-h LC_{50}	125	Hirsch (1963)
(*Idus idus melanotus*)		Static	18-20			C_9	48-h LC_{50}	88	
		Static	18-20			C_{10}	48-h LC_{50}	16.6	

Table 28 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Golden orfe (contd)		Static	18–20			C_{11}	48-h LC_{50}	6.6	
		Static	18–20			C_{12}	48-h LC_{50}	2.6	
		Static	18–20			C_{13}	48-h LC_{50}	0.57	
		Static	18–20			C_{15}	48-h LC_{50}	0.23	
		Static	18–20			C_{16}	48-h LC_{50}	0.68	
	1.2–1.8	Static	20			NS	48-h LC_{50}	3.94	Mann (1976)
	g	Static	20			NS	48-h LC_{50}	1.85	
		Static	20			NS	48-h LC_{50}	1.24	
		Flow	20	150		NS	48-h LC_{50}	4.9	Reiff et al. (1979)
		Flow	20	150		NS	48-h LC_{50}	2.4	
		Flow	20	150		NS	48-h LC_{50}	1.2	
		Flow	20	268		NS	48-h LC_{50}	2.1–2.9	
		Flow	20	268		NS	96-h LC_{50}	1.9–2.9	
		Flow	20	268		NS	48-h LC_{50}	1.3–1.7	
		Flow	20	268		NS	96-h LC_{50}	1.2–1.3	
		Flow	20	268		NS	48-h LC_{50}	0.8–0.9	
		Flow	20	268		NS	96-h LC_{50}	0.4–0.6	
Himedaka (killifish) (*Oryzias latipes*)	4–5 wk	Static[f]	23			C_8–C_{14}	96-h LC_{50}	10–18	Canton & Slooff (1982)
	323 mg	Static[f]	23–24		5.6–6.1	$C_{11.7}$	48-h LC_{50}	15	Kikuchi et al.
	323 mg	Static[f]	23–24		5.6–6.1	$C_{11.7}$	48-h LC_{50}	10	(1976)
	~262 mg		Static[f]	21–22	6.7–7.1	C_{12}	48-h LC_{50}	12	Kikuchi &
	~262 mg		Static[f]	21–22	6.7–7.1	NS	48-h LC_{50}	10	Wakabayashi (1984)

Table 28 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Himedaka (contd)						C_{161}	48-h LC_{50}	> 50	Tomiyama (1974)
						C_6	48-h LC_{50}	> 50	
						C_8	48-h LC_{50}	> 50	
						C_{10}	48-h LC_{50}	> 50	
						C_{12}	48-h LC_{50}	4	
						C_{14}	48-h LC_{50}	4	Iimori & Takita (1979)
			25		7.2–7.9		48-h LC_{50}	7.6	Hidaka et al. (1984)
			25		7.2–7.9		96-h LC_{50}	7.3	
	Adult	Static[c]	20	5		$C_{11.7}$	96-h LC_{50}	13	Wakabayashi & Onizuka (1986)
	Adult	Static[c]	20	25		$C_{11.7}$	96-h LC_{50}	8.8	
	Adult	Static[c]	20	125		$C_{11.7}$	96-h LC_{50}	4.8	
	Adult	Static[c]	20	625		$C_{11.7}$	96-h LC_{50}	3.2	
	Adult	Static[c]	20	0		$C_{11.7}$	48-h LC_{50}	6.7	Wakabayashi & Onizuka (1986)
	Adult	Static[c]	20	1		$C_{11.7}$	48-h LC_{50}	4.8	
	Adult	Static[c]	20	5		$C_{11.7}$	48-h LC_{50}	4.7	
	Adult	Static[c]	20	10		$C_{11.7}$	48-h LC_{50}	3.5	
	Adult	Static[c]	20	15		$C_{11.7}$	48-h LC_{50}	3.8	
	Adult	Static[c]	20	20		$C_{11.7}$	48-h LC_{50}	2.5	
	Adult	Static[c]	20	25		$C_{11.7}$	48-h LC_{50}	1.9	
	Adult	Static[c]	20	30		$C_{11.7}$	48-h LC_{50}	1.6	
	Adult	Static[c]	20	35		$C_{11.7}$	48-h LC_{50}	1.4	

Table 28 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Cobalt suzume (*Chrysiptera hollisi*)							48-h LC_{50}	1.3	Iimori & Takita (1979)
Smallmouth bass (*Micropterus dolomieu*)		Flow	15	43	7.2–7.9	NS	96-h LC_{50}	3.7	McKim et al. (1975)
Black bullhead (*Ictalurus melas*)		Flow	23	50	7.5	NS	96-h LC_{50}	6.4	Thatcher & Santner (1967)
Common shiner (*Notropis cornutus*)		Flow	23	50	7.5	NS	96-h LC_{50}	4.9	Thatcher & Santner (1967)
Emerald shiner (*Notropis atherinoides*)		Flow	23	50	7.5	NS	96-h LC_{50}	3.3	Thatcher & Santner (1967)
Bleeker (*Puntius gonionotus*)	0.3 g	Static	28			NS	96-h LC_{50}	11.8	Eyanoer et al. (1985)
	0.3 g	Static	35			NS	96-h LC_{50}	11.5	
	0.3 g	Static		50		NS	96-h LC_{50}	13.6	
	0.3 g	Static		110		NS	96-h LC_{50}	11.8	
	0.3 g	Static		260		NS	96-h LC_{50}	11.4	

Table 28 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	LAS chain length	End-point	Concn (mg/litre)	Reference
Ayu	0.26 mg		1163			NS	48-h LC_{50}	0.86	Sueishi et al.
(*Plecoglossus altivelis*)	0.29 g		1163			NS	48-h LC_{50}	0.53	(1988)
	1.24 g		1163			NS	48-h LC_{50}	0.77	
	6.51 g		1163			NS	48-h LC_{50}	1.45	
	27.98 g		1163			NS	48-h LC_{50}	1.17	

Flow, flow-through conditions: LAS concentration in water maintained continuously; NS, not specified; static[r], static renewal: water changed periodically; static, water unchanged for the duration of test; finger, fingerling

[a] mg/litre $CaCO_3$

[b] Based on nominal concentration

[c] Based on measured concentrations

Table 29. Toxicity of linear alkylbenzene sulfonates (LAS) to marine fish

Organism	Size or age	Static or flow	Temp. (°C)	Salinity (‰)	LAS chain length	End-point	Concn (mg/litre)	Reference
Cod (*Gadus morhua*)	30 cm	Static	6–8	32–34	C_{12}	96-h LC_{50}	1[a]	Swedmark et al. (1971)
	30 cm	Static	15–17	32–34	C_{12}	96-h LC_{50}	< 1[a]	
Flounder (*Pleuronectes flesus*)		Static	6–8	32–34	C_{12}	96-h LC_{50}	1.5[a]	
		Static	15–17	32–34	C_{12}	96-h LC_{50}	< 1[a]	
Plaice (*Pleuronectes platessa*)		Static	6–8	32–34	C_{12}	96-h LC_{50}	> 1 –< 5[a]	
Mosbled sole (*Limanda yokohamae*)	Newly hatched				NS	48-h LC_{50}	0.5–1	Yasunaga (1976)
	10 days				NS	48-h LC_{50}	0.1–0.5	
	30 days				NS	48-h LC_{50}	0.5–1	
	40 days				NS	48-h LC_{50}	< 0.1	
	Newly hatched				NS	48-h LC_{50}	0.05–0.1	
Olive flounder (*Paralichtys olivaceus*)	5 days				NS	48-h LC_{50}	< 0.1	
	10 days				NS	48-h LC_{50}	0.1–0.5	
	30 days				NS	48-h LC_{50}	0.1–0.5	
Himedaka (killifish) (*Oryzias latipes*)	Adult	Static[c]	20	0	$C_{11.7}$	48-h LC_{50}	6.7	Wakabayashi & Onizuka (1986)
	Adult	Static[c]	20	1	$C_{11.7}$	48-h LC_{50}	4.8	
	Adult	Static[c]	20	5	$C_{11.7}$	48-h LC_{50}	4.7	

Table 29 (contd)

Organism	Size or age	Static or flow	Temp. (°C)	Salinity (‰)	LAS chain length	End-point	Concn (mg/litre)	Reference
Himedaka (contd)	Adult	Static[r]	20	10	$C_{11.7}$	48-h LC_{50}	3.5	
	Adult	Static[r]	20	15	$C_{11.7}$	48-h LC_{50}	3.8	
	Adult	Static[r]	20	20	$C_{11.7}$	48-h LC_{50}	2.5	
	Adult	Static[r]	20	25	$C_{11.7}$	48-h LC_{50}	1.9	
	Adult	Static[r]	20	30	$C_{11.7}$	48-h LC_{50}	1.6	
	Adult	Static[r]	20	35	$C_{11.7}$	48-h LC_{50}	1.2	

Static: water unchanged for duration of test; static[r], static renewal: water changed periodically; NS, not specified
[a] Based on nominal concentration

A concentration of 36 mg/litre LAS severely affected the viability of the perfused gills of rainbow trout (*Oncorhynchus mykissi*). Vascular resistance increased gradually during perfusion, with a concomitant decrease in oxygen transfer. LAS at 0.05 mg/litre more than doubled cadmium transfer (0.8 µg/litre) through the perfused gills; and at concentrations of 36 mg/litre LAS and 0.9 mg/litre cadmium, there was a marked reduction in cadmium transfer (Pärt et al., 1985).

A9.3.3.4 Behavioural effects

Hidaka and co-workers have reported several studies of the avoidance of surfactants by fish (Hidaka et al., 1984; Hidaka & Tatsukawa, 1989; Tatsukawa & Hidaka, 1978). The results should be interpreted with caution, since the environmental relevance and the reproducibility and sensitivity of these tests is unclear; furthermore, no effect was seen after removal of the olfactory organs. Another study (Maki, 1979a) showed no adverse toxicological effects at concentrations two times greater than those reported to cause avoidance reactions.

Hidaka et al. (1984) found that the minimal avoidance concentration of LAS, i.e. the concentration at which fish spent 65% of a 5-min period in clean water in order to avoid LAS, was 13.5 µg/litre for medakas (*Oryzias latipes*). Medakas exposed to LAS at concentrations of 5–50 µg/litre for 10 min showed significant avoidance to 10, 20, and 30 µg/litre. No significant avoidance of concentrations of 10–50 µg/litre LAS was found after removal of the olfactory organs (Hidaka & Tatsukawa, 1989).

The threshold concentrations for avoidance of LAS by ayu (*Plecoglossus altivelis*) were 0.11 µg/litre of a formulation and 1.5 µg/litre of pure reagant LAS (Tatsukawa & Hidaka, 1978).

A9.3.3.5 Interactive effects with other chemicals

The chronic toxicity of *para, para*-DDT (50 mg/litre) to goldfish (*Carassius auratus*) was increased by prior exposure to LAS at 4 mg/litre for 37 days (Dugan, 1967).

The toxicity of 1 mg/litre LAS solution to mosquito fish exposed under static conditions was not affected by allowing the LAS solution to react with excess chlorine (Katz & Cohen, 1976).

A concentration of 1 mg/litre LAS significantly increased the toxicity of fuel oil to bluegill (*Lepomis macrochirus*), reducing the 24-h

LC_{50} from 91 to 51 mg/litre. The authors concluded that sublethal concentrations of LAS increased the toxicity of fuel oil by increasing its penetration (Hokanson & Smith, 1971). The toxicity of No. 2 and No. 4 fuel oils in six species of freshwater fish was increased in the presence of 1-5 mg/litre LAS (Rehwoldt et al. , 1974).

The uptake of cadmium by freshwater trout (*Salmo gairdneri*) exposed to 0.14 μmol/litre LAS was more than two times greater than in controls. Reduced cadmium uptake was reported in fish exposed to 100 μmol/litre LAS. The authors reported that trout exposed to low levels of both LAS and cadmium could take up lethal cadmium concentrations. LAS were reported to interact with the gill proteins involved in cadmium transport, resulting in increased permeability to cadmium (Pärt et al., 1985).

Fathead minnows (*Pimepheles promelas*)were exposed to various pesticides in the presence and absence of 1 mg/litre LAS. Parathion acted synergistically with LAS, reducing the 96-h LC_{50} from 1410 to 720 μg /litre. Endrin and LAS showed no synergism, and no consistent results were obtained for DDT (Solon et al., 1969). Methyl parathion, ronnel, trithion, and trichloronat were also found to act synergistically with LAS, but neither *ortho*-ethyl-*ortho*-4-nitrophenyl phenylphosphonothioate nor dicapthon exhibited synergism. The synergistic relationship does not appear to be exclusive to a general structural group (Solon & Nair, 1970).

Goldfish (*Carassius auratus*) were exposed to mixtures of LAS and chloramines and LAS and copper at ratios of 1:1, 2:1, and 1:2, and toxicity curves and 24-h and 96-h LC_{50} values were compared. LAS and chloramines had an additive effect at a ratio of 1:1, but at 2:1 and 1:2 synergistic effects were seen. LAS and copper at ratios of 1:1 and 2:1 had additive effects; however, at 1:2, high concentrations and longer exposure times had additive effects, and low concentrations and shorter exposure times had synergistic effects (Tsai & McKee, 1978).

When eggs of cod (*Gadus morhua*) were exposed to mixtures of LAS and zinc or copper from fertilization to hatching, zinc had a weak synergistic affect on the toxicity of LAS, but LAS had a strong synergistic affect on the toxicity of copper (Swedmark & Granmo, 1981).

In a study of the effect of polyoxyethylene (20) on the acute toxicity of C_{12} LAS, red killifish (*Oryzias latipes*) and carp (*Cyprinus carpio*) were exposed to the 48-h LC_{50} of LAS for the respective species and to

5–40 mg/litre of either a polyoxyethylene sorbitan ester, a polyethylene glycol, a polypropylene glycol, or a protein (albumin, kaolin, or bentonite). Addition of most of these substances decreased mortality. No mortality was observed in carp exposed to LAS and 14 or 28 mg/litre polyoxyethylene (20) sorbitan monooleate (SMOE20) or to other nonionic surfactants with a similar polyoxyethylene sorbitan ester structure—polyoxyethylene (6) sorbitan monolaurate, polyoxyethylene (6) sorbitan monooleate, polyoxyethylene (20) sorbitan monolaurate, and polyoxyethylene (20) sorbitan monostearate—or to albumin. No significant effect on mortality induced by LAS was reported after simultaneous exposure to polyoxyethylene (6) sorbitan monostearate, polyethylene glycol, polypropylene glycol, kaolin, or bentonite. The authors also examined the histological effects of these chemicals on the gills of carp exposed to high concentrations of LAS, including the 48-h LC_{50} of 3.5 mg/litre and the LC_{100} of 7 mg/litre. Histological changes in fish exposed only to 3.5 mg/litre LAS included the appearance of mucous cells and agglutination of the secondary lamellae; carp exposed to a mixture of LAS and SMOE20 showed only slight swelling of the secondary lamellae and slight proliferation of the gill epidermal cells. Exposure only to LAS at 7 mg/litre resulted in marked proliferation of the epidermal cells and agglutination of secondary lamellae; exposure to both LAS and SMOE20 induced only swelling of the secondary lamellae. No effects were reported on the gills of control fish or on other organs of the exposed fish; and no significant differences from controls were reported in haematological or serum biochemical parameters for fish exposed to either LAS or the LAS:SMOE20 mixture. When the distribution of LAS in the tissues and organs of carp was examined, higher levels were found in the blood and most organs after exposure to LAS only than after exposure to the mixture; the differences were statistically significant in blood, muscle, and gill but not in spleen or gall-bladder. Adsorption of the 5- and 6-phenyl isomers of LAS was similar when they were given alone or in conjunction with SMOE20, but more of the 4- and (especially) the 2-phenyl isomers was adsorbed by fish receiving LAS alone, indicating that SMOE20 decreases the acute toxicity of LAS to fish by decreasing the adsorption on the gills of the more toxic isomer (Toshima et al., 1992).

A9.3.4 *Amphibia*

No reliable data were available.

A9.3.5 *Studies of the mesocosm and communities*

Diversity and similarity indices were used in many studies to assess the effects of LAS on phytoplankton communities, usually on the basis of taxonomy, mean number of species, and density. Mean density and similarity indices were then compared with those of controls. In general, these indices are not sensitive to change, as the densities of some species may decrease while the indices do not.

The effects of C_{12} and C_{13} LAS on short-term photosynthetic activity were studied in plankton sampled from Acton Lake, Ohio, United States, during May–October in the laboratory and *in situ*. Toxicity increased with the temperature of the water, the most sensitive period being June–August, and LAS were less toxic during periods of diatom dominance and low phytoplankton density. Thus the density of diatoms decreased during June–August, and that of green and blue algae increased. The comparison of the results of laboratory and field tests was highly dependent on species and the in-situ end-point. Short-term tests for photosynthetic activity *in situ* gave 3-h EC_{50} values of 0.2–8.1 mg/litre (mean, 1.9 mg/litre) for C_{13} LAS and 0.5–8.0 mg/litre (mean, 3.4 mg/litre) for C_{12} LAS (Lewis & Hamm, 1986). (See also section 9.3.1.1.)

In another study of the effect of LAS on phytoplankton communities in Acton Lake, Ohio (Lewis, 1986), phytoplankton were exposed *in situ* to LAS at a concentration of 0.01, 0.02, 0.24, 0.80, 27, or 108 mg/litre for 10 days. The LOEL for LAS, based on community similarity indices and the mean number of species, was 108 mg/litre. The similarity index (coefficient of community) decreased as the concentration of LAS increased, with calculated values of 0.62 at 0.01 mg/litre and 0.43 at 108 mg/litre. No significant effects were seen on the community diversity index or phytoplankton density. Green algae were the species least affected, on the basis of abundance, followed in order of decreasing tolerance by blue-green algae and diatom species. *Chlorophyta* species were the most abundant at higher concentrations of LAS, comprising 74% of the total cell volume at 108 mg/litre; their abundance tended to increase to a maximum at this concentration and then decrease to values similar to those of the controls. *Chlorophyta* species of the genera *Chlamydomonas, Oocystis,* and *Sphaerocystis* were not found after exposure to higher concentrations of LAS. *Chlamydomonas* was found only in waters with a concentration of LAS ≤ 0.8 mg/litre, and *Oocystis* and *Sphaerocystis* were found only at concentrations ≤ 27 mg/litre. The peak density of blue–green phytoplankton (56% of cell volume) was

achieved at 0.24 mg/litre LAS, declining to 17% at 108 mg/litre. The density of the major species, *Schizothrix calcicola*, was greatest at 27 mg/litre LAS but declined to a level below that of controls at 108 mg/litre LAS. The abundance of diatoms was low at all concentrations of LAS. At concentrations ≤ 0.24 mg/litre, the average density of diatoms was 23% of the total cell volume, similar to that of controls; at concentrations of 0.24-0.8 mg/litre, the diatom density was 10% of the cell volume. The mean densities of the major diatom species, such as *Cyclotella glomerata*, *Cyclotella pseudostelligera*, and *Nitzschia frustulum v. perminuta*, followed the overall trend for diatoms, reaching a peak at low LAS concentrations and declining to control values at higher concentrations.

In the same study, the laboratory-based 96-h EC_{50} values for exposure to $C_{11.8}$ LAS were calculated to be 29.0 mg/litre for *Selenastrum* and 0.0096 mg/litre for *Microcystis*, on the basis of population growth. The lowest concentration of LAS that produced a significant effect on algal growth in the laboratory was 0.05–1.0 mg/litre, which is considerably lower than the 27–108 mg/litre value found to be the lowest that altered the structure of a natural phytoplankton community. The differences between the results of laboratory and field tests were smaller for a laboratory-based EC_{50} than for an LOEL. Calculations based on the EC_{50} give a 30-fold difference for *Microcystis* but essentially no difference for *Selenastrum* (Lewis, 1986).

The toxic effects of LAS were also examined on periphyton communities above and below the outfall of a wastewater treatment plant on Little Miami River, Ohio, United States. The dominant species at both test sites were diatoms, *Amphora perpusilla* and *Navicula minima* accounting for at least 80% of the total cell volume. The tests were conducted *in situ*, with 21-day continous-flow exposure to LAS (average chain length, $C_{11.9}$) in river water entering submerged exposure tubes at a concentration of 0.2, 1.1, 9.8, or 28.1 mg/litre, after a four-week colonization period. The delivery rate of LAS was adjusted daily according to measurements of river flow in order to maintain the desired test concentrations. The periphyton at the site below the treatment plant outfall were exposed to LAS in the presence of 20–30% treated municipal effluent. No effects on the structure or function of the periphyton community above the outfall were reported after exposure to an average concentration of LAS ≤ 1 mg/litre. The lowest concentration that had an effect on the upstream periphyton community was 9.8 mg/litre, which reduced photosynthesis by 16%; a concentration of 28.1 mg/litre reduced photosynthesis by 64%, with a noticeable reduction in chlorophyll *a*. No effects on community similarity or

diversity were reported in comparison with control communities. Mean cell densities were increased by 26% after exposure to 0.2 mg/litre LAS and by 17% after exposure to 1.1 mg/litre; exposure to 28.1 mg/litre reduced mean cell density by 28%. Exposure to LAS had no significant effects on the abundance of the three main species in the upstream community. Increased photosynthesis (by 12–39%) and chlorophyll *a* (50–51%), were reported after exposure to 1.1 or 9.8 mg/litre LAS, but exposure to 28.1 mg/litre resulted in a 52% decrease in photosynthesis and a 71% decrease in chlorophyll *a*. No effects on the similarity or diversity of the periphyton community were reported at any concentration of LAS tested. Cell densities of periphyton were increased by 34% after exposure to 9.8 mg/litre LAS and by 13% after exposure to 28.1 mg/litre. The abundance of the three main species in the downstream periphyton community was not affected. The lowest concentration of LAS that induced an effect was 3.3 mg/litre for the upstream periphyton community and 16.6 mg/litre for the downstream community. The authors suggested that the difference between the two values was due to the presence of 20–30% sewage downstream, which reduced the bioavailability of LAS (Lewis et al., in press).

When an aquatic ecosystem was exposed to LAS at concentrations of 0.25–1.1 mg/litre for 90 days, the numbers of phytoplankton were unaffected but primary productivity was significantly reduced at all concentrations. The zooplankton population showed a more variable response: the number of rotifers was reduced at all concentrations, and those of *Diaptomus* and *Cyclops* were reduced at ≥ 0.51 mg/litre. The number of ostracods was decreased at 0.38 mg/litre but was increased at 0.51 and 1.1 mg/litre. The chironomid population was significantly reduced at concentrations ≥ 0.38 mg/litre (Chattopadhyay & Konar, 1985). Exposure of an aquatic ecosystem consisting of phytoplankton, zooplankton, and benthic organisms to 1 mg/litre of a preparation of LAS for 90 days significantly reduced the numbers of phytoplankton and zooplankton per litre but did not significantly affect the numbers of chironomid larvae (Panigrahi & Konar (1986).

The effect of C_{12} LAS on microbial communities was studied in a model ecosystem consisting of a 19-litre glass tank containing sediment from Winton Lake, Ohio, United States, and several trophic levels, comprising bacteria, algae, macrophytes (*Elodea canadensis*, *Lemna minor*), macroinvertebrates (*Daphnia magna*, *Parantanytarsus parthenogenica*), and blugill sunfish (*Lepomis macrochirus*). After a four-week equilibrium period, LAS were added at 0.5 or 5.0 mg/litre to a flow-through system with six to 10 replacements per day for 26 days. The structure of the

microbial communities was not affected, and no differences were reported in mean biomass or number of colony-forming units between the microorganisms exposed at the two levels. The function of the microbial communities, assayed by measuring the degradation of both LAS and D-glucose, was reduced only at 5.0 mg/litre. In a similar system, in which the same concentrations of LAS were added in the form of sewage effluent, no effect was seen on the structure of the microbial community or on their function, measured only as the degradation of LAS (Larson & Maki, 1982).

Addition of LAS (average chain length, $C_{11.8}$) at a measured, relatively uniform concentration of 0.36 ± 0.05 mg/litre to 50-m outdoor experimental streams had no effect on total density, species richness, percentage similarity, or dominance of macroinvertebrates or periphyton or on the processing of organic matter of leaf discs. Fathead minnows (*Pimephales promelas*) and amphipods (*Hyallela azteca*) were exposed in groups of 10 and 20 per box placed in the streams at three locations. The mortality rates of the amphipods were 17–25% after exposure to LAS and 47% among controls; no effects were seen on the survival or weights of the fish, although minor effects were found on length (Fairchild et al., 1993).

A study of the fate and effects of surfactants in outdoor artificial streams addressed the effect of LAS on drift and population densities of macroinverebrates, the reproductive behaviour of an amphipod, the scud (*Gammarus pulex*), the survival of a fish, the three-spined stickleback (*Gasterosteus aculeatus*), and photosynthesis by the community. The concentration of LAS in sediment was reported to increase with increasing water concentration, and selective adsorption of longer-chain LAS homologues to sediment was reported. The microbial populations of both the water and the sediment adapted to LAS, resulting in a reduction in its half-life during the test. LAS at concentrations < 1.5 mg/litre did not affect macroinverebrate drift, population density, or community photosynthesis. Survival of the fish and reproduction by the amphipod were affected at concentrations of 1.5–3.0 mg/litre (Mitchell & Holt, 1993).

A9.3.6 *Field studies*

The effect of LAS downstream of a sewage outflow was studied by monitoring sediment, water, and the distribution of invertebrates at an upstream control site, a site near the discharge point, and a site 200 m downstream of the outflow. The concentrations of LAS in sediment were 1–40 mg/kg dry weight, with concentrations < 2 mg/kg at the

control site and 200 m downstream. No effect of LAS in the effluent or in the streambed sediments could be discerned on the invertebrate populations (Ladle et al., 1989).

A9.3.7 *Toxicity of biodegradation intermediates and impurities of linear alkylbenzene sulfonates*

Tests of degradation products and impurities of LAS show that they are less toxic than LAS themselves.

A9.3.7.1 *Individual compounds*

The 48-h LC_{50} values in *Daphnia magna* were 208 ± 85 mg/litre for sulfophenylundecanoic acid, disodium salt (mixed isomers, 6–10 phenyl); about 6000 mg/litre for 3-(sulfophenyl) butyric acid, disodium salt; and about 5000 mg/litre for 4-(sulfophenyl) valeric acid, disodium salt. The equivalent 48-h LC_{50} values in the fathead minnow (*Pimephales promelas*) were 77 ± 12, about 10 000, and about 10 000 mg/litre, respectively (Kimerle & Swisher, 1977).

The 24-h LC_{50} values in *Daphnia* were about 22 000 mg/litre for 3-sulfophenylbutyric acid, disodium salt; about 12 000 mg/litre for 3-sulfophenylheptanoic acid, disodium salt; > 22 000 mg/litre for 3-sulfophenylbutyric acid, disodium salt; and 2 000 mg/litre for sulfophenylundecanoic acid, disodium salt. Other tests were carried out with the last two compounds, giving 96-h LC_{50} values of about 28 000 and 1200 mg/litre in fathead minnows (*Pimephales promelas*); 28-day NOELs of > 2000 and > 200 mg/litre for survival and reproduction of *Daphnia*; and 30-day NOECs of > 1400 and > 52 mg/litre for survival of the fry of fathead minnows (egg hatchability and fry growth were less sensitive) (Swisher et al., 1978).

The 96-h LC_{50} for mixed isomers of sulfophenylundecanoic acid disodium salt in bluegill (*Leponis macrochirus*) was 75 mg/litre (Swisher et al., 1964). The 24–96-h LC_{50} values in fathead minnows were 1000–1500 mg/litre for sulfophenylundecanoic acid (C_{11}) and 25 000– 32 000 mg/litre for sulfophenyl butyrate (C_4) (Swisher et al., 1978).

The 48-h LC_{50} for the alkanoic acid derivatives of 2-sulfophenyl C_{13} LAS and 4-sulfophenyl C_{13} LAS in nearly pure form was > 800 mg/litre in goldfish (*Carassius auratus*) (Divo & Cardini, 1980).

The 24-h LC_{50} values for *Daphnia magna* exposed to dialkyltetralin sulfonates, which are trace contaminants of LAS, were 420, 195, 110, 50,

and 27 mg/litre for tetralin sodium sulfonates of chain lengths C_{10}, C_{11}, C_{12}, and C_{13}, respectively (Arthur D. Little Inc., 1991).

A9.3.7.2 Effluents

Interpretation of tests on effluents must take into account the following:

- As concentrations arwe often reported as MBAS, testing of effluent from a sewage treatment plant may result in overestimation of the actual concentrations of LAS, owing to interference (see section 2.3).
- The bioavailability of LAS is decreased by the presence of high concentrations of suspended solids; thus, as effluents are diluted in the environment, availability is usually increased, although biodegradation occurs.

Addition of LAS (C_{10}–C_{15}) to detergent-free activated sludge plant effluent (95% was removed as MBAS) gave a nominal 96-h LC_{50} in rainbow trout (*Oncorhynchus mykiss*)of 0.36 mg/litre. After treatment, the 96-h LC_{50} was 29.5 mg/litre, expressed in terms of the concentration of the surfactant in the influent (Brown et al., 1978).

When bluegill were exposed to effluent from continuous-flow activated sludge units fed 100 mg/litre LAS, none died during 4–11-day exposure (Swisher et al., 1964).

A9.4 Terrestrial organisms

A9.4.1 Terrestrial plants

Young seedlings of tomato, lettuce, radish, pea, cucumber, and barley were grown in a soil-based compost and were watered and given a foliar spray of a preparation of LAS. No effects were noted at concentrations up to 100 mg/litre (Gilbert & Pettigrew, 1984). In another study, barley, tomato, and bean plants were grown from seed and watered with a solution containing LAS at a concentration of 10, 25, or 40 mg/litre. Plants that received the lowest dose germinated at the same time as controls, but plants watered at 25 or 40 mg/litre germinated three days later. The growth of barley plants was inhibited at all three concentrations; however, the dose of 25 mg/litre increased the growth rate of beans, and the highest dose increased the growth rate of both tomatoes and beans (Lopez-Zavala et al., 1975).

The 21-day EC_{50} values for LAS (C_{10}–C_{13}), based on the emergence of seedlings and early stages of growth, were 167 mg/litre in sorghum, 289 mg/litre in sunflower, and 316 mg/kg in mung bean. The highest concentration that caused no significant reduction in the growth of any of the three species was 100 mg/kg (Holt et al., 1989; Mieure et al., 1990). In a second study, 407 mg/kg $C_{11.36}$ or 393 mg/kg $C_{13.13}$ LAS were mixed with sewage sludge, and nine common plant species, including five crop plants, were exposed as seed either at the same time or two weeks after application of the sludge to soil at a rate of 9000 kg/ha. There was no significant effect on seed germination and no significant inhibition of growth (Mieure et al., 1990).

Orchid seedlings (*Phalaenopsis* or *Epidendrum* sp.) were grown in culture media containing either the sodium or the ammonium salt of LAS at a concentration of 10, 100, or 1000 mg/litre. The lowest dose had no effect on growth, and that of ammonium LAS had no effect on germination. At 100 mg/litre, survival was halved and germination completely inhibited (Ernst et al., 1971). A concentration of 1000 mg/litre caused drastic changes in morphology, loss of membranes, swelling of thylakoids, and the appearance of dense osmophilic granules in chloroplasts (Healey et al., 1971).

The growth of pea seedlings grown for 26 days in quartz sand to which 0.005% (50 mg/kg) LAS had been added was significantly reduced, as measured by the fresh weight of roots and the length and fresh weight of pea greens (Lichtenstein et al., 1967).

LAS were not toxic with respect to growth at the early life stages of radish, Chinese cabbage, and rice when added in hydroponic culture at concentrations of 10, 20, and 20 mg/litre, respectively; concentrations of 20, 35, and 35 mg/litre were toxic (Takita, 1982).

When seeds of *Pisum sativum* and *Crotolaria juncea* were exposed to LAS for 24 h before sowing, the percentage germination was reduced at concentrations of 1 ml/litre for *P. sativum* and 10 ml/litre for *C. juncea*, although no statistical analysis was presented. No germination occurred after exposure to LAS at concentrations of 20 ml/litre for *P. sativum* and 40 ml/litre for *C. juncea*. Radicle length was reduced at ≥ 0.1 ml/litre in both species (Sharma et al., 1985).

Application of LAS at 50 g/m^2 under field conditions to loamy and sandy soils (corresponding to 0.47–1 mg/kg dry weight, respectively) led to considerable physiological damage, including leaf necrosis,

chlorosis, and turgescence, to ryegrass (*Lolium perenne*) after 14 days; however, there was no difference in the fresh weight yield after harvesting at 45–54 days (Litz et al., 1987).

A9.4.2 Terrestrial invertebrates

When the earthworm *Eisenia foetida* was exposed to $C_{11.36}$ LAS incorporated into soil at nominal concentrations of 63–1000 mg/kg dry weight, the 14-day LC_{50} was > 1000 mg/kg. On the basis of a statistical analysis of body weights, the no-effect concentration was 250 mg/kg; this was confirmed by HPLC to be 235 mg/kg. In a second study, $C_{11.36}$ and $C_{13.13}$ LAS were incorporated into sludge and applied to soil, and the earthworm *Lumbricus terrestris* was exposed to the subsequent mixture, which contained LAS at concentrations of 84–1333 mg/kg. The 14-day LC_{50} was again found to be greater than the highest concentration (> 1333 mg/kg). The no-effect concentration, based on weight and burrowing behaviour, was the nominal concentration of 667 mg/kg, measured by HPLC as 613 mg/kg. The worms were exposed, however, to LAS under conditions of continuous light, which would inhibit them from surfacing to feed and thus increase their exposure to and the toxicity of the test over that of the same concentration in the field (Mieure et al., 1990).

Topical application to house flies (*Musca domestica*) of LAS at the same time as parathion, diazinon, or dieldrin in ratios of 1:1 and 1:10 had no effect on the toxicity of the insecticides. When LAS were added to soil treated with parathion or diazinon, however, a significant synergistic effect was observed on the toxicity of the insecticides to the fruit fly *Drosophila melanogaster*. The optimal concentration of LAS that resulted in synergy was 23 mg/kg (Lichtenstein, 1966).

A9.4.3 Birds

No significant effect on egg quality was found after Leghorn chickens were fed a diet containing 200 mg/kg LAS for 45 days (Lopez-Zavala et al., 1975).

B. α-Olefin sulfonates

B1. SUMMARY

See Overall Summary, Evaluation, and Recommendations (pp. 7–21).

B2. IDENTITY, PHYSICAL AND CHEMICAL PROPERTIES, AND ANALYTICAL METHODS

B2.1 Identity

Chemical formula:	$C_nH_{2n}O_3S$ Na, $C_nH_{2n+1}O_4S$ Na (n = 14–18)
Chemical structure:	$CH_3(CH_2)_jCH{:}CH(CH_2)_kSO_3^-$ Na^+ $CH_3(CH_2)_mCH(CH_2)_nSO_3^-$ Na^+ OH (m,n, integers)
Common names:	Sodium alpha-olefinsulfonate, alpha-olefin-sulfonic acid sodium salt, AOS sodium salt
Common trade names:	Bioterge AS 40 F, Elfan OS 46, Geropon MLS/A, Hostapur OS Brands, Lipolan, Lipomix G, Lipon PB-800, Lutensit A-PS, Nansa LSS38/AS, Sawaclean, Sermul EA 214, Sulframin AOS, Witconate (McCutcheon, 1989)
Abbreviations:	AOS, AOS-Na
CAS Registry numbers:	29963-33-5 Sodium 1-tetradecenesulfonate 29734-60-9: Sodium hexadecenesulfonate 13513-23-0: Sodium 3-hydroxyhexadecyl-1-sulfonate 26446-92-4: Octadecene-1-sulfonic acid sodium salt 13513-42-3: 3-Hydroxy-1-octadecanesulfonic acid, sodium salt

Specifications: AOS are mixtures consisting of about 60-65% alkene sulfonates, 30–35% hydroxylalkane sulfonates, and 5–10% disulfonates. Various positional isomers of alkene sulfonates and hydroxyalkane sulfonates have been reported (Gentempo et al., 1985; Williamson, 1993). Sodium C_{14}–C_{16} AOS are typically shipped as solutions containing 35–40% active matter in water. Sodium C_{16}–C_{18} AOS are typically slurries containing 28–30% active matter in water at ambient temperature.

B2.2 Physical and chemical properties

AOS are white crystalline solids consisting of various chemical compounds and their isomers, with different properties. Typical properties of AOS are given in Table 30. Two ranges are usually offered; the commonest are based on C_{14}–C_{16} olefin and the other on C_{16}–C_{18} olefin. Detergency is maximal with alkyl chain lengths of C_{15}–C_{18}. Maximal detergency is also obtained with the same range of alkyl chain lengths in a detergent formulation that includes alkali builders and chelating agents (Yamane et al., 1970). AOS are stable, even in hot acidic media.

B2.3 Analytical methods

There is no officially recognized specific procedure for the analysis of AOS in environmental samples. The methods commonly used to analyse anionic surfactants are also used for AOS, except those involving high-performance liquid chromatography (HPLC), which has limited use in environmental analyses for AOS, because they do not absorb ultra-violet radiation as effectively as do linear alkylbenzene sulfonates (LAS). A modified version of the methylene blue-active substance (MBAS)–HPLC method described in the monograph on LAS has been developed (Takita & Oba, 1985).

Nonspecific methods used in the analysis of anionic surfactants in general, such as the MBAS method, can be used to analyse materials for AOS (see section 2.3 of the monograph on LAS).

Table 30. Relationship between alkyl chain length, Krafft point, critical micelle concentration (CMC), and surface tension of α-olefin sulfonates

Alkyl chain length	Krafft point[a] (°C)	CMC[a] (g/litre)	Surface tension (dyne/cm)
12	–	4.0	–
14	–	1.0	30
16	10	0.3	33
18	30	0.1	35
20	40	–	–
		(25 °C)	(25 °C)

From Ohki & Tokiwa (1970)
[a]The solubility of surfactants in water, defined as the concentration of dissolved molecules in equilibrium with a crystalline surfactant phase, increases with rising temperature. For surfactants, there is a distinct, sharp bend (break-point) in the solubility-temperature curve. The steep increase in solubility above the sharp bend is caused by micelle formation. The point of intersection of the solubility and critical micelle curves, plotted as a function of temperature, is referred to as the Krafft point. This is a triple point at which surfactant molecules coexist as monomers, micelles, and hydrated solids. The temperature corresponding to the Krafft point is called the Krafft temperature. At above the Krafft temperature and critical micelle concentration, a micellar solution is formed. Under these conditions, higher levels than the aqueous solubility may be obtained.

B3. SOURCES OF HUMAN AND ENVIRONMENTAL EXPOSURE

B3.1 Natural occurrence

AOS do not occur naturally.

B3.2 Anthropogenic sources

B3.2.1 Production levels and processes

AOS are synthesized industrially. Although they have been available since the 1930s, production for use in commercial surfactant formulations was somewhat limited until recently owing to a lack of suitable feedstock. Development of continuous and short-contact sulfur trioxide sulfonation processes and the increased availability of highly pure Ziegler-derived α-olefin feedstock has recently made AOS surfactants competitive with other surfactants on the market (Arthur D. Little Inc., 1977, 1981).

The estimated world consumption of AOS in 1988 was 50 200 tonnes (Colin A. Houston & Associates Inc., 1990). In 1990, that group estimated that world consumption would be 51 900 tonnes; an alternative estimate (Hewin International Inc. 1992) was 76 000 tonnes (Table 31).

Table 31. Estimated worldwide consumption of α-olefin sulfonates (tonnes)

Region	Household products	Personal care products	Industrial and institutional use	All uses
North America	3 000	7 000	4 000	14 000
Western Europe	2 000	3 000	3 000	8 000
Japan	24 000	7 000	2 000	33 000
Rest of the world	18 000	3 000	–	21 000
Total	57 000	20 000	9 000	76 000

From Hewin International Inc. (1992)

AOS are prepared commercially by direct sulfonation of linear α-olefins with a dilute stream of vaporized sulfur trioxide in a continuous thin-film reactor. The olefin is obtained by wax cracking or ethylene polymerization with a Ziegler-type catalyst (Tomiyama, 1970). The reaction is complex and follows several paths, forming large amounts of various sultones as intermediates which hydrolyse during subsequent quenching and neutralization. Commercial AOS products contain a mixture of two major components, alkene sulfonate and hydroxyalkane sulfonate, with smaller amounts of alkene disulfonates, hydroxyalkane disulfonates, and saturated sultones.

B3.2.2 *Uses*

AOS are good detergents, have good foaming characteristics in hard water and are used in heavy-duty laundry detergents, light-duty dishwashing detergents, shampoos, and cosmetics. Table 31 indicates the use patterns for AOS.

B4. ENVIRONMENTAL TRANSPORT, DISTRIBUTION, AND TRANSFORMATION

Section summary

It can be inferred that AOS are transported into the environment in a similar manner to that established for LAS, alkyl sulfates, and other detergent surfactants. Fewer data are available on the environmental transport, distribution, and transformation of AOS than for LAS. The environmental fate of AOS is similar to that of LAS and alkyl sulfates: it is readily biodegraded under aerobic conditions, and primary biodegradation is complete within 2–10 days, depending on the temperature. At temperatures below 5–10 °C, biodegradation kinetics are reduced, owing to a reduction in microbial activity. No data were available on abiotic degradation. There was no evidence of bioaccumulation or bioconcentration in a study of fish in which the uptake and distribution of AOS were examined.

B4.1 Transport and distribution between media

In the same manner as other detergent compounds, AOS are discharged into the environment in wastewater. The wastewater may undergo sewage treatment if such facilities are available. In countries where there are no adequate wastewater treatment facilities, AOS released to the environment are removed by biodegradation and adsorption mechanisms (see section 4.2 of the monograph on LAS).

Limited studies of the adsorption of AOS are available. In a study of the adsorption of C_{12} AOS on river sediments, the equilibrium quantities adsorbed were proportional to the organic carbon content of the sediments, with a sorption coefficient K_{oc} (dimensionless; normalized for the level of organic matter) of 0.65. This indicates that adsorption of C_{12}AOS is slightly weaker, than, for example, that of C_{12}LAS or C_{12}alkyl sulfonates (Urano et al., 1984). Like other detergent chemicals, AOS are adsorbed onto sewage sludge and river sediments in the environment.

B4.2 Biotransformation

B4.2.1 Biodegradation

B4.2.1.1 Aerobic biodegradation

Primary biodegradation of AOS, studied in die-away tests in water from various sites on the Tama River, Japan, was complete within three

to five days when measured by the MBAS method; however, total organic carbon was completely removed after an incubation time of 20 days. In a study of AOS in seawater collected from the mouth of the Tama River, 99% of MBAS was removed within one day, and 90% of organic carbon was removed within five days (Sekiguchi et al., 1975b).

In a comparison of the MBAS and total organic carbon methods for measuring biodegradation with the shake-culture method, AOS lost 99% of their activity as measured by the MBAS method and 90% of total carbon within one day; 100% was lost within five days (Sekiguchi et al., 1975a). In another study, complete loss of parent AOS (initial concentration, 100 mg/litre) as determined by the MBAS method was seen within 15 days, and 90% of total organic carbon was removed within eight days (Miura et al., 1979). In a static die-away test system, 90% biodegradation of three commercial AOS products, comprising 100% C_{14}–C_{16} AOS and > 95% C_{15}–C_{18} AOS (determined as MBAS), was reported within four days (Gafa & Lattanzi, 1974).

In a shake-culture test in Bunch-Cambers medium, C_{15}–C_{18} AOS were degraded by 99% (determined as MBAS) or 90% (removal of total organic carbon) within one day; 100% total organic carbon was removed within five days. The authors did not verify whether the removal was the result of adsorption or mineralization (Sekiguchi et al., 1972). The biodegradation of C_{15} AOS and three C_{15}–C_{18} compounds with disulfonate contents of < 4, 15, and 50% in a shake-flask culture system was reported to be 96% (determined as MBAS), with no significant difference between compounds (Oba et al., 1968b).

In a modified OECD screening test, 85% of C_{14}–C_{18} AOS (measured as chemical oxygen demand) was removed. Measurement of MBAS in the same test indicated 99% removal (Gerike, 1987).

The aerobic biodegradation of 20 mg/litre AOS at 27 °C was followed during a 10-day incubation period. Primary degradation, measured by the MBAS method, was complete within 10 days. The theoretical CO_2 production had reached 30-40% within that time (Itoh et al., 1979).

The oxygen uptake of C_{14}–C_{18} AOS was reported to be 85% of the theoretical oxygen demand in a closed-bottle test (Gerike, 1987). The average biochemical oxygen demand for C_{12}–C_{18} AOS containing up to 40% hydroxylalkane sulfonates was 51.6% at five days, while glucose under the same conditions had a biochemical oxygen demand of 69.6% (Procter & Gamble Co, unpublished data).

The primary and ultimate biodegradability of a series of pure AOS homologues (C_{12}, C_{14}, C_{16}, and C_{18}) was determined by measuing CO_2 production. Primary biodegradation was 98-99% within three days, the rate of degradation varying with chain length. Degradation of C_{12} and C_{14} AOS occurred at a similar rate (65% within 30 days), but C_{18} AOS degraded more slowly. Mineralization of all AOS samples was reported to be at least 50% within two weeks, whereas mineralization of glucose during that time was 75-80% (Kravetz et al., 1982). In a study of the biodegradation of the two major breakdown products of AOS, alkene sulfonate and hydroxyalkane sulfonate, AOS homologues (C_{15}, C_{16}, C_{17}, C_{18}) were degraded to about 50%, and in each case the alkene sulfonate was degraded at least twice as fast as the hydroxyalkane sulfonate (Sekiguchi et al., 1975c).

The biodegradation of C_{18} AOS at a concentration of 28 mg/litre was studied in activated sludge (concentration, 100 mg dry matter per litre) over 12 days: 90% was lost within eight days, as measured by removal of chemical oxygen demand. The specific rate of biodegradation was calculated to be 5.3 mg/g per h (Pitter & Fuka, 1979).

In the OECD confirmatory test with activated sludge, 20 mg/litre AOS were degraded, as follows: 97% C_{14} AOS within 17 days, 98% C_{16} AOS within seven days, and 94% C_{14}-C_{18} AOS within eight days (Maag et al., 1975).

Primary biodegradation of C_{15}–C_{18} AOS was dependent on incubation temperature in die-away tests with water from the Tama River, Japan. Primary biodegradation was complete within two days at 27 °C, within five days at 15 °C, and within two days at 21 °C; however, at a water temperature of 10 °C about 20% of the AOS had still not been degraded within the nine-day test (Kikuchi, 1985).

When C_{15}–C_{18} AOS were added to seawater, no MBAS activity was present after five days (Marquis et al., 1966).

B4.2.1.2 *Anaerobic degradation*

The primary anaerobic biodegradation of C_{15}–C_{18} AOS (measured as MBAS) by bacteria on sludge sampled from a sewage treatment plant was 19% within 14 days and 31% within 28 days. More parent AOS were degraded by bacteria from the bottom of a private cesspool, with 34% lost within 14 days and 43% within 28 days. The anaerobic degradation reported may have been due to the presence of hydroxyalkane sulfonate

compounds (Oba et al., 1967). AOS and LAS were reported to be the two surfactants that were least degraded anaerobically (Itoh et al., 1987).

B4.2.2 *Abiotic degradation*

No information was available.

B4.2.3 *Bioaccumulation and biomagnification*

Rapid, significant absorption of ^{14}C-AOS by the gills of goldfish (*Carassius auratus*) was seen after exposure to AOS at a concentration of 10 mg/litre. The concentration of AOS in the gills increased from 0.3 mg/kg after 0.5 h of exposure to 48.3 mg/kg after 3 h. AOS were not detected in the alimentary canal (Tomiyama, 1975). Three hours is a relatively short exposure, and the authors did not determine whether a steady state of adsorption had been achieved. Tomiyama (1978) reported that AOS accumulated to the greatest extent in the gills of exposed fish, with additional accumulation in the gall-bladder. Only limited conclusions can be drawn from this study, however, owing to the short exposure period

B4.3 Interaction with other physical, chemical, and biological factors

No information was available.

B5. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

Few data are available on environmental concentrations of AOS because of the lack of an accepted analytical method for this purpose. A modified analytical method based on MBAS–HPLC measurement has been used to measure AOS (Takita & Oba, 1985). The concentration in the Tama River, Japan, was calculated to be < 0.0016–0.002 mg/litre.

The annual average concentration of AOS in wastewater was 0.160–0.164 mg/litre on the basis of total MBAS concentrations of 8.4 and 8.2 mg/litre. AOS was not detected in the effluent from a treatment plant outfall (Oba et al., 1976).

AOS can be expected to mineralize rapidly in all environmental compartments and to be removed to a large extent during sewage treatment. Environmental concentrations in receiving surface waters, sediments, soils, estuaries, and the marine environment can also be expected to be low.

B6. KINETICS

Section summary

AOS administered orally are readily absorbed by the gastrointestinal tract of rats and are distributed throughout the body; they are eliminated primarily in the urine and, to a lesser extent, in the faeces within 24 h of administration. AOS applied dermally are absorbed only minimally by intact skin. Several metabolites have been isolated, but their chemical structures have not been identified.

B6.1 Absorption, distribution, and excretion

^{14}C-AOS were synthesized by sulfonation and hydrolysis of tetradecene-1-^{14}C. The labelled compound was composed of a mixture of about 55% sodium 3-hydroxyalkane sulfonate [$C_{11}H_{23}CH(OH)$-CH_2SO_3Na] and about 45% sodium 2-^{14}C alkenyl sulfonate [$C_{11}H_{23}CH_2CH_2{}^{14}CH_2SO_3Na$]. After oral administration of 100 mg /kg ^{14}C-AOS (50 µCi/kg) in water to rats, the level of radiolabel in blood reached a peak at 3 h (0.08% of the dose/ml) and then rapidly decreased, since little radioactivity was detected 24 h after the administration. At 4 h after administration, 0.45% of the dose per gram of tissue was detected in liver and 0.65% in kidney, but the levels in tissues other than the gastrointestinal tract were < 0.1%. Thereafter, the radiolabel in organs and tissues decreased rapidly, and 24 h after administration, about 0.8% was detected in the caecal contents and < 0.02% in other tissues. No specific accumulation was observed in any tissue. Within 24 h of administration, 72% of the dose was excreted in urine and 22% in faeces. At the end of the experiment, after four days, no ^{14}C residue (< 0.1% of the dose) was detected in urine or faeces. Cumulative excretion in the bile within 12 h after administration was about 4.3% of the radioactivity administered (Inoue et al., 1982).

The biological half-lives of AOS and their metabolites in blood after intravenous administration of 10 mg/kg ^{14}C-AOS in rats were 15 and 1 h, respectively. The marked difference in half-life can be accounted for by the fact that the binding of AOS to plasma proteins, especially serum albumin, increased in proportion to its concentration while that of the metabolites did not increase to any appreciable extent. The volume of distribution of AOS was 8 litres/kg, and that of the metabolites was 0.5 litres/kg (Inoue et al., 1982).

A dose of 0.5 ml of a 0.2% aqueous solution of ^{14}C-AOS was applied to the dorsal skin (4 x 3 cm) of rats with bile-duct and bladder cannulae. The total amount absorbed through the skin was estimated to be about 0.6% on the basis of the recoveries of ^{14}C in urine, bile, and the main organs over 24 h. At that time, the level of radiolabel was higher in the liver (0.123% of dose) than in the kidney (0.059%), spleen (0.004%), brain (0.01%), or lung (0.012%). A total of about 0.24% of the applied dose was recovered in these organs. After 24 h, 0.33% of the radiolabel was excreted in the urine and 0.08% in the bile. When the solution was painted on skin damaged by 20 applications of cellophane adhesive tape to remove the stratum corneum, the rates of excretion were 36.3% in the urine and 1.8% in the bile (Minegishi et al., 1977).

B6.2 Biotransformation

AOS and its metabolites were investigated in tissues and excrement after oral administration of 100 mg/kg ^{14}C-AOS to rats. AOS and a metabolite more polar than AOS were detected in blood, liver, kidney, bile, and urine by thin-layer chromatography. As most of the ^{14}C-labelled compounds in urine were alcoholic, unsaturated, and of sulfonic functionality, the metabolite may be a hydroxylated or polyhydroxylated sulfonic acid with a shorter chain than AOS, although the precise chemical structure remains to be elucidated (Inoue et al., 1982).

B7. EFFECTS ON LABORATORY MAMMALS AND *IN VITRO* TEST SYSTEMS

Section summary

The oral LD_{50} for AOS sodium salt in mice was 3000 mg/kg. AOS are skin and eye irritants. Data from studies in experimental animals are limited, but no effects were observed in a long-term study in which oral doses of 250 mg/kg body weight per day were administered to rats. Fetotoxicity was observed in the progeny of rabbits administered a maternally toxic dose of 300 mg/kg body weight per day.

The available long-term studies are inadequate to evaluate the carcinogenic potential of AOS in experimental animals; however, in the limited studies available in which animals were administered AOS orally or on the skin, there was no evidence of carcinogenicity.

The limited data available also indicate that AOS are not genotoxic *in vivo* or *in vitro*.

B7.1 Single exposures

The LD_{50} values for AOS (sodium salt of sulfonated C_{15}–C_{18} *n*-olefin) in male ddy mice were 3000 mg/kg body weight by oral administration, 1660 mg/kg by subcutaneous injection, 170 mg/kg by intraperitoneal injection, and 90 mg/kg by intravenous injection. The toxic effects seen at high oral doses were reduced voluntary activity, diarrhoea, anaemia, dyspnoea, and respiratory collapse. Clonic convulsions followed by respiratory collapse were seen in animals given the material intravenously (Oba et al., 1968a).

B7.2 Short-term exposure

No data were available.

B7.3 Long-term exposure; carcinogenicity

B7.3.1 Mouse

The skin of Swiss-Webster mice was painted with 20% C_{14}–C_{18} AOS, 25% C_{14}–C_{18} AOS, 20% C_{14}–C_{16} AOS, 25% C_{14}–C_{16} AOS, 6.7 or 8.3%

C_{16} 1,4-sultone, water, or acetone, or remained untreated. Animals were treated with 0.02 ml of test material on about 1 cm^2 of exposed skin three times per week for 92 weeks. Final necropsies were conducted when the survival of each group reached 30% (~ 19 months). Histopathological examination showed no evidence of carcinogenicity with any test material (Haar, 1983).

B7.3.2 *Rat*

AOS (97.93% of a 60.4:39.6% (w/w) mixture of alkenyl sulfonate and hydroxyalkane sulfonate; chain-length distribution, 25% C_{14}, 45% C_{16}, 30% C_{18}) were fed to four groups of 50 male and 50 female CFY rats at a dietary level of 0, 1000, 2500, or 5000 ppm, corresponding to 49, 122, or 245 mg/kg body weight per day, for two years. No adverse clinical signs were seen, and survival rates were not affected by treatment. The rate of body weight gain was marginally lower during the second trimester of the study in both males and females receiving 5000 ppm, and food intake was marginally lower during the first year among females receiving 5000 ppm. During the remainder of the study, body weight gain and food consumption were similar to those of the control animals. Investigation of the eyes, blood, and urine of controls and of those receiving 5000 ppm several times during the experiment revealed no reaction to treatment; and no changes related to treatment were seen in gross appearance or organ weights of rats in any group killed after 104 weeks. Histological examination of a limited range of tissues did not provide evidence of toxicity or tumour induction that could be attributed to treatment (Hunter & Benson, 1976).

Groups of 40 male and 40 female Wistar rats were fed the following materials in the diet for 24-27 weeks: 1, 0.75, or 0.5% C_{14}–C_{18} AOS (corresponding to 500, 375, or 250 mg/kg body weight per day); 1, 0.75, or 0.5% C_{14}–C_{18} AOS (corresponding to 500, 375, or 250 mg/kg body weight per day); or 0.33, 0.25, or 0.16% C_{16} 1,4-sultone (corresponding to 165, 125, or 80 mg/kg body weight per day). One control group consisted of 100 males and 100 females and another of 40 males and 40 females. No excess of tumours over that in controls was observed with any treatment (Haar, 1983).

In 70-week studies on Wistar rats, 0.5 ml of a 1.0, 10, or 30% aqueous solution of AOS or 0.5 ml of a 50% aqueous solution of a detergent based on AOS was applied dermally; 24 h after the application, each site was washed with warm water. No abnormal gross or histopathological findings were reported (Tomizawa, 1978).

These studies are summarized in Table 32.

B7.4 Skin and eye irritation; sensitization

AOS (C_{10}; purity, 99.21%) were applied as 0.5 g of a 20 or 30% solution once a day for 15 days to the backs of three male Wistar rats. The skin at the application site and the tissues of the tongue and oral mucosa of animals receiving the 30% solution were examined histologically 16 days after application. Body weight gain was reduced in the group given the 20% solution, and body weight was decreased in the group at 30%. Macroscopically, there were no abnormalities at the application site. Histologically, although atrophy of the stratum spinosum was noted, neither necrosis nor inflammatory cell infiltration was present. No abnormalities of the tongue were observed, but severe atrophy was observed in the mucosa of the oral cavity. The local lesions caused by application of AOS were reported to be minimal in comparison with those induced by application of linear dodecylbenzenesulfate or lauryl sulfate (Sadai & Mizuno, 1972).

Solutions of 0.05–4% AOS (sodium salt of sulfonated C_{15}–C_{18} *n*-α-olefin) were instilled at a dose of 0.1 ml into the eyes of one to three rabbits, and the eyes were examined after 24 h. No abnormal findings were observed with the 0.05% solution, but slight congestion was observed with 0.1% and marked reactions, including severe congestion and oedema, increased secretion, opacity of the cornea, and absence of the corneal reflex, were observed at $\geq$ 1% (Oba et al., 1968a).

Solutions of C_{14}–C_{19} olefin (84% C_{15}–C_{17}) and five other solutions consisting mainly of C_{10}, C_{12}, C_{14}, C_{16}, or C_{18} were instilled into the eyes of three rabbits at one of six concentrations ranging from 0.01 to 5%. The rabbits were examined over a period of 168 h. The materials elicited similar reactions. No abnormal reaction was seen with 0.05%; slight congestion was observed with 0.1% within 2 h after application of the solution; and marked congestion or oedema was observed with 0.5%, which disappeared by 24 h. In the groups treated with 1 or 5%, marked reactions, including severe congestion and oedema, increased secretion, turbidity of the cornea, and disappearance of the corneal reflex, continued for 24 h but had usually completely disappeared by 120 h (Iimori et al., 1972).

In 1973, the apparent sensitizing potential of AOS attracted attention (Haar, 1983). AOS can contain unsaturated γ-sultones when

Table 32. Carcinogenicity of α-olefin sulfonates (AOS) after long-term exposure

Species, strain, numbers per group	Test material (specification)	Route	Dosage	Results	Reference
Mouse, Swiss-Webster 40 M, 40 F	AOS, C_{14}–C_{18}	Dermal	0, 200, 250 mg/kg (water, acetone) 3 times/week, 92 weeks	No gross or histopathological adverse effects on skin	Haar (1983)
Mouse, Swiss-Webster 40 M, 40 F	AOS, C_{14}–C_{16}	Dermal	0, 200, 250 mg/kg (water, acetone) 3 times/week, 92 weeks	No gross or histopathological adverse effects on skin	Haar (1983)
Rat, CFY, 50 M, 50 F	AOS, C_{14}–C_{18} (a.i., 97–93%)	Oral (diet)	0, 0.1, 0.25, 0.5%,	No adverse effects 2 years	Hunter & Benson (1976)
Rat, X-MRC, 40 M, 40 F	AOS, C_{14}–C_{18}	Oral (diet)	0, 0.5, 0.75, 1.0%, 24-27 months	No excess of tumours in comparison with controls	Haar (1983)
Rat, X-MRC, 40 M, 40 F	AOS, C_{14}–C_{16}	Oral (diet)	0, 0.5, 0.75, 1.0%, 24-27 months	No excess of tumours in comparison with controls	Haar (1983)
Rat, Wistar, 10 M, 10 F	AOS, C_{15}–C_{19}	Dermal	0, 250, 2500, 7500 mg/kg bw, 3 times per week, 70 weeks	No gross or histopathological abnormalities	Tomizawa (1978)
Rat, Wistar, 10 M, 10 F	AOS-based detergent	Dermal	12.5 g/kg bw 3 times/week, 70 weeks	No gross or histopathological abnormalities	Tomizawa (1978)

M, male; F, female; a.i., active ingredient

manufactured under certain conditions, and these are strong sensitizers in guinea-pigs (Haar, 1983; Roberts & Williams, 1983; Roberts et al., 1990). When the levels of these sultones were reduced to low levels by altering the manufacturing techniques, AOS no longer caused sensitization (Haar, 1983; Oba et al., 1985; Roberts et al., 1990).

Skin sensitization was studied in guinea-pigs with pastes made of C_{14}–C_{16} AOS, a light-duty dishwashing detergent containing AOS, some consumer products containing AOS, and mixtures of these products with alkyl unsaturated sultone in sodium lauryl sulfate or hypochlorite bleach. The pastes, the dishwashing detergent, most of the consumer products, and the mixtures with hypochlorite bleach induced sensitization, the degree of response being related to the amount of unsaturated γ-sultone present in the material tested (Bay & Danneman, 1985).

B7.5 Reproductive toxicity, embryotoxicity, and teratogenicity

AOS (C_{14}–C_{18}) were administered at a concentration of 0.2, 2, 300, or 600 mg/kg body weight per day to CD rats, CD-1 mice, and NZW rabbits orally once a day by gavage. Groups of 20 rats and mice were given AOS on days 6–15 of pregnancy, and groups of 13 rabbits were treated on days 6–18 of pregnancy. The doses of 0.2 and 2 mg/kg were estimated to be equivalent to 1–2 and 10–20 times the maximal amount of AOS to which humans are exposed. No adverse effects were seen in rat dams, even at the maximal dose. Mouse dams given 300 or 600 mg/kg showed piloerection, decreased movement, and inhibition of body weight gain; six dams at 600 mg/kg died. All rabbits given 600 mg/kg and one given 300 mg/kg died; anorexia and decreased body weight were seen initially in surviving dams given 300 mg/kg. Both mouse and rabbit dams given 0.2 or 2 mg/kg showed only initial inhibition of body weight gain. No adverse effects were seen on litter parameters of rats at any dose. In mice, total litter loss was found in five dams given 600 mg/kg and in six dams given 300 mg/kg; however, the average number of live fetuses in the other dams was no different from that in controls. The average body weights of the fetuses of dams given 300 or 600 mg/kg was significantly lower than that of controls. The incidence of major malformations was not significantly increased in rats, mice, or rabbits. There were no significant minor anomalies or skeletal variations (extra ribs) in rats at any dose. The offspring of mice at 600 mg/kg had a significant increase in delayed ossification. Those of rabbits at 300 mg/kg had a significant increase in skeletal anomalies and variations, although the incidence of skeletal variations was within

the normal background range, and there was no delayed ossification. The effects of AOS on the fetuses, such as changes in litter parameters and delayed ossification, were considered to reflect the effects of AOS on the dams. There were no adverse effects on fetuses of mouse or rabbit dams given 0.2 or 2 mg/kg or on fetuses of rat dams given 0.2, 2, 300, or 600 mg/kg, where effects on the dams were either not observed or were minimal (Palmer et al., 1975b).

AOS and AOS-S (a synthetic detergent with AOS as the main ingredient) were applied to the shaven dorsal skin of mice at a dose of 0.5 ml/mouse per day of a 0.1% (the concentration of AOS usually found in detergents), 1%, or 5% aqueous solution of AOS or a 0.5% (equivalent to 0.1% AOS), 5%, or 25% aqueous solution of AOS-S on days 0-14 of pregnancy. Adverse effects on the dams and fetuses were found in a few cases. None of the dams died; the viability, body weight, and sex ratio of the fetuses did not differ from those of controls; and there were no malformations (Sawano, 1978).

B7.6 Mutagenicity and related end-points

AOS did not cause differential toxicity in *Bacillus subtilis rec* at a concentration of 20 µg/disc or reverse mutation in *Salmonella typhimurium* TA98 or TA100 at 10–100 µg/disc, in the presence or absence of metabolic activation (Oda et al., 1980).

One batch of AOS (C_{14}–C_{16}; 28.4% active ingredient) induced host-mediated mutagenicity at 283 mg/kg body weight in rats inoculated with *S. typhimurium* TA1530 but not in an assay with TA1534 or in plate incorporation assays with either strain (Arthur D. Little Inc., 1993).

B7.7 Special studies

Rabbit erythrocytes were mixed with solutions containing various concentrations of AOS (sodium salt of sulfonated C_{15}–C_{16} *n*-α-olefin; average relative molecular mass, 338.5) at room temperature for 3 h. The 50% haemolytic concentration was 1.5 mg/litre (Oba et al., 1968a). The effects of AOS on methaemoglobin formation were studied in groups of three male mice given an oral or intraperitoneal dose of 0.3 or 3.0 g/kg body weight C_{15}–C_{18} AOS. The level of methaemoglobin in blood was measured 0.5, 1, 2, 3, and 24 h after administration of AOS. No significant increase was observed (Tamura & Ogura, 1969).

In an immunological study of AOS, a complex (HA) prepared by mixing AOS with human serum albumin (HSA) containing 30 mg of total protein was injected subcutaneously or intravenously into rabbits during a period of 2.5 months, and the anti-serum produced was subjected to the ordinary precipitation reaction. As a control, anti-AOS-serum, similarly prepared, was subjected to the same reaction. Minor positive reactions were seen in the HA-anti-HA and HSA-anti-HSA systems but not in the AOS-anti-HA or AOS-anti-AOS systems (Iimori & Ushiyama, 1971).

B8. EFFECTS ON HUMANS

Section summary

In patch tests, human skin can tolerate contact to solutions containing up to 1% AOS for 24 h with only mild irritation. AOS can cause delipidation of the skin surface, elution of natural moisturizing factor, denaturation of the outer epidermal layer proteins, and increased permeability and swelling of the outer layer. AOS did not induce skin sensitization in volunteers. There is no conclusive evidence that AOS induce eczema. No serious injuries or fatalities have been reported following accidental ingestion of detergent formulations that could contain AOS.

B8.1 Exposure of the general population

AOS surface-active agents are found in shampoos, dishwashing products, household cleaners, and laundry detergents. The composition of nonionic and ionic surfactants in these products varies between 10 and 30%. Surface-active agents can affect human skin and eyes.

B8.2 Clinical studies

B8.2.1 Skin irritation and sensitization

AOS are mildly to moderately irritating to human skin, depending on the concentration.

The relative intensity of skin roughness induced on the surface of the forearm was evaluated in volunteers by a circulation method consisting of contact with 1% solutions of C_{12}, C_{14}, C_{16}, and C_{18} AOS for 10 min. The skin response was characterized mainly on the basis of gross visible changes. C_{12} AOS induced more skin roughening than compounds with longer or shorter alkyl chains. The relative degree of skin roughening *in vivo* was correlated with the extent of protein denaturation measured *in vitro* (Imokawa et al., 1975a).

Primary skin irritation induced by a 1% aqueous solution (pH 6.8) of AOS containing 27% C_{15}, 25% C_{16}, 28% C_{17}, and 18% C_{18} (relative molecular mass, 338.5) was studied in a 24-h closed-patch test on the forearms of seven male volunteers. The intensity of skin irritation was scored by grading erythema, fissuring, and scales. The average score

for AOS was 3.97 and that for a control (water) was 1.79. The same compound was evaluated at 0.3% for the relative intensity of skin lesions produced on the surface of the hands by an immersion test involving 30 repetitions of a 1-min dip and 1-min dry. The average score for AOS with regard to erythema, irritation, fissuring, scaling, and loss of suppleness was 5.75, while that for the water control was 2.5 (Oba et al., 1968a).

Skin irritation induced by a 1% aqueous solution of C_{14}, C_{16}, and C_{18} AOS was studied in a 24-h closed-patch test on the forearm and in a test in which the compound was dripped onto the interdigital surface for 40 min once daily for two consecutive days at a rate of 1.2–1.5 ml/min. Skin reactions were scored by grading erythema in the patch test and by grading scaling in the drip test. The score for AOS was 1 (slight erythema) in the patch test and 0.35 (minimal scaling) in the drip test (Sadai et al., 1979).

In sensitization tests on volunteers, AOS in a paste or in a detergent mixture containing up to 0.06% AOS and up to 0.002 ppm unsaturated γ-sultone did not produce sensitization, although one subject had a strong dermal response, which was considered to be due to pre-existing sensitization. Two out of 264 subjects using a light-duty detergent containing AOS developed hand dermatitis and had positive reactions to AOS paste and/or unsaturated γ-sultone in sodium lauryl sulfate in a patch test. Use of a hand dishwashing liquid containing AOS did not cause sensitization provided the level of unsaturated γ-sultones was kept low (Bay & Danneman, 1985). Patch tests on 790 volunteers after four months' use of a dishwashing liquid showed no evidence of sensitization (Oba et al., 1985).

B8.2.2 *Effects on the epidermis*

The effects of AOS on the epidermal outer layer (stratum corneum) are similar to those of other surface-active agents (see section 8.2.2 of the monograph on LAS), including delipidation of the skin surface, elution of natural moisturizing factor, denaturation of stratum corneum protein, increased permeability, swelling of the stratum corneum, and inhibition of enzyme activities in the epidermis (Wood & Bettley, 1971; Imokawa et al., 1974; Okamoto, 1974; Imokawa et al., 1975a,b).

The effects of anionic surfactants on various types of proteins were studied using skin keratin as a filamentous protein, bovine serum

albumin as a globular protein, acid phosphatase as an enzyme protein, and membrane lysosome as a membrane protein. The denaturing effects of surfactants were measured as liberation of sulfhydryl groups and enzyme inhibition. AOS were less potent than LAS or alkyl sulfates. A relationship was observed between denaturing potency, skin irritant action, and alkyl chain length (Imokawa & Katsumi, 1976; Imokawa & Mishima, 1976).

B8.2.3 *Hand eczema*

Skin reactions to a 0.04, 0.4, or 4.0% aqueous solution of AOS (25.0% C_{14}, 45.0% C_{16}, 30.0% C_{18}) were evaluated in a 24-h closed-patch test on the lower back of 10 healthy volunteers and 11 patients with hand eczema (progressive keratosis palmaris). The incidence and intensity of skin reactions were significantly higher in the group with hand eczema than in a control group with normal skin (Okamoto & Takase, 1976a,b).

B8.2.4 *Accidental or suicidal ingestion*

No data were available that related specifically to AOS.

B9. EFFECTS ON OTHER ORGANISMS IN THE LABORATORY AND THE FIELD

Section summary

Limited data are available on the effects of AOS on environmental organisms. The 24-h LC_{50} values for daphnids were 19–26 mg/litre; the 48-h LC_{50} values ranged from 0.3 mg/litre for brown trout (*Salmo trutta*) to 6.8 mg/litre for golden orfe (*Idus idus melanotus*), and the 96-h LC_{50} was 0.5–5.0 mg/litre for brown trout. One study suggested that AOS have little toxicity for birds.

B9.1 Microorganisms

No information was available.

B9.2 Aquatic organisms

B9.2.1 Aquatic plants

The EC_{50} values for $C_{16.4}$ AOS in the green alga *Selenastrum capricornutum* exposed for two to three days, based on growth, fell within the range 45–65 mg/litre (Yamane et al., 1984). The EC_{50} for C_{16}–C_{18} AOS on the growth of *S. capricornutum* was > 20 mg/litre (Konno & Wakabayashi (1987).

B9.2.2 Aquatic invertebrates

Daphnia magna and *Dapghnia pulex* less than 24 h old were exposed to C_{16}–C_{18} AOS under static conditions, in which the water was unchanged for the duration of the test, at a temperature of 20 °C and a water hardness of 25 mg/litre $CaCO_3$. The 6-h LC_{50} values were > 64 and > 130 mg/litre, and the 24-h LC_{50} values were 19 and 26 mg/litre, for the two species respectively (Wakabayashi et al., 1988).

B9.2.3 Fish

The acute toxicity of AOS to fish is summarized in Table 33. The 48-h LC_{50} values ranged from 0.3 mg/litre for brown trout (*Salmo trutta*) to 6.8 mg/litre for golden orfe (*Idus idus melanotus*); the 96-h LC_{50} for brown trout was 0.5–5.0 mg/litre. Acute toxicity tended to increase with carbon chain length.

When eggs of rainbow trout (*Oncorhynchus mykiss*) and carp (*Cyprinus carpio*) were exposed to C_{16}–C_{18} AOS, the EC_{50} values, based on hatchability, were 4.9 for rainbow trout and 3.0 mg/litre for carp (Wakabayashi & Onizuka, 1986). In one-month old rainbow trout under semi-static conditions, the 14- and 28-day LC_{50} values for C_{16}–C_{18} AOS were 0.62 and 0.58 mg/litre. The EC_{50} based on growth was 0.35 mg/litre (Wakabayashi & Mizorogi, 1989).

The time to lethality in goldfish (*Carassius auratus*) exposed to AOS was 2 h at a concentration of 5 mg/litre and 1 h at 10 mg/litre. Addition of 2100 mg/litre egg albumin increased the time to 100% lethality to 3 h and addition of 4200 mg/litre albumin increased the time to 6 h (Tomiyama, 1974).

B9.3 Terrestrial organisms

B9.3.1 Terrestrial plants

AOS were not toxic with respect to growth at the early life stages of radish, Chinese cabbage, and rice when added in hydroponic culture at concentrations of 56, 56, and 32 mg/litre, respectively; concentrations of 100, 100, and 56 mg/litre were toxic (Takita, 1982).

B9.3.2 Terrestrial invertebrates

No information was available.

B9.3.3 Birds

No significant effect on egg quality was found after Leghorn chickens were fed a diet containing 200 mg/kg AOS for 45 days (Lopez-Zavala et al., 1975).

Table 33. Toxicity of α-olefin sulfonates (AOS) to fish

Species	Length, weight, or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	AOS chain length	End-point	Concn (mg/litre)	Reference
Masu trout (*Oncorhynchus masou*)	2 mo	Static[d]	8.5–9.6	30	NS	C_{16}–C_{18}	96-h LC_{50}	0.56	Wakabayashi et al. (1984)
Rainbow trout (*Oncorhynchus mykiss*)	40 d	Static[d]	8.8–10.9	25	NS		96-h LC_{50}	0.78	Wakabayashi et al. (1984)
	4 d	Static[d]	10	25	NS	C_{16}–C_{18}	96-h LC_{50}	0.61	Wakabayashi & Onizuka (1986)
	19 d	Static[d]	10	25	NS	C_{16}–C_{18}	96-h LC_{50}	0.98	
Brown trout (*Salmo trutta*)	2.8–5.8 cm	Flow	15	26–30	NS	C_{14}–C_{16}	48-h LC_{50}	2.5–5.0[b]	Reiff et al. (1979)
	2.8–5.8 cm	Flow	15	26–30	NS	C_{14}–C_{16}	96-h LC_{50}	2.5–5.0[b]	
	2.8–5.8 cm	Flow	15	26–30	NS	C_{16}–C_{18}	48-h LC_{50}	0.6[b]	
	2.8–5.8 cm	Flow	15	26–30	NS	C_{15}–C_{18}	96-h LC_{50}	0.5[b]	
	2–4 cm	Flow	15	250	NS	C_{14}–C_{16}	48-h LC_{50}	3.5[b]	
	2–4 cm	Flow	15	250	NS	C_{16}–C_{18}	96-h LC_{50}	3.1[b]	
	2–4 cm	Flow	15	250	NS	C_{14}–C_{16}	48-h LC_{50}	0.3–0.5[b]	
Goldfish (*Carassius auratus*)		Static	20		NS	C_{12}–C_{16}	6-h LC_{50}	11.2[c]	Gafa (1974)
		Static	20		NS	C_{14}–C_{18}	6-h LC_{50}	3.0[c]	

Table 33 (contd)

Species	Length, weight, or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	AOS chain length	End-point	Concn (mg/litre)	Reference
Golden orfe	1.2–1.8 g	Static	20		NS	C_{14}–C_{16}	48-h LC_{50}	5.08	Mann (1976)
(*Idus idus*	1.2–1.8 g	Static	20		NS	C_{16}–C_{18}	48-h LC_{50}	1.44	
melanotus)	5–7 cm	Flow	20	150	NS	C_{14}–C_{16}	48-hLC_{50}	5.7[b]	Reiff et al.
	5–7 cm	Flow	20	150	NS	C_{16}–C_{18}	48-hLC_{50}	1.9[b]	(1979)
		Flow	20	268	NS	C_{14}–C_{16}	48-h LC_{50}	3.7–6.8[b]	
		Flow	20	268	NS	C_{16}–C_{18}	48-h LC_{50}	1.0[b]	
		Flow	20	268	NS	C_{14}–C_{16}	96-h LC_{50}	3.4–4.9[b]	
		Flow	20	268	NS	C_{16}–C_{18}	96-h LC_{50}	0.9[b]	
Harlequin fish		Flow	20	20	NS	C_{14}–C_{16}	48-h LC_{50}	4.8[b]	Reiff et al.
(*Rasbora*		Flow	20	20	NS	C_{16}–C_{18}	48-h LC_{50}	0.9[b]	(1979)
heteromorpha)		Flow	20	20	NS	C_{14}–C_{16}	96-h LC_{50}	3.3[b]	
		Flow	20	20	NS	C_{16}–C_{18}	96-h LC_{50}	0.5[b]	
Medaka	175–332 mg	Static	21-22	25	6.7–7.1	C_{14}–C_{18}	6-h LC_{50}	6.2[b]	Kikuchi &
(*Oryzias latipes*)	175–332 mg	Static	21-22	25	6.7–7.1	C_{14}–C_{18}	48-h LC_{50}	1.8[b]	Wakabayashi
	175–332 mg	Static	21-22	25	6.7–7.1	C_{16}–C_{18}	6-h LC_{50}	2.7[b]	(1984)
	175–332 mg	Static	21-22	25	6.7–7.1	C_{16}–C_{18}	48-h LC_{50}	0.81[b]	
Carp	3.5–5.5 cm	Static	21		7.5–7.8	Technical	24-h LC_{50}	3.2[c]	Lopez-Zavala
(*Cyprinus carpio*)	3.5–5.5 cm	Static	21		7.5–7.8	Technical	96-h LC_{50}	3.0[c]	et al. (1975)
	2 d	Static[d]	20	25	NS	C_{16}–C_{18}	96-h LC_{50}	> 1.4	Wakabayashi
	15 d	Static[d]	20	25	NS	C_{16}–C_{18}	96-h LC_{50}	1.5	& Onizuka (1986)

Table 33 (contd)

Species	Length, weight, or age	Static or flow	Temp. (°C)	Hardness (mg/litre)[a]	pH	AOS chain length	End-point	Concn (mg/litre)	Reference
Carp (contd). (*Cyprinus carpio*)	50 d	Static[r]	21	75	NS		96-h LC_{50}	1.0	Wakabayashi et al. (1984)
		Static[r]							
White tilapia (*Tilapia melan opleura*)	5–7 cm	Static	21		7.5–7.8	Technical	24-h LC_{50}	2.0[c]	Lopez-Zavala et al. (1975)
	5–7 cm	Static	21		7.5–7.8	Technical	96-h LC_{50}	2.0[c]	
Grey mullet (*Mugil cephalus*)		Static	20.6-22.0				96-h LC_{50}	0.70	Wakabayashi et al. (1984)

Static[r], static renewal: water changed at regular intervals; flow, flow-through conditions: concentration in water maintained continuously; static: water unchanged for duration of test

[a] mg/litre $CaCO_3$

[b] Measured concentration

[c] Nominal concentration

C. Alkyl sulfates

C1. SUMMARY

See Overall Summary, Evaluation, and Recommendations (pp. 7–21)

C2. IDENTITY, PHYSICAL AND CHEMICAL PROPERTIES, AND ANALYTICAL METHODS

C2.1 Identity

Chemical formula:	$C_nH_{2n+1}O_4S$ Na (n = 10–8)
Chemical structure:	$C_nH_{2n+1}OSO_3^-$ Na^+ (n, integer)
Common names:	Sodium alkylsulfate, sulfuric acid alkyl ester sodium salt, alkylsulfate sodium salt, alcohol sulfuric ester sodium salt, sodium dodecyl sulfate, sodium lauryl sulfate
Common trade names:	Akyporox SAL SAS, Akyposal, Alphenate TFC 76, Alscoap LN, Aremsol, Berol, Cosmopon, Dehydag, Elfan, Emal, Empicol, Gardinol, Genapol CRT 40, Manro, Marlinat KT 50, Melanol LP 1, Monogen, Montopol CST, Montovol, Neopon LT, Nikkol, Nissan Persoft SK, Perlankrol ATL-40, Perlankrol, Polystep B, Rewopol, Sactipon, Sactol, Sandopan KD, Sermul, Stepanol WA 100, Sufatol, Sufetal, Sulfopon, Sunnol, Surfax, Swascol, Teepol HB 7, Tensopol Tesapon, Texapon, Ufarol AM 70, Zoharpon, Zorapol LS-30, (McCutcheon, 1993)
Abbreviations:	AS, AS-Na, SDS
CAS Registry numbers:	151-21-3 (C_{12} AS), 1120-04-3 (C_{18} AS), 68130-43-8 (C_8-C_{18} AS)

Specification: AS are higher alcohol sulfuric ester salt types of anionic surfactants. Depending on which precursor alcohol is used as the raw material, the alkyl group is linear or branched, may contain a single homologue or a mixture of chain lengths, and is usually primary. The data presented are applicable mainly to linear alcohol sulfates and AS with predominantly single or similar type of branching.

C2.2 Physical and chemical properties

AS are white crystalline powders. Their physical properties differ widely depending on their alkyl groups, and they are usually produced and used as mixtures. The relationships between the critical micelle concentration, solubility, and alkyl chain length are shown in Table 34.

Table 34. Relationships between alkyl chain length, critical micelle concentration (CMC), and solubility

Alkyl chain length	CMC x 10^{-3} mol/litre[a,c]	Solubility/°C[b,c]
8	136	–
12	8.6	15
14	2.4	28
16	0.58	42
18	0.16	55

[a] From Evans (1956)

[b] Temperature at which 10 g of AS dissolve in 1 litre of water (Gotte, 1954)

[c] The solubility of surfactants in water, defined as the concentration of dissolved molecules in equilibrium with a crystalline surfactant phase, increases with rising temperature. For surfactants, there is a distinct, sharp bend (break-point) in the solubility–temperature curve. The steep increase in solubility above the sharp bend is caused by micelle formation. At above the critical micelle concentration, a micellar solution is formed. Under these conditions, higher levels than the aqueous solubility may be obtained.

AS are readily hydrolysed in hot acidic media. Compounds with an alkyl chain length of C_{10} (27 °C), C_{12} (25 °C), C_{14} (40 °C), or C_{16} (40 °C) have a surface tension of 40 dyne/cm at the temperatures shown in parentheses at concentrations greater than the critical micelle

concentration, indicating a good ability to reduce surface tension (Dreger et al., 1944).

Cleansing capacity at 25 °C increases with alkyl chain length up to C_{13} and then becomes constant up to C_{16}. In an actual detergent containing alkali builders and chelating agents, however, maximal detergency was obtained with C_{14} compounds (Yamane et al., 1970).

C2.3 Analysis

C2.3.1 Isolation

Since AS are readily susceptible to hydrolysis in acidic media, special attention is required.

C2.3.2 Analytical methods

There is no officially recognized, specific procedure for the analysis of AS in environmental samples. The methods used for analysing linear alkylbenzene sulfonates (LAS) are commonly used for AS, except those involving high-performance liquid chromatography (HPLC), which is of limited use for detecting AS in environmental samples because AS do not effectively absorb ultra-violet radiation. An HPLC method for the analysis of AS after its conversion by derivatization into an ultra-violet-active species has been proposed (Utsunomiya et al., 1982). A modified analytical method has been developed that is based on measurement of methylene blue-active substances (MBAS) by HPLC. This method permits determination of AS at concentrations as low as 0.05 mg/litre (Takita & Oba, 1985). Trace enrichment followed by gas chromatography and flame ionization detection have been proposed for the sensitive determination of AS as their trimethylsilyl ethers in environmental samples (Fendinger et al., 1992a).

Non-specific methods used in the analysis of anionic surfactants in general, such as the methylene blue method, may be used for the analysis of AS (see also section 2.3 of the monograph on LAS).

C3. SOURCES OF HUMAN AND ENVIRONMENTAL EXPOSURE

Section summary

Few quantitative data are available on AS in the environment, but AS can be expected to mineralize rapidly in all environmental compartments and to be removed to a large extent during sewage treatment. Environmental concentrations in receiving surface waters, sediments, soils, estuaries, and the marine environment can be expected to be low.

C3.1 Natural occurrence

AS do not occur naturally.

C3.2 Anthropogenic sources

C3.2.1 Production levels and processes

AS are synthesized industrially. Worldwide consumption of AS in 1987 was about 117 000 tonnes in the United States, 56 000 tonnes in western Europe, and 46 000 tonnes in Japan (Richtler & Knaut, 1988). In western Germany in 1987, some 10 000 tonnes of AS and 87 000 tonnes of LAS were used (Schöberl et al., 1988). Worldwide consumption was estimated to be 289 000 tonnes in 1990 (Hewin International Inc., 1992; see Table 35).

Table 35. Estimated worldwide use of alkyl sulfates in 1990 (tonnes)

Region	Household products	Personal care products	Industrial and institutional use
North America	140 000	33 000	9 000
Western Europe	49 000	12 000	7 000
Japan	21 000	6 000	4 000
Rest of the world	–	4 000	4 000
Total	210 000	55 000	24 000

From Hewin International Inc. (1992)

AS were originally made by the sulfation of natural fatty alcohols. They are currently produced from both natural and synthetic fatty alcohols. Primary AS are usually manufactured by conventional sulfation of the parent alcohol with either sulfur trioxide or chlorosulfonic acid. The product of this reaction is then neutralized with an appropriate base (NaOH, Na_2CO_3, NH_4OH, or triethanolamines).

C3.2.2 *Uses*

Initially, AS were used as washing agents for wool or as active ingredients in heavy-duty laundry detergents. They are now used mainly in personal care products (shampoos, toothpastes, toiletries), household detergents (light-duty dishwashing detergents, heavy-duty laundry detergents), and industrial applications.

C4. ENVIRONMENTAL TRANSPORT, DISTRIBUTION, AND TRANSFORMATION

Section summary

AS can be expected to be transported into the environment by mechanisms similar to those that operate for LAS and α-olefin sulfonates (AOS). AS are readily biodegradable under aerobic conditions, both in laboratory tests and under environmental conditions, and primary biodegradation is complete within two to five days. Less information is available on the effect of temperature on the biodegradation of AS than for LAS. The biodegradation kinetics of AS appear to be less affected by temperature than those of other surfactants. The whole-body bioconcentration factors are 2–73, depending on chain length. AS are taken up by fish mainly through the gills and are subsequently distributed to the liver and gall-bladder. After biotransformation, AS are excreted rapidly. They are not bioconcentrated or biomagnified in aquatic organisms.

C4.1 Transport and distribution between media

After use, AS are discharged into the environment in wastewater, like other detergent compounds, where they can undergo sewage treatment if such facilities are available. In countries where adequate wastewater treatment facilities are not available, AS released to the environment are removed by biodegradation and adsorption in the receiving surface water (see section 4.2 of the monograph on AOS).

Sorption equilibria were obtained rapidly (within 20 min) for pure homologues of AS (> 99%) with chain lengths of C_8, C_9, C_{10}, C11, C_{12}, C_{13}, and C_{14}, suggesting that sorption is due to a hydrophobic bonding mechanism, as has been observed for other surfactants. Thus, sorption of AS to sediment is likely to be stronger for longer chain homologues than for shorter ones. The K_D values for C_{12}AS were 70 and 100 for two river sediments, whereas for C_{12} LAS on the same sediments they were 310 and 330 (Marchesi et al., 1991). Adsorption of AS therefore competes in kinetic terms with biodegradation as a mechanism for removal of AS from the environment, as is seen for surfactants in general.

C4.2 Biotransformation

C4.2.1 Biodegradation

C4.2.1.1 Biodegradation pathway; mechanism

Several species of bacteria have been found that can mineralize AS. AS with chains longer than six carbons are degraded by the initial action of a sulfatase enzyme, producing sulfate and the corresponding alcohol. The alcohol is readily oxidized by formation of an aldehyde, to produce carboxy acid, which can be further oxidized by β-oxidation and in the citric acid cycle. Secondary ketones and hydroxy ketones of AS are produced as metabolites but have not been detected in simulated activated sludge. Biodegradation of short-chain homologues of AS may proceed by oxidation of the chain before hydrolysis of the ester bond by the sulfatase enzyme.

The metabolic pathway for biodegradation of C_{12} AS by *Pseudomonas* strains has been described (Hsu, 1965; Thomas & White, 1989). Initial liberation of the sulfate head produces dodecanol, which is further transformed into more polar metabolites, including dodecanal and dodecanoic acid. These products may be further metabolized by β-oxidation, or they may be elongated to C_{14}, C_{16}, or C_{18} fatty acyl residues, which are then incorporated into lipid fractions such as phospholipids (Thomas & White, 1989).

C4.2.1.2 Biodegradation in the environment

The aerobic biodegradation of 20 mg/litre AS at 27 °C was followed during a 10-day incubation period. Primary degradation, measured by the MBAS method, was complete within five days. The theoretical production of CO_2 reached 60–90% within 10 days (Itoh et al., 1979).

The biodegradation of AS at a concentration of 30 mg/litre was studied in a vessel containing activated sludge at a concentration of 100 mg/litre over a period of 12 days, by measuring chemical oxygen demand. All of the AS were lost within two days; the specific rate of biodegradation was calculated to be 20 mg/g per h (Pitter & Fuka, 1979).

The biodegradation of an initial concentration of 6 mg/litre C_{12} AS was studied by the die-away method, in which disappearance of the compound is followed over a given period. Less than 10% of the

original amount remained in river water in the test vessel after 12 days' exposure, and complete degradation was reported within 21 days (Okpokwasili & Nwabuzor, 1988).

The capacity of epilithic (sampled from the surface of pebbles) and planktonic river bacterial populations to degrade C_{12} AS was studied under simulated environmental conditions. Samples were collected from four polluted sites and one clean site in a polluted river in South Wales, United Kingdom. In die-away tests, AS were degraded after an apparent lag at all four polluted sites, but degradation by the bacterial populations at the clean site was relatively slow. Quantification of the kinetic components that contributed to the die-away curves demonstrated that biodegradation of AS occurred at concentrations below its K_m by bacteria with exponential growth that are unaffected by addition of the test substrate. Degradation of AS in the clean sample followed a different pattern, but there was generally little or no growth on endogenous carbon. The authors concluded that the capacity of epilithic bacterial populations to degrade C_{12} AS is more stable than that of planktonic populations (Anderson et al., 1990).

Riverine bacteria that can grow in the presence of 0.5 mmol/litre C_{12} AS are widespread, and a greater incidence of isolates resistant to C_{12} AS was recorded at a polluted site than in clean water. The ability of each culture to produce alkyl sulfatases, the enzymes that initiate degradation of AS, was also determined. Bacteria containing alkyl sulfatases were widespread, but a greater alkyl sulfatase yield was obtained from polluted site. The authors concluded that more strains at the polluted site had constitutive rather than inducible enzymes. An increased incidence of strains containing multiple alkyl sulfatases was also recorded at the polluted site (White et al., 1985).

In another study in South Wales, the distribution of planktonic bacteria capable of degrading 98.5% C_{12} was examined in water samples at sites along a river. The annual mean prevalence of such bacteria was 8.1-16.0% of the total number of isolates. The proportion of isolates that degrade AS in clean water was no different from that at polluted sites, and a lower density was recorded at the source owing to a reduction in overall numbers. A higher percentage of bacteria capable of degrading C_{12} AS was recorded in estuarine samples than in samples from the middle of the polluted river; however, when cell numbers were taken into account, the cell density was similar at all polluted sites on the river, including the estuary. The incidence of these isolates was not correlated with either biochemical oxygen demand or oxygen concentration, but

the incidence tended to increase at the end of the summer. More than half of the isolates contained constitutive alkyl sulfatase enzymes, while they were induced or repressed in the remainder after exposure to AS. No variation in the proportions of type of enzyme regulation was seen between sampling sites or times (White et al., 1989).

The biodegradation of AS was also examined at three sites, above, at, and below a sewage works outfall on the South Wales river. Samples capable of degrading C_{12} AS after only one day's exposure were found at each site. No biodegradation of AS was reported at a pristine source site. The onset of biodegradation was more rapid following longer exposure of the river, suggesting the existence of an adaptive mechanism. A model of the die-away kinetics of degradation suggested that C_{12} AS were biodegraded by a bacterial population growing at the expense of endogenous carbon. The activity of the epilithic samples in degrading AS increased during the first four days of exposure at each site. The stabilized values (days 4–14) increased from the upstream site to the outfall, decreasing to intermediate values downstream. The sewage input had less effect on activities in degrading AS than on bacterial cell densities. Little variation in growth characteristics was seen throughout colonization at the three sites. The authors concluded that the adaptation seen during exposure in the river was attributable to colonization of the epilithon by an existing population that degraded C_{12} AS and not to acquisition or adaptation of biodegrading capacity (Russell et al., 1991).

The half-life for primary degradation of 20 mg/litre C_{12} AS in seawater varied over a range of 0.26 to 0.34 days, and degradation was reported to follow first-order kinetics. Primary degradation was followed by an immediate increase in bacterial number and thymidine incorporation (Vives-Rego et al., 1987). C_{12} AS was found to be degraded rapidly in seawater, and 250 g/litre were found in sediment; at 25 °C, 90% was degraded within five days. No lag phase was reported, and the degradation kinetics were reported to be first-order (Sales et al., 1987).

C_{15}–C_{16} AS were 98% removed at 15 °C and 99% removed at 8 °C (Gilbert & Pettigrew, 1984). Similarly, C_{12}–C_{15} and C_{12}–C_{14} AS were found (by the MBAS method) to be biodegraded during winter and spring in a trickling filter sewage treatment plant (Mann & Reid, 1971). These results suggest that temperature has no major effect on the removal of alkyl sulfates under environmental conditions.

Primary biodegradation of C_{12} AS was less affected by incubation temperature than that of other anionic surfactants in die-away tests

with water from the Tama River, Japan. Primary biodegradation was complete within one day at temperatures of 21 and 27 °C, within two days at 15 °C, and within three days at 10 °C (Kikuchi, 1985).

Over 99% of MBAS activity in activated sludge was lost in a 19-day OECD screening test and in the 28-day OECD confirmatory test. Mineralization of both C_{12}–C_{14} and C_{16}–C_{18} AS was complete, with 90–95% degradation for C_{12}–C_{14} AS and 77–88% for C_{16}–C_{18} AS in the two test systems (Steber & Wierich, 1987).

C4.2.1.3 *Anaerobic degradation*

Biodegradation of AS under anaerobic conditions has been reported in several studies, with 88% degradation of stearyl sulfate containing C_{14} AS in an anaerobic screening test (Birch et al., 1989) and 95% ultimate degradation of the same compound (Steber & Wierich, 1987).

C4.2.2 *Abiotic degradation*

No information was available.

C4.2.3 *Bioaccumulation and biomagnification*

Carp (*Cyprinus carpio*) were exposed to ^{35}S-C_{12} AS at a concentration of 0.85 mg/litre for up to 24 h. Within 1 h, AS was concentrated in the gills, hepatopancreas, and kidneys with concentration factors of 1.6, 1.4, and 1.5, respectively. After the initial uptake in the gills, the levels of AS fell, and other organs and tissues, such as the skin surface, muscle, brain, kidney, hepatopancreas, and gall-bladder showed gradual uptake over the exposure period. The concentration factors after 24 h ranged from 2.0 for the skin surface to 43 for gall-bladder. Blood and kidney also showed uptake, but the levels after 24 h were less than those after 4 and 8 h, respectively. AS were lost rapidly from all tissues except the gall-bladder when the fish were kept in 'clean' water for 48 h (Kikuchi et al., 1978).

Carp maintained in water containing 0.5 mg/litre ^{35}S-C_{12} AS absorbed the compound within 1 h, and an equilibrium for the whole body and gall-bladder was reached within 24 h, with concentration factors of about 4 and 700, respectively. After 24 h, the levels of AS in hepatopancreas had decreased from the initial level. When the fish were transferred to 'clean' water, 50% of the AS was still present after 72 h (Wakabayashi et al., 1978).

When carp were exposed to ^{35}S-C_{12} AS at concentrations between 2.7 µg/litre and 40 mg/litre for up to 120 h, equilibrium was reached within 72 h, at concentration factors of 3.9-5.3, which were independent of the concentration of AS in solution (Wakabayashi et al., 1981).

In a study of the effect of chain length on the uptake of AS, carp were exposed to 0.5 mg/litre of ^{35}S-C_{12}, ^{35}S-C_{14}, or ^{35}S-C_{16} AS for 24 h. Absorption of AS reached a maximum within the exposure period. The whole-body concentration factors were 2.1, 11, and 73 for the three surfactants respectively, and thus increased with alkyl chain length. This tendency was also observed in gills and hepatopancreas, but the factors in the gall-bladder were almost the same for the three homologues. When fish were transferred to 'clean' water, the elimination rate decreased with increasing carbon chain length, and 50% of C_{16} AS was retained after 120 h (Wakabayashi et al., 1980).

The absorption, tissue distribution, metabolism, and route of excretion of 50 mg/litre C_{12} AS were studied in goldfish (*Carassius auratus*) exposed for 24 h. AS was absorbed mainly through the gills and was distributed rapidly throughout the body; it was absorbed to a lesser extent (20% of total) by cutaneous absorption and orally (8%). The highest concentration of AS was measured in the gall-bladder, mainly because of its small size. The greatest proportion of the absorbed AS was located in the body, gut, liver, and gall-bladder. The level of AS in the tissues fell by 38% over 24 h in unfed fish and by 68% in fed fish. The high concentration of AS in the liver and gall-bladder was thought to indicate metabolism of the compound in the liver. The metabolites of AS that were identified included successive products of ß-oxidation of the alkyl chain and butyric-4-sulfate (Tovell et al., 1975).

C4.3 Interaction with other physical, chemical, and biological factors

The presence of 1 mg/litre AS (chain length unspecified) had no significant effect on the uptake of mercury by phytoplankton (*Diogenes* sp.) or mussels (*Mytilus* sp.) (Laumond et al., 1973).

Exposure of bacteria to 20 mg/litre phenol and 0.5 mg/litre C_{12} AS resulted in a directly additive effect. Exposure to 2.5 mg/litre phenol and 0.5 mg/litre AS resulted in a synergistic effect. No interactive effects were reported between sodium cyanide and AS in the same test protocol (Dutka & Kwan, 1982).

C4.4 Ultimate fate following use

As no specific analytical method is available for AS, their concentrations in environmental samples have not been established. Like detergent compounds, AS are present in wastewater after use. A large proportion is removed during treatment of wastewater, mainly as a result of a combination of biodegradation and adsorption processes. As for other surfactants, these processes continue when AS are released into the environment.

C5. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

Section summary

Data on the environmental concentrations of AS are limited. At sewage treatment plants where the influent concentrations of AS were < 0.01–0.7 mg/litre, the effluent contained predominantly C_{12} AS, at concentrations of < 0.005–0.1 mg/litre. Surface waters receiving treated wastewater contained AS at concentrations below the detection limit of 0.005 mg/litre.

Environmental levels

AS were measured at two sewage treatment plants in the United States where the influent concentrations were < 0.01–0.7 mg/litre, which were at least 2.4 times lower than those predicted on the basis of use of AS and per-capita wastewater in the United States. The predominant homologues of AS in untreated wastewater were C_{12}, C_{14}, and C_{15}. The effluent contained predominantly C_{12} AS, at concentrations of < 0.005–0.1 mg/litre, showing that removal exceeded 98% during rotating biological contact and activated sludge treatment. Surface waters receiving treated wastewater contained AS at concentrations below the detection limit of 0.005 mg/litre (Fendinger et al., 1992a,b).

C6. KINETICS

Section summary

AS are readily absorbed by the gastrointestinal tract after oral administration and are excreted principally in the urine, only minor amounts being eliminated in the faeces. Penetration of AS through intact skin appears to be minimal. AS are extensively metabolized in various species to several metabolites. Butyric acid-4-sulfate has been identified as their major metabolite.

C6.1 Absorption, distribution, and excretion

In a study of the absorption of higher alcohol sulfates, ^{14}C-hexadecyl sulfate salts were administered orally to humans and dogs. After a single dose of 14.4 mg/kg bw of the salts to dogs, the maximal plasma concentration of hexadecyl sulfate (1.22–2.45 μg/ml) was reached within 30–60 min; 6 h later, the plasma concentration had decreased to about one-tenth of the peak value. Within 72 h, 50–79% of the administered dose had been excreted in the urine and 12-41% in the faeces. After a single dose of 360 mg to humans, the maximal plasma concentration was reached at 2 h, although there was marked variation between individuals (range, about 3.1–23 μmol/ml) (Merits, 1975).

Potassium dodecyl ^{35}S-sulfate was injected intravenously or intraperitoneally at 1 mg/ml to male and female rats. The proportions of the administered dose excreted in the urine and faeces and the amounts retained in the carcass after 24 h are shown in Table 36. Most of the radiolabel appeared in the urine of both male and female rats, although some was present as inorganic ^{35}S-sulfate. The intestinal flora do not play a significant role in the metabolism of potassium dodecyl ^{35}S-sulfate, since the distribution of radiolabel in the urine and faeces was similar in rats pretreated with antibiotics and in untreated rats. Whole-body autoradiograms of rats killed 5 min after administration of the compound by intraperitoneal injection showed significant amounts of radiolabel in the liver; the concentrations increased up to 30 min and then gradually declined, only trace amounts remaining after 4 h. The kidney was the only other organ in which any appreciable accumulation was reported (quantitative data not presented) (Denner et al., 1969).

In order to investigate the percutaneous absorption of AS, 0.5 ml of 25 mmol/litre sodium ^{14}C-dodecyl sulfate in water was applied to the

Table 36. Excretion of ^{14}C-alkyl sulfates by rats after injection of 1 mg/ml

Route of administration	Sex	Excretion (%)		
		Urine (total ^{35}S)	Faeces	
			Inorganic ^{35}S	Total ^{35}S
Intraperitoneal	Male	86.3	14.4	0.2
	Female	93.2	18.1	0.9
Intravenous	Male	95.6	23.5	–
	Female	97.4	11.4	–

From Denner et al. (1969)

dorsal skin (10 cm^2) of rats for 15 min. Heavy deposition of the surfactant on the skin surface and in the upper regions of the hair follicles was observed. The ^{14}C level in urine was calculated to be equivalent to a penetration of 0.26 $\mu g/cm^2$ per 24 h (Howes, 1975).

In young swine administered sodium dodecyl ^{35}S-sulfate (3.3 mmol/animal) orally, the labelled compound was well absorbed from the intestine. Traces of radiolabelled sulfur were found only in bristles, bones, and bone marrow. The total amounts of ^{35}S retained in organs and tissues were 1.7% of the dose at 82 h, 0.6% at 200 h, and 0.18% at 10 weeks. About 90% of the sodium dodecyl sulfate was recovered in urine and about 10% in faeces at 140 h (Havermann & Menke, 1959).

Similar results were obtained in guinea-pigs in a study of the percutaneous absorption of 3 µmol sodium lauryl ^{35}S-sulfate in water through skin *in vivo*. Less than 0.4% of the dose was found to have penetrated the skin, on the basis of recovery of radiolabel in the urine, faeces, and expired air. The permeability constant was calculated to be 0.65×10^{-6} cm/min (Prottey & Ferguson, 1975).

In a study of the dermal absorption of some homologues of AS, ranging from octyl to octadecyl sulfate, by isolated human abdominal skin, no penetration of the dermis was detected (Blank & Gould, 1961).

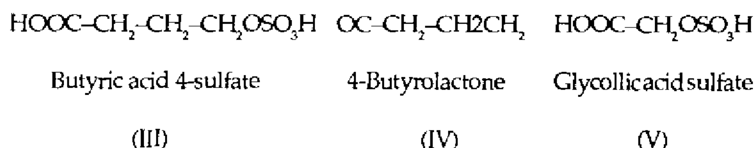
The rates of excretion in urine and faeces after oral, intravenous, or intraperitoneal administration of ^{14}C- or ^{35}S-labelled C_{10}–C_{18} AS to rats, dogs, and humans are summarized in Table 37.

C6.2 Biotransformation

Potassium dodecyl ^{35}S-sulfate was extensively metabolized in rats to yield a single ester sulfate, identified as butyric acid 4-^{35}S-sulfate (III in scheme below), and inorganic ^{35}S-sulfate. These compounds were degraded by a process involving initial ω-oxidation followed by β-oxidation of fatty acids with successive elimination of a C_2 fragment. The final product of degradation of potassium dodecyl ^{35}S-sulfate was potassium butyric acid 4-^{35}S-sulfate, which was excreted in urine. When this product was injected intraperitoneally into rats, it was mostly eliminated unchanged in the urine, but about 20% of the dose was present as an inorganic ^{35}S-sulfate. These findings suggest that the sulfate ester is hydrolysed *in vivo* (Denner et al., 1969).

Butyric acid 4-sulfate was hydrolysed nonenzymatically *in vitro* at pH 5.0 and above, and the 4-butyrolactone (IV) and inorganic SO_4^{2-} ion were liberated in approximately equimolar amounts (Ottery et al., 1970).

After administration of ^{14}C-hexadecyl sulfate to rats, dogs, and humans, the main metabolite was identified as the sulfate ester of 4-hydroxybutyric acid. A minor metabolic product, *tert*-^{14}C-butyrolactone, was also isolated from the urine of rats, dogs, and humans. The urine of dogs contained still another metabolite, which was isolated and identified as glycollic acid sulfate (V) (Merits, 1975).



Qualitative analysis of ^{35}S in the urine of rats administered potassium decyl ^{35}S-sulfate or potassium octadecyl ^{35}S-sulfate intravenously showed that butyric acid 4-^{35}S-sulfate was the major metabolite and inorganic ^{35}S-sulfate a minor metabolite; no unchanged compound was detected (Burke et al., 1975).

Similar results were obtained when sodium octadecyl ^{35}S-sulfate was administered orally to rats . It was suggested that alkylsulfates with even-numbered carbons, like C_{10}, C_{12}, C_{16}, and C_{18}, are degraded by a common pathway involving ω-oxidation followed by β-oxidation, and finally excreted in urine as metabolized forms with C_4 or C_2 (Adachi et al., 1979).

Table 37. Excretion of alkyl sulfates (AS) in the urine and faeces of rats, dogs, and humans

AS[a]	Species	Treatment	Length of treatment (h)	Excretion (%)		Reference
				Urine	Faeces	
^{35}S-AS(C_{10})-K	Rat	1 mg/rat ip	48	82.9 79.5	1.2 1.0	Burke et al. (1975)
^{35}S-AS(C_{11})-K	Rat	1 mg/200 g ip	48	98.2 90.6	2.5 7.3	Burke et al. (1976)
	Rat	1 mg/200 g po	48	75.1 88.7	14.3 5.7	
	Rat	1 mg/200 g iv	48	85.9 74.8	5.9 18.5	
^{35}S-AS(C_{12})-K	Rat	1 mg/rat ip	48	86.3 93.2	0.2 0.9	Denner et al. (1969)
	Rat	1 mg/rat po	48	98.7 106.9	0.7 0.5	
^{35}S-AS(C_{16})-EM	Rat	14.4 mg/kg po	96	94	5	Merits (1975)
^{14}C-AS(C_{16})-EM	Rat	14.4 mg/kg po	72	87	3	
^{35}S-AS(C_{16})-Na	Dog	2.9 mg/kg iv	72	83	3	
^{14}C-AS(C_{16})-TMA	Dog	4.4 mg/kg iv	48	50	41	

Table 37 (contd)

AS[a]	Species	Treatment	Length of treatment (h)	Excretion (%)		Reference
				Urine	Faeces	
^{35}S-AS(C_{16})-EM	Dog	14.4 mg/kg po	72	52	37	
^{14}C-AS(C_{16})-EM	Dog	14.4 mg/kg po	72	65	26	
^{14}C-AS(C_{16})-EM	Human	250 mg po	72	80 20	7 73	
^{35}S-AS(C_{18})-K	Rat	1 mg/rat ip	48	77.1 73.9	1.1 2.6	Burke et al. (1975)
	Rat	1 mg/200 g po	48	76.7 68.8	4.1 6.1	
^{35}S-AS(C_{18})-Na	Rat	4 mg/rat po	48	95.3	2.2	Adachi et *al*. (1979)

[a] K, potassium salt; EM, erythromycin salt; TMA, trimethylammonium salt

The metabolism of surfactants with odd-numbered carbon chains, like C_{11} potassium undecyl ^{35}S-sulfate, was also investigated in rats. Propionic acid 3-^{35}S-sulfate was identified as the major metabolite in urine; pentanoic acid 5-^{35}S-sulfate and inorganic ^{35}S-sulfate were identified as minor metabolites (Burke et al., 1975, 1976).

C7. EFFECTS ON LABORATORY MAMMALS AND *IN VITRO* TEST SYSTEMS

Section summary

The oral LD_{50} values for AS in rats ranged from 1000 to 4120 mg/kg bw. AS irritate the skin and eye at concentrations of about 0.5% or more. Although the effects of short- and long-term exposure to AS in animals have been investigated, most of the studies are limited by inadequate histopathological examination or small group size. Toxic effects have been reported in rats administered AS in the diet or drinking-water at concentrations equivalent to ≥ 200 mg/kg per day.

Maternal toxicicity and fetotoxic effects have been observed at a dose equivalent to 200 mg/kg per day.

The available long-term studies are inadequate to evaluate the carcinogenic potential of AS in experimental animals; however, in the limited studies available, in which animals were administered AS in the diet, there was no evidence of carcinogenicity.

On the basis of limited data, AS also do not appear to be genotoxic *in vivo* or *in vitro*.

7.1 Single exposures

The oral, intraperitoneal, intravenous, and dermal LD_{50} values for AS are summarized in Table 38. The acute oral toxicity of AS in rats and guinea-pigs may vary with the length of the alkyl chain, and compounds with shorter chains are less toxic. The low LD_{50} value for sodium lauryl sulfate after dermal application to rabbits may indicate rapid skin penetration.

Table 38. Acute toxicity of alkyl sulfates (AS)

Species	Sex	Route	LD_{50}[a]	Test material	Reference
Mouse	NS	po	2900	C_8 sodium AS	Gloxhuber (1974)
			2200	C_{10} sodium AS	
			2700	C_{12} sodium AS	
			3000	C_{14} sodium AS	
			> 8000	C_{16} sodium AS	
			> 8000	C_{18} sodium AS	

Table 38 (contd)

Species	Sex	Route	LD_{50}[a]	Test material	Reference
Rat	M	po	4120	40% solution of sodium 2-ethylhexanol sulfate	Smyth et al. (1941)
	M	po	1250	25% solution of sodium 7-ethyl-2-methyl undecanol-4 sulfate	
	M	po	1425	25% solution of sodium 3,9-diethyl tridecanol-6 sulfate	
	M	po	2730	30% solution of sodium lauryl sulfate	
	F,M	po	1280 (C_{12}–C_{15})	86% sodium laury sulfate	Walker et al. (1967)
	F,M	po	1000–2000	Sodium coconut alcohol sulfate (mainly C_{12})	Brown & Muir (1970)
	F,M	ip	210	Sodium lauryl sulfate	Epstein et al. (1939)
	F,M	Dermal	2000 (100% deaths)	30% slurry of sodium lauryl sulfate	Carson & Oser (1964)
Guinea-pig	F,M	po	1520	40% solution of sodium 2-ethylhexanol sulfate	Smyth et al., (1941)
	F,M	po	650	25% solution of sodium 7-ethyl-2-methyl undecanol-4 sulfate	
	F,M	po	425	25% solution of sodium 3,9-diethyl tridecanol-6 sulfate	
	F,M	Dermal	1200 (no deaths)	33% slurry of sodium lauryl sulfate	Carson & Oser (1964)
	F,M	Dermal	2000 (100% deaths)	33% slurry of sodium lauryl sulfate	
Rabbit	F,M	Dermal	580	33% slurry of sodium lauryl sulfate	
	F,M	iv	121 (100% deaths)	33% slurry of sodium lauryl sulfate	

M, male; F, female
[a] As active ingredient

There were no overt signs of poisoning, except diarrhoea in rats given sodium coconut alcohol sulfate orally (Brown & Muir, 1970); however, signs of central nervous stimulation, including tremors, tonic–clonic convulsions, and respiratory collapse, were observed in

rabbits, guinea-pigs, and rats given lauryl sulfate dermally and in rabbits given the compound intravenously (Carson & Oser, 1964).

In animals that died after receiving large doses of AS, the main gross pathological findings were haemorrhage and congestion of the stomach wall and bloodstained urine. Histopathological examination of rats given the sodium sulfate derivative of 3,9-diethyltridecane-4-ol orally revealed congestion, cloudy swelling of convoluted tubules with marked toxic degeneration of the cells, and granular detritus in the kidneys of animals killed by the LD_{50}, whereas only congestion and cloudy swelling were seen in the kidneys of animals that survived the LD_{50}. At larger doses, similar severe kidney injury and necrosis of the intestinal villi of the entire mucosal surface of the small intestine were observed. Only minor injury was seen in the liver, and the other organs examined were normal (Smyth et al., 1941).

C7.2 Short-term exposure

The results of short-term tests for toxicity with repeated doses are summarized in Table 39.

C7.2.1 Rat

C7.2.1.1 Administration in the diet

Groups of five male and five female Wistar rats were fed diets containing technical-grade sodium lauryl sulfate (purity, 98%) at a concentration of 0, 0.5, 1, or 2% (equivalent to 245, 490, or 980 mg/kg of diet per day) for two or four weeks. No abnormalities were seen in behaviour or food intake; but body weight gain was significantly suppressed in females at the highest dose, and haematological examination revealed a significant decrease in red blood cells at two weeks. Biochemical examination of the serum revealed a significant increase in the glucose level at two weeks in males given 2%, a significant increase in glutamate–oxalate transaminase at two weeks in females given 1 or 2%, significant increases in glutamate–pyruvate transaminase and alkaline phosphatase activities at four weeks in all females, and a significant decrease in cholinesterase activity at four weeks in females given 2%. Both the absolute and relative weights of the liver and thyroid were increased at two weeks in males and females given 2%, and those of the liver and left kidney were increased at four weeks in all females; the weights of the thymus were decreased in males given 2% at four weeks. Histopathological examination of rats with

Table 39. Results of short-term exposure of experimental animals to alkyl sulfates (AS)

Species, strain, numbers per group	Material	Route	Dosage	Results	Reference
Rat, Wistar, 10	AS, C_{12} (a.i. 98%)	Diet	0, 0.5, 1.0, 2.0%, 4 weeks	Changes in haematological parameters, serum enzyme activities, and liver; depressed body weight gain in females at highest dose; increased weights of liver, thyroid and kidney at highest dose; decreased thymus weight in males	Oishi et al. (1974)
Rat, 25	AS, C_{12} (Irium®)	Diet	30, 60 mg/rat per day, 5 weeks	Dose-related hepatic effects	Hatton et al. (1940)
Rat, Wistar, 5	AS, C_{12}	Diet	1.5%, 12 weeks	Changes in serum, renal, and hepatic enzyme activities; depressed body weight gain; increased liver weight	Ikawa et al. (1978)
Rat, Osborne-Mendel, 5 M	AS, C_{12}	Diet	9, 2, 4, 8%, 4 months	Diarrhoea, abdominal bloating; depressed body weight gain	Fitzhugh & Nelson (1948)
Rat, Carworth, 24	AS, C_{12}–C_{15} (a.i. 86%)	Diet	9, 0.04, 0.02, 0.1, 0.5%, 13 weeks	Increased liver weight in females at highest dose	Walker et al. (1967)
Rat, Wistar, 5, 10	AS	Drinking-water	0, 0.25, 0.5, 1.0, 2.0, 4.0%, 30 days	Renal changes; proteinuria; depressed body weight gain at 4% sodium 2-ethylhexanol sulfate	Smyth et al. (1941)
Rat, Wistar 15 M	AS (a.i.22.5%)	Dermal	5 mg/kg per day, 30 days	Dermal irritation; hepatic effects	Sakashita et al. (1974)

Table 39 (contd)

Species, strain, numbers per group	Material	Route	Dosage	Results	Reference
Rat, Wistar (NS) M	AS (a.i. 22.5%)	Dermal	5 mg/kg per day, 30 days	Hepatic degeneration	Sakashita (1979)
Rabbit 3 M, 3 F	AS, sodium lauryl sulfate	Dermal	6, 60, 150 mg/kg, 5 times/week, 3 months	Dermal irritation	Carson & Oser (1964)

a.i., active ingredient; M, male; F, female; NS, not specified

increased liver weight revealed slight swelling of liver cells and increased numbers of dividing liver cells. This finding was considered to be an adaptation to administration of the test material. Cylinders in the renal tubules, vacuolar degeneration of the epithelial cells of the renal tubules, periodic acid-Schiff stain-positive substances in the renal tubules, and atrophy of the renal glomeruli were observed mainly in rats given 1 or 2% (Oishi et al., 1974).

Groups of 25 albino rats (sex not specified) were given diets containing a sodium lauryl sulfate formulation (Irium®) at a dose of 0, 30, or 60 mg/animal per day for eight weeks. The only abnormal sign in the experimental groups was soft stools. Histological examinations of the livers of four rats in each group revealed swelling of liver cells, compression of cellular cords, and prominent nuclei. These effects were particularly marked in rats given the high dose (Hatton et al., 1940).

Groups of five male Wistar SPF rats were fed a diet containing sodium dodecyl sulfate at a concentration of 1.5% (equivalent to 750 mg/kg of diet per day) for 2, 4, or 12 weeks, and were compared with a control group. Body weight gain was suppressed and relative liver weight significantly increased from two weeks. Biochemical analysis of serum revealed increased activities of alkaline phosphatase and glutamate–pyruvate transaminase and a decreased level of cholesterol. Enzymatic examinations of the liver showed decreased activity of glucose-6-phosphatase at 12 weeks, decreased activity of glucose-6-phosphate dehydrogenase and increased activity of lactate dehydrogenase at each observation time, and increased isocitrate dehydrogenase activity at 4 and 12 weeks. Examination of the renal cortex showed decreased activities of 5′-nucleotidase and Mg-ATPase at 12 weeks and increased isocitrate dehydrogenase activity at 4 and 12 weeks. Examination of the renal medulla showed decrease activities of Mg- and Na,K-ATPases and increased isocitrate dehydrogenase activity at 12 weeks (Ikawa et al., 1978).

Groups of five male Osborne-Mendel rats were given diets containing sodium lauryl sulfate at a concentration of 0, 2, 4, or 8% (equivalent to 1000, 2000, or 4000 mg/kg of diet per day) for four months. Significant inhibition of growth was observed with 4%; severe diarrhoea and marked abdominal bloating were noted at 8%, and all the rats died within two weeks. Autopsy revealed irritation of the gastrointestinal tract in rats fed 8% (Fitzhugh & Nelson, 1948).

Technical-grade sodium lauryl sulfate (86% w/w active ingredient; chain length distribution, C_{12}–C_{15}) was fed to four groups of 12 male and

12 female Carworth Farm 'E' rats at a dietary level of 0, 40, 200, 1000, or 5000 ppm (corresponding to 2, 10, 50, or 250 mg/kg bw per day) for 13 weeks. No abnormalities were observed in behaviour, body weight, food intake, haematological parameters, urinary pH or osmolality, serum urea or protein, or organ weights, except for a significant increase in the absolute weight of the liver in females fed 5000 ppm (Walker et al., 1967).

C7.2.1.2 *Administration in the drinking-water*

Groups of five or 10 male Wistar rats were given water containing sodium 2-ethylhexanol sulfate, sodium 7-ethyl-2-methyl undecanol-4 sulfate, or sodium 3,9-diethyl tridecanol-6 sulfate at a concentration of 0, 0.25, 0.5, 1, 2, or 4% for 30 days. Water intake was decreased at concentrations ≥ 2% of sodium 2-ethylhexanol sulfate and sodium 7-ethyl-2-methyl undecanol-4 sulfate and at ≥ 1% sodium 3,9-diethyl tridecanol-6 sulfate. Body weight gain was suppressed at 4% sodium 2-ethylhexanol sulfate. None of the rats died, and no haematological abnormalities were observed during the experiment. Proteinurea was seen at 2 and 4% sodium 2-ethylhexanol sulfate. The major histopathological findings were renal changes, including light cloudy swelling and secretion in the renal tubules and congestion or dilation of Bowman's capsule. The no-effect doses were 0.44 g/kg bw per day of sodium 2-ethylhexanol sulfate, 0.1 g/kg bw per day of sodium 7-ethyl-2-methyl undecanol-4 sulfate, and 0.25 g/kg bw per day of sodium 3,9- diethyl tridecanol-6 sulfate (Smyth et al., 1941).

C7.2.1.3 *Dermal application*

A group of 15 male Wistar rats received 2 ml of a commercial preparation of AS (22.5% active ingredient) on their backs, and the livers of three rats were examined under the electron microscope three and 30 days later; a control group was available. Redness of the skin and wrinkles were observed in treated animals at 24 h; the redness subsequently increased, the dermis became lacerated, and bleeding occurred. These lesions reached a peak at 57 days but tended to regress about 10 days later. Five rats died within the first 19 days. Electron microscopy at three days revealed separation of the intercellular space, cells with a high electron density, elongation of mitochondria, swelling of the smooth-surfaced endoplasmic reticulum, and a decreased prevalence of fatty droplets. Electron microscopy at 30 days showed liver parenchymal cells filled with mitochondria, apparently abnormally

divided and proliferated smooth-surfaced endoplasmic reticulum, abnormally rough-surfaced cells, a typical Golgi apparatus, myelin-like structures in bile canaliculi, and extracellular prolapse of mitochondria (Sakashita et al., 1974).

Electron microscopy of the liver was also performed after dermal application of a commercial preparation of AS (22.5% active ingredient) to male Wistar rats (number not specified) at a dose of 5 mg/kg active ingredient once a day for 30 days. Hepatic degeneration, seen as atrophy and a high density of liver cells, was observed; in cells, there was deformation of nuclei, mitochondria, and the Golgi apparatus, an increased number of lysosomes, and swelling of endoplasmic reticula (Sakashita, 1979).

As no information was given on the method of application (occluded or non-occluded), these results were not interpretable in terms of risk to human health.

C7.2.2 Rabbit

Sodium lauryl sulfate was applied dermally to three groups, consisting of two male and two female rabbits with intact skin and one male and one female rabbit with abraded skin, at a dose of 6, 60, or 150 mg/kg bw five times per week for three months. A control group consisted of one male and one female with intact skin and one male with abraded skin. Dose-related irritation of the skin was observed in all treated animals (Carson & Oser, 1964).

C7.3 Long-term exposure; carcinogenicity

C7.3.1 Mouse

In a study of the effects of AS on the carcinogenicity of benzo[*a*]pyrene (BaP), a 10% AS solution, a 0.3% BaP solution, and a 10% AS:0.3% BaP solution were applied to the backs of groups of 10 male and 20 female mice twice a week for one year. Skin tumours appeared in all mice treated with BaP or AS:BaP. The average ages at the appearance of skin tumours were 119 days in the group exposed to BaP and 102 days in that exposed to AS:BaP. It was concluded that AS accelerated the induction of tumours by BaP ($p < 0.1$). Untreated mice and vehicle (acetone) controls had no skin tumours; one female exposed to AS had a skin tumour, but this finding was not considered to be related to treatment (Yamamoto, 1977).

C7.3.2 Rat

C7.3.2.1 Administration in the diet

Three groups of 12 weanling male Osborne-Mendel rats were given food containing sodium lauryl sulfate at a concentration of 0.25, 0.5, or 1.0% for two years; there was a similar sized control group. No effects attributable to the test material were observed on growth, mortality, or the macroscopic or histopathological appearance of organs. No tumours were reported (Fitzhugh & Nelson, 1948). As there were few animals per group and no toxic effects at any dose, the observations are considered to be of limited value.

C7.3.2.2 Administration in the drinking-water

Groups of 4–11 white rats were given drinking-water containing sodium lauryl sulfate at a concentration of 0, 0.1, 0.25, 0.5, 1, 5, or 10% for 120 or 160 days. Dose-related increases in mortality occurred at doses ≥ 0.25%; at doses ≥ 5%, all rats died. Histological examination of rats exposed to doses ≥ 0.25% revealed marked inflammatory changes of the lumen of the oesophagus in those that died, but the changes were slight in surviving animals. No abnormalities were seen in the liver, kidney, or intestine. The intake of the materials was about 30 mg/animal per day in those given 0.1% and 150 mg/animal per day in those given 1.0% (Epstein et al., 1939).

Groups of 9 or 10 weanling male Wistar rats were given drinking-water containing technical-grade sodium lauryl sulfate at a concentration of 0, 0.05, or 0.25% for five months. Growth was not suppressed, even at the higher concentration, and the activities of serum enzymes, including glutamate–oxalate and glutamate–pyruvate transaminases, alkaline phosphatase and cholinesterase, were not affected. At 0.25%, the triglyceride level increased in the liver but decreased in serum, while hepatic and serum levels of cholesterol, phospholipids, and free fatty acids were unchanged. Increased weights of spleen, lung, and kidney were noted at 0.25%. Histopathologically diagnosed bronchopneumonia, observed in all animals given 0.25% and two animals given 0.05%, was considered to be a characteristic effect of the test material (Fukazawa et al., 1978).

The results of long-term studies are shown in Table 40.

Table 40. Results of long-term exposure of experimental animals to alkyl sulfates (AS)

Species, strain, numbers per group	Material	Route	Dosage	Results	Reference
Mouse, ddy/SLC 10 M, 20 F	10% AS, 3% benzo[*a*]pyrene	Dermal	Twice per week, 1 year	Skin tumours	Yamamoto (1977)
Rat, Osborne-Mendel 10–12 M	1.0%AS, C_{12}	Diet	0, 0.25, 0.5, 1.0%, 2 years	No effects	Fitzhugh & Nelson (1948)
Rat, Wistar, 9–10	AS, C_{12}	Diet	0, 0.05, 0.25%, 5 months	Increased weights of spleen, lung, and liver at highest dose	Fukuzawa et al. (1978)
Rat, 4–11	AS, C_{12}	Diet	0, 0.1, 0.25, 0.5 1.0, 5.0%, 160 days	Oesophageal irritation	Epstein et al. (1939)

C7.4 Skin and eye irritation; sensitization

C7.4.1 Local irritation

C7.4.1.1 Skin

Groups of two to six white rats received a subcutaneous injection of 1 ml of one of 10 solutions of sodium AS, ranging from 0.125 to 10% and were observed for one week after the injection. No reactions occurred at 0.125%, but sloughing and subcutaneous lumps in the skin appeared in rats given doses ≥ 0.19%. In a study in which the diffusibility of trypan blue was used as an index of irritation, groups of five to nine white rats were given subcutaneous injections of 0.2 ml sodium AS at one of six concentrations ranging from 0.15 to 5%. Two hours after the injection, slight reactions were seen in animals given 0.15% and marked reactions in those given 2.5 or 5% (Epstein et al., 1939).

Groups of three albino rabbits received closed-patch applications of 5 ml of 1, 5, or 25% sodium lauryl sulfate solution on intact and abraded areas of shaven abdominal skin. Over a 14-day period, 10 applications were made to intact skin and three to abraded skin; additionally, small amounts of the material were applied daily to the intact ears of groups of three rabbits. Occluded application to the abdomen produced erythema and blistering, which was more severe on abraded skin. Application to the intact ear resulted in very slight erythema at the 1% concentration, very slight to slight erythema at 5%, and slight erythema with moderate to severe burns at 25% (Olson et al., 1962).

Sodium alcohol (coconut alcohol, mainly C_{12}) sulfate solutions of 0.1, 1.0, and 2.5% were applied in occluded tests in rabbits as 1 ml of each solution on the back three times on three days. Macroscopic and histological examination seven days after application revealed no abnormalities at 1.0% and moderate irritation at 2.5%. In open tests, 1 ml of each of the solutions was applied to the backs of rabbits and 0.5 ml to the backs of guinea-pigs five times a week for 4.5 weeks. No abnormal findings were seen in animals receiving 0.1 or 1.0% groups, but there was moderate irritation at 2.5% (Brown & Muir, 1970).

Groups of three male Wistar rats received applications of 0.5 g of a 20 or 30% solution of linear lauryl sulfate (C_{12}; purity, 98.91%) on the back once a day for 15 days. The skin at the application site and the tissues of the tongue and oral mucosa (to determine the effects of licking) of animals receiving the 30% solution were examined histologically 16 days after application. Body weight gain was inhibited

in the group given the 20% solution; body weight was decreased in the group at 30%, and two rats had died by the end of the experiment. A dry, thick, yellowish-white or reddish-brown crust was observed after two to three days in animals given 20% and after one to two days in those given 30%. When the crust was abraded several days later, ulcers occurred at the abraded site, which remained unchanged for 16 days in animals at 20% group and were aggravated in those at 30%. Histological examination of the application site revealed severe necrosis extending from the epidermis to the upper layer of the dermis, dense inflammatory-cell infiltration into the upper layer of the dermis just below the necrotic area, diffuse inflammatory-cell infiltration throughout the dermis, swelling of collagenous fibres in the dermis, and sloughing. Histological examination of the tongue revealed necrosis extending from the surface to the middle epithelial layer of the mucosa, inflammatory-cell infiltration into the upper layer of the dermis, and sloughing. Histological examination of the mucosa of the oral cavity revealed thickening of the stratum corneum and germinative and slight degeneration (pale staining) of epithelial cells (Sadai & Mizuno, 1972).

The effects of sodium lauryl sulfate on oesophageal and gastric mucosa were studied in cats by irrigation and pledget techniques. In the irrigation technique, the stomachs and oesophaguses of two cats were filled with 10 and 20% solutions of sodium lauryl sulfate, respectively, for 15 min, and then tissues were taken for histological examination. Pledgets soaked in 10 or 20% sodium lauryl sulfate solution were applied to the exposed oesophageal and gastric mucosa of two other cats for 10 min, and specimens were taken 90 min later. The 10% solution produced moderate injury to the oesophagus, consisting of intramucosal oedema and congestion and loss of superficial epithelial layers; in the stomach, there was hydropic degeneration, loss of surface mucosal cells, vascular congestion with submucosal oedema, and occasional focal ulceration. Treatment with the 20% solution resulted in more extensive damage, and particularly extensive submucosal oedema and disruption and erosion of the superficial mucosa of both the oesophagus and stomach (Berensen & Temple, 1976).

C7.4.1.2 *Eye*

Three drops of one of nine solutions of sodium lauryl sulfate ranging from 0.019 to 5.0% were instilled into the eyes of rabbits three times at 10-min intervals, and the rabbits were observed for 48 h. There were no abnormal findings at 0.038%, but slight chemosis and redness were seen at 0.075% and marked chemosis and redness at 5% (Epstein et al., 1939).

The minimal concentration of sodium lauryl sulfate that caused corneal necrosis (detected by fluorescein staining) after instillation into the eyes of rabbits was 0.1% (Smyth et al., 1941). In another study, two drops of a 1, 5, or 25% solution of sodium lauryl sulfate were instilled into both sides of the eyes of groups of three rabbits; 30 min later, one of the eyes was washed. Moderate corneal injury was observed in unwashed eyes of animals receiving the 5 or 25% solution; in washed eyes, either slight conjunctivitis or moderate corneal injury was observed at 25%, slight conjunctivitis at 5%, and only very slight conjunctivitis at 1% (Olson et al., 1962).

In an irritation test based on a method developed by the United States Food and Drug Administration, 0.1, 1, or 25% solutions of sodium coconut alcohol sulfate were instilled into the eyes of rabbits. No reaction was seen at 0.1%; mild conjunctivitis lasting for 48 h was seen at 1%, and severe conjunctivitis lasting for 72 h was observed at 25% group, but there was no permanent damage (Brown & Muir, 1970). Solutions of a synthetic alkyl sulfate and five AS consisting mainly of C_{10}, C_{12}, C_{14}, C_{16}, or C_{18}, were instilled at concentrations of 0.01–5% into the eyes of three rabbits, which were observed for 168 h. The materials caused similar reactions. No abnormalities were seen at 0.01%. Slight congestion and marked congestion or oedema were observed at 0.05 and 0.1% within 2 h, but these effects had disappeared 24 h later. In the groups given ≥ 0.5%, marked reactions were seen for 24 h, including severe congestion and oedema, increased lachrymal secretion, turbidity of the cornea, and disappearance of the corneal reflex, but these tended to regress and had disappeared completely by 120 h (Iimori et al., 1972).

C7.4.2 *Skin sensitization*

A 0.1% solution of a sodium lauryl sulfate derivative of coconut alcohol was applied to the skin or injected intradermally into groups of 10 guinea-pigs three times per week for three weeks. Ten days later the animals received challenge doses and were observed for 48 h. No reaction occurred in the group treated dermally, but a slight reaction was observed 24 h after the challenge in some of the guinea-pigs treated intradermally (Brown & Muir, 1970).

C7.5 Reproductive toxicity, embryotoxicity, and teratogenicity

Daily doses of 0.2, 2, 300, or 600 mg/kg bw of AS were administered by gavage to CD rats, CD-1 mice, and NZW rabbits. Groups of 20 rats and mice were given AS on days 6–15 of pregnancy, and groups of 13 rabbits were treated on days 6–18 of pregnancy. The doses of 0.2 and

2 mg/kg bw per day were estimated to be equivalent to 1–2 and 10–20 times the maximal amount of AS to which humans are exposed. Three rats given 600 mg/kg bw died during the study, but the surviving rats and those given 300 mg/kg bw had only mild to moderate inhibition of body weight gain. Mice given 600 mg/kg bw showed severe effects, including anorexia and inhibition of body weight gain, and four animals died during the study; in those given 300 mg/kg bw, inhibition of body weight gain was mild to moderate. Rabbits given 600 mg/kg bw showed severe effects, including diarrhoea, anorexia, and reduced rate of body weight gain, and 11 died during the study; those given 300 mg/kg bw showed mild to moderate reduction of body weight gain. No toxic effects were seen in any of the animals given 0.2 or 2 mg/kg bw. No adverse effects were seen on litters of rats at any dose. Some mice and rabbits at each dose had total litter loss, but the other litter parameters did not differ from those of controls. No major malformations were seen at any dose in offspring of rats, mice, or rabbits, and the incidence of skeletal variations in offspring of rats given 600 mg/kg bw was significantly low. A high incidence of skeletal anomalies was seen in litters of mice given 600 mg/kg bw, and those of rabbits at 2.0 mg/kg bw had a significantly higher incidence of skeletal variations; however, the incidences of anomalies and variations were within the background range (Palmer et al., 1975a).

Groups of 21 ICR mice received applications of 15 mg/kg bw per day of a 0.4, 4, or 6% aqueous solution of AS (98% sodium dodecyl sulfate, 0.5% N_2SO_4, 0.1% NaCl, and 0.1% H_2O) to a 3 x 3-cm^2 area of shaven dorsal skin on days 6–13 of pregnancy. The 0.4% solution was equivalent to about 10–12 times the specified concentration used by humans, and the application area was equivalent to about one-seventh of the total surface area of the mouse. The body weight gain of dams exposed to the 4 or 6% solution was reduced; there were no deaths. The numbers of dams with surviving young were 19/21 in the control group, 20/20 at 0.4%, 17/20 at 4%, and 11/21 at 6%; the decrease in dams at 6% was significant. Fetal weights were significantly lower in dams at 4 and 6%, but there were no other differences from the control values. The incidence of cleft palate was fairly high in offspring of dams exposed to the 4 or 6% solution, and a tendency to delayed ossification was seen; however, none was significant (Takahashi et al., 1976).

A dose of 0.1 ml/day of a 2% aqueous solution of AS was applied to a 2 x 3-cm^2 area of shaven dorsal skin in groups of 20–26 ICR mice on days 1–17 of pregnancy. The same dose of a 20% solution was applied to a similar group up to the 10th day of pregnancy, and implantation was examined on the 11th day. In addition, 14 mice were injected

subcutaneously with 2 mg/kg bw per day of AS on days 8–10 of pregnancy. The numbers of dams with implantations were 18/20 controls, 14/22 at 2%, 1/26 at 20%, and 13/14 at 2 mg/kg bw; the decrease at 20% was significant. There were no significant changes in litter parameters and no significant changes in the incidences of major malformations, minor anomalies, or skeletal variations. AS thus disturbed implantation and caused abortion at maternally toxic doses, but in surviving litters it had no effect on the size or numbers of fetuses, although low fetal weight and delayed ossification were observed. At doses that had no or only mild effects on the dams, no adverse effects were seen on the fetuses. The effects of AS on the fetus therefore appear to be secondary to the toxic effects on the dams (Nomura et al., 1980).

C7.6 Mutagenicity and related end-points

Sodium lauryl sulfate did not cause differential toxicity in *Bacillus subtilis* H17 (*rec*$^+$) or M45 (*rec*$^-$) at concentrations of 20–2000 µg/plate, and it did not induce reverse mutations in *Salmonella typhimurium* TA98 or TA100 at 1–500 µg/plate or in *Escherichia coli* WP2 *trp* at 10–1000 µg per plate (Inoue & Sunakawa, 1979).

Sodium lauryl sulfate, Dobanol 25 sulfate LCU, and Dobanol 25 sulfate HCB (aliphatic alcohol sulfates with chain lengths of C_{10}–C_{15}) were fed in the diet to groups of six male and six female Colworth/ Wistar rats for 90 days at a concentration of 0.56 or 1.13%, the latter being the maximal tolerated dose. No effect was seen on chromosomes in bone-marrow cells (Hope, 1977).

After dodecyl sulfate was administered to male ddY mice intraperitoneally at 50 mg/kg bw, the incidence of polychromatic erythrocytes with micronuclei in the bone marrow was similar in treated and control groups (Kishi et al., 1984).

C7.7 Special studies

Intravenous injection of 1 mg/min sodium decyl sulfate or 5.7 mg/min sodium dodecyl sulfate to cats increased pulmonary arterial pressure, caused a small increase in systemic vascular resistance, and reduced the ventilation volume per minute after about 5 min. Intravenous injection of 4.6 mg/min sodium octyl sulfate or 6.3 mg/min sodium tetradecyl sulfate had similar effects. The increase in pulmonary arterial pressure was considered to be due to a direct effect on the smooth muscle of blood vessels and bronchi. The blood sugar level was unchanged (Schumacher et al., 1972).

The effects of sodium lauryl sulfate on histamine release from mast cells were studied *in vitro* in peritoneal mast cells isolated from rats. Histamine was released at a concentration of 0.03 mmol/litre, and the critical micelle concentration in buffer at 22 °C was 1.0 mmol/litre. Sodium lauryl sulfate and its mono- and tri-ethoxy derivatives had the most potent histamine releasing capacity of nine surfactants with a chain length of C_{12} (Prottey & Ferguson, 1975).

C8. EFFECTS ON HUMANS

Section summary

In patch tests, human skin can tolerate contact with solutions containing up to 1% AS for 24 h with only mild irritation. AS caused delipidation of the skin surface, elution of natural moisturizing factor, denaturation of the proteins of the outer epidermal layer, and increased permeability and swelling of the outer layer. They did not induce skin sensitization in volunteers, and there is no evidence that they induce eczema. No lasting injuries or fatalities have been reported following accidental ingestion of detergent formulations containing AS.

C8.1 Exposure of the general population

Surface-active agents are found in shampoos, dishwashing products, household cleaners, and laundry detergents, and AS are major components of these products. The composition of nonionic and ionic surfactants varies between 10 and 30%. Surface-active agents can affect human skin and eyes.

C8.2 Clinical studies

C8.2.1 Skin irritation and sensitization

AS can be mildly to moderately irritating to human skin. No data were available on sensitization.

The relative intensity of skin erythema produced on the lower back of volunteers was evaluated by applying concentrations of 0.2–5.4% of C_8, C_{10}, C_{12}, C_{14}, or C_{16} AS under a closed patch for 24 h or under a closed patch re-applied once daily for 10 days. C_{12} AS were more potent than AS with other alkyl chain lengths (Kligman & Wooding, 1967).

A circulation method was used to evaluate the relative intensity of skin roughness induced on the surface of the forearms of volunteers after application for 1 min of 1% aqueous solutions of AS with an alkyl chain length of C_8, C_{10}, C_{12}, or C_{14}. The potential to cause skin roughness increased with alkyl chain length, reaching maximal intensity at C_{12} (Imokawa et al., 1974, 1975a). In other studies, the relative degree of skin roughening was correlated with the extent of protein denaturation but not with irritating potential determined in a closed-patch test (Imokawa et al., 1975b).

Primary skin irritation induced by a 1% aqueous solution (pH 6.8) of dodecyl sulfate (relative molecular mass, 288.5) was studied in a 24-h closed-patch test on the forearms of seven male volunteers. The relative intensity of skin irritation was scored by grading erythema, fissuring, and scaling. The average score for AS was 4.86, whereas that for a water control was 1.79. Dodecyl sulfate was more irritating than either LAS or AOS (Oba et al., 1968a).

The intensity of skin irritation produced by a 1% aqueous solution of sodium AS was studied in a 24-h closed-patch test on the forearm and in a 40-min drip test on the interdigital surface in which the compound was dripped once daily for two consecutive days at a rate of 1.2–1.5 ml/min. Skin reactions were scored by grading erythema in the patch test and by grading scaling in the drip test. The average scores were 2.5 for primary skin irritation at 24 h in the patch test and 1 for scaling at two days in the drip test; in both tests, the control value was 0. AS was more irritating than LAS or AOS in the patch test, whereas the score of AS for skin scaling in the drip test was similar to that of LAS but higher than that of AOS (Sadai et al., 1979).

Moderate to intense erythema was produced on the forearms of 10 volunteers in a 24-h closed-patch test by a 10% aqueous solution of AS with an average chain length of C_{12}. The mean irritation scores were significantly higher at 26 h (2.85 out of 8 possible points) and at 28 h (2.88) than at 24 h (2.00), when the patches were removed. Irritation had decreased by 48 h, and a significant decrease in the intensity of inflammation was apparent at 96 h (Dahl & Trancik, 1977).

In a 48-h patch test on the upper arms of 100 pairs of twins (54 monozygotic, 46 dizygotic) with a solution of 0.5% C_{12} AS, no reaction was seen in 50% of the subjects, and slight reaction, ranging from noninflammatory changes to mild erythema, in the other 50%. The response was not related to the type of twin (Holst & Moller, 1975).

Application of aqueous 0.5, 1, or 2% solutions of AS with an average chain length of C_{12} to the backs of healthy male volunteers produced epidermal hyperplasia. Treatment with the 1% solution induced an approximately 30-fold increase in mitotic activity, which peaked 48 h after treatment. Application of either the 0.5 or the 2% solution induced similar but milder changes (Fisher & Maibach, 1975).

Skin permeability to C_8, C_{10}, C_{12}, C_{14}, C_{16}, and C_{18} AS prepared as 0.02, 0.5, and 1% solutions (0.58% C_8 and 0.74% C_{18}) was studied by a circulation method on the forearms of healthy male and female

volunteers. C_{12} AS attained maximum permeation, whereas the permeation of C_8 and C_{18} AS was of the same order as that of water. The authors pointed out the close relationship between permeation and irritation (Szakall & Schulz, 1960).

C8.2.2 *Effects on the epidermis*

The effects of AS on the stratum corneum include delipidation of the skin surface, elution of natural moisturizing factor, denaturation of protein of the stratum corneum, increased permeability, swelling of the stratum corneum, and inhibition of enzyme activities in the epidermis. These effects, and some others, constitute a potential hazard to the epidermis.

The water-holding capacity of thin sheets of callus isolated from the plantar surface of the human foot, with relative moisture contents of 76, 88, and 97%, was compared before and after immersion in water, AS, or soap solution. Water-holding capacity was measured as the weight of water taken up from each solution. The relative moisture content decreased after treatment with AS or soap solution (Blank & Shappirio, 1955).

Elution of natural moisturizing factor was compared for nine kinds of surfactants, including AS, in the arm immersion test, in patch tests, and by measuring eluted amino acids and protein, skin permeation, and freeing of sulfhydryl groups. AS induced a strong reaction in the immersion test and relatively strong reactions in the other tests. The author concluded that the immersion test was the best simulation of actual use (Polano, 1968).

A detergent consisting of long-chain AS was shown to denature stratum corneum protein and thus expose enclosed sulfhydryl groups (Anson, 1941). AS readily released sulfhydryl groups from stratum corneum obtained from abdominal skin taken at autopsy within 12 h of death, but there was no correlation between changes in epidermal permeability and the amounts of sulfhydryl released (Wood & Bettley, 1971). AS were the most effective surfactants with regard to denaturation of protein, measured as inhibition of invertase activity (Imokawa et al., 1974; Okamoto, 1974). AS were found to denature skin keratin (a filamentous protein), bovine serum albumin (a globular protein), acid phosphatase (an enzyme protein), and membrane lysozymes (membrane protein) (Imokawa & Katsumi, 1976). Sodium laurate was reported to produce swelling of the stratum corneum (Putterman et al., 1977).

AS with a hydrophobic chain length of C_{12} were maximally absorbed on human callus. Extraction of proteins from human callus was also a function of chain length: C_{12} and C_{14} AS were much more active than C_8, C_{10}, and C_{18} AS (Dominguez et al., 1977).

C8.2.3 *Hand eczema*

In a 24-h closed-patch test of 0.2–0.5% aqueous solutions of AS on the fingers of nine women with hand eczema, skin lesions were not exacerbated, although four women felt slight itching at the patch site (Sasagawa, 1963).

C8.2.4 *Accidental or suicidal ingestion*

Four members of a family accidentally ingested unknown quantities of a household detergent containing 24% lauryl sulfate, 60% sodium tripolyphosphate, and 16% anhydrous soap. Shortly after ingestion, all of the family members experienced abdominal pain and nausea. The 10-year-old daughter and 13-year-old son felt oropharyngeal pain, and the son was found at endoscopic examination to have a 2.5 x 2 cm oropharyngeal burn in the right posterior pharynx and first-degree burns of the oesophagus. The mother had erythema, friability, erythema and a few superficial erosions of the distal oesophagus, and gastritis evidenced by exudate and petechial lesions on the mucosa. The father had haematemesis on a few occasions. The mother, father, and son were examined about one month after the incident by an X ray examination after a barium meal; no strictures were found (Berenson & Temple, 1974).

C9. EFFECTS ON OTHER ORGANISMS IN THE LABORATORY AND THE FIELD

Section summary

AS have been studied in short- and long-term studies in the laboratory and in one study carried out under more realistic conditions. Their toxicity is dependent on alkyl chain length, but no data were available on the differential toxicity of linear and branched AS.

In aquatic organisms, the EC_{50} values for C_{12} AS in a community of marine microorganisms were 2.1–4.1 mg/litre. The NOEC values were 35–550 mg/litre (C_{16}/C_{18}) for *Pseudomonas putida* and 14–26 mg/litre (C_{12}–C_{16}/C_{18}) for green algae; and the EC_{50} values were > 20–65 mg/litre (C_{12}–C_{13}) for green algae and 18–43 mg/litre (C_{12}) for macrophytes.

In aquatic invertebrates, the $L(E)C_{50}$ values were 4–140 mg/litre (C_{12}/C_{15}–C_{16}/C_{18}) for freshwater species and 1.7-56 mg/litre (all C_{12}) for marine species. The long-term NOECs were 16.5 mg/litre (C_{16}–C_{18}) for *Daphnia magna* and 0.29–0.73 mg/litre (chain length not specified) for marine species.

In fish, the LC_{50} values were 0.5–5.1 mg/litre (C_{12}–C_{16} or chain length not specified) for freshwater species and 6.4–16 mg/litre (all C_{12}) for marine species. In a 48-h study of *Oryzias latipes*, chain length influenced LC_{50} values, the measured concentrations being 46 mg/litre for C_{12}, 2.5 mg/litre for C_{14}, and 0.61 mg/litre for C_{16}. This and other studies indicate that toxicity differs by a factor of five for two units of chain length.

In a flow-through study of the effect of C_{16}–C_{18} AS on a biocenosis, an NOEC of 0.55 mg/litre was observed. Many of the studies of toxicity in aquatic environments were carried out under static conditions. As AS are readily biodegraded, this design may result in underestimates of toxicity.

Few data were available on the effects of AS on terrestrial organisms. An NOEC of > 1000 mg/kg (C_{16}–C_{18}) was reported for earthworms and turnips.

C9.1 Microorganisms

During tests of biodegradation, marine bacteria used 20 mg/litre AS as a nutrient source. It was therefore concluded that its toxicity for

the bacterial community studied is nil or very low (Vives-Rego et al., 1987). In a study of the effect of C_{12} AS on the metabolic activity of a marine microbial community, the EC_{50} values for toxic effects on thymidine incorporation and glucose metabolism were reported to be 4.1 and 2.1 mg/litre, respectively. AS also increased exoproteolytic activity (Vives-Rego et al., 1986).

The 30-min EC_{50} for C_{16}–C_{18} AS, based on oxygen consumption, was 35 mg/litre in *Pseudomonas putida* (Robra, 1976). The NOEC for cell reproduction in *Pseudomonas putida* exposed to C_{16}–C_{18} AS was 550 mg/litre (Bringmann & Kühn, 1977).

C9.2 Aquatic organisms

C9.2.1 Aquatic plants

C9.2.1.1 Freshwater algae

The phytoflagellate alga *Poterioochromonas malhamensis* was exposed to C_{12} AS at sublethal concentrations of 28.8, 57.6, 72, 86.4, 100.8, and 115.2 mg/litre (100, 200, 250, 300, 350, and 400 µmol/litre), being transferred every three to four days into fresh medium with a higher test concentration. The initial cell density in each medium was 0.1×10^6 cells/ml; the final cell density, after exposure to the highest concentration of AS, was 0.05×10^6 cells/ml, which was similar to that reached after exposure of unacclimatized algal cultures to 200 µmol/litre AS. Exposure to AS at 57.6 mg/litre (240 µmol/litre) was reported to affect mitosis and cytokinesis, with the formation of cells containing up to 12 nuclei. Exposure of the alga to 50.4 mg/litre (175 µmol/litre) AS resulted in a 24% increase in telophases (binucleated cells). Cells with eight nuclei were also reported in this culture (Röderer, 1987).

The green alga *Selenastrum capricornutum* was exposed to analytical grade C_{12} AS at a concentration of 10, 20, 30, 40, 50, or 100 mg/litre in synthetic medium for three weeks. Growth was reduced by 30% at the lowest concentration (Nyberg, 1988).

The green alga *Chlamydomonas reinhardi* was exposed to 0.02, 0.2, or 2.0 mmol/litre of C_{10}, C_{12}, C_{14}, C_{16}, or C_{18} AS for 7–10 days. Photometric absorption (652 nm) by the exposed cultures was no different from that by controls for the first six days of exposure, although it was reduced slightly at 2 mmol/litre. The authors concluded that the AS were present at below the critical micelle concentration at all concentrations tested (Ernst et al., 1983).

The EC_{50} for growth of the green alga *Selenastrum capricornutum* exposed to C_{12} AS for two to three days was within the range 45–65 mg/litre (Yamane et al., 1984). An EC_{50} of 9 mg/litre C_{14} AS was found for growth of *S. capricornutum* (Konno & Wakabayashi, 1987).

C9.2.1.2 Macrophytes

The seven-day EC_{50} values for C_{12} AS in the duckweed *Lemna minor* under flow-through conditions were 43 mg/litre for frond count, 29 mg/litre for dry weight, and 18 mg/litre for root length. The time-independent EC_{50} for growth rate/doubling time was 44 mg/litre (Bishop & Perry, 1981).

C9.2.2 Aquatic invertebrates

The acute toxicity of AS to aquatic invertebrates is summarized in Table 41. The 48-h LC_{50} values were 8–60 mg/litre for daphnids; the 96-h LC_{50} values ranged from 3.2 to 4.2 mg/litre for marine invertebrates.

The 48-h LC_{50} for lugworms (*Arenicola marina*) exposed to AS was calculated to be 15.2 mg/litre (95% confidence interval, 13.2–17.6). Tissues from lugworms exposed to AS at a concentration close to that of the LC_{50} were examined for changes in morphology by both light and electron microscopy: serious damage was found in the epidermic receptors and less serious damage in the caudal epidermis and gills. No morphological effects were reported on the thoracic epidermis or intestine. AS caused separation inside the caudal epithelial layer, resulting in holes in some caudal papillae. Deciliation of the epidermic receptors was also reported. The authors concluded that the physiological response of damaged epidermic receptors was reduced or blocked after exposure to AS. AS also induced fissures in the epithelial layer of the gills (Conti, 1987).

Caeriodaphnia dubia were exposed to C_{12} AS for three generations under static renewal conditions, with the following mean water parameters: temperature, 26.2 °C; pH, 8.2; hardness, 94.4 mg/litre $CaCO_3$; and alkalinity, 82.2 mg/litre $CaCO_3$. The water was changed every second day. The LC_{50} for survival of three broods of *C. dubia* was calculated to be 41 ± 3.2 mg/litre. The mean EC_{50}, based on progeny produced, was calculated to be 36 ± 3.2 mg/litre. No statistically significant effects were reported after exposure to 83 mg/litre AS, although the size of later broods was reduced (Cowgill et al., 1990).

The effect of 0.25–10 mg/litre AS was studied on the growth and survival of eggs and larvae of oysters (*Crassostrea virginica*) and clams (*Mercenaria mercenaria*). The minimal concentrations that caused a significant reduction in the number of fertilized eggs which developed into normal larvae two days after hatching were 0.73 mg/litre for clams and 0.29 mg/litre for oysters. The minimal concentration that caused a significant reduction in growth and survival between two and 12 or 14 days after hatching was 1.46 mg/litre for both species. The EC_{50} values, based on the development of fertilized clam and oyster eggs to normal straight-hinge larvae after 48 h, were calculated to be 0.47 mg/litre for clams and 0.37 mg/litre for oysters (Hidu, 1965).

After snails (*Lymnaea peregra*) were exposed to C_{12} AS at measured concentrations of 0.6-12 mg/litre for six days, a significant, dose-related reduction in the dry weight of shells was observed, but the organic content of shells was not significantly affected at any concentration (Tarazona & Nunez, 1987).

C9.2.3 *Fish*

The acute toxicity of AS to fish is also summarized in Table 41. The 48-h LC_{50} values were 0.5–51 mg/litre for medaka (*Oryzias latipes*). A 96-h LC_{50} value of 1.7 mg/litre was reported for both rainbow trout (*Salmo gairdneri*) and sheepshead minnow (*Cyprinodon variegatus*). The acute toxicity of AS to fish tends to increase with increasing carbon-chain length.

Rainbow trout (*Oncorhynchus mykiss*) and goldfish (*Carrasius auratus*) were exposed to C_{12} AS at a concentration of 70 mg/litre at different levels of water hardness. Trout treated in hard water (300 mg/litre $CaCO_3$) died within 40–45 min; those treated in soft water (60 mg/litre $CaCO_3$) died after 3 h. Goldfish treated in hard water died within 90–110 min, whereas those treated in distilled water (no $CaCO_3$) were alive and apparently normal after 24 h (Tovell et al., 1974). When yearling rainbow trout were maintained in water containing C_{12} AS at a concentration of 100 mg/litre, the time to 50% lethality was calculated to be 4.9 h. The changes seen in the gills were typical of an acute inflammatory reaction: The gill epithelium was lifted away from the underlying tissue, and lymphocytes and granulocytes invaded the subepithelial spaces. Large numbers of epithelial cells died, but the epithelium was not punctured (Abel & Skidmore, 1975).

After exposure of the eggs of carp (*Cyprinus carpio*) to AS of various chain lengths from spawning to hatching, the LC_{50} values were calculated

Table 41. Toxicity of alkyl sulfates (AS) to aquatic organisms

Species	Size or age	Static or flow	Temp. (°C)	Hardness or salinity	pH	AS chain length	End-point	Concn (mg/litre)	Reference
Eastern oyster (*Crassostrea virginica*)	Embryo	Static	20	25[a]		C_{12}	48-h LC_{50}	1.7[b]	Mayer (1987)
Mysid shrimp (*Metamysidopsis swifti*)	Juvenile	Static	25	30[a]		C_{12}	96-h LC_{50}	3.2[b]	
Mysid shrimp (*Mysidopsis bahia*)	Juvenile	Static	25	20[a]		C_{12}	96-h LC_{50}	4.2[b]	
	Adult	Static	22			C_{12}	96-h LC_{50}	6.62	Roberts et al. (1982)
Shrimp (*Neomysis americana*)	Adult	Static	22	20.0 ± 0.5[a]		C_{12}	96-h LC_{50}	7.24	
Copepod (*Eurytemora affinis*)	Adult	Static		10[a]		C_{12}	96-h LC_{50}	2.6	
(*Acartia tonsa*)	Adult	Static				C_{12}	96-h LC_{50}	0.55	
Scud (*Gammarus pulex*)						NS	72-h LC_{50}	9–46	Gilbert & Pettigrew (1984)
Water flea (*Daphnia magna*)		Static	20			C_{12}	24-h EC_{50}	17.4	Snell & Persoone (1989)
		Static	20			C_{12}	24-h EC_{50}	27.5	Persoone et al. (1989)
						C_{16}–C_{18}	24-h EC_{50}	27.5	Steber et al. (1988)
						C_{12}	24-h EC_{50}	10.5–24.3	Cowgill et al. (1990)

Table 41 (contd)

Species	Size or age	Static or flow	Temp. (°C)	Hardness or salinity	pH	AS chain length	End-point	Concn (mg/litre)	Reference
Water flea (*Daphnia pulex*)						C_{12}	24-h LC_{50}	15.0	Snell & Persoone (1989)
						C_{12}	24-h LC_{50}	9.5–20.5	Cowgill et al. (1990)
Mosquito (*Aedes aegypti*)	2nd/3rd stage	Static	25			C_{12}–C_{15}	24-h LC_{50}	4	van Emden et al. (1974)
Rainbow trout (*Salmo gairdneri*)		Flow	15	350–375[c]	8.3–8.5	NS	96-h LC_{50}	4.62	Fogels & Sprague (1977)
						NS	96-h LC50	1.7	Gilbert & Pettigrew (1984)
Atlantic silverside (*Menidia menidia*)	59 mm	Static	22	10[a]		C_{12}	96-h LC_{50}	6.4	Roberts et al. (1982)
Medaka (killifish) (*Oryzias latipes*)							48-h LC_{50}	10	Tomiyama (1974)
	323 mg	Static[f]	23–24		5.6–5.8	C_{12}	24-h LC_{50}	70[b]	Kikuchi et al. (1976)
	323 mg	Static[f]	23–24		5.6–5.8	C_{12}	48-h LC_{50}	51[b]	
	323 mg	Static[f]	19–21		5.6–5.8	C_{14}	24-h LC_{50}	5.9[b]	
	323 mg	Static[f]	19–21		5.6–5.8	C_{16}	24-h LC_{50}	0.78[b]	
	323 mg	Static[f]	19–21		5.6–5.8	C_{12}	48-h LC_{50}	0.5[b]	
	~262 mg	Static[f]	21–22		6.7–7.1	C_{12}	48-h LC_{50}	46[d]	Kikuchi & Wakabayashi (1984)
	~262 mg	Static[f]	21–22		6.7–7.1	C_{12}	48-h LC_{50}	2.5[d]	
	~262 mg	Static[f]	21–22		6.7–7.1	C_{12}	48-h LC_{50}	0.61[d]	

Table 41 (contd)

Species	Size or age	Static or flow	Temp. (°C)	Hardness or salinity	pH	AS chain length	End-point	Concn (mg/litre)	Reference
Sheepshead minnow (*Cyprinodon variegatus*)	Juvenile	Static	25	20[a]		C_{12}	96-h LC_{50}	1.7[b]	Mayer (1987)
Fathead minnow (*Pimephales promelas*)	NS	Static	NS	80–400	7.4–8.2	NS	96-h LC_{50}	5–6	Henderson et al. (1959)
	< 30 d	Static	20			C_{15}	48-h LC_{50}	7.8	Cowgill et al. (1990)
	<30 d		17			C_{12}	24-h LC_{50}	7.7–9.7	
	<30 d		17			C_{12}	96-h LC_{50}	7.0-9.0	
	30±2 d		20			C_{12}	48-/96-h LC_{50}	38.0	
Carp (*Cyprinus carpio*)	4.4 mg	Static	22	25	7	C_{10}	12-h LC_{50}	180[b]	Kikuchi et al. (1976)
	4.4 mg	Static	22	25	7	C_{10}	48-h LC_{50}	13[b]	
	4.4 mg	Static	22	25	7	C_{12}	12-h LC_{50}	46[b]	
	4.4 mg	Static	22	25	7	C_{14}	48-h LC_{50}	5.0[b]	
	4.4 mg	Static	22	25	7	C_{16}	12-h LC_{50}	0.69[b]	
	4.4 mg	Static	22	25	7	C_{16}	48-h LC_{50}	0.69[b]	

Static, water unchanged for duration of test; NS, not specified; flow, flow-through conditions: AS concentration in water maintained continuously; static[r], static renewal: water changed at regular intervals

[a] Salinity (‰)

[b] Based on nominal concentrations

[c] Hardness expressed as mg/litre $CaCO_3$

[d] Based on measured concentrations

to be 18 mg/litre for C_{12} AS, 2.9 mg/litre for C_{14} AS, and > 1.6 mg/litre for C_{16} AS (Kikuchi et al., 1976).

The minimal avoidance concentration of AS, i.e. the concentration at which fish spend 65% of a 5-min period in clean water in order to avoid AS, was 7.1 µg/litre for medakas (*Oryzias latipes*) (Hidaka et al., 1984). The threshold concentrations for avoidance of AS by ayu (*Plecoglossus altivelis*) were 4.0 µg/litre of a formulation and 8.4 µg/litre of pure reagant AS (Tatsukawa & Hidaka, 1978). The environmental relevance of avoidance studies is questionable (see also section A9.3.3.4 of the monograph on LAS).

Larvae of the fathead minnow (*Pimephales promelas*) were exposed to C_{12} AS at a concentration of 1.2, 2.3, 4.6, 9.2, or 18.4 mg/litre for seven days under static renewal conditions. Survival and final dry weight were not significantly affected at concentrations up to and including 4.6 mg/litre; however, at 9.2 and 18.4 mg/litre, no fish survived. When the test was repeated over an eight-day period, significantly reduced survival was seen at 4.6 mg/litre, but this result was variable, as some replicates did not show significant effects. The mean of the geometric means of the NOEC and LOEC values for the embryo-larval test was 3.8 mg/litre; the mean LC_{50} value was 5.5 mg/litre (Pickering, 1988).

An LC_{50} value of 38 mg/litre was reported for fathead minnows exposed to C_{12} AS for either 48 or 96 h. The authors suggested that the same value was obtained because the tests were not carried out aseptically and the C_{12} AS had degraded completely within 48 h (Cowgill et al., 1990).

C9.2.4 *Tests in biocenoses*

In a flow-through biocenosis test, 13 species of aquatic organisms were exposed to C_{16}–C_{18}AS. The species used represented several trophic levels: seven species of algae, four species of protozoa, and two species of rotifers. An NOEC of 0.55 mg/litre was reported for 'biocenotic toxicity'. The lowest concentration at which biocenotic toxicity was reported was 1.65 mg/litre (Guhl, 1987).

C9.3 Terrestrial organisms

No information was available.

APPENDIX I

Reference values for intakes and body weights of laboratory animals, with conversion factors for deriving no-observed-adverse-effect levels (NOAELs) in milligrams per kilogram per day from doses administered as parts per million

Species	Body weight (kg)	Inhalation rate	Water consumption	Food consumption	Dose conversion[a]		
					Air (m^3/day)	Water (litres/day)	Food (g/day)
Mouse	0.03[b]	0.04[b]	0.006[b]	4[b]	1.33	0.20	0.13
Rat	0.35[b]	0.11[d]	0.05[b]	18[b]	0.31	0.14	0.05
Hamster	0.14[b]	0.13[b]	0.03[b]	12[b]	0.93	0.21	0.09
Guinea-pig	0.84[b]	0.40[b]	0.20[b]	34[b]	0.48	0.24	0.04
Rabbit	3.8[b]	2.0[b]	0.41[b]	186[b]	0.53	0.11	0.05
Rhesus monkey	8.0[b]	5.4[c]	0.53[b]	320[b]	0.68	0.07	0.04
Dog	12[b]	4.3[b]	0.61[b]	300[b]	0.36	0.05	0.03
Cat	1.5[d]	0.75[d]	0.15[e]	168[e]	0.50	0.10	0.11
Pig	80[e]	–	5.5[e]	2250[e]		0.07	0.03

From Health Canada (in press); most values have been rounded to two significant figures.

[a] Air: 1 mg/m^3 in air = *x* in mg/kg bw per day; water: 1 ppm (mg/litre) = *x* in mg/kg bw per day; food: 1 ppm in food = *x* in mg/kg bw per day

[b] From Calabrese & Kenyon (1991)

[c] Calculated from the minute volume of 220 ml/kg bw reported by Flecknell (1987)

[d] From Flecknell (1987); values are average of the ranges reported.

[e] From Canadian Council on Animal Care (1980–84); values are average of the ranges reported.

REFERENCES

Abbott DC (1962) The colorimetric determination of anionic surface-active materials in water. Analyst, **87**: 286–293.

Abdel-Shafy HI, Azzam AM, & El-Gamal IM (1988) Studies on the degradation of synthetic detergents by sewage. Bull Environ Contam Toxicol, **41**: 310–316.

Abe S (1984) [Biodegradation of sodium alkylbenzenesulfonates by a soil perfusion method.] Nippon Kagaku Kaishi, **9**: 1465–1470 (in Japanese).

Abe S & Seno M (1987) Biodegradation of sodium linear alkylbenzenesulfonates evaluated with a soil perfusion method. J Am Oil Chem Soc, **64**: 148–152.

Abel PD & Skidmore JF (1975) Toxic effects of an anionic detergent on the gills of rainbow trout. Water Res, **9**: 759–765.

Acher AJ & Yaron B (1977) Behavior of anionic surfactants in a soil-sewage effluent system. J Environ Qual, **6**: 418–420.

Adachi A & Kobayashi T (1982) [Atomic absorption spectrophotometric determination of anionic surfactants in water.] Eisei Kagaku, **28** (2): 99–105 (in Japanese).

Adachi T, Tanaka A, & Yamaha T (1979) [Metabolism of octadecyl sulfate in rats.] Shokuhin Eiseigaku Zasshi, **20** (2): 120–126 (in Japanese).

Amano K & Fukushima T (1993) Partitioning of linear alkylbenzenesulfonates in natural water and sediment. J Environ Sci Health, **28**: 683–696.

Amano K, Fukushima T, Inaba K, & Nakasugi O (1989) Adsorption of linear alkylbenzenesulfonates with the suspended solids in natural aquatic systems. Jpn J Water Pollut Res, **12**: 506–515.

Amano K, Fukushima T, & Nakasugi O (1991) Fate of linear alkylbenzene sulfonates in a lake estuary. Water Sci Technol, **23**: 497–506.

Ambe Y (1973) Determination of alkylbenzene sulfonate (ABS) in bottom sediment. Environ Sci Technol, **7**: 542–545.

Ambe Y & Hanya T (1972) [Determination of alkylbenzene sulfonate in polluted water by a combined method of methylene blue colorimetry with infrared spectrometry.] Bunseki Kagaku, **21**: 252–256 (in Japanese).

Anderson DJ, Day MJ, Russell NJ, & White GF (1990) Die-away kinetic analysis of the capacity of epilithic and planktonic bacteria from clean and polluted river water to biodegrade sodium dodecyl sulfate. Appl Environ Microbiol, **56**: 758–763.

Anson ML (1941) The sulfhydryl groups of egg albumin. J Gen Physiol, **24**: 399–421.

Antal M (1972) Changes in blood glucose level induced by sodium dodecylbenzene sulfonate (SDBS) in rats. Z Ernährwiss, **12** (2): 144–151.

Arima T, Takahashi K, Kawana T, Wakabayashi M, & Kikuchi M (1981) Toxicity of detergent to aquatic organisms. Aquaculture, **29**: 30–37.

Arthur JW (1970) Chronic effects of linear alkylate sulfonate detergent on *Gammarus pseudolimnaeus, Campeloma decisum* and *Physa integra*. Water Res, **4**: 251–257.

Arthur D Little Inc. (1977) Human safety and environmental aspects of major surfactants. A Report to the Soap and Detergent Association, New York, 31 May 1977. Cambridge, Massachusetts, Arthur D. Little Inc., 201 pp.

Arthur D Little Inc. (1981) Human safety and environmental aspects of major surfactants. A Report to the Soap and Detergent Association, New York, 20 February 1981. Cambridge, Massachusetts, Arthur D. Little Inc., 291 pp.

Arthur D Little Inc. (1991) Environmental and human safety of major surfactants: Vol. 1. Anionic surfactants: Part 1. Linear alkylbenzene sulfonates. Final Report to the Soap and Detergent Association, New York, February 1991. Cambridge, Massachusetts, Arthur D. Little Inc., 244 pp.

Arthur D Little Inc. (1993) Environmental and human safety of major surfactants: Vol. 1. Anionic surfactants: Part 4. Alpha olefin sulfonates. Final Report to the Soap and Detergent Association, New York, August 1993. Cambridge, Massachusetts, Arthur D. Little Inc., 84 pp.

Bae KS, Lee HJ, Hah YC, & Hong SW (1982) [The distribution and annual variation of detergent-degrading bacteria in the lower Han river.] Korean J Microbiol, **20**: 113–118 (in Korean).

Barera Y & Adams WJ (1983) Resolving some practical questions about Daphnia acute toxicity tests. In: Bishop WE, Cardwell RD, & Heidolph BB ed. Aquatic toxicology and hazard assessment. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 509–518 (ASTM STP 802).

Bay PHS & Danneman PJ (1985) Skin sensitization potential of alpha-olefin sulfonate (AOS) and a prototype dishwashing detergent containing AOS. J Am Oil Chem Soc, **62** (4): 646.

Berenson MM & Temple AR (1974) Detergent ingestion; unique experience of a family. Clin Toxicol, **7** (1): 25–28.

Berenson MM & Temple AR (1976) Detergent toxicity: effects on esophageal and gastric mucosa. Clin Toxicol, **8**: 399–404.

Berna JL, Ferrer J, Moreno A, Prats D, & Ruiz Bevia F (1989) The fate of LAS in the environment. Tenside Surfactants Deterg, **26**: 101–107.

Berna JL, Moreno A, & Ferrer J (1991) The behaviour of LAS in the environment. J Chem Technol Biotechnol, **50**: 387–398.

Berna JL, Moreno A, Banerji A, Fritsch TR, & Vora BV (1993a) Growth and developments in LAB technologies: 30 years of innovation and more to come. Presentation at the 1993 World Surfactant Congress, 23 September 1993, Montreux, Switzerland. New York, Soap and Detergent Association, 27 pp.

Berna JL, Moreno A, & Ferrer J (1993b) An assessment of the ultimate biodegradation of LAS. Tenside Surfactants Deterg, 30 (3): 217–222.

Birch RR, Biver C, Campagna R, Gledhill WE, Pagga U, Steber J, Reust H, & Bentinck WJ (1989) Screening of chemicals for anaerobic biodegradability. Chemosphere, **19**: 1527–1550.

Bishop WE & Maki AW (1980) A critical comparison of two bioconcentration test methods. In: Eaton JG, Parrish PR, & Hendricks AC ed. Aquatic toxicology. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 61–77 (ASTM STP 707).

Bishop WE & Perry RL (1981) Development and evaluation of a flow-through growth inhibition test with duckweed (*Lemna minor*). In: Branson DR & Dickson KL ed. Aquatic toxicology and hazard assessment. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 421–435 (ASTM STP 737).

Blank IH & Gould E (1961) Penetration of anionic surfactants into skin: (II) study of mechanisms which impede the penetration of synthetic anionic surfactants into skin. J Invest Dermatol, **37** (6): 311–315.

Blank IH & Shappirio EB (1955) The water content of the stratum corneum. J Invest Dermatol, **25**: 391–401.

Blohm SG (1957) The connection between skin-irritating and protein-denaturing effects of some surface-active agents. Acta Dermatol Venereol, 37 (4): 269–275.

von Bock KJ & Mann H (1971) [Anionic surfactants in the coastal areas of the North Sea.] Arch. Fischereiwiss, **22**: 287–292 (in German).

Bornmann G, Loeser A, & Stanisic M (1963) [Study of a detergent based on dodecylbenzene sulfonate.] Fette Seifen Anstrichm, **65** (10): 818–824 (in German).

Borstlap (1967) Intermediate biodegradation products of anionic detergents; their toxicity and foaming properties. In: Proceedings of the International Congress on Surface Active Substances, Brussels, September 1964, Vol. 3, 891–901.

Brenner TE (1968) The impact of biodegradable surfactants on water quality. J Am Oil Chem Soc, **45**: 433–436.

Bressan M, Brunetti R, Casellato S, Fava GC, Giro P, Marin M, Negrisolo P, Tallandini L, Thomann S, Tosoni L, Turchetto M, & Campesan GC (1989) Effects of linear alkylbenzene sulfonate (LAS) on benthic organisms. Tenside Surfactants Deterg, **26**: 148–158.

Bressan M, Marin MG, & Brunetti R (1991) Effects of linear alkylbenzene sulphonate (LAS) on skeletal development of sea urchin embryos (*Paracentrotus lividus* Lmk). Water Res, **25**: 613–616.

Bringmann G & Kühn R (1977) [Limiting values for noxious effects of water pollutants on bacteria (*Pseudomonas putida*) and green algae (*Scendesmus quadricauda*) in the cell multiplication test.] Z Wasser Abwasser Forsch, **10** (3): 87–98 (in German).

Brown VKH & Muir CMC (1970) The toxicities of some coconut alcohol and dobanol-23 derived surfactants. Tenside Surfactants Deterg, **7** (3): 137–139.

Brown VM, Abram FSH, & Collins LJ (1978) The acute lethal toxicity to rainbow trout of an LAS surfactant and of its residues and degradation products. Tenside Surfactants Deterg, **15** (2): 57–59.

Brunner PH, Capri S, Marcomini A, & Giger W (1988) Occurrence and behaviour of linear alkylbenzene sulphonates, nonylphenol diethoxylates in sewage and sewage sludge treatment. Water Res, **22**: 1465–1472.

Buehler EV, Newmann EA, & King WR (1971) Two-year feeding and reproduction study in rats with linear alkylbenzene sulfonate (LAS). Toxicol Appl Pharmacol, **18**: 83–91.

Burke B, Olavesen AH, Curtis CG, & Powell GM (1975) The biodegradation of some anionic detergents in the rat: a common metabolic pathway. Xenobiotica, **5** (9): 573–584.

Burke B, Olavesen AH, Curtis CG, & Powell GM (1976) The biodegradation of the surfactant undecyl sulphate. Xenobiotica, **6** (11): 667–678.

Cain RB, Willats AJ, & Bird JA (1971) Surfactant biodegradation: metabolism and enzymology. In: Biodegradation of materials. New York, John Wiley and Sons, vol 2, pp 136–144.

Calabrese A & Davis HC (1967) Effects of 'soft' detergents on embryos and larvae of the American oyster (*Crassostrea virginica*). Proc Natl Shellfish Assoc, **57**: 11–16.

Calabrese EJ & Kenyon EM (1991) Air toxics and risk assessment. Chelsea, Michigan, Lewis Publishers Inc., 612 pp.

Canadian Council on Animal Care (1980-1984) Guide to the care and use of experimental animals: Vols 1 and 2. Ottawa, Canadian Council on Animal Care.

Canton JH & Slooff W (1982) Substitutes for phosphate containing washing products: their toxicity and biodegradability in the aquatic environment. Chemosphere, **11**: 891–907.

Carson S & Oser BL (1964) Dermal toxicity of sodium lauryl sulfate. J Soc Cosmet Chem, **15**: 137–147.

Castles MA, Moore BL, & Ward SR (1989) Measurement of linear alkylbenzenesulfonates in aqueous environmental matrices by liquid chromatography with fluorescence detection. Anal Chem, **61** (22): 2534–2540.

Cavalli L, Gellera A, Lazzarin A, Nucci GC, Romano P, Ranzani M, & Lorenzi E (1991) Linear alkylbenzene sulphonate removal and biodegradation in a metropolitan plant for water treatment. Riv Ital Sostanze Grasse, **68**: 75–81.

Cavalli L, Divo C, Giuffrida G, Pellizzon T, Radici P, Valtorta L, & Zatta A (1993a) Producing linear alkyl benzene (LAB) from linear olefins using an AlCl3 catalyst. Spec Chem, August: 228–231.

Cavalli L, Gellera A, & Landone A (1993b) LAS removal and biodegradation in a wastewater treatment plant. Environ Toxicol Chem, **12**: 1777–1788.

CEFIC (1992) Linear alkylbenzene. Report of a working group. Brussels, European Chemical Industry Council, 6 pp.

Chattopadhyay DN & Konar SK (1985) Chronic effects of linear alkyl benzene sulfonate on aquatic ecosystem. Environ Ecol, **3**: 428–433.

Colin A Houston & Associates Inc. (1990) Higher alcohols world markets 1988-2000. A multiclient study: January 1990. Mamaroneck, New York, Colin A. Houston & Associates, 21 pp.

Comotto RM, Kimerle RA, & Swisher RD (1979) Bioconcentration and metabolism of linear alkylbenzene sulphonate by daphnids and fathead minnows. In: Marking LL & Kimerle RA ed. Aquatic toxicology. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 232–250 (ASTM STP 667).

Conti E (1987) Acute toxicity of three detergents and two insecticides in the lugworm, *Arenicola marina* (L.): a histological and a scanning electron microscopic study. Aquat Toxicol, **10**: 325–334.

Cook TM & Goldman CK (1974) Degradation of anionic detergents in Chesapeake Bay. Chesapeake Sci, **15**: 52–55.

Cordon TC, Maurer EW, & Stirton AJ (1972) The biodegradation of some sulfated alkanolamides. J Am Oil Chem Soc, **49**: 174–177.

Cowgill UM, Milazzo DP, & Landenberger BD (1990) The reproducibility of the three brood Ceriodaphnia test using the reference toxicant sodium lauryl sulphate. Arch Environ Contam Toxicol, **19**: 513–517.

Cresswell DG, Baldock GA, Chasseaud LF, & Hawkins DR (1978) Toxicology studies of linear alkylbenzene sulphonate (LAS) in rhesus monkeys: (II) The disposition of [^{14}C]LAS after oral or subcutaneous administration. Toxicology, **11**: 5–17.

Dahl MV & Trancik RJ (1977) Sodium lauryl sulfate irritant patch tests: degree of inflammation at various times. Contact Dermatitis, **3**: 263–266.

Daly LW, Schroeder RE, & Killeen JC (1980) A teratology study of topically applied linear alkylbenzene sulphonate in rats. Food CosmetToxicol, **18**: 55–58.

De Henau H, Matthijs E, & Hopping WD (1986) Linear alkylbenzene sulphonates (LAS) in sewage sludges, soils and sediments: analytical determination and environmental safety considerations. Int J Environ Anal Chem, **26**: 279–293.

De Henau H, Matthijs E, & Namkung E (1989) Trace analysis of LAS by HPLC. Detailed results from two municipal sewage treatment plants. In: Quaghebeur D, Temmerman I, & Angeletti G ed. Organic contaminants in waste water, sludge and sediment: Occurrence, fate and disposal. London, Elsevier Science Publishers, pp 5–18.

Denner WHB, Olavesen AH, Powell GM, & Dodgson KS (1969) The metabolism of potassium dodecyl [^{35}S] sulphate in the rat. Biochem J, **111**: 43–51.

Devi Y & Devi S (1986) Effect of synthetic detergents on germination of fern spores. Bull Environ Contam Toxicol, **37**: 837–843.

Di Corcia A, Marchetti M, Samperi R, & Marcomini A (1991) Liquid chromatographic determination of linear alkylbenzenesulfonates in aqueous environmental samples. Anal Chem, **63**: 1179–1182.

Divo C & Cardini G (1980) Primary and total biodegradation of linear alkylbenzene sulphonates. Tenside Surfactants Deterg, **17**: 30–36.

Dolan JM & Hendricks AC (1976) The lethality of an intact and degraded LAS mixture to bluegill sunfish and a snail. J Water Pollut Control Fed, **48**: 2570–2577.

Dolan JM, Gregg BC, Cairns J, Dickson KL, & Henricks AC (1974) The acute toxicity of three new surfactant mixtures to a mayfly larvae. Arch Hydrobiol, **74**: 123–132.

Dominguez JG, Parra JL, Infante MR, Pelejero CM, Balaguer F, & Sastre T (1977) A new approach to the theory of adsorption and permeability of surfactants on keratinic proteins: the specific behaviour of certain hydrophobic chains. J Soc Cosmet Chem (UK), **28**: 165–182.

Dreger EE, Keim GI, Miles GD, Shedlovsky L, & Ross J (1944) Sodium alcohol sulfates: properties involving surface activity. Ind Eng Chem, **36** (7): 610–617.

Dugan PR (1967) Influence of chronic exposure to anionic detergents on toxicity of pesticides to goldfish. J Water Pollut Control Fed, **39**: 63–71.

Dutka BJ & Kwan KK (1982) Application of four bacterial screening procedures to assess changes in the toxicity of chemicals in mixtures. Environ Pollut, **A29**: 125–134.

Eganhouse RP, Blumfield DL, & Kaplan IR (1983) Long-chain alkylbenzenes as molecular tracers of domestic wastes in the marine environment. Environ Sci Technol, **17**: 523–530.

Eggert CR, Kaley RG, & Gledhill WE (1979) Application of a laboratory freshwater lake model in the study of linear alkylbenzene sulfonate (LAS) biodegradation. In: Bourquin AW & Pritchard PH ed. Proceedings of the Workshop on Microbial Degradation of Pollutants in Marine Environments, Pensacola Beach, Florida, 9–14 April 1978. Washington, DC, US Environmental Protection Agency, pp 451-461 (EPA-600/9-79-012).

Endo T, Furuido Y, Namie K, Yamamoto N, Hasunuma H, & Ueda K (1980) [Studies of the chronic toxicity and teratogenicity of synthetic surfactants.] Ann Rep Tokyo Metrop Res Inst Environ Prot [Tokyo Kogai Kenkyujo Nempo]: 236–246 (in Japanese).

Environment Agency Japan (1978) Chemicals in the environment. Tokyo, 81 pp.

Epstein S, Throndson AH, Dock W, & Tainter ML (1939) Possible deleterious effects of using soap substitutes in dentifrices. J Am Dental Assoc, **26**: 1461–1471.

Ernst R, Arditti J, & Healey PL (1971) Biological effects of surfactants I. Influence on the growth of orchid seedlings. New Phytol, **70**: 457–475.

Ernst R, Gonzales CJ, & Arditti J (1983) Biological effects of surfactants: Part 6— effects of anionic, non-ionic and amphoteric surfactants on a green alga (*Chlamydomonas*). Environ Pollut, **A31**: 159–175.

European Committee of Organic Surfactants and Their Intermediates (1990) Classification and labelling of surfactants. Brussels, 38 pp.

Eurostat (1991) Environment statistics. Annex to overview of the state of the environment in the European Communities 1992: COM(92) 23 Final—Volume III. Brussels, Commission of the European Communities, 102 pp.

Evans HC (1956) Alkyl sulphates (Part 1); critical micelle concentrations of the sodium salts. J Soc Cosmet Chem, **7**: 579–586.

Eyanoer HF, Upatham ES, Duangsawasdi M, & Tridech S (1985) Effects of water hardness and temperature on the toxicity of detergents to the freshwater fish, *Puntius gonionotus*, bleeker. J Sci Soc Thailand, **11**: 67–77.

Fairchild JF, Dwyer FJ, La Point T, Burch SA, & Ingersoll CG (1993) Evaluation of a laboratory-generated NOEC for linear alkylbenzene sulfonate in outdoor experimental streams. Environ Toxicol Chem, **12**: 1763–1775.

Federle TW & Pastwa GM (1988) Biodegradation of surfactants in saturated subsurface sediments: a field study. Ground Water, **26**: 761–770.

Federle TW & Schwab BS (1989) Mineralization of surfactants by microbiota of aquatic plants. Appl Environ Microbiol, **55**: 2092–2094.

Federle TW & Schwab BS (1992) Mineralisation of surfactants in anaerobic sediments of a laundromat wastewater pond. Water Res, **26**: 123–127.

Federle TW & Ventullo RM (1990) Mineralization of surfactants by microbiota of submerged plant detritus. Appl Environ Microbiol, **56**: 333–339.

Fendinger NJ, Begley WM, McAvoy DC, & Eckhoff WS (1992a) Determination of alkyl sulfate surfactants in natural waters. Environ Sci Technol, **26**: 2493–2498.

Fendinger NJ, Begley WM, McAvoy DC, & Eckhoff WS (1992b) Monitoring of alkyl sulfate in wastewater treatment systems and surface water. Presented before the Division of Environmental Chemistry, American Chemical Society, San Francisco, April 1992. Washington, DC, American Chemical Society, p 2.

Field JA, Barber EM, Thurman EM, Moore BL, Lawrence DA, & Peake DA (1992) Fate of alkylbenzenesulfonates and dialkyltetralinsulfonates in sewage-contaminated groundwater. Environ Sci Technol, **26** (6): 1140–1148.

Figge K & Schoberl P (1989) LAS and the application of sewage sludge in agriculture. Tenside Surfactants Deterg, **26**: 122–128.

Fischer WK (1980) [Development of detergent concentration in German rivers 1960-1980.] Tenside Surfactants Deterg, **17**: 250–261 (in German).

Fischer WK & Gerike P (1975) Biodegradability determinations via unspecific analyses (chemical oxygen demand, dissolved organic carbon) in coupled units of the OECD confirmatory test—II: Results. Water Res, **9**: 1137–1141.

Fisher LB & Maibach HI (1975) Effect of some irritants on human epidermal mitosis. Contact Dermatitis, **1**: 273–276.

Fitzhugh OG & Nelson AA (1948) Chronic oral toxicity of surface-active agents. J Am Pharm Assoc, **37**: 29–32.

Flecknell PA (1987) Laboratory animal anaesthesia. An introduction for research workers and technicians. Toronto, Academic Press, pp 118–132.

Fogels A & Sprague JB (1977) Comparative short-term tolerance of zebrafish, flagfish, and rainbow trout to five poisons including potential reference toxicants. Water Res, **11**: 811–817.

Froebe CL, Simion FA, Rhein LD, Cagan RH, & Kligman A (1990) Stratum corneum lipid removal by surfactants: relation to *in vivo* irritation. Dermatologica, **181** (4): 277–283.

Fujii T, Sakamoto Y, Abe Y, Mikurita H, Yuzawa K, & Hiraga K (1977) [Pathological examination of rats fed with linear alkylbenzene sulfonate for their lifespan.] Ann Rep Tokyo Metrop Res Lab Public Health, **28** (2): 85–108 (in Japanese).

Fujita H, Kojima A, & Hiraga K (1977) [Screening tests of mutagenicity of linear alkylbenzene sulfonate (LAS) in a microbial system.] Ann Rep Tokyo Metrop Res Lab Public Health, **28** (2): 112–114 (in Japanese).

Fujiwara K, Sugiyama T, Koike K, & Oba K (1975) [Biodegradation of linear alkylate sulfonates in a river model.] Jpn J Hyg, **29**: 552–557 (in Japanese).

Fukuzawa K, Tokumura A, Yamada S, & Tsukatani H (1978) [Oral toxicity of sodium dodecyl sulfate.] Eisei Kagaku, **24** (2): 107–110 (in Japanese).

Gafa S (1974) Studies on relationship between acute toxicity to fish and surface activity of anionic surfactants. Riv Ital Sostanze Grasse, **51**: 182–192.

Gafa S & Lattanzi B (1974) Evaluation of some 'third generation' anionic surfactants in comparison with a linear alkylbenzene sulfonate. 12th World Congress on Soaps. Milan, International Society for Fat Research.

Gagnon MJ (1979) Note on a rapid and sensitive method for the determination of anionic detergents in natural waters at the ppb level. Water Res, **13**: 53–56.

Games LM (1983) Practical applications and comparisons of environmental exposure assessment models. In: Bishop WE, Cardwell RD, & Heidolph BB ed. Aquatic toxicity and hazard assessment: Sixth ASTM Symposium. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 282–299.

Gard-Terech A & Palla JC (1986) Comparative kinetics study of the evolution of freshwater aquatic toxicity and biodegradability of linear and branched alkylbenzene sulfonates. Ecotoxicol Environ Saf, **12**: 127–140.

Gentempo PG, Dickson MK, & Guin KF (1985) Analysis of alpha-olefin sulfonates via quantitative carbon-13 NMR. J Am Oil Chem Soc, **62** (4): 645.

Gerike P (1987) Environmental impact. In: Falbe J ed. Surfactants in consumer products. Berlin, Springer-Verlag, pp 450–474.

Gerike P, Winkler K, Schneider W, & Jakob W (1989) Residual LAS in German rivers. Tenside Surfactants Deterg, **26**: 136–140.

Giger W, Alder AC, Brunner PH, Marcomini A, & Siegrist H (1989) Behaviour of LAS in sewage and sludge treatment and in sludge-treated soil. Tenside Surfactants Deterg, **26**: 95–100.

Gilbert PA & Pettigrew R (1984) Surfactants and the environment. Int J Cosmet Sci, **6**: 149–158.

Gledhill WE (1975) Screening test for assessment of ultimate biodegradability: Linear alkylbenzene sulfonates. Appl Microbiol, **30**: 922–929.

Gloxhuber C (1974) Toxicological properties of surfactants. Arch Toxicol, **32**: 245–269.

Gotte E (1954) [Properties of salts of primary unbranched alkylsulfates and their solutions with regard to their technical use, Part 1.] Fette Seifen Anstrichm, **56** (8): 583–587 (in German).

Granmo A (1972) Development and growth of eggs and larvae of *Mytilus edulis* exposed to a linear dodecylbenzenesulphonate, LAS. Mar Biol, 15: 356–358.

Guhl W (1987) [Biotic similarity indices in the comparison of waters from identical sources.] Limnologica (Berlin), **18** (1): 1–13 (in German).

Haar GT (1983) Safety of alpha olefin sulfonates. J Am Oil Chem Soc, **60** (4): 876–881.

Halvorson H & Ishaque M (1969) Microbiology of domestic wastes; III. Metabolism of LAS-type detergents by bacteria from a sewage lagoon. Can J Microbiol, **15**: 571–576.

Hamano Y, Yamamoto H, Inoue K, Oda Y, Kuwano A, Mitsuda B, & Kunita N (1976) [Studies on the toxicity of commercial detergents (4th report); fetal toxicity (fetal median lethal dose) in mice.] Ann Rep Osaka Prefect Inst Public Health, 7: 147–152 (in Japanese).

Hand VC, Rapaport RA, & Pittinger CA (1990) First validation of a model for the adsorption of linear alkylbenzenesulfonate (LAS) to sediment and comparison to chronic effects data. Chemosphere, **21**: 741–750.

Harrold SP (1959) Denaturation of epidermal keratin by surface active agents. J Invest Dermatol, **32**: 581–588.

Hasegawa H & Sato M (1978) [Permeation of LAS through rat skin.] Tokyo, Science and Technology Agency, Research Cordination Bureau, pp 172–175 (in Japanese).

Hashimoto S, Sakurai K, & Nagai T (1976) [Determination of trace amounts of alkylbenzene sulfonate in the river water by high performance liquid chromatography.] Bunseki Kagaku, **25**: 639–643 (in Japanese).

Hatton EH, Fosdick LS, & Calandra J (1940) The toxicity and rubefacient action of sulphated higher alcohols. J Dent Res, **19**: 87–92.

Hattori M, Seno K, Harada S, Ishizu Y, & Goto M (1984) [Effects of some environmental chemicals in the Daphnia reproduction test.] Jpn Ecol Chem, **6**: 23–27 (in Japanese).

Havermann H & Menke KH (1959) [Biological study of the water-soluble surface-active substances.] Fette Seifen Anstrichm, **61** (6): 429–434 (in German).

Hayashi M (1980) [Toxicological studies on detergents containing linear alkylbenzene sulfonate (LAS) in rats and mice.] Shikoku Med J, **36** (1): 37–44 (in Japanese).

Healey PL, Ernst R, & Arditti J (1971) Biological effects of surfactants: II. Influence on the ultrastructure of orchid seedlings. New Phytol, **70**: 477–482.

Health Canada (in press) Canadian Environmental Protection Act. Human health risk assessment for priority substances; Appendix E. Reference values for intakes and body weights of laboratory animals. Ottawa, Health and Welfare Canada.

Hellmann H (1978) [Anionic detergents in the Rhine 1971-1977, their degradation and biodegradation potential of the river.] Tenside Surfactants Deterg, **15**: 291–294 (in German).

Henderson C, Pickering QH, & Cohen JM (1959) The toxicity of synthetic detergents and soaps to fish. Sew Ind Wastes, **31**: 295–306.

Hennes EC & Rapaport RA (1989) Calculation and analytical verification of LAS concentrations in surface waters, sediment and soil. Tenside Surfactants Deterg, **26**: 141–147.

Hewin International Inc. (1992) The technology and markets for anionic surfactants: North America, western Europe and Japan in the global perspective 1991–1995 and scenario 2000. Amsterdam, Hewin International Inc., 53 pp.

Heywood R, James RW, & Sortwell RJ (1978) Toxicology studies of linear alkylbenzene sulphonate (LAS) in rhesus monkeys; (I) simultaneous oral and subcutaneous administration for 28 days. Toxicology, **11**: 245–250.

Hidaka H & Tatsukawa R (1989) Avoidance by olfaction in a fish, medaka (*Oryzias latipes*), to aquatic contaminants. Environ Pollut, **56**: 299–309.

Hidaka H, Suga M, & Tatsukawa R (1984) [Avoidance of anionic surfactants in medakas (*Oryzias latipes*).] Nippon Nogeikagaku Kaishi, **58**: 1–7 (in Japanese).

Hidaka H, Kubota H, Gratzel M, Serpone N, & Pelizzetti E (1985) Photodegradation of surfactants;I. Degradation of sodium dodecylbenzene sulfonate in aqueous semiconductor dispersions. Nouv J Chim, **9**: 67–69.

Hidu H (1965) Effects of synthetic surfactants on the larvae of clams (*M. mercenaria*) and oysters (*C. virginica*). J Water Pollut Control Fed, **37**: 262–270.

Higuchi K, Shimoishi Y, Miyata H, & Toei K (1982) Spectrophotometric determination of sodium 4-dodecylbenzenesulfonate with 1-(N-methylpyridinium-4-yl-azo)-4-(4-diethylaminophenylazo)naphthalene iodide. Bull Chem Soc Jpn, **55** (2): 621–622.

Hironaga K (1979) [Case reports; 29 emergency cases of children (non-fatal cases of drinking household products).] Yakuji Shinpo, **36**: 27–29 (in Japanese).

Hirsch E (1963) [Structure of alkylbenzenesulfonates and impact on behaviour of fish.] Vom Wasser, 30: 249–259 (in German).

Hitchcock WS & Martin DF (1977) Effects and fate of a surfactant in cultures of the red tide organisms, *Gymnodinium breve*. Bull Environ Contam Toxicol, **18**: 291–296.

Hokanson KEF & Smith LL (1971) Some factors influencing toxicity of linear alkylate sulfonate (LAS) to the bluegill. Trans Am Fish Soc, **100**: 1–12.

Hollis CG, Phillips WK, & Johnson JE (1976) Thermal effects on biodegradation of linear alkyl benzenesulfonate. Dev Ind Microbiol, **17**: 345–350.

Holman WF & Macek KJ (1980) An aquatic safety assessment of linear alkylbenzene sulfonate (LAS): chronic effects on fathead minnows. Trans Am Fish Soc, **109**: 122–131.

Holst R & Moller H (1975) One hundred twin pairs patch tested with primary irritants. Br J Dermatol, **93**: 145–149.

Holt MS, Matthijs E, & Waters J (1989) The concentrations and fate of linear alkylbenzene sulphonate in sludge amended soils. Water Res, **23**: 749–759.

Holysh M, Paterson S, Mackay D, & Bandurraga MM (1986) Assessment of the environmental fate of linear alkylbenzenesulphonates. Chemosphere, **15**: 3–20.

Hong SD, Lee SH, Hah SC, & Ha HP (1984) [Biodegradation of synthetic detergents by *Klebsiella pneumoniae* L-127.] Korean J Appl Microbiol Bioeng, **12**: 91–97 (in Korean).

Hon-Nami H & Hanya T (1980a) Linear alkylbenzene sulfonates in river, estuary and bay water. Water Res, **14**: 1251–1256.

Hon-Nami H & Hanya T (1980b) Differences in the composition of linear alkylbenzene sulfonate homologues in river sediment and river water. Jpn J Limnol, **41**: 1–4.

Hope J (1977) Absence of chromosome damage in the bone marrow of rats fed detergent actives for 90 days. Mutat Res, **56**: 47–50.

Howes D (1975) The percutaneous absorption of some anionic surfactants. J Soc Cosmet Chem, **26**: 47–63.

Hrsak D, Bosnjak M, & Johanides V (1981) Kinetics of linear alkylbenzene sulphonate and secondary alkane sulphonate biodegradation. Tenside Surfactants Deterg, **18**: 137–140.

Hrsak D, Bosnjak M, & Johanides V (1982) Enrichment of linear alkylbenzenesulphonate (LAS) degrading bacteria in continuous culture. J Appl Bacteriol, **53**: 413–422.

Hsu YC (1965) Detergent-splitting enzyme from pseudomonas. Nature, **207**: 385–388.

Huber L (1989) Conclusions for an ecological evaluation of LAS. Tenside Surfactants Deterg, **26** (2): 71–74.

Huddleston RL & Allred RC (1963) Microbial oxidation of sulfonated alkylbenzenes. In: Vol 4. Developments in industrial microbiology. Washington, DC, American Institute of Biological Sciences, pp 24–38.

Huddleston RL & Nielsen AM (1979) LAS biodegradation: the fate of the aromatic ring. Soap Cosmet Chem Spec, March: 34–44.

Hunter B & Benson HG (1976) Long-term toxicity of the surfactant alpha-olefin sulphonate (AOS) in the rat. Toxicology, **5**: 359–370.

Husmann W (1968) Solving the detergent problem in Germany. Water Pollut Control, **3**: 80–90.

Ichihara Y, Yoshiue S, Omure K, Fujimoto T, Moritomo M, & Yoshikawa J (1967) [A case study of anionic surfactant poisoning.] J Jpn Soc Int Med [Nichi Naikaishi], **56** (11): 128 (in Japanese).

Ihara I, Ozaki M, Nakamura F, Yokota I, Nakaura H, Aso T, Ozaki M, Kaneko Y, Miyauchi S, Miura K, Hayakawa Y, Moriyama S, Ishiguro T, & Yoshida Y. (1970) [ABS in the water of the Tama River; V. Data from February 1968 to May 1970.] J Jpn Oil Chem Soc, **19**: 1043–1048 (in Japanese).

Iimori M & Takita Y (1979) [A study of the effect of linear alkylbenzenesulfonate on fish.] J Jpn Oil Chem Soc, **28**: 185–189 (in Japanese).

Iimori M & Ushiyama S (1971) [Serological studies on alpha-olefin sulfonate protein complexes.] J Jpn Oil Chem Soc, **20** (2): 24–26 (in Japanese).

Iimori M, Ogata T, & Kudo K (1972) [Eye irritation testing of surface active agents in experimental animals.] J Jpn Oil Chem Soc, **21** (6): 334–337 (in Japanese).

Iimori M, Inoue S, & Yano K (1973) [Effects of skin application of detergents on pregnant mice.] J Jpn Oil Chem Soc [Yukagaku], **22** (12): 807–813 (in Japanese).

Ikawa M, Yoneyama M, Nakao T, & Hiraga K (1978) [Uptake of organic acid and organic base by renal cortical slices of rats treated with LAS and ABS.] Ann Rep Tokyo Metrop Res Lab Public Health., **29** (2): 51–54 (in Japanese).

Ikeda Y (1965) [Food additives and synthetic detergents.] Sogo-Rinsyo, **14** (4): 621–626 (in Japanese).

Imahori A, Kitagawa T, & Shiobara S (1976) [Effects of linear alkylbenzene sulfonate (LAS) applied dermally to pregnant mice on the pregnant mice and their fetuses.] Jpn J Public Health [Nippon Koshueisei Zasshi], **23** (2): 68–72 (in Japanese).

Imokawa G & Katsumi M (1976) [Denaturing action of typical anionic surfactants on several proteins.] J Jpn Oil Chem Soc [Yukagaku], **25** (1): 24–30 (in Japanese).

Imokawa G & Mishima Y (1976) [The cumulative effect of surface active agents on human cutaneous horny layers.] Nichihikaishi, **86** (8): 473–481 (in Japanese).

Imokawa G & Mishima Y (1979) Cumulative effect of surfactants on cutaneous horny layers: lysosome labilizing action. Contact Dermatitis, **5**: 151–162.

Imokawa G, Sumura K, & Katsumi M (1974) [A correlation between adsorption of a surfactant onto callus and resulting skin-roughness.] J Jpn Oil Chem Soc [Yukagaku], **23** (11): 719–725 (in Japanese).

Imokawa G, Sumura K, & Katsumi M (1975a) A study of skin roughness caused by surfactants: (I) A new method *in vivo* for evaluation of skin roughness. J Am Oil Chem Soc, **52**: 479–483.

Imokawa G, Sumura K, & Katsumi M (1975b) Study of skin roughness caused by surfactants: (II) Correlation between protein denaturation and skin roughness. J Am Oil Chem Soc, **52**: 484–489.

Inaba K & Amano K (1988) HPLC determination of linear alkylbenzenesulfonate (LAS) in the aquatic environment. Seasonal changes in LAS concentration in polluted lake water and sediment. Int J Environ Anal Chem, **34**: 203–213.

Inoue K & Masuda F (1976) Effects of detergents on mouse fetuses. J Jpn Food Hgy Soc [Shoku Eishi], **17** (2): 158–169.

Inoue K & Sunakawa T (1979) [Mutagenicity tests of surfactants.] Jpn Fragr J, **38**: 67–75 (in Japanese).

Inoue K, Shibata T, Hamano Y, Oda Y, Kuwano A, Yamamoto H, Mitsuda B, & Kunita N. (1977) [*In vivo* cytogenetic tests of some synthetic detergents in mice.] Ann Rep Osaka Prefect Inst Public Health, **8**: 17–24 (in Japanese).

Inoue K, Kaneko K, & Yoshida M (1978) Adsorption of dodecylbenzenesulfonates by soil colloids and influence of soil colloids on their degradation. Soil Sci Plant Nutr, **24**: 91–102.

Inoue K, Sunakawa T, & Takayama S (1979) [Studies of *in vitro* cell transformation and mutagenicity by surfactants and other compounds.] J Jpn Soc Cosmet Chem [Shogishi], **13** (2): 50–60 (in Japanese).

Inoue K, Sunakawa T, & Takayama S (1980) Studies of *in vitro* cell transformation and mutagenicity by surfactants and other compounds. Food Cosmet Toxicol, **18**: 289–296.

Inoue S, O'Grodnick JS, & Tomizawa S (1982) Metabolism of alpha-olefin sulfonate (AOS) in rats. Fundam Appl Toxicol, **2**: 130–138.

Ishihara M & Kinebuchi S (1967) [Causes of hand eczema, and their examination methods.] Rinsho Hifuka, **21** (3): 241–251 (in Japanese).

Ito T & Kakegawa S (1972) [Development of mild detergent formulations.] Baioteku, **3** (2): 157–164 (in Japanese).

Ito R, Kawamura H, Chang HS, Kudo K, Kajiwara S, Toida S, Seki Y, Hashimoto M, & Fukushima A (1978) [Acute, subacute and chronic toxicity of magnesium linear alkylbenzene sulfonate (LAS-Mg).] J Med Soc Toho Univ [Toho Igakukai Zasshi], **25** (5-6): 850–875 (in Japanese).

Itoh S-I, Setsuda S, Utsonomiya A & Naito S (1979) [Studies on the biodegradation test method for chemical substances: II. Ultimate biodegradabilities of some anionic, nonionic, and cationic surfactants estimated by CO_2 production.] J Jpn Oil Chem Soc [Yukagaku], **28**: 199–204 (in Japanese).

Itoh S, Naito S, & Unemoto T (1987) [Comparative studies on anaerobic biodegradation of anionic and nonionic surfactants.] Eisei Kagaku, **33**: 415–422 (in Japanese).

Janicke W (1971) On the behaviour of synthetic organic substances during the treatment of waste liquors— IV. Progress of the biodegradation of 'soft' anionic synthetic detergents. Water Res, **5**: 917–931.

Janicke W & Hilge G (1979) [Biodegradability of anionic/cationic surfactant complexes under the aerobic and anaerobic conditions of effluent and sludge treatment.] Tenside Surfactants Deterg, **16**: 117–122 (in German).

Janicke W & Niemitz W (1973) [Comparative investigations of detergent raw materials and commercial detergents and biological sewage treatments.] Vom Wasser, **40**: 369–389 (in German).

Japanese Soap & Detergent Association (1992) [Annual report of water quality in the environment.] Tokyo, Japanese Soap and Detergent Association (in Japanese).

Javed S, Misra V, & Viswanathan PN (1988) In vitro studies on the effect of linear alkyl benzene sulphonate on serum albumin. Int J Cosmet Sci, **10**: 241–246.

Jimenez L, Breen A, Thomas N, Federle TW, & Sayler GS (1991) Mineralization of linear alkylbenzene sulfonate by a four-member aerobic bacterial consortium. Appl Environ Microbiol, **57**: 1566–1569.

Jones JH (1945) General colorimetric method for determination of small quantities of sulfonated or sulfated surface active compounds. J Assoc Off Agric Chem, **28** (2): 398–408.

Kantin R, Baumgarten MGZ, Cabeda M, Beaumord AC, & de Almeida TL (1981) Concentration of anionic detergents in Rio Grande water (South Brazil). Mar Pollut Bull, **12**: 50–53.

Katsuno T, Tsukioka T, & Kono Y (1983) [LAS in river sediments, algae and water plants.] Bull Nagano Res Inst Health Pollut, **6**: 23–25 (in Japanese).

Katz BM & Cohen GM (1976) Toxicities of excessively chlorinated organic compounds. Bull Environ Contam Toxicol, **15** (6): 644–650.

Kawamura T, Sasagawa S, Masuda T, Honda S, Kinoshita M, Harada S, Ishizaki T, Nagai R, Hirokawa K, Anzai T, Anekoji K, Hidano A, Kawano T, Ikegami I, Sato S, & Aoyama T (1970) [Basic studies on the standardization of a skin patch test.] Jpn J Dermatol [Nihon Hifu Kagakukai Zasshi], **80** (5): 301–314 (in Japanese).

Kawashima K & Takeno T (1982) [Fate of linear alkylbenzenesulfonates (^{14}C-LAS-Ca, ^{14}C-LAS-Na) in soils and plants.] J Jpn Oil Chem Soc [Yukagaku], **31**: 944–948 (in Japanese).

Kay JH, Kohn FE, & Calandra JC (1965) Subacute oral toxicity of a biodegradable, linear alkylbenzene sulfonate. Toxicol Appl Pharmacol, **7**: 812–818.

Kikuchi K (1978) Observations on some tissues of mice injected with LAS synthetic detergent for varying periods from the day of birth. Kawasaki Med J, **4** (3): 193–201.

Kikuchi M (1985) [Biodegradation of some surfactants in river water with relation to chemical structure and water temperature.] Bull Jpn Soc Sci Fish, **51**: 1859–1864 (in Japanese).

Kikuchi M & Wakabayashi M (1984) Lethal response of some surfactants to medaka (*Oryzias latipes*) with relation to chemical structure. Bull Jpn Soc Sci Fish, **50**: 1235–1240.

Kikuchi M, Wakabayashi M, Nakamura T, Inoue W, Takahashi K, Kawana T, Kawahara H, & Koido Y (1976) A study of detergents. II. Acute toxicity of anionic surfactants on aquatic organisms. Ann Rep Tokyo Metrop Res Inst Environ Prot: 57–69.

Kikuchi M, Wakabayashi M, Kojima H, & Yoshida T (1978) Uptake, distribution, and elimination of sodium alkyl sulfate in carp. Ecotoxicol Environ Saf, **2**: 115–127.

Kikuchi M, Tokai A, & Yoshida T (1986) Determination of trace levels of linear alkylbenzenesulfonates in the marine environment by high-performance liquid chromatography. Water Res, **20** (5): 643–650.

Kimerle RA & Swisher RD (1977) Reduction of aquatic toxicity of linear alkylbenzene sulfonate (LAS) by biodegradation. Water Res, **11**: 31–37.

Kimerle RA, Macek KJ, Sleight BH, & Burrows ME (1981) Bioconcentration of linear alkylbenzene sulfonate (LAS) in bluegill (*Lepomis macrochirus*). Water Res, **15**: 251–256.

Kishi M, Satoh S, Horiguchi, Y, & Ito K (1984) [Effects of surfactants on bone marrow cells.] Bull Kanagawa Public Health Lab, **14**: 57–58 (in Japanese).

Kligman AM & Wooding WM (1967) A method for the measurement and evaluation of irritants on human skin. J Invest Dermatol, **49** (1): 78–94.

Knaebel DB, Federle TW, & Vestal JB (1990) Mineralization of linear alkylbenzene sulfonate LAS) and linear alcohol ethoxylate (LAE) in 11 contrasting soils. Environ Toxicol Chem, **9**: 981–988.

Knopp PV, Uhren LJ, Rohlich GA, & Nichols MS (1965) Field study of the removal of linear alkylate sulfonate detergents by the activated sludge process. J Am Oil Chem Soc, **42**: 867–873.

Kobayashi H, Ichikawa H, Fujii T, Yano N, Konno T, Hiraga K, Nakamura H, Watanabe Y, & Mimura S (1972) [Studies on toxicity of synthetic detergents. (I) Acute oral toxicity of linear and branched alkyl benzene sulfonate.] Ann Rep Tokyo Metrop Res Lab Public Health, **24**: 397–408 (in Japanese).

Kobayashi T, Adachi A, Iida H, Oba K, Oda K, Okumura H, Tsuji K, Numata H, & Morimoto Y (1986) [Collaborative studies on determination of anionic surfactants in water using Co-PADAP.] Hyg Chem [Eisei Kagaku], **32** (5): 391–396 (in Japanese).

Kobuke Y (1985) [Concentration of linear alkylbenzene sulfonates (LAS) and its composition in the river waters of Hyogo Prefecture.] Jpn J Limnol [Rikusuigaku Zasshi], **46**: 279–286 (in Japanese).

Koizumi N, Ninomiya R, Inoue Y, Tsukamoto T, Fujii M, & Yamamoto Y (1985) Implantation disturbance studies with linear alkylbenzene sulphonate in mice. Arch Environ Contam Toxicol, **14**: 73–81.

Kojima S (1989) [Pollution by fluorescent whitening agents (FWA) and linear alkylbenzene sulfonates (LAS) in environmental water.] Ann Rep Environ Pollut Res Inst Nagoya, **19**: 61–68 (in Japanese).

Kondo M, Yamamoto H, & Arakawa Y (1983) [Effects of detergents on algae growth and physiological activities.] Bull Aichi Environ Center, **11**: 10–21 (in Japanese).

Konno R & Wakabayashi M (1987) [Effects of chemical compounds on the growth of planktonic algae.] Ann Rep Tokyo Metrop Res Inst Environ Protect [Tokyo Kogai Kenkyujo Nempo]: 113–116 (in Japanese).

Kravetz LH, Chung JC, & Rapean JC (1982) Ultimate biodegradation studies of alpha olefin sulfonates. J Am Oil Chem Soc, **59** (4): 206–210.

Krishna Murti GSR, Volk VV, & Jackson ML (1966) Soil adsorption of linear alkylate sulfonate. Soil Sci Soc Am Proc, **30**: 685–688.

Kutt E & Martin DF (1977) Effect of selected surfactants on the growth characteristics of *Gymnodinium breve*. Mar Biol, **28**: 253–259.

Kuwano A, Yamamoto H, Inoue K, Hamano Y, Oda Y, Mitsuda B, & Kunita N (1976) [Studies on toxicity of commercial detergents (2nd report). Acute toxicity in mice.] Ann Rep Osaka Prefect Inst Public Health, **7**: 137–140 (in Japanese).

Ladle M, House WA, Armitage PD, & Farr IS (1989) Faunal characteristics of a site subject to sewage plant discharge. Tenside Surfactants Deterg, **26**: 159–168.

Lal H, Misra V, Viswanathan PN, & Krishna Murti CR (1983) Comparative studies on ecotoxicology of synthetic detergents. Ecotoxicol Environ Saf, **7**: 538–545.

Lal H, Misra V, Viswanathan PN, & Krishna Murti CR (1984a) The water flea (*Daphnia magna*) as a sensitive indicator for the assessment of toxicity of synthetic detergents. Ecotoxicol Environ Saf, **8**: 447–450.

Lal H, Misra V, Viswanathan PN, & Krishna Murti CR (1984b) Effect of synthetic detergents on some of the behavioral patterns of fish fingerlings (*Cirrhina mrigala*) and its relation to ecotoxicology. Bull Environ Contam Toxicol, **32**: 109–115.

Larson RJ (1990) Structure-activity relationships for biodegradation of linear alkylbenzenesulfonates. Environ Sci Technol, **24**: 1241–1246.

Larson RJ & Maki AW (1982) Effect of linear alkylbenzene sulfonate on the structure and function of microbial communities in model ecosystems. In: Pearson JG, Foster RB, & Bishop WE ed. Aquatic toxicology and hazard assessment. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 120–136 (ASTM STP 766).

Larson RJ & Payne AG (1981) Fate of the benzene ring of linear alkylbenzene sulphonate in natural waters. Appl Environ Microbiol, **41**: 621–627.

Larson RJ, Federle TW, Shimp RJ, & Ventullo RM (1989) Behaviour of linear alkylbenzene sulfonate (LAS) in soil infiltration and groundwater. Tenside Surfactants Deterg, **26**: 116–121.

Laumond F, Neuberger M, Oonier B, Fourey A, Bittel R, & Aubert M (1973) Experimental investigations at the laboratory on the transfer of mercury in marine trophic chains. Rev Int Oceanogr Med, **31/32**: 47–53.

Lay JP, Klein W, & Korte F (1983) Elimination and biodistribution studies of [^{14}C]dodecylbenzene sulfonate in rats, following low dosing in the daily diet and a single i.p. administration. Toxicol Lett, **17**: 187–192.

Lee S-M & Park C-K (1984) [Acute toxicity of onchean stream water to the sea urchin, *Hemicentrotus pulcherrimus*.] Bull Korean Fish Soc, **17**: 414–422 (in Korean).

Leidner H, Gloor R, & Wuhrmann K (1976) [The kinetics of degradation of linear alyklbenzene sulfonate.] Tenside Surfactants Deterg, **13**: 122–130 (in German).

Leo AJ & Hansch C (1979) Substituent constants for correlation. Analysis in chemistry and biology. New York: Wiley, 98 pp.

Lewis MA (1983) Effect of loading density on the acute toxicities of surfactants, copper, and phenol to *Daphnia magna* Straus. Arch Environ Contam Toxicol, **12**: 51–55.

Lewis MA (1986) Comparison of the effects of surfactants on freshwater phytoplankton communities in experimental enclosures and on algal population growth in the laboratory. Environ Toxicol Chem, 5: 319–332.

Lewis MA & Hamm BG (1986) Environmental modification of the photosynthetic response of lake plankton to surfactants and significance to a laboratory-field comparison. Water Res, 20: 1575–1582.

Lewis MA & Suprenant D (1983) Comparative acute toxicities of surfactants to aquatic invertebrates. Ecotoxicol Environ Saf, 7: 313–322.

Lewis MA, Pittinger CA, Davidson DH, & Ritchie CJ (in press) In-situ response of natural periphyton to an anionic surfactant and an environmental risk assessment for phytotoxic effects. Environ Toxicol Chem.

Lichtenstein EP (1966) Increase of persistence and toxicity of parathion and diazinon in soils with detergents. J Econ Entomol, 59: 985–993.

Lichtenstein EP, Fuhremann TW, Scopes NEA, & Skrentny RF (1967) Translocation of insecticides from soils into pea plants. Effects of the detergent LAS on translocation and plant growth. J Agric Food Chem, 15: 864–869.

Litovitz TL, Schmitz BF, & Bailey KM (1990) 1989 Annual Report of the American Association of Poison Control Centers National Data Collection System. Am J Emerg Med, 8: 394–445.

Litz N, Doering HW, Thiele M, & Blume H-P (1987) The behavior of linear alkylbenzenesulfonate in different soils: A comparison between field and laboratory studies. Ecotoxicol Environ Saf, 14: 103–116.

Longwell J & Maniece WD (1955) Determination of anionic detergents in sewage, sewage effluents and river waters. Analyst, 80: 167–171.

Lopez-Zavala A, Aluja AS de, Elias BL, Manjarrez L, Buchmann A, Mercado L, & Caltenco S (1975) The effects of the ABS, LAS and AOS detergents on fish, domestic animals and plants. Prog Water Technol, 7: 73–82.

Maag H, Praun F, & Schoberl P (1975) [Recent discoveries in the olefin sulfonate field. I. Effect of carbon number and branching of the alkyl chain on the application and environmental characteristics of olefin sulfonates.] Tenside Surfactants Deterg, 12: 11–15 (in German).

Maki AW (1979a) Correlations between *Daphnia magna* and fathead minnow (*Pimephales promelas*) chronic toxicity values for several classes of test substance. J Fish Res Board Can, 36: 411–421.

Maki AW (1979b) Respiratory activity of fish as a predictor of chronic fish toxicity values for surfactants. In: Marking LL & Kimerle RA ed. Aquatic toxicology. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 77–95 (ASTM STP 667).

Maki AW & Bishop WE (1979) Acute toxicity studies of surfactants to *Daphnia magna* and *Daphnia pulex*. Arch Environ Contam Toxicol, **8**: 599–612.

Mancini P, Bellandi S, La Conca P, & Pantani F (1984) [Microdetermination of anionic surface active agents in drinking water and in waters that can be made potable.] Riv Merceol, **23**: 143–149 (in Italian).

Mann H (1976) [Golden orfe fish comparative testing of acute toxicity of substances in water and wastewater: results of a ring test.] Wasser Abwasser-Forsch, **9**: 103–109 (in German).

Mann AH & Reid VW (1971) Biodegradation of synthetic detergents evaluation by community trials: I. Linear alkylbenzene sulphonates. J Am Oil Chem Soc, **48**: 588–594.

Marchesi JR, House WA, White GF, Russell NJ, & Farr IS (1991) A comparative study of the adsorption of linear alkyl sulphates and alkylbenzene sulphonates on river sediments. Colloids Surf, **53**: 63–78.

Marcomini A & Giger W (1987) Simultaneous determination of linear alkylbenzenesulfonates, alkylphenol polyethoxylates, and nonylphenol by high-performance liquid chromatography. Anal Chem, **59**: 1709–1715.

Marin MG, Bressan M, & Brunetti R (1991) Effects of linear alkylbenzene sulphonate (LAS) on two marine benthic organisms. Aquat Toxicol, **19**: 241–248.

Marquis DM, Sharman SH, House R, & Sweeney WA (1966) Alpha olefin sulfonates from a commercial SO_3-air reactor. J Am Oil Chem Soc, **43**: 607–614.

Martinez J, Vives-Rego J, & Sanchez-Leal J (1989) The effect of chemical structure and molecular weight of commercial alkylbenzenes on the toxic response of *Daphnia* and naturally occurring bacteria in fresh and seawater. Water Res, **23**: 569–572.

Martinez J, Soto Y, Vives-Rego J, & Bianchi M (1991) Toxicity of Cu, Ni and alkylbenzene sulfonate (LAS) on the naturally occurring bacteria in the Rhone River plume. Environ Toxicol Chem, **10**: 641–647.

Masubuchi M, Takahashi A, Takahashi O, & Hiraga K (1976) [Cytogenetic studies and dominant lethal tests with long term administration of butylated hydroxytoluene (BHT) and linear alkylbenzene sulfonate (LAS) in mice and rats.] Ann Rep Tokyo Metrop Res Lab Public Health, **27** (2): 100–104 (in Japanese).

Masuda F & Inoue K (1974) [Effects on fetuses of linear alkylbenzene sulfonate administered subcutaneously to pregnant mice.] Congenit Anom [Senten Ijo], **14** (4): 309–318 (in Japanese).

Masuda F, Okamoto K, & Inoue K (1973) [Effects on fetuses of linear alkylbenzene sulfonate applied to mouse maternal skin during pregnancy.] J Food Hyg Soc Jpn [Shokuhin Eiseigaku Zasshi], **14** (6): 580–582 (in Japanese).

Masuda F, Okamoto K, & Inoue K (1974) [Effects of linear alkylbenzene sulfonate applied dermally to pregnant mice on fetal development.] J Food Hyg Soc Jpn [Shokuhin Eiseigaku Zasshi], **15** (5): 349–355 (in Japanese).

Mathur AK, Narang S, Gupta BN, Singh A, Singh S, & Shanker R (1992) Effect of dermal exposure to LAS detergent and HCH pesticide in guinea pigs: biochemical and histopathologic changes in liver and kidney. J Toxicol Cutan Ocular Toxicol, **11** (1): 3–13.

Matsueda T, Osaki Y, & Shigee S (1982) [Determination of alkylbenzenesulfonate in water by high-performance liquid chromatography.] Jpn Anal (Bunseki Kagaku), **31**: 59–62 (in Japanese).

Matsuura T & Smith JM (1970) Kinetics of photodecomposition of dodecyl benzene sulfonate. Ind Eng Chem Fundam, **9**: 252–260.

Matthijs E & De Henau H (1987) Determination of LAS. Tenside Surfactants Deterg, **24**: 193–199.

Maurer EW, Cordon TC, & Stirton AJ (1971) Microaerophilic biodegradation of tallow-based anionic detergents in river water. J Am Oil Chem Soc, **48**: 163–165.

Mayer FL (1987) Acute toxicity handbook of chemicals to estuarine organisms. Washington, DC, US Environmental Protection Agency, p 178 (EPA/600/8-87/017; PB-87-188686).

McAvoy DC, Eckhoff WS, & Rapaport RA (1993) Fate of linear alkylbenzene sulfonate in the environment. Environ Toxicol Chem, **12**: 977–987.

McCutcheon (1989) McCutcheon's emulsifiers and detergents: International edition. Glen Rock, New Jersey, McCutcheon's Division, Manufacturing Confectioner Publishing Co., 240 pp.

McCutcheon (1993) Volume 1: McCutcheon's emulsifiers & detergents: North American edition-International edition. Glen Rock, New Jersey, McCutcheon's Division, Manufacturing Confectioner Publishing Co. 295 pp.

McEvoy J & Giger W (1985) Accumulation of linear alkylbenzenesulphonate surfactants in sewage sludges. Naturwissenschaften, **72**: 429–431.

McEvoy J & Giger W (1986) Determination of linear alkylbenzenesulfonates in sewage sludge by high-resolution gas chromatography/mass spectrometry. Environ Sci Technol, **20**: 376–383.

McKim JM, Arthur JW, & Thorslund TW (1975) Toxicity of a linear alkylate sulfonate detergent to larvae of four species of freshwater fish. Bull Environ Contam Toxicol, **14**: 1–7.

Merits I (1975) The metabolism of labelled hexadecyl sulphate salts in the rat, dog and human. Biochem J, 148: 219–228.

Michael WR (1968) Metabolism of linear alkylate sulfonate and alkyl benzene sulfonate in albino rats. Toxicol Appl Pharmacol, 12: 473–485.

Mieure JP, Waters J, Holt MS, & Matthijs E (1990) Terrestrial safety assessment of linear alkylbenzene sulfonate. Chemosphere, 21: 251–262.

Mikami Y, Nagai H, Sakai Y, Fukushima S, & Nishino T (1969) [Effects of a detergent on the development of the mouse embryo.] Congenit Anom (Senten Ijo), 9: 230 (in Japanese).

Minegishi K, Osawa M, & Yamaha T (1977) Percutaneous absorption of alpha-olefin sulfonate (AOS) in rats. Chem Pharm Bull, 25 (4): 821–825.

Ministry of Health and Welfare (1992) [Trend analysis of water works in Japan.] J Jpn Water Works Assoc, 61: 41–78 (in Japanese).

Misra V, Lal H, Viswanathan PN & Krishna Murti CR (1984) ^{45}Ca uptake from water by snails (*Lymnaea vulgaris*) in control and detergent-polluted samples. Ecotoxicol Environ Saf, 8: 97–99.

Misra V, Lal H, Chawla G, & Viswanathan PN (1985) Pathomorphologic changes in gills of fish fingerlings (*Cirrhina mrigala*) by linear alkyl benzene sulfonate. Ecotoxicol Environ Saf, 10: 302–308.

Misra V, Chawla G, Kumar V, Lal H, & Viswanathan PN (1987) Effect of linear alkyl benzene sulfonate in skin of fish fingerlings (*Cirrhina mrigala*): Observations with scanning electron microscope. Ecotoxicol Environ Saf, 13: 164–168.

Misra V, Javed S, Pandey DP, & Viswanathan PN (1989a) Biochemical and pathomorphologic studies for safety evaluation of synthetic detergents. J Toxicol Cutan Ocular Toxicol, 8: 271–278.

Misra V, Kumar V, Pandey SD, & Viswanathan PN (1989b) Uptake and distribution of ^{203}Hg by fish fingerlings, *Cirrhina mrigala*, exposed to linear alkyl benzene sulphonate. Bull Environ Contam Toxicol, 43: 139–143.

Misra V, Kumar V, Pandey SD, & Viswanathan PN (1991) Biochemical alterations in fish fingerlings (*Cyprinus carpio*) exposed to sublethal concentration of linear alkyl benzene sulphonate. Arch Environ Contam Toxicol, 21: 514–517.

Mitchell GC & Holt MS (1993) The fate and effects of linear alkylbenzene sulphonate (LAS) in outdoor artificial streams. Presented at the First SETAC World Congress: Ecotoxicology and Environmental Chemistry—A Global Perspective, Lisbon, Portugal, 28–31 March 1993. New York, Society of Environmental Toxicology and Chemistry.

Miura KIV, Suzuki K, Tsuchikura A, Baba Y, Hayakana T, Hayashi S, Moriyama T, Ishiguro T, & Yoshida Y (1968) [Alkylbenzene sulphonate in the water of the Tama River.] J Jpn Oil Chem Soc [Yukagaku], 17: 635–637 (in Japanese).

Miura K, Yamanaka K, Sangai T, Yoshimura K, & Hayashi N (1979) [Application of the biological oxygen consumption measurement technique in the biodegradation testing of surfactants.] J Jpn Oil Chem Soc [Yukagaku], **28** (5): 351–355 (in Japanese).

Moreno A, Ferrer J, & Berna JL (1990) Biodegradability of LAS in a sewer system. Tenside Surfactants Deterg, **27** (5): 312–315.

Motomizu S, Fujiwara S, Fujiwara A, & Toei K (1982) Solvent extraction-spectrophotometric determination of anionic surfactants with ethyl violet. Anal Chem, **54**: 392–397.

Motoyama S & Mukai H (1981) [Synthetic detergents in the environment.] Niigota Rikagaku, **7**: 21-29 (in Japanese).

Nakae A, Tsuji K, & Yamanaka M (1980) Determination of trace amounts of alkylbenzenesulfonates by high-performance liquid chromatography with fluorimetric detection. Anal Chem, **52** (14): 2275–2277.

Nakahara M, Ishibashi M, Asao T, Tsukamoto T, Shinagawa K, Kinoshita Y, Yamamoto H, Mitsuda B, & Kunita N (1976) [Studies on toxicity of commercial detergents; 1st report.] Ann Rep Osaka Prefect Inst Public Health, **7**: 131–135 (in Japanese).

Nakamura E, Fujisawa I, & Namiki H (1983) [Determination of anionic surfactant in water by indirect atomic absorption spectrometry after solvent extraction with potassium-dibenzo-18-crown-6.] Jpn Anal [Bunseki Kagaku], **32**: 332–336 (in Japanese).

Nielsen AM & Huddleston RL (1981) Ultimate biodegradation of linear alkylbenzene sulfonate alkyl and ring carbon. Dev Ind Microbiol, **22**: 415–424.

Nielsen AM, Meyers JD, Bleckmann CA, & Huddleston RL (1980) LAS biodegradation: Ultimate fate of alkyl and ring carbon. Househ Pers Prod Ind, March: 68–87.

Nishimura H (1976) [Could skin application of cleansing detergents be teratogenic cause in successive joint studies and interpretation of the results?] Congenit Anom (Senten Ijo), **16**: 175–185 (in Japanese).

Nolen GA, Klusman LW, Patrick LF, & Geil RG (1975) Teratology studies of a mixture of tallow alkyl ethoxylate and linear alkylbenzene sulfonate in rats and rabbits. Toxicology, **4**: 231–243.

Nomura T, Kimura S, Hata S, Kanzaki T, & Tanaka H (1980) The synthetic surfactants AS and LAS interrupt pregnancy in mice. Life Sci, **26**: 49–54.

Nonaka K, Hattori Y, & Nakamoto M (1989) [Determination of LAS in environmental and domestic waste waters.] Suishitsu Odaku Kenkyo, **12** (3): 194–200 (in Japanese).

Nonaka K, Hayashi Y, & Nakamoto M (1990) [The distribution and fate of LAS in some river waters of Osaka.] Ann Rep Environ Pollut Control Cent Osaka Prefect Gov, **12**: 68–79 (in Japanese).

Nusair TL, Danneman PJ, Stotte J, & Bay PJ iS (1988) Consumer products: risk assessment process for contact sensitization. Toxicologist, **8**: 258.

Nyberg H (1988) Growth of *Selenastrum capricornutum* in the presence of synthetic surfactants. Water Res, **22**: 217–223.

Oba K & Yoshida Y (1965) [Determination of ABS in well water.] J Jpn Water Works Assoc [Suido Kyokai Zasshi], **371**: 16–21 (in Japanese).

Oba K, Yoshida Y, & Tomiyama S (1967) [Studies on the biodegradation of synthetic detergents. I. Biodegradation of anionic surfactants under aerobic and anaerobic conditions.] J Jpn Oil Chem Soc [Yukagaku], **16**: 517–523 (in Japanese).

Oba K, Mori A, & Tomiyama S (1968a) [Biochemical studies of n-alpha-olefin sulfonates: (II) Acute toxicity, skin and eye irritation, and some other physiological properties.] J Jpn Oil Chem Soc [Yukagaku], 17 (11): 628–634 (in Japanese).

Oba K, Mori A, & Tomiyama S (1968b) [Biochemical studies of n-alpha-olefin sulfonate. (I) Biodegradability under aerobic conditions.] J Jpn Oil Chem Soc [Yukagaku], **17** (9): 517–520 (in Japanese).

Oba K, Yagi R, & Miura K (1975) [Conversion of detergents to biodegradable compounds and their removal in municipal sewage treatment plants.] Yosui To Haisui, **17**: 1385–1391 (in Japanese).

Oba K, Miura K, Sekiguchi H, Yagi R, & Mori A (1976) Microanalysis of anionic surfactants in waste water by infrared spectroscopy. Water Res, **10**: 149–155.

Oba K, Mori A, & Nagai T (1985) Safety in use of AOS materials in Japan. J Am Oil Chem Soc, **62**: 645.

Oda Y, Kuwano A, Inoue K, Hamano Y, Yamamoto H, Mitsuda B, & Kunita N (1977) [Studies on the toxicity of commercial detergents. 6th report: mutagenicity study in bacteria.] Ann Rep Osaka Prefect Inst Public Health, **8**: 11–16 (in Japanese).

Oda Y, Yamamoto H, & Kunita N (1980) [Studies on the mutagenicity of surfactants.] Abstracts of 9th Annual Meeting of the Japanese Environmental Mutagen Society. J Jpn Mutag Soc, **37**: 11–16 (in Japanese).

Ohki K & Tokiwa F (1969) [Physicochemical properties of sulfate-type and ether sulfate-type surfactants: effects of alkyl chain structure.] J Chem Soc Jpn Chem Ind Chem [Nihon Kagakuzasshi], **90** (11): 1114–1118 (in Japanese).

Ohki K & Tokiwa F (1970) [Physicochemical properties of alpha-olefin-sulfonate sodium salts.] J Chem Soc Jpn Chem Ind Chem [Nihon Kagakuzasshi], **91** (6): 534–539 (in Japanese).

Ohkuma T (1981) [Determination of sodium linear alkylbenzene sulfonate in rivers of Fukuoka City.] Fukuoka-shi Eisei Skikenshoho, **7**: 75–80 (in Japanese).

Oishi S, Takahashi O, Masubuchi M, Fukuda H, Kamiya N, Yoshida S, Mikuriya H, Yuzawa K, & Hiraga K (1974) [The toxicity of the higher alcoholic detergents; (II), Subacute toxicity of sodium dodecyl sulfate.] Ann Rep Tokyo Metrop Res Lab Public Health, **25**: 669–675 (in Japanese).

Okamoto K (1974) [Action of the surfactants upon the skin, desquamative change of the skin surface, irritation of the skin and the inhibition of invertase activity by surfactants.] J Jpn Oil Chem Soc [Yukagaku], **23** (11): 726–729 (in Japanese).

Okamoto T & Shirane Y (1982) [Pollution by linear alkylbenzene sulfonates (LAS) in river and coastal areas in Hiroshima Prefecture.] Hiroshima-Ken Kankyo Sata Kenkyu Hokoku, **3**: 60–65 (in Japanese).

Okamoto K & Takase Y (1976a) [Studies on keratodermia tylodes palmaris progressiva.] Shinshu Med J [Shinshu Ishi], **24** (2): 131–141 (in Japanese).

Okamoto K & Takase Y (1976b) [Action of surfactants upon the skin; (IV), Effect of sodium alkyl sulfates with lipophilic groups of different alkyl chain length on the skin.] Shinshu Med J [Shinshu Ishi], **24** (3): 243–250 (in Japanese).

Okpokwasili GC & Nwabuzor CN (1988) Primary biodegradation of anionic surfactants in laundry detergents. Chemosphere, **17**: 2175–2182.

Olson KJ, Dupree RW, Plomer ET, & Rowe VK (1962) Toxicological properties of several commercially available surfactants. J Soc Cosmet Chem (USA), **13**: 469–482.

Osburn QW (1986) Analytical methodology for linear alkylbenzene sulfonate (LAS) in waters and wastes. J Am Oil Chem Soc, **63**: 257–263.

Oser BL & Morgareidge K (1965) Toxicologic studies with branched and linear alkyl benzene sulfonates in rats. Toxicol Appl Pharmacol, **7**: 819–825.

Ottery J, Olavesen AH, & Dodgson KS (1970) Metabolism of dodecyl sulphate in the rat: non-enzymic liberation of sulphate and gamma-butyrolactone from the major metabolite, butyric acid 4-sulphate. Life Sci, **9** (2): 1335–1340.

Painter HA (1992) Anionic surfactants. In: de Oude NT ed. The handbook of environmental chemistry. Vol 3, Part F: Anthropogenic compounds. Berlin, Springer-Verlag, pp 1–88.

Painter HA & Mobey FE (1992) The question of the anaerobic biodegradability of linear alkylbenzene sulfonates (LAS). Proceedings of the European Committee of Organic Surfactants and Their Intermediates (CESIO) Surfactant Congress, London, June 1992. Brussels, CESIO, pp 34–41.

Painter HA & Zabel TF (1988) Review of the environmental safety of LAS. Report to the Council of European Federations of Chemical Industry (CEFIC). Medmenham, Water Research Centre, 245 pp (CO 1659-M/1/EV 8658).

Painter HA & Zabel T (1989) The behaviour of LAS in sewage treatment. Tenside Surfactants Deterg, **26**: 108–115.

Palmer AK, Readshaw MA, & Neuff AM (1975a) Assessment of the teratogenic potential of surfactants (Part I)—LAS, AS and CLD. Toxicology, **3**: 91–106.

Palmer AK, Readshaw MA, & Neuff AM (1975b) Assessment of the teratogenic potential of surfactants (Part II)—AOS. Toxicology, **3**: 107–113.

Palmisano AC, Schwab BS, Maruscik DA, & Ventullo RM (1991) Seasonal changes in mineralisation of xenobiotics by stream microbial communities. Can J Microbiol, **37**: 939–948.

Panigrahi AK & Konar SK (1986) Effects of mixture of petroleum refinery effluent and an anionic linear alkyl benzene sulfonate detergent on aquatic ecosystem. Environ Ecol, **4**: 434–438.

Pärt P, Svanberg O, & Bergstrom E (1985) The influence of surfactants on gill physiology and cadmium uptake in perfused rainbow trout gills. Ecotoxicol Environ Saf, **9**: 135–144.

Persoone G, Van de Vel A, Van Steertegem M, & De Nayer B (1989) Predictive value of laboratory tests with aquatic invertebrates: influence of experimental conditions. Aquat Toxicol, **14**: 149–167.

Pickering QH (1966) Acute toxicity of alkyl benzene sulfonate and linear alkylate sulfonate to the eggs of the fathead minnow, *Pimephales promelas*. Air Water Pollut Int J, **10**: 385–391.

Pickering QH (1988) Evaluation and comparison of two short-term fathead minnow tests for estimating chronic toxicity. Water Res, **22**: 883–893.

Pickering QH & Thatcher TO (1970) The chronic toxicity of linear alkylate sulfonate (LAS) to *Pimephales promelas*, Rafinesque. J Water Pollut Control Fed, **42**: 243–254.

Pitter P & Fuka T (1979) The problem of ultimate biodegradability of linear alkylbenzene sulphonates. Tenside Surfactants Deterg, **16**: 298–302.

Pittinger CA, Woltering DM, & Masters JA (1989) Bioavailability of sediment-sorbed and aqueous surfactants to *Chironomus riparius* (midge). Environ Toxicol Chem, **8**: 1023–1033.

Polano MK (1968) The interaction of detergents and the human skin. J Soc Cosmet Chem, **19**: 3–20.

Prats D, Ruiz F, Vazquez B, Zarzo D, Berna JL, & Moreno A (1993) LAS homolog distribution shift during wastewater treatment and composting: ecological implications. Environ Toxicol Chem, **12**: 1599–1608.

Price KS, Waggy GT, & Conway RA (1974) Brine shrimp bioassay and seawater BOD of petrochemicals. J Water Pollut Control Fed, **46**: 63–77.

Prottey C & Ferguson T (1975) Factors which determine the skin irritation potential of soaps and detergents. J Soc Cosmet Chem, **26**: 29–46.

Putterman GT, Wolejsza NF, Wolfram MA, & Laden K (1977) The effect of detergents on swelling of stratum corneum. J Soc Cosmet Chem (USA), **28**: 521–532.

Rama Bhat S, Crisp PT, Eckert JM, & Gibson NA (1980) A spectrophotometric method for the determination of anionic surfactants at µg-l[1] levels. Anal Chem Acta, **116**: 191–193.

Rapaport RA & Eckhoff WS (1990) Monitoring linear alkyl benzene sulfonate in the environment: 1973-1986. Environ Toxicol Chem, **9**: 1245–1257.

Rapaport RA, Hopping WD, & Eckloff WS (1987) Monitoring LAS in the environment: abstracts and programme. Eighth Annual Meeting of the Society of Environmental Toxicology and Chemistry (SETAC), Pensacola, Florida. New York, Society of Environmental Toxicology and Chemistry.

Rehwoldt R, Lasko L, Shaw C, & Wirhowski E (1974) Toxicity study of two oil spill reagents toward Hudson River fish species. Bull Environ Contam Toxicol, **11**: 159–162.

Reiff B, Lloyd R, How MJ, Brown D, & Alabaster JS (1979) The acute toxicity of eleven detergents to fish: results of an interlaboratory exercise. Water Res, **13**: 207–210.

Rennison PM, Powell GM, Olavesen AH, Howes D, & Black JG (1987) Absorption and excretion of linear alkylbenzene sulphonates in rats. Biochem Soc Trans, **15**: 456.

Renzoni A (1974) Influence of toxicants on marine invertebrate larvae. Thalassia Jugosl., **10**: 197–211.

Richtler HJ & Knaut J (1988) World prospects for surfactants. In: Proceedings of the Second World Surfactants Congress: Surfactants in Our World Today and Tomorrow, Paris, 24–27 May 1988. Paris, Syndicat National des Fabricants d'Agents de Surface et de Produits Auxiliaires Industriels.

Richtler HJ & Knaut J (1991) Environment and oleochemicals—a challenge. Fett Wissensch Technol, **93**: 1–13.

Roberts DW (1989) Aquatic toxicity of linear alkyl benzene sulphonates (LAS)—A QSAR analysis. In: Turner JE, England MW, Schultz TW, & Kwaak NJ ed. QSAR 88. Proceedings of the Third International Workshop on Quantitative Structure-Activity Relationships in Environmental Toxicology, Knoxville, Tennessee, 22–26 May 1988. Springfield, Virginia, US Department of Commerce, National Technical Information Service, pp 91–98.

Roberts DW (1991) QSAR issues in aquatic toxicology of surfactants. Sci Total Environ, **109/110**: 557–568.

Roberts DW & Williams DL (1983) Sultones as by-products in anionic surfactants. Tenside Surfactants Deterg, **20** (3): 109–111.

Roberts MH, Warinner JE, Tsai CF, Wright D, & Cronin E (1982) Comparison of estuarine species sensitivities to three toxicants. Arch Environ Contam Toxicol, **11**: 681–692.

Roberts DW, Lawrence JG, Fairweather IA, Clemett CJ, & Saul CD (1990) Alk-1-ene-1,3-sultones in a-olefin sulphonates. Tenside Surfactants Deterg, **27** (2): 82–86.

Robinson EC & Schroeder RE (1992) Reproductive and developmental toxicity studies of a linear alkylbenzene mixture in rats. Fundam Appl Toxicol, **18** (4): 549–556.

Robra KH (1976) [Detection of toxic water contaminants by inhibition of substrate oxidation in *Pseudomonas* using polarography.] Wasser / Abwasser, **117**: 80–86 (in German).

Röderer G (1987) Toxic effects of tetraethyl lead and its derivatives on the chrysophyte *Poterioochromonas malhamensis*. VIII. Comparative studies with surfactants. Arch Environ Contam Toxicol, 16: 291–301.

Rohrer DM & Woodbridge DD (1975) Reduction in surfactants by irradiation. Bull Environ Contam Toxicol, **13**: 31–36.

Rosner E, Sawinsky A, Molnar A, Pasztor G, & Mihak I (1973) [Laboratory tests on workers exposed to dodecyl benzolsulfonate.] Egeszsegtudomany, **17**: 163–165 (in Hungarian).

Russell NJ, Anderson DJ, Day MJ, & White GF (1991) Colonization of biofilms by bacteria capable of biodegrading sodium dodecyl sulphate (SDS) at clean and polluted sites. Microbiol Ecol, **22**: 85–98.

Sadai M & Mizuno N (1972) [Effect of long term topical application of some anionic surfactants on the skin, oral mucous membrane, and tongue.] Jpn J Dermatol [Nihon Hifuka Gakkaishi], **82** (4): 207–221 (in Japanese).

Sadai M, Sato T, Arima M, Adachi K, & Tamai H (1979) [Effect of some surfactants on the skin.] J Jpn Cosmet Sci Soc [Kousho Kaishi], **3** (1): 60–62 (in Japanese).

Saito T & Hagiwara K (1982) Analysis of traces of surfactants in water with anion-exchange resin and polymeric adsorbent. Fresenius Z Anal Chem, **312**: 533–535.

Saito T, Higashi K, & Hagiwara K (1982) Determination of traces of sodium alkylbenzenesulphonate by high-performance liquid chromatography: application to water. Fresenius Z Anal Chem, **313**: 21–23.

Sakashita S (1979) Electron microscopic study of liver tissue after cutaneous administration of detergents: multiple application of 1% solution. Jpn J Clin Electron Microsc [Nihon Rinsho Denshikenbikyo Gakkaishi], **12** (1-2): 189–216.

Sakashita S, Agata I, & Akiyoshi S (1974) [An electron microscopic study on the liver after the dermal application of various detergents.] Mie Med J [Mie Igaku], **18**: 124–143 (in Japanese).

Sales D, Quiroga JM, & Gomez-Parra A (1987) Primary biodegradation kinetics of anionic surfactants in the marine environment. Bull Environ Contam Toxicol, **39**: 385–392.

Samejima Y (1991) [Effects of synthetic surfactants and natural soap on the development of mouse embryos *in vitro* and the fertilizing capacity of mouse and human sperm.] J Osaka Univ Med Sch, **3** (12): 675–682 (in Japanese).

Sasagawa S (1963) [The present status of housewives' hand dermatitis in Japan and the role of detergents as possible causes.] Hifukano Rinsho, **5**: 47-55 (in Japanese).

Sasagawa S, Takase Y, & Ishihara M (1978) [A study on the irritation effects of detergent components on human skin by the patch test: A report of studies on synthetic detergents.] Tokyo, Science and Technology Agency of Japan, Research Coordination Bureau pp 243–266 (in Japanese).

Sato K, Ando H, Yuzawa K, & Hiraga K (1972) [Studies on toxicity of synthetic detergents; (III), Examination of teratogenic effects of alkyl benzene sulfonate spread on the skin of mice.] Ann Rep Tokyo Metrop Res Lab Public Health, **24**: 441–448 (in Japanese).

Sawada K, Inomata S, Gobara B, & Suzuki T (1983) Extraction and determination of anionic surfactants with copper (II) ethylenediamine derivative complexes. Talanta, **30** (3): 155–159.

Sawano J (1978) [Teratogenicity studies of anionic surfactants (AOS, AOS-S) absorbed through skin: A report of studies on synthetic detergents.] Tokyo, Science and Technology Agency of Japan, Research Coodination Bureau, pp 112–113 (in Japanese).

Schöberl P (1989) Basic principles of LAS biodegradation. Tenside Surfactants Deterg, **26**: 86–94.

Schöberl P, Bock KJ, & Huber L (1988) Surfactant biodegradation. Tenside Surfactants Deterg, **25**: 2–7.

Schumacher KA, Classen HG, Marquardt P, & Spath L (1972) [Acute pulmonary hypertonus in cats after parenteral application of sodium decylsulfate and sodium dodecyl sulfate.] Arzneim Forsch, **22** (9): 1489–1491 (in German).

Sedlak RI & Booman KA (1986) A study of LAS and alcohol ethoxylate removal at a municipal wastewater treatment plant. Chim Oggi, **9**: 21-24.

Sekiguchi HK, Miura K, Oba K, & Mori A (1972) Biodegradation of AOS and other surfactants. J Jpn Oil Chem Soc, **21**: 451-455.

Sekiguchi H, Miura K, Oba K, & Mori A (1975a) [Biodegradation of a-olefin sulfonates (AOS) and other surfactants.] J Jpn Oil Chem Soc [Yukagaku], **24**: 145-148 (in Japanese).

Sekiguchi H, Miura K, & Oba K (1975b) [Biodegradation of some anionic surfactants in the river die-away test.] J Jpn Oil Chem Soc [Yukagaku], **24**: 451-455 (in Japanese).

Sekiguchi H, Oba K, Masuda M, & Sugiyama T (1975c) [Studies on the relative biodegradation rate of alkene vs hydroxyalkane sulfonates in a-olefin sulfonate.] J Jpn Oil Chem Soc [Yukagaku], **24**: 675-679 (in Japanese).

Setzkorn EA & Huddleston RL (1965) Ultraviolet spectroscopic analysis for following the biodegradation of hydrotopes. J Am Oil Chem Soc, **42**: 1081-1084.

Sharma AK, Bandre TR, Srinivasu T, & Chandra N (1985) Deleterious effects of detergent on plants. Environ Ecol, **3**: 444-445.

Shiga Prefecture (1988) [Water quality of Lake Biwa in 1988.] Shiga Prefect Inst Public Health: 22 (in Japanese).

Shimp RJ (1989) LAS biodegradation in estuaries. Tenside Surfactants Deterg, **26** (6): 390-393.

Shiobara S & Imahori A (1976) [Effects of linear alkylbenzenesulfonate orally administered to pregnant mice on the pregnant mice and their fetuses.] J Food Hyg Soc Jpn [Shokuhin Eiseigaku Zasshi], **17** (4): 295-301 (in Japanese).

Smyth HF, Seaton J, & Fischer L (1941) Some pharmacological properties of the 'Tergitol' penetrants. J Ind Hyg Toxicol, **23** (10): 478-483.

Snell TW & Persoone G (1989) Acute toxicity bioassays using rotifers. II. A freshwater test with *Brachionus rubens*. Aquat Toxicol, **14**: 81-92.

Solon JM & Nair JH (1970) The effect of a sublethal concentration of LAS on the acute toxicity of various phosphate pesticides to the fathead minnow (*Pimephales promelas* Rafinesque). Bull Environ Contam Toxicol, **5**: 408-413.

Solon JM, Lincer JL, & Nair JH (1969) The effect of sublethal concentration of LAS on the acute toxicity of various insecticides to the fathead minnow (*Pimephales promelas* Rafinesque). Water Res, **3**: 767-775.

Stalmans M, Matthijs E, & de Oude NT (1991) Fate and effects of detergent chemicals in the marine and estuarine environment. Water Sci Technol, **24**: 115-126.

Standing Technical Committee on Synthetic Detergents (1978) Annual report of the UK Standing Technical Committee on Synthetic Detergents. London, Her Majesty's Stationery Office.

Standing Technical Committee on Synthetic Detergents (1989) Annual report of the UK Standing Technical Committee on Synthetic Detergents. London, Her Majesty's Stationery Office.

Steber J (1979) Investigations into the biodegradation of ^{14}C-labelled linear alkyl benzene sulphonate in models of surface waters and sewage purification plants. Tenside Surfactants Deterg, **16**: 140–145.

Steber J & Wierich P (1987) The anaerobic degradation of detergent range fatty alcohol ethoxylates. Studies with ^{14}C-labelled model surfactants. Water Res, **21**: 661–667.

Steber J, Gode P, & Guhl W (1988) Fatty alcohol sulfates: The ecological evaluation of a group of important detergent surfactants. Fett Wiss Technol, **90**: 32–38.

Stenton SC, Dennis JH, Walters EH, & Hendrick DJ (1990) Asthmagenic properties of a newly developed detergent ingredient: sodium isononanoyl oxybenzene sulphonate. Br J Ind Med, **47** (6): 405–410.

Sterzel W (1992) Daily human intake. In: Gloxhuber C & Künstler K ed. Anionic surfactants; biochemistry, toxicology, dermatology, 2nd ed. New York, Marcel Dekker, Inc., pp 411–417.

Sueishi T, Morioka T, Kaneko H, Kusaka M, Yagi S, & Chikami S (1988) Environmental risk assessment of surfactants: fate and environmental effects in Lake Biwa basin. Regul Toxicol Pharmacol, **8**: 4–21.

Sullivan WT & Evans RL (1968) Major US river reflects surfactant changes. Environ Sci Technol, **2**: 194–200.

Sullivan WT & Swisher RD (1969) MBAS and LAS surfactants in the Illinois River, 1968. Environ Sci Technol, **3**: 481–483.

Sunakawa T, Ikida Y, & Okamoto K (1979) [Absorption, distribution, metabolism, and xcretion of linear alkylbenzene sulfonate in rats.] J Jpn Oil Chem Soc [Yukagaku], **39** (2): 59–68 (in Japanese).

Sunakawa T, Inoue K, & Okamoto K (1981) [Studies on the mutagenicity of surfactants; mutagenicity of surfactants following activation with various liver homogenates (S-9) and mutagenicity in the presence of norharman.] Hyg Chem [Eisei Kagaku], **27** (4): 204–211 (in Japanese).

Swedmark M & Granmo A (1981) Effects of mixtures of heavy metals and a surfactant on the development of cod (*Gadus morhua* L.). Rapp P-V Reun Cons Int Explor Mer, **178**: 95–103.

Swedmark M, Braaten B, Emanuelsson E, & Granmo A (1971) Biological effects of surface active agents on marine animals. Mar Biol, **9**: 183–201.

Swisher RD (1963) The chemistry of surfactant biodegradation. J Am Oil Chem Soc, **40**: 648–656.

Swisher RD (1967) Biodegradation of LAS-benzene rings in activated sludge. J Am Oil Chem Soc, **44**: 717–724.

Swisher RD (1970) Surfactant biodegradation. New York, Marcel Dekker, Inc., 469 pp (Surfactant Science Series, Vol 3).

Swisher RD (1972) Linear alkylbenzene sulfonate (LAS) benzene rings: Biodegradation and acclimation studies. J Jpn Oil Chem Soc, **21**: 130–142.

Swisher RD (1976) Surfactants: from recalcitrant to docile. In: Proceedings of the 3rd International Biodegradation Symposium. London, Applied Science Publishers, pp 853–865.

Swisher RD (1981) The problem of ultimate biodegradability of linear alkylbenzene sulfonates: an extension. Tenside Surfactants Deterg, **18**: 57–63.

Swisher RD (1987) Surfactant biodegradation. New York, Marcel Dekker, Inc., 1085 pp (Surfactant Science Series, Vol 18).

Swisher RD, O'Rourke JT, & Tomlinson HD (1964) Fish bioassays of linear alkylate sulfonates (LAS) and intermediate biodegradation products. J Am Oil Chem Soc, **41**: 746–752.

Swisher RD, Gledhill WE, Kimerle RA, & Taulli TA (1978) Carboxylated intermediates in the biodegradation of linear alkylbenzene sulfonates (LAS). In: Proceedings of the 7th International Congress on Surface Active Substances, Moscow, June 1976. Moscow, Teknol Vody, Vol 4, pp 218–230.

Szakall A & Schulz KH (1960) [Permeation of fatty alcohol sulfate and sodium soap of defined chain length (C_8-C_{18}) in intact human skin, and its relation to irritant activity.] Fette Seifen Anstrichm, **62** (3): 170–175 (in German).

Tabor CF, Barber LB, & Runnells DD (1993) Anionic surfactants in the Mississippi River: A detailed examination of the occurrence and fate of linear alkylbenzene sulfonate. Presented at the Division of Environmental Chemistry, American Chemical Society, Denver, Colorado, 28 March–2 April 1993.

Taguchi S, Tonoshima T, Kasahara I, & Goto K (1981) [Extraction and spectrophotometric determination of anionic surfactants with [2-(5-chloro-2-pyridylazo)-5-diethylaminophenolato] cobalt (III) chloride.] Kogyo Yosui, **278**: 23–27 (in Japanese).

Takada H & Ishiwatari R (1987) Linear alkylbenzenes in urban riverine environments in Tokyo: distribution, source, and behavior. Environ Sci Technol, **21**: 875–883.

Takada H, Ishiwatari R, & Ogura N (1992a) Distribution of linear alkylbenzenes (LABs) and linear alkylbenzene-sulphonates (LAS) in Tokyo Bay sediments. Estuar Coast Shelf Sci., **35**: 141–156.

Takada H, Ogura N, & Ishiwatari R (1992b) Seasonal variations and modes of riverine input of organic pollutants to the coastal zone: 1. Flux of detergent-derived pollutants to Tokyo Bay. Environ Sci Technol, **26**: 2517–2523.

Takahashi O, Shiba H, Nakao T, & Hiraga K (1974) [Inhibition of thrombin by linear alkylbenzene sulfonate (LAS).] Ann Rep Tokyo Metrop Res Lab Public Health, **25**: 637–645 (in Japanese).

Takahashi A, Sato K, Ando H, Kubo Y, & Hiraga K (1975) [Teratogenicity of some synthetic detergent and linear alkylbenzene sulfonate (LAS).] Ann Rep Tokyo Metrop Res Lab Public Health, **26** (2): 67–78 (in Japanese).

Takahashi A, Ando H, Kubo Y, & Hiaga K (1976) [Effects of dermal application of sodium dodecyl sulfate (SDS) on pregnant mice and their fetuses.] Ann Rep Tokyo Metrop Res Lab Public Health, **27** (2): 113–118 (in Japanese).

Takami K, Kamo T, Mochizuki K, Sugimae A, & Nakamoto M (1987) [Rapid HPLC determination of linear alkylbenzenesulfonates in environmental water by using concentration technique with a mini column.] Jpn Anal [Bunseki Kagaku], **36**: 276–281 (in Japanese).

Takano S, Yagi N, & Kunihiro K (1975) [Determination of trace amounts of alkyl benzenesulfonates in water: determination by methylene blue spectrophotometry and high performance liquid chromatography.] J Jpn Oil Chem Soc [Yukagaku], **24** (6): 389–394 (in Japanese).

Takeshita R & Yoshida H (1975) [Studies of anion active surfactants; (I), Determination of anion active surfactants in natural and waste waters using cleanup by Amberlite XAD2 column chromatography.] J Hyg Chem [Eisei Kagaku], **21** (4): 209–215 (in Japanese).

Takita Y (1982) [The effects of some detergents and anionic surfactants on the early stages of growth of some agricultural plants.] J Jpn Oil Chem Soc [Yukagaku], **31**: 507–510 (in Japanese).

Takita Y (1985) [Effects of biodegradation intermediates of linear alkylbenzenesulfonate on the growth of sporelings of *Porphyra yeroensis*.] J Jpn Oil Chem Soc [Yukagaku], **34**: 198–201 (in Japanese).

Takita Y & Oba K (1985) [Determination of trace amounts of anionic surfactants in the water of Tama River.] Jpn J Water Pollut Res, **8** (11): 752–754 (in Japanese).

Tamura J & Ogura Y (1969) [Pharmacological studies on surface active agents. 9: Anionic surface active agents and methaemoglobin formation in the mouse.] Med Biol [Igakuto Seibutugaku], 8 (2): 41–44 (in Japanese).

Tanaka Y & Nakanishi H (1981) [Purification on Sephadex LH-20 and high performance liquid chromatographic determination of dodecylbenzenesulfonate in sediment and biological samples.] Bunseki Kagaku, 30: 567–73 (in Japanese).

Tang NH (1974) Relationship between BOD removal and LAS detergent removal. Springfield, Virginia, National Technical Information Service, 66 pp (NTIS Report PB-232 977/7WP).

Taniguchi S, Yamada A, Morita S, Ogaki S, & Noda T (1978) Results of studies on synthetic detergents. Tokyo, Science and Technology Agency, Research Coordination Bureau, pp 18–54.

Tarazona JV & Nunez O (1987) Acute toxicity of synthetic detergents to snails: Effect of sodium lauryl sulfate on *Limnaea peregra* shells. Bull Environ Contam Toxicol, 39: 1036–1040.

Tatsukawa R & Hidaka H (1978) [The avoidance test of chemical substances by fish: avoidance of detergents by ayu (*Plecoglossus altivelis*).] Nippon Nosei Kasaku Kaishi, 52: 263–270 (in Japanese).

Taylor MJ (1985) Effect of diet on the sensitivity of *Daphnia magna* to linear alkylbenzene sulfonate. In: Cardwell RD, Purdy R, & Bahner RC ed. Aquatic toxicology and hazard assessment: Seventh Symposium. Philadelphia, Pennsylvania, American Society for Testing and Materials, pp 53–72 (ASTM STP 854).

Terzic S & Ahel M (1993) Determination of linear alkylbenzene sulphonates in Krka River estuary. Bull Environ Contam Toxicol, 50: 241–246.

Thatcher TO & Santner JF (1967) Acute toxicity of LAS to various fish species. Proceedings of 21st Industrial Waste Conference, Purdue University. Washington, Industrial Waste Association, pp 996–1002.

Thomas ORT & White GF (1989) Metabolic pathway for the biodegradation of sodium dodecyl sulfate by pseudomonas sp. C12B. Biotechnol Appl Biochem, 11: 318–327.

Thurman EM, Barber LB, & LeBlanc D (1986) Movement and fate of detergents in groundwater: a field study. J Contam Hydrol, 1: 143–161.

Tiba S (1972) [Studies on the acute and chronic toxicity of linear alkylbenzene sulfonate.] J Food Hyg Soc Jpn [Shokuhin Eiseigaku Zasshi], 13 (6): 509–516 (in Japanese).

Tiba S, Shiobara S, Imahori A, & Kitagawa T (1976) [Effects of linear alkylbenzene sulfonate on dam, fetus, and newborn rat.] J Food Hyg Soc Jpn [Shokuhin Eiseigaku Zasshi], 17 (1): 66–71 (in Japanese).

Tokai A, Kikuchi M, Wakabayashi M, & Yoshida T (1990) [Environmental behavior of selected chemicals in Tokyo Bay with relation to their physicochemical and biochemical properties.] J Chem Soc Jpn Chem Ind Chem [Nippon Kagaku Kaishi], 9: 982–991 (in Japanese).

Tokyo Metropolitan Government (1974) [A study on detergents.] Tokyo, Tokyo Metropolitan Government, 10 pp (Report No. 49-5-29) (in Japanese).

Tomiyama S (1970) [Alpha-olefin sulfonate: commercial production and performance.] J Jpn Oil Chem Soc [Yukagaku], 19 (6): 359–368 (in Japanese).

Tomiyama S (1972) [Current problems of detergents, mainly related to water pollution.] J Jpn Oil Chem Soc [Yukagaku], 21 (1): 2–8 (in Japanese).

Tomiyama S (1974) [Effects of surfactants on fish.] Bull Jpn Soc Sci Fish, 40: 1291–1296 (in Japanese).

Tomiyama S (1975) Fundamental study of biochemical behavior of anionic sulfonate- and sulfate-type surfactants. J Am Oil Chem Soc, 52: 135–138.

Tomiyama S (1978) [Possible mechanisms of the effects on fish and shellfish of household detergents.] J Jpn Oil Chem Soc [Yukagaku], 27: 347–353 (in Japanese).

Tomizawa S (1978) Results of studies on synthetic detergents. Tokyo, Science and Technology Agency, Research Coordination Bureau, pp 55–88.

Toshima Y, Moriya T, & Yoshimura K (1992) Effects of polyoxyethylene (20) sorbitan monooleate on the acute toxicity of linear alkylbenzenesulfonate (C12LAS) to fish. Ecotoxicol Environ Saf, 24: 26–36.

Tovell PWA, Newsome C, & Howes D (1974) Effect of water hardness on the toxicity of an anionic detergent to fish. Water Res, 8: 291–296.

Tovell PWA, Howes D, & Newsome CS (1975) Absorption, metabolism and excretion by goldfish of the anionic detergent sodium lauryl sulphate. Toxicology, 4: 17–29.

Trehy ML, Gledhill WE, & Orth RG (1990) Determination of linear alkylbenzenesulfonates and dialkyltetrasulfonates in waters and sediment by gas chromatography/mass spectrometry. Anal Chem, 62: 2581–2586.

Tsai CF & McKee RA (1978) The toxicity to goldfish of mixtures of chloramines, and LAS (linear alkylate sulfonate). College Park, Maryland, University of Maryland, Water Resources Research Center, 36 pp (Technical Report No. 44; PB-280-554).

Tsukioka T & Murakami T (1983) [Determination of linear-alkylbenzenesulfonate by gas chromatography.] Jpn Anal [Bunseki Kagaku], 32: 723–728 (in Japanese).

Uchiyama M (1977) Determination of alkyl benzenesulfonate in water by the UV method. Water Res, 11: 205–207.

Urano K & Saito M (1985) Biodegradability of surfactants and inhibition of surfactants to biodegradation of other pollutants. Chemosphere, **14**: 1333–1342.

Urano K, Saito M, & Murata C (1984) Adsorption of surfactants on sediments. Chemosphere, **13**: 293–300.

United States Environmental Protection Agency (1981) Mono(C_{10-16}) alkylbenzenesulfonate acids and their sodium salts. Draft hazard information review for TSCA Interagency Testing Committee (EPA Contract No. 68-01-5789). Rockville, Maryland, Enviro Control, 101 pp.

Utsunomiya A, Itoh SI, Setsuda S, Naito S, & Shimozato T (1980) [Studies on sodium linear alkylbenzenesulfonate (LAS). I. Distribution of LAS in the sediments of the Sagami River estuary.] Eisei Kagaku, **26**: 159–166 (in Japanese).

Utsunomiya A, Ikeda T, Takamatsu K, & Naito S (1982) [Determination of sodium alkylsulfates as 3,5-dinitrobenzoate derivatives.] Jpn Anal [Bunseki Kagaku], **31**: 115–119 (in Japanese).

Van Emden HM, Kroon CCM, Schoeman EN, & Van Seventer HA (1974) The toxicity of some detergents tested on *Aedes aegypti* L., *Lebistes reticulatus* Peters, and *Biomphalaria glabrata* (Say). Environ Pollut, **6**: 297–308.

Van Scott EJ & Lyon JB (1953) A chemical measure of the effect of soaps and detergents on the skin. J Invest Dermatol, **21**: 199–203.

Verge C, Moreno A, Bravo J, Ferrer J, & Bengoechea C (1993) Toxicity of LAS vs activated sludge of a WWTP and microalgae (*Scenedesmus subspicatus*). Presented at Society of Environmental Toxicology and Chemistry (SETAC) 1st World Congress, Lisbon, March 1993. New York, Society of Environmental Toxicology and Chemistry.

Vives-Rego J, Vaque D, & Martinez J (1986) Effect of heavy metals and surfactants on glucose metabolism, thymidine incorporation and exoproteolytic activity in sea water. Water Res, **20**: 1411–1415.

Vives-Rego J, Vaque MD, Leal JS, & Parra J (1987) Surfactants biodegradation in sea water. Tenside Surfactants Deterg, **24**: 20–22.

Volk VV & Jackson ML (1968) Alkyl benzene sulfonate and linear alkylate sulfonate adsorption by hydroxy iron and aluminium systems. J Water Pollut Control Fed, **40**: 205–213.

Wagner R (1978) [The content of MBAS and BiAS in a communal sewage treatment plant.] Wasser/Abwasser, **119**: 235–242 (in German).

Wakabayashi M & Mizorogi N (1989) [Subacute toxicity of chemical substances on rainbow trout.] Ann Rep Tokyo Metrop Res Inst Environ Prot [Tokyo Kogai Kenkyujo Nempo], 1988: 167–169 (in Japanese).

Wakabayashi M & Onizuka S (1986) [Some factors affecting toxicity of chemicals to fishes.] Ann Rep Tokyo Metrop Res Inst Environ Prot [Tokyo Kogai Kenkyujo Nempo], 1986: 102–104 (in Japanese).

Wakabayashi M, Kikuchi M, Kojima H, & Yoshida T (1978) Bioaccumulation profile of sodium linear alkylbenzene sulfonate and sodium alkyl sulfate in carp. Chemosphere, **11**: 917–924.

Wakabayashi M, Kikuchi M, Kojima H, & Yoshida T (1980) Effect of alkyl chain on the uptake, distribution, and excretion of ^{35}S-labelled alkyl sulfates in carp. Ecotoxicol Environ Saf, **4**: 195–206.

Wakabayashi M, Kikuchi M, Sato A, & Yoshida T (1981) [The relationship between exposure concentration and bioaccumulation of surfactants.] Bull Jpn Soc Sci Fish, **47**: 1383–1387 (in Japanese).

Wakabayashi M, Kikuchi M, Naganuma Y, & Kawakara H (1984) [Acute toxicity of some detergents in fishes.] Ann Rep Tokyo Metrop Res Inst Environ Prot [Tokyo Kogai Kenkyujo Nempo], 1984: 114–118 (in Japanese).

Wakabayashi M, Konno R, & Nishiido T (1988) [Relative lethal sensitivity of two daphnid species to some chemicals.] Ann Rep Tokyo Metrop Res Inst Environ Prot [Tokyo Kogai Kenkyujo Nempo], 1988: 126–128 (in Japanese).

Waldmeyer T (1968) Analytical records of synthetic detergent concentrations, 1956-1966. Water Pollut Control, **67** (2): 66–79.

Walker AIT, Brown VKH, Ferrigan LW, Pickering RG, & Williams DA (1967) Toxicity of sodium lauryl sulphate, sodium lauryl ethoxysulphate and corresponding surfactants derived from synthetic alcohols. Food Cosmet Toxicol, **5**: 763–769.

Ward TE (1986) Biodegradation of LAS and model chemicals in aerobic and anaerobic sewage treatment processes and sludge-amended soils. Abstracts of the 7th Annual Meeting of the Society of Environmental Toxicology and Chemistry (SETAC), Arlington, USA, 6-9 November 1983. New York, Society of Environmental Toxicology and Chemistry (Abstract 300).

Ward TE & Larson RJ (1989) Biodegradation kinetics of linear alkylbenzene sulfonate in sludge-amended agricultural soils. Ecotoxicol Environ Saf, **17**: 119–130.

Watanabe Y, Nagashima T, & Imai N (1968) [Effect of detergents on human skin in relation to hand eczema.] Saigai Igaku, **11** (9): 933–940 (in Japanese).

Watari N, Torizawa K, Kanai M, & Suzuki Y (1977) Ultrastructural observations of the protective effect of glycyrrhizin for mouse liver injury caused by oral administration of detergent ingredient(LAS). Jpn J Clin Electron Microsc [Nihon Rinsho Denshikenbikyo Kaishi]), 10 (1-2): 121–139.

Waters J (1976) The contribution of linear alkylbenzene sulphonate to the MBAS level of some UK and Dutch surface waters. Vom Wasser, **47**: 131–140.

Waters J & Garrigan JT (1983) An improved microdesulphonation/gas liquid chromatography procedure for the determination of linear alkylbenzene sulphonates in UK rivers. Water Res, **17**: 1549–1562.

Waters J, Holt MS, & Matthijs E (1989) Fate of LAS in sludge amended soils. Tenside Surfactants Deterg, **26**: 129–135.

Welp G & Brummer G (1985) [The Fe(III)-reduction test: a simple procedure to determine the effects of environmental chemicals on microbial activity in soils.] Z Pflanzenernähr Bodenkd, **148**: 10–23 (in German).

White GF, Russell NJ, & Day MJ (1985) A survey of sodium dodecyl sulphate (SDS) resistance and alkylsulphatase production in bacteria from clean and polluted river sites. Environ Pollut, **A37**: 1–11.

White GF, Anderson DJ, Day MJ, & Russell NJ (1989) Distribution of planktonic bacteria capable of degrading sodium dodecyl sulphate (SDS) in a polluted South Wales river. Environ Pollut, **57**: 103–115.

Williamson R (1993) Alpha olefin sulfonate composition. Chem Week, 27 January: 13.

Wood DCF & Bettley FR (1971) The effect of various detergents on human epidermis. Br J Dermatol, **84**: 320–325.

Yakabe Y, Etoh C, Matsunobo Y, Katsuura H, Miura K, & Yoshimura K (1992) Kinetic study on the biodegradation of linear alkybenzenesulfonates (LAS) in well water. Chemosphere, **24** (8): 969–977.

Yamamoto H (1977) [Carcinogenic and co-carcinogenic testing of detergents (alkyl-sulfonic-natrium, polyoxy-ethylene-alkylether and potassium soap) on the skin of mice.] J Nara Med Assoc [Nara Igaku Zasshi], **28**: 307–327 (in Japanese).

Yamamoto K & Motomizu S (1987) [Solvent extraction with spectrophotometric determination of anionic surfactants in seawater.] Jpn Anal [Bunseki Kagaku], **36**: 335–338 (in Japanese).

Yamamoto H, Inoue K, Hamano Y, Oda Y, Kuwano A, Mitsuda B, & Kunita N (1976) [Studies on toxicity of commercial detergents. 3rd Report. Teratogenic effects in mice administered orally.] Ann Rep Osaka Prefect Inst Public Heath, **7**: 141–145 (in Japanese).

Yamane I, Nagayama M, Kashiwa I, Kuwamura H, Ando S, & Mori A (1970) [Olefin sulfonates. IV. Correlation between carbon chain length and surface-active properties of alpha-olefin sulfonates.] J Chem Soc Jpn [Kogyo Kagaku Zasshi], **73** (4): 723–727 (in Japanese).

Yamane AN, Okada M, & Sudo R (1984) The growth inhibition of planktonic algae due to surfactants used in washing agents. Water Res, **18**: 1101–1105.

Yanagisawa F, Watanabe S, & Yamagishi T (1964) [Biochemical studies of dodecylbenzene sulfonates; differences between soft and hard detergents.] Jpn J Public Health [Nihon Koshueisei Zasshi], **11** (13): 859–864 (in Japanese).

Yasuda H (1980) [Determination of anionic surfactants in sea and river waters by anion exchange method.] Jpn J Public Health [Nihon Koshueisei Zasshi], **27** (8): 373–378 (in Japanese).

Yasunaga Y (1976) [The influence of some pollutants on the survival of eggs and larvae of two species of flatfish, *Limanda yokohamae* and *Paralichtys olivaceus*.] Bull Tokai Reg Fish Res Lab, **86**: 81–111 (in Japanese).

Yediler A, Zhang Y, Cai JP, & Korte F (1989) Effect of the microbial population size on the degradation of linear alkylbenzene sulfonate in lake water. Chemosphere, **18**: 1589–1597.

Yokoyama Y & Sato H (1991) Reversed-phase high-performance liquid chromatographic determination of linear alkylbenzenesulponates in river water at ppb levels by precolumn concentration. J Chromatogr, **555**: 155–162.

Yoneyama M & Hiraga K (1977) [Effect of linear alkylbenzene sulfonate on serum lipid in rats.] Ann Rep Tokyo Metrop Res Lab Public Health, **28** (2): 109–111 (in Japanese).

Yoneyama M, Fujii T, Ikawa M, Shiba H, Sakamoto Y, Yano N, Kobayashi H, Ichikawa H, & Hiraga K (1972) [Studies on the toxicity of synthetic detergents. (II) Subacute toxicity of linear and branched alkyl benzene sulfonates in rats.] Ann Rep Tokyo Metrop Res Lab Public Health, **24**: 409–440 (in Japanese).

Yoneyama M, Mabuchi Y, Ikawa M, Kobayashi H, & Ichikawa H (1976) [Subacute toxicity of linear alkylbenzene sulfonate.] Ann Rep Tokyo Metrop Res Lab Public Health, **27** (2): 105–112 (in Japanese).

Yoneyama M, Masubuchi M, Oishi S, Takahashi O, Ikawa M, Yoshida S, Oishi H, Mikuriya H, Yuzawa K, & Hiraga K (1977) [Toxicity of linear alkylbenzene sulfonate by dietary administration for life-span to rats.] Ann Rep Tokyo Metrop Res Lab Public Heath, **28** (2): 73–84 (in Japanese).

Yoneyama M, Ikawa M, Nakao T, & Hiraga K (1978) [Effects of LAS on incorporation of acetate-1-^{14}C in liver lipids in rats.] Ann Rep Tokyo Metrop Res Lab Public Health, **29** (2): 55–57 (in Japanese).

Yoshida H & Takeshita R (1978) [Studies on anion active surfactants: II; Contamination of anionic surfactants in the Inland Sea of eastern Seto.] Eisei Kagaku, **24**: 78–82 (in Japanese).

Yoshikawa S, Sano H, & Harada T (1984) [Determination of LAS in river water by high-performance liquid chromatography.] Suishitsu Odaku Kenkyu, 7: 191–194 (in Japanese).

Yoshikawa S, Sano H, & Harada T (1985) [Distribution of LAS in river water and sediment at Tsurumi River.] Jpn J Water Pollut Res, 8: 755–758 (in Japanese).

Yoshimura H, Tanaka S, Fujiyama Y, Sugiyama T, & Nagai T (1984a) [Determination of ionic surfactants by high performance liquid chromatography.] J Chem Soc Jpn [Nihon Kagaku Kaishi], 3: 445–451 (in Japanese).

Yoshimura K, Hayashi K, Kawase J, & Tsuji K (1984b) [Presence of anionic surfactants in rivers.] Jpn J Limnol [Rikusuigaku Zasshi], 45: 51–60 (in Japanese).

Yushi (1978) Good decomposition of LAS together with detection of ABS. Water quality and synthetic detergents from the standpoint of a 1977 study. Yushi, 31: 30–33 (in Japanese)

Zaccone G, Fasulo S, Lo Cascio P, & Licata A (1985) Patterns of enzyme activities in the gills of the catfish *Heteropneustes fossilis* (Bloch) exposed to the anion-active detergent Na-alkyl-benzenesulphonate (LAS). Histochemistry, 82: 341–343.

Zoller U (1985) The 'hard' and 'soft' surfactant profile of Israel municipal wastewaters. J Am Oil Chem Soc, 62: 1006–1009.

ALKYLBENZENESULFATES A CHAINE DROITE ET COMPOSES VOISINS

1. RECAPITULATION, EVALUATION ET RECOMMANDATIONS GENERALES

1.1 Identité, propriété et méthodes d'analyse

Les alkylbenzènesulfonates à chaîne droite (appelé aussi alkylbenzènesulfonates linéaires ou ASL, les α-oléfine-sulfonates (AOS) et les alkylsulfates (AS)) sont des tensio-actifs anioniques dont les molécules sont caractérisées par la présence d'un groupement hydrophobe et d'un groupement hydrophile (polaire). Les produits du commerce sont des mélanges d'isomères et d'homologues de produits voisins, qui different par leurs propriétés physicochimiques et qui, sous leurs diverses formes, ont des applications variées.

L'analyse des ASL, des AOS et des AS peut se faire par des méthodes non spécifiques. On utilise généralement l'essai au bleu de méthylène, qui permet de mettre en évidence tout composé contenant un groupement anionique et un groupement hydrophobe. On peut donc être gêné par la présence d'autres substances lorsqu'on travaille sur des échantillons prélevés dans l'environnement; en outre, la sensibilité de la méthode n'est que de 0,02 mg/litre. On a mis au point d'autres méthodes non spécifiques qui peuvent se substituer à celles-ci mais on ne les utilise guère. En ce qui concerne les échantillons prélevés dans l'environnement, il n'existe de méthodes spécifiques que pour les ASL et les AS. En ce qui concerne les AOS, on dispose d'une méthode améliorée qui repose sur la réaction au bleu de méthylène et la chromatographie en phase liquide à haute performance (HPLC).

Les ASL sont des composés non volatils que l'on obtient par sulfonation des alkylbenzènes à chaîne droite. Les produits du commerce sont toujours constitués de mélanges d'homologues ayant des chaînes alkylées de différentes longueurs (C_{10}–C_{13} ou C_{14}) et d'isomères qui différent par la position du point d'attache de la chaine sur le noyau phényle (positions 2 à 5). Tous les homologues et isomères des ASL peuvent être dosés dans des échantillons environnementaux ou d'autres matrices au moyen de méthodes d'analyse spécifiques comme la HPLC, la chromatographie en phase gazeuse et la chromatographie en phase gazeuse couplée à la spectrométrie de masse.

Les AOS sont également des dérivés non volatils produit par sulfonation des α-oléfines. Ils consistent dans le mélange de deux types de composés, les alcène-sulfonates de sodium et les hydroxyalcane-sulfonates de sodium, avec une chaine alkylée en C_{14}–C_{18}.

Egalement non volatils, les AS s'obtiennent en traitant par l'acide sulfurique, les alcools d'origine oléochimique ou pétrochimique. Ce sont des mélanges d'homologues avec une chaîne alkylée en C_{10}–C_{18}. On met actuellement au point des méthodes d'analyse spécifiques pour la surveillance de l'environnement.

1.2 Sources d'exposition humaine et environnementale

On utilise les ASL, les AOS et les AS comme principes actifs de divers produits d'entretien ou d'hygiène personnelle, ou encore, pour certaines applications spéciales. Après usage, ces détergents sont rejetés dans l'environnement avec les eaux usées.

Il peut y avoir exposition professionnelle à ces composés. Quant à l'exposition de la population humaine en général et des êtres vivants dans leur milieu naturel, elle dépend du type d'application de ces substances (ou d'autres tensio-actifs), des pratiques locales en matière de traitement des effluents et des caractéristiques du milieu récepteur.

En 1990, la consommation mondiale de ces produits d'établissait à 2 millions de tonnes pour les ASL, 86 000 tonnes pour le AOS et 289 000 tonnes pour les AS.

1.3 Concentrations dans l'environnement

1.3.1 Alkylbenzènesulfonates à chaîne droite

On peut doser les ASL à l'aide de méthodes spécifiques et sensibles dans pratiquement tous les compartiments du milieu où ils sont susceptibles de se trouver. Leur concentration diminue progressivement selon la séquence suivante: eaux usées > effluents traités > eaux de surface > mer.

Dans les zones où l'on utilise principalement des ASL comme tensio-actifs, leur concentration est généralement de 1–10 mg/litre dans les eaux usées, de 0,05–0,1 mg/litre dans les effluents traités par voie biologique, de 0,05–0,6 mg/litre dans les effluents traités sur lit filtrant, de 0,005–0,05 mg/litre dans les eaux de surface situées au-

dessous de déversoirs d'égouts (avec des concentrations qui tombent rapidement à 0,01 mg/litre en aval du déversoir), de < 1–10 mg/kg dans les sédiments de cours d'eau (≤ 100 mg/kg dans les sédiments très pollués à proximité des zones de décharge), de 1–10 g/kg dans les boues d'égouts, de < 15 mg/kg dans les sols amendés à l'aide de boues d'égouts (initialement 5–10 mg/kg; on a trouvé des concentrations ≤ 50 mg/kg après d'importants épandages de boues, d'ailleurs non représentatifs). La concentration des ASL dans les eaux estuarielles varie de 0,001 à 0,01 mg/litre, mais elle peut être beaucoup plus élevée là où il y a déversement direct d'eaux usées. En mer, à distance du rivage, les concentrations vont de < 0,001 à 0,002 mg/litre.

Il est à noter que la concentration de ASL varie considérablement dans l'environnement. Ces variations sont dues à la diversité des méthodes d'analyse, des points de prélèvement (qui vont de zones très polluées où sont déversés des effluents insuffisamment traités à des secteurs où l'effluent a subi un traitement intensif), des périodes de prélèvement (ce qui selon le cas peut signifier une différence du simple au double) et enfin, des volumes de ASL consommés.

La surveillance de l'environnement montre que les ASL ne s'accumulent pas au cours du temps dans les différents compartiments du milieu. La concentration dans le sol, loin d'augmenter, diminue au contraire par suite de la minéralisation. Comme les ASL ne se décomposent pas en anaérobiose stricte (pour donner naissance à du méthane), on ne peut pas en conclure qu'ils subissent une minéralisation dans les sédiments anaérobies. Au taux actuel d'utilisation, les ASL parviennent dans les différents compartiments de l'environnement à un rythme sensiblement égal à celui de leur assimilation, ce qui crée les conditions d'un état stationnaire.

1.3.2 *Les α-oléfine-sulfonates et alkyle-sulfates*

Les données dont on dispose sur la concentration des AOS dans l'environnement sont limitées en raison de la difficulté à analyser les échantillons prélevés dans le milieu. En général, on peut déceler la présence des tensio-actifs anioniques au moyen de méthodes colorimétriques non spécifiques (comme celles qui sont basées sur la réaction au bleu de méthylène), mais la présence d'autres substances est gênante et ces méthodes ne permettent pas de procéder à un dosage spécifique des α-oléfine-sulfonates. Une méthode spécifique de dosage des AS dans l'environnement est en cours de mise au point.

Les études effectuées en laboratoire indiquent que les AOS et les AS sont rapidement minéralisés dans tous les compartiments de l'environnement et presque totalement éliminés des effluents au cours du traitement de ces derniers. Leur concentration dans les eaux de surface, les sédiments, le sol, les eaux estuarielles et le milieu marin est probablement faible. C'est précisément ce que l'on a constaté pour la concentration des AOS dans l'eau des rivières.

1.4 Transport, distribution et transformation dans l'environnement

Aux températures inférieures à 5–10 °C, la cinétique de biodégradation des ASL, des AOS et des AS est ralentie en raison de la réduction de l'activité microbienne.

1.4.1 Alkylbenzène-sulfonates à chaîne droite

Les voies de pénétration des ASL dans l'environnement varient selon les pays, mais la porte d'entrée principale est constituée par la décharge des stations d'épuration des eaux usées. Lorsque ces stations sont inexistantes ou fonctionnent mal, il peut y avoir décharge directe dans les rivières, les lacs et la mer. L'épandage de boues d'égout sur les terrains agricoles peut également constituer une voie de pénétration de ASL dans l'environnement.

A mesure qu'ils pénètrent dans l'environnement, les ASL en sont éliminés par divers mécanismes qui vont de l'adsorption à la biodégradation ultime. Les ASL sont adsorbés sur les surfaces colloïdales et les particules en suspension, et l'on a mesuré des coefficients d'adsorption de 40–5200 litres/kg selon le milieu et la structure des ASL en cause. Ils subissent une biodécomposition dans les eaux de surface (demi-vie 1–2 jours), dans les sédiments aérobies (1–3 jours) ainsi que dans les écosystèmes marins et estuariels (5–10 jours).

Lors du traitement primaire des effluents, environ 25% des ASL (de 10–40%) s'adsorbent sur les boues résiduelles et sont rejetés avec elles. Ils ne sont pas éliminés au cours de la digestion anaérobie des boues mais au cours du traitement aérobie, leur demi-vie étant alors de 10 jours. Après épandage des boues sur le sol, les ASL sont généralement décomposés à hauteur de 90% en l'espace de trois mois, la demi-vie étant de l'ordre de 5–30 jours.

Le facteur de concentration des ASL dans le corps entier varie de 100 à 300 pour l'ensemble des ^{14}C-ASL et ^{14}C-métabolites. Ils sont captés par les poissons essentiellement à travers les branchies et se répartissent ensuite dans le foie et la vésicule après biotransformation. Les ASL sont rapidement excrétés et rien n'indique par conséquent qu'ils subissent une bioamplification.

1.4.2 *α-Oléfine-sulfonates*

Les données relatives au transport, à la distribution et à la transformation des AOS dans l'environnement sont encore moins nombreuses que dans le cas des ASL. On peut toutefois penser que les AOS sont transportées dans l'environnement à peu près comme les ASL, les AS et les autres détergents tensio-actifs et que leur destinée y est analogue à celles des ASL et des AS. En aérobiose, elles subissent une biodécomposition rapide et cette biodécomposition primaire est achevée en 2 à 10 jours, en fonction de la température. On ne dispose que de données limitées sur la bioaccumulation des AOS; en tout état de cause elles ne s'accumulent pas chez les poissons. On ne dispose d'aucune donnée sur leur décomposition en milieu abiotique.

1.4.3 *Alkylsulfates*

Les AS sont transportés dans l'environnement par des mécanismes analogues à ceux qui sont à l'oeuvre dans le cas des ASL et des AOS. Ils sont facilement biodégradable en aérobiose ou en anaérobiose, que ce soit au laboratoire ou dans l'environnement; la biodécomposition primaire est achevée en l'espace de 2 à 5 jours. Les facteurs de bioconcentration pour le corps entier varient de 2 à 73 ainsi qu'avec la longueur de la chaîne des différents homologues. Chez les poissons, les AS sont captés, distribués, biotransformés et excrétés de la même manière que les ASL et ne se concentrent pas dans les autres organismes aquatiques.

1.5 Cinétique

Les ASL, les AOS et le AS sont facilement résorbés dans les voies digestives, après quoi ils se répartissent dans l'ensemble de l'organisme où ils sont largement métabolisés. Les ASL subissent une ω- et une β-oxydation. Les composés initiaux et leurs métabolites sont principalement excrétés par la voie rénale, encore qu'une certaine proportion de la dose absorbée puisse l'être également pas la voie fécale, après métabolisation et passage dans les voies biliaires. Les ASL,

les AOS et les AS ne sont absorbés qu'en quantités minimes par voie percutanée lorsque la peau est intacte, mais un contact prolongé peut altérer l'intégrité de la barrière épidermique, ce qui permet une résorption plus importante; à fortes concentrations, il peut y avoir réduction du temps de pénétration.

1.6 Effets sur les animaux de laboratoire et sur les systèmes d'épreuve *in vitro*

On a relevé, pour la DL_{50} des sels de sodium des ASL, des valeurs allant de 404 à 1470 mg/kg de poids corporel chez le rat et de 1259 à 2300 mg/kg de poids corporel chez la souris, ce qui incite à penser que les rats sont plus sensibles que les souris à l'action toxique des ASL. Chez la souris, on a obtenu une DL_{50} de 3000 mg/kg de poids corporel pour un sel de sodium d'AOS. Chez le rat, les valeurs de la DL_{50} par voie orale allaient de 1000 à 4120 mg/kg de poids corporel pour les AS. Les ASL, les AOS et les AS sont irritants pour la peau et les yeux.

Lors d'études subchroniques au cours desquelles on a administré à des rats des ASL dans leur nourriture ou leur eau de boisson à des concentrations quotidiennes correspondant à plus de 120 mg/kg de poids corporel, on a observé des effets minimes, qui consistaient notamment en modifications des paramètres biochimiques et altérations histopathologiques au niveau du foie. Bien que lors d'une étude, on ait observé des modifications ultrastructurales dans les hépatocytes à des doses plus faibles, ces modifications se sont révélées réversibles. D'ailleurs, les autres études n'ont pas révélé de tels effets aux mêmes doses, mais il n'est pas exclu que lors de l'étude initiale, les organes aient fait l'objet d'un examen plus minutieux. Des effets ont également été observés sur la fonction de reproduction chez des animaux auxquels on avait administré des doses quotidiennes > 300 mg/kg; il s'agissait d'une réduction du taux de grossesse et d'une certaine mortalité dans les portées. Après application cutanée de longue durée à des rats de solutions de ASL à plus de 5% et application, également cutanée, du même type de solution à des cobayes à raison de 60 mg/kg de poids corporel pendant 30 jours, on a observé des modifications biochimiques et histopathologiques. Des applications cutanées répétées de solutions de teneur ≥ 0,3% de ASL ont produit des effets toxiques sur les foetus ainsi que sur la reproduction, mais les doses étaient également toxiques pour les femelles gestantes.

On n'a guère de données résultant d'études sur des animaux de laboratoire qui permettraient d'évaluer les effets potentiels des AOS

chez l'homme. Aucun effet n'a été observé sur des rats ayant reçu, pendant une longue durée, des doses quotidiennes de 250 mg/kg de poids corporel en administration orale; toutefois une dose quotidienne de 300 mg/kg de poids corporel, toxique pour les femelles gestantes, a entraîné des effets foetotoxiques chez des lapins. L'application topique d'AOS sur la peau et les yeux de divers animaux de laboratoire a produit des effets localisés.

Les effets d'une exposition à long et à court terme aux AS ont été étudiés à plusieurs occasions sur l'animal mais la plupart des études en question pêchent par les insuffisances des examens histopathologiques ou la trop petite taille des groupes; en outre, les doses les plus élevées utilisées dans les études à long terme n'ont pas produit le moindre effet toxique, de sorte qu'il n'a pas été possible d'établir la valeur de la dose sans effets nocifs observables. Cependant, lorsqu'on a administré à des rats des concentrations quotidiennes de ces substances correspondant à 200 mg/kg de poids corporel ou davantage, par incorporation à leur nourriture ou à leur eau de boisson, on a systématiquement observé un certain nombre d'effets. En outre, l'application topique sur la peau ou les yeux d'AS à des concentrations égales ou supérieures à environ 0,5%, a donné lieu à une irritation localisée. Par ailleurs à fortes concentrations, on observe des effets toxiques sur les femelles gestantes ainsi que sur les foetus.

La plupart des études à long terme ne se prêtent pas à l'évaluation du pouvoir cancérogène des ASL, des AOS et des AS chez l'animal de laboratoire en raison de facteurs tel que le nombre trop faible d'animaux, un nombre de doses limité, la non détermination de la dose tolérée maximale, et, en outre, un examen histopathologique limité dans la majorité des cas. Dans les travaux où les effets anatomo-pathologiques ont été convenablement étudiés, on n'a pas déterminé la dose tolérée maximale et les doses employées n'ont pas produit d'effets toxiques. Toutefois et compte tenu de ces réserves, on peut retenir que les études au cours desquelles on a administré à des animaux des ASL, des AOS et des AS par voie orale, n'ont pas révélé de signes de cancérogénicité; quant aux études à long terme consistant en applications topiques d'AOS par badigeonnage cutané, elles n'ont pas non plus révélé la présence d'effets imputables à ces substances.

Sur la base de ces données limitées, il ne semble pas que ces composés soient génotoxiques *in vivo* ou *in vitro*.

1.7 Effets sur l'homme

L'application d'un timbre cutané imprégné de solution contenant jusqu'à 1% de ASL, d'AOS ou d'AS pendant 24 heures montre que la peau humaine supporte le contact avec cette substance au prix d'une légère irritation. Ces tensio-actifs provoquent une délipidation de l'épiderme, une élution du facteur d'humidification naturelle, ainsi qu'une dénaturation des protéines de la couche épidermique externe, dont ils augmentent la perméabilité et dont ils provoquent le gonflement. Ni les ASL, ni les AOS, ni les AS n'ont provoqué de sensibilisation cutanée chez les volontaires et rien n'indique de façon concluante qu'ils puissent provoquer un eczéma. On n'a pas signalé de lésions graves ou mortelles consécutives à l'ingestion accidentelle de ces tensio-actifs.

1.8 Effets sur l'environnement

1.8.1 *Alkylbenzène-sulfonates à chaîne droite*

1.8.1.1 *Milieu aquatique*

Les ASL ont été très largement étudiés tant au laboratoire (études à court et à long terme) que dans des conditions plus proches de la réalité (études sur le micro- et le mésocosme et études en situation réelle). En général, la diminution de la longueur de la chaîne alkylée ou une plus grande intériorisation du groupement phényle s'accompagnent d'une diminution de la toxicité. Les observations effectuées sur des poissons et sur des daphnies montrent que lorsque la longueur de la chaîne diminue d'une unité (par exemple lorsqu'elle passe de C_{12} à C_{11}), la toxicité est approximativement divisée par deux.

Les résultats des tests en laboratoire sont les suivants:

— *Microorganismes*: Les résultats sont très variables en raison de l'utilisation de systèmes d'épreuve très divers (par exemple inhibition des boues activées, cultures mixtes et espèces individuelles). Les valeurs de la CE_{50} vont de 0,5 mg/litre (une seul espèce) à > 1000 mg/litre. Dans le cas des microorganismes, il n'existe pas de relation linéaire entre la longueur de la chaîne et la toxicité.

— *Plantes aquatiques*: Les résultats dépendent largement de l'espèce. En ce qui concerne les plantes d'eau douce, les valeurs de la CE_{50} se situent entre 10 et 235 mg/litre (C_{10}–C_{14}), dans le cas des algues vertes; entre 5 et 56 mg/litre ($C_{11,1}$ –C_{13}), dans le cas des algues bleu-vert; entre 1,4 et

50 mg/litre ($C_{11,6}$–C_{13}) pour les diatomées et entre 2,7 et 4,9 mg/litre ($C_{11,8}$) pour les macrophytes. Il semble que les algues marines soient même encore plus sensibles. Dans le cas des algues, il n'y a probablement pas non plus de relation linéaire entre la longueur de la chaine et la toxicité.

— *Invertébrés:* Les valeurs de la CE_{50} et de la CL_{50} (exposition aiguë) pour au moins 22 espèces d'eau douce se situent entre les limites suivantes: 4,6–200 mg/litre (longueur de chaine non précisée; C_{13}) dans le cas des mollusques; 0,12–27 mg/litre (longueur de chaine non précisée; $C_{11,2}$–C_{18}) dans le cas des crustacés; 1,7–16 mg/litre (longueur de chaine non précisée; $C_{11,8}$) dans le cas des vers et enfin 1,4–270 mg/litre (C_{10}–C_{15}) dans le cas des insectes. Dans le cas d'une exposition chronique, les valeurs de la CE_{50} et de la CL_{50} sont de 2,2 mg/litre ($C_{11,8}$) pour les insectes et de 1,1–2,3 mg/litre ($C_{11,8}$–C_{13}) pour les crustacés. La concentration sans effets chroniques observables (basée sur la mortalité ou des effets sur la fonction de reproduction) est de 0,2 à 10 mg/litre (longueur de chaîne non précisée; $C_{11,8}$) pour les crustacés. Il semble que les invertébrés marins soient plus sensibles, avec des valeurs de la CL_{50} allant de 1 à plus de 100 mg/litre (dans presque tous les cas, C_{12}) pour 13 espèces et avec une concentration sans effets observables de 0,025 à 0,4 mg/litre (longueur de chaine non précisée dans l'ensemble des tests) dans le cas des sept espèces étudiées

— *Poissons*: Pour 21 espèces d'eau douce, les valeurs de la CL_{50} aiguë se situent entre 0,1 et 125 mg/litre (C_8–C_{15}); les valeurs de la CE_{50} et/ou de la CL_{50} pour une exposition chronique sont, pour deux espèces, respectivement égales à 2,4 et à 11 mg/litre (longueur de chaîne non spécifiée; $C_{11,7}$); quant à la concentration sans effets observables, elle va de 0,11–8,4 à 1,8 mg/litre (longueur de chaine non précisée; $C_{11,2}$–C_{13}) pour deux espèces. Dans ce cas encore, les poissons de mer se révèlent plus sensibles, avec des valeurs de la CL_{50} aiguë allant de 0,05 à 7 mg/litre (longueur de chaîne non spécifiée; $C_{11,7}$) pour six espèces et des valeurs de la CL_{50} chronique allant de 0,01 à 1 mg/litre (longueur de chaîne non précisée) pour deux espèces. Dans la plupart des publications, la longueur de la chaine n'est pas précisée. Pour des espèces marines, on a également fait état d'une concentration sans effets observables $< 0,02$ mg/litre (C_{12}).

Les produits communément utilisés dans le commerce ont en moyenne, une chaîne latérale en C_{12}. Des composés ayant diverses longueurs de chaîne ont été étudiés sur *Daphnia magna* et sur des poissons, mais dans le cas des autres organismes d'eau douce, c'est en général des composés dont la longueur de chaine moyenne est de $C_{11,8}$

qui ont été utilisés. Les valeurs caractéristiques de la CE_{50} et de la CL_{50} aiguë pour les ASL en C_{12} sont 3–6 mg/litre chez *Daphnia magna* et 2–15 mg/litre chez les poissons d'eau douce; celles de la concentration sans effets observables pour une exposition chronique sont de 1,2 à 3,2 mg/litre pour *Daphnia magna* et de 0,48–0,9 mg/litre pour les poissons d'eau douce. Chez les poissons de mer, les valeurs caractéristiques de la CL_{50} aiguë pour des ASL en C_{12} sont de < 1–6,7 mg/litre.

Les organismes halophiles et en particulier les invertébrés, se révèlent être plus sensibles aux ASL que les organismes d'eau douce. Chez les invertébrés, l'action séquestrante des ASL sur le calcium peut affecter la biodisponibilité de cet ion pour la morphogénèse. Les ASL exercent un effet général sur le transport ionique. Les produits de biodécomposition et les sous-produits des ASL sont 10 à 100 fois plus toxiques que les composés de départ.

Les résultats obtenus dans des conditions plus proches de la réalité sont les suivants: on a étudié les ASL au moyen de toute sorte de tests en eau douce et à plusieurs niveaux trophiques, notamment dans des enceintes lacustres (organismes inférieurs), dans des écosystèmes modèles (sédiments et réseaux hydrographiques), des cours d'eau en aval et en amont des déversoirs de stations d'épuration des eaux usées et enfin, des cours d'eau expérimentaux. Dans presque tous les cas on a utilisé des ASL en C_{12}. Les algues se sont révélées être plus sensibles en été qu'en hiver, les valeurs de la CL_{50} à 3 heures étant de 0,2 à 8,1 mg/litre après la photosynthèse, alors que dans les écosystèmes modèles, on n'observait aucun effet sur l'abondance relative des populations d'algues à la concentration de 0,35 mg/litre. Selon ces études, la valeur de la concentration sans effets observables se situe de 0,24 à 5 mg/litre selon l'organisme et le paramètre étudié. Ces résultats sont en assez bon accord avec ceux des épreuves en laboratoire.

1.8.1.2 *Milieu terrestre*

On dispose de données sur les végétaux et les lombrics. Pour sept espèces de plantes étudiées dans des solutions nutritives, on a obtenu des valeurs de la concentration sans effets observables qui se situent dans les limites < 10–20 mg/litre; pour trois espèces étudiées sur sol d'après leur croissance, on a obtenu 100 mg/kg (C_{10}–C_{13}). Pour les lombrics, la CL_{50} à 14 jours était > 1000 mg/kg.

1.8.1.3 *Oiseaux*

Une étude sur des poulets qui recevaient une nourriture contenant de ces substances, a permis de fixer à > 200 mg/kg la dose sans effets observables (d'après la qualité des oeufs).

1.8.2 *α-Oléfine-sulfonates*

On dispose de données limitées concernant les effets des AOS sur les organismes aquatiques et terrestres.

1.8.2.1 *Milieu aquatique*

On ne dispose que des résultats des épreuves en laboratoire:

— *Algues*: Valeur de la CE_{50} : > 20–65 mg/litre (C_{16}–C_{18}) pour les algues vertes

— *Invertébrés*: Valeur de la CL_{50} : 19 et 26 mg/litre (C_{16}–C_{18}) pour la daphnie

— *Poissons*: Pour neuf espèces de poissons on a obtenu des valeurs de la CL_{50} aiguë de 0,3–6,8 mg/litre (C_{12}–C_{18}). Sur la base d'études à court terme effectuées sur la truite de mer (*Salmo trutta*), l'ide rouge (*Idus melanotus*) et le rasbora (*Rasbora heteromorpha*), on peut conclure que la toxicité des composés en C_{14}–C_{16} est environ cinq fois plus faible que celle des composés en C_{16}–C_{18}, avec des valeurs de la CL_{50} (à toutes les concentrations mesurées) de 0,5–3,1 (C_{16}–C_{18}) et de 2,5–5,0 mg/litre (C_{14}–C_{16}). Deux études à long terme effectuées sur la truite arc-en-ciel ont montré que le paramètre le plus sensible était la croissance, et qu'il permettait d'obtenir une CE_{50} de 0,35 mg/litre. Pour ce qui est des poissons de mer, on a obtenu pour le mulet gris ou muge (*Mugal cephalus*), une valeur de la CL_{50} à 96 heures de 0,70 mg/litre.

1.8.2.2 *Milieu terrestre*

Une étude portant sur des végétaux en solution nutritive a montré que la concentration sans effets observables se situait dans les limites 32–56 mg/litre. Dans une autre étude, portant cette fois sur des poulets qui recevaient les AOS dans leur nourriture, on a obtenu une valeur > 200 mg/kg pour la concentration sans effets observables (d'après la qualité des oeufs).

1.8.3 Alkyl-sulfates

1.8.3.1 Organismes aquatiques

Les AS ont fait l'objet d'études à court et à long terme et d'une étude dans des conditions plus proches de la réalité. On constate encore que leur toxicité dépend de la longueur de la chaîne latérale alkylée; par contre on ne dispose d'aucune donnée qui indiquerait l'existence d'une différence de toxicité entre les AS à chaine droite et les AS à chaîne ramifiée.

Les résultats des épreuves de laboratoire sont les suivants:

— *Microorganismes*: Les valeurs de la CE_{50} dans une communauté marine étaient de 2,1–4,1 mg/litre (C_{12}). Pour *Pseudomonas putida*, les concentrations sans effets observables étaient de 35–550 mg/litre (C_{16}–C_{18}).

— *Végétaux aquatiques*: Les valeurs de la CE_{50} s'établissaient comme suit: > 20–65 mg/litre (C_{12}–C_{13}) pour les algues vertes et 18–43 mg/litre (C_{12}) pour les macrophytes. Les concentrations sans effets observables s'établissaient à 14–26 mg/litre (C_{12}–C_{16}/C_{18}) chez les algues vertes.

— *Invertébrés*: Les valeurs de la CE_{50} et de la CL_{50} se situaient entre 4 et 140 mg/litre (C_{12}/C_{15}–C_{16}/C_{18}) pour les espèces d'eau douce et entre 1,7 et 56 mg/litre (tous les composés en C_{12}) chez les espèces marines. La concentration sans effets observables pour *Daphnia magna* était de 16,5 mg/litre (C_{16}/C_{18}) en exposition chronique, les valeurs se situant entre 0,29 et 0,73 mg/litre (longueur de chaine non précisée) pour les espèces marines.

— *Poissons*: Les valeurs de la CL_{50} se situaient entre 0,5 et 5,1 mg/litre (longueur de chaine non précisée ou C_{12}–C_{16}) pour des espèces d'eau douce et entre 6,4 et 16 mg/litre (tous les composés en C_{12}) pour les espèces marines. On n'a pas eu connaissance d'études à long terme.

Il est à noter que nombre de ces travaux ont été effectués dans des conditions statiques. Comme les AS sont facilement biodégradables, il est possible qu'on en ait sous estimé la toxicité. Lors d'une étude de 48 heures sur *Oryzias latipes*, on a obtenu pour la CL_{50} des valeurs respectivement égales à 46, 2,5 et 0,61 mg/litre (mesures de concentrations) pour des composés en C_{12}, C_{14} et C_{16}. Cette étude et d'autres, montrent que la toxicité s'accroît d'un facteur 5 lorsque la longueur de la chaîne augmente de deux unités. Une étude dynamique

sur une biocénose, avec des composés en C_{16}–C_{18} a permis d'obtenir une concentration sans effets observables de 0,55 mg/litre.

1.8.3.2 *Organismes terrestres*

On a fait état, pour les lombrics et les navets, de concentrations sans effets observables de valeur > 1000 mg/kg (C_{16}–C_{18}).

1.9 Evaluation des risques pour la santé humaine

Les ASL sont les tensio-actifs les plus largement utilisés pour la fabrication de détergents et de produits de nettoyage; les AOS et les AS entrent également dans la composition des détergents et des produits destinés à l'hygiène personelle. La principale voie d'exposition humaine est donc le contact cutané. Cependant de petites quantités de ASL, d'AOS et d'AS peuvent être ingérées avec l'eau de boisson ou sous forme de résidus subsistant sur les ustentsiles de cuisine et dans les aliments. Bien que les données sur ce point soient limitées, on peut estimer à environ 5 mg/personne la quantité de ASL ingérée quotidiennement de cette manière. Quant à l'exposition professionnelle à ces trois catégories de produits, elle peut intervenir lors de la préparation des différentes substances qui en contiennent, mais on ne dispose d'aucune donnée sur les effets qu'une exposition chronique à ces composés pourrait avoir sur l'homme.

Les ASL, les AOS et les AS peuvent irriter la peau par suite d'un contact répété ou prolongé aux concentrations qui sont celles des produits non dilués. Chez le cobaye, les AOS peuvent provoquer une sensibilisation cutanée lorsque la concentration en sultone γ-insaturée dépasse environ 10 ppm.

Les études à long terme sur animaux de laboratoire dont on connaît les résultats sont insuffisantes pour permettre d'évaluer le pouvoir cancérogène des ASL, des AOS et des AS, et ce, pour différentes raisons: conception même de ces études, trop petit nombre d'animaux utilisés et doses administrées trop faibles, enfin examens histopathologiques trop succints. Compte tenu de ces réserves, les résultats fournis par les études au cours desquelles les animaux ont reçu des ASL, des AOS ou des AS par voie orale, ne comportent aucun signe de cancérogénicité; par ailleurs l'application d'AOS aux animaux par badigeonnage cutané, a également donné des résultats négatifs. Ces composés ne se révèlent pas non plus génotoxiques *in vivo* ou *in vitro*, encore que peu d'études aient été publiées sur ce point.

Des études sub-chroniques au cours desquelles des rats avaient reçu des ASL dans leur nourriture ou leur eau de boisson à des concentrations quotidiennes correspondant environ à 120 mg/kg de poids corporel, ont révélé la présence d'effets minimes, notamment des altérations biochimiques et des modifications histopathologiques au niveau du foie; toutefois d'autres études au cours desquelles des animaux avaient été exposés plus longtemps à des doses plus élevées n'ont pas mis d'effets en évidence. L'application cutanée de ASL a provoqué une intoxication générale ainsi que des effets localisés.

La dose journalière moyenne de ASL absorbée par la population générale, telle qu'on peut l'évaluer sur la base d'estimations de l'exposition de cette population par l'intermédiaire de l'eau de boisson, des ustensiles de cuisine et des aliments, est probablement beaucoup plus faible (de l'ordre de trois ordres de grandeur) que les concentrations qui se révèlent produire des effets insignifiants sur les animaux de laboratoire.

Les effets des AOS observés sur l'homme à l'occasion des quelques études dont on a connaissance, rapellent ceux qui ont été mis en évidence chez des animaux de laboratoire exposés aux ASL. Comme on ne dispose pas de données suffisantes pour évaluer la dose journalière moyenne d'AOS absorbée par la population générale ni sur les concentrations susceptibles de produire des effets chez l'homme et l'animal, il n'est pas possible de savoir avec certitude si l'exposition aux AOS présentes dans l'environnement représente un risque pour la santé humaine. Les concentrations d'AOS présentes dans les milieux auxquels l'homme pourrait être exposé, sont de toute manière plus faibles que celles des ASL, du fait de la moindre utilisation des AOS.

Des effets ont été observés systématiquement à l'occasion de quelques études à portée limitée effectuées sur des rats à qui l'on avait fait ingérer quotidiennement des AOS soit avec leur nourriture, soit dans leur eau de boisson à des concentrations supérieures ou égales à 200 mg/kg de poids corporel. Des applications topiques répétées ou prolongées produisent également des effets localisés sur la peau et les yeux. On ne dispose pas non plus de données suffisantes pour évaluer la dose journalière moyenne d'AS absorbée par la population générale. Toutefois, étant donné que les tensio-actifs à base d'AS ne sont pas utilisés aussi abondamment que ceux qui contiennent des ASL, il est probable que la dose d'AS absorbée est au moins mille fois plus faible que celle qui produit des effets sur l'animal.

1.10 Evaluation des effets sur l'environnement

Les ASL, les AS et les AOS sont utilisés en grandes quantités et rejetés dans l'environnement avec les eaux usées. Pour évaluer le risque qui leur est attaché, il faut comparer les concentrations auxquelles l'exposition peut se produire avec celles qui ne provoquent aucun effet indésirable, cette comparaison pouvant être faite pour un certain nombre de milieux présents dans l'environnement. En ce qui concerne les tensio-actifs anioniques en général, les plus importants de ces milieux sont constitués par les stations de traitement des eaux usées, les eaux de surface, les sols amendés au moyen de sédiments et de boues d'égout, ainsi que les eaux estuarielles et marines. Les composés subissent une biodécomposition (depuis les premiers stades jusqu'à leur dégradation ultime) ainsi qu'une adsorption, qui réduisent leur concentration dans l'environnement ainsi que leur biodisponibilité. Le racourcissement de la chaîne latérale alkylée et la disparition de la structure du composé initial conduisent à des composés qui sont moins toxiques que les molécules de départ. Il importe d'en tenir compte lorsqu'on compare les résultats des épreuves en laboratoire aux effets qui pourraient se produire dans l'environnement. En outre, lorsqu'on évalue le risque associé à l'exposition, dans l'environnement, à ces trois types de tensio-actifs anioniques, il faut que les comparaisons entre les différentes épreuves de toxicité portent sur des composés dont la chaîne latérale à la même longueur.

Les effets des ASL sur les organismes aquatiques ont été très largement étudiés. Lors des épreuves de laboratoire effectuées en eau douce, ce sont les poissons qui se sont révélés les plus sensibles; ainsi la concentration sans effets observables pour un cyprin d'Amérique du Nord, *Pimephales promelas*, est d'environ 0,5 mg/litre (C_{12}); tous ces résultats ont été confirmés lors d'épreuves effectuées dans des conditions plus proches de la réalité. Pour ce qui est du phytoplancton, des épreuves de toxicité aiguë sur trois heures ont donné, pour la CE_{50}, des valeurs de 0,2–0,1 mg/litre (C_{12}–C_{13}), alors qu'on n'a constaté aucun effet sur l'abondance relative du plancton dans d'autres tests effectués à la concentration de 0,24 mg/litre ($C_{11,8}$). Il semble que les espèces marines soient légèrement plus sensibles que la plupart des autres groupes taxonomiques.

Ces trois types de composés anioniques se retrouvent dans l'environnement à des concentrations qui varient dans de larges limites. De ce fait, il n'est pas possible de procéder à une évaluation du risque écologique qui soit d'une portée générale. Toute évaluation du risque

doit s'appuyer sur une connaissance suffisante de l'exposition et des concentrations agissantes dans l'écosystème étudié.

Pour ce qui est de l'évaluation du risque imputable à la présence d'AS et d'AOS dans l'environnement, il faudra encore réunir des données précises sur l'exposition à ces composés. C'est pourquoi on utilise des modèles pour étudier l'exposition à ces produits dans les différents milieux qui en sont les récepteurs. En ce qui concerne les organismes aquatiques, les données toxicologiques sur les AS et les AOS sont relativement rares, notamment dans les cas d'exposition chronique à des concentrations constantes. Celles dont on dispose montrent que cette toxicité est analogue à celle des autres tensio-actifs anioniques.

Les organismes aquatiques halophiles se révèlent plus sensibles que les organismes dulçaquicoles à ces composés; toutefois leur concentration est plus faible dans l'eau de mer, sauf au débouché des émissaires d'eaux usées. La destinée et les effets de ces composés, qui sont présents dans les effluents déversés en mer, n'ont pas été étudiés en détail.

Pour évaluer la sûreté écologique de tensio-actifs tels que les ASL, les AOS et les AS, il faut comparer les concentrations effectives dans l'environnement à celles qui ne produisent aucun effet. Les besoins en matière de recherche sont déterminés non seulement par les propriétés intrensèques de tel ou tel produit chimique mais aussi par les modalités ou les tendances de sa consommation. Tous ces facteurs peuvent varier fortement d'une région à l'autre, aussi l'appréciation et l'évaluation des risques doivent-elles être effectuées région par région.

1.11 Recommandations pour la protection de la santé humaine et de l'environnement

1. Comme il peut y avoir exposition à des poussières sur les lieux de travail (au cours de la fabrication et de la préparation des différentes formules), il faut veiller à ce que les précautions habituelles d'hygiène et sécurité du travail soient respectées afin d'assurer la protection des travailleurs.

2. La composition des préparations destinées à la consommation des ménages et à l'usage industriel doit être étudiée pour éviter tout danger, en particulier lorsqu'il s'agit de produits destinés au nettoyage ou au lavage du linge à la main.

3. L'exposition à ces produits dans l'environnement et les effets qu'ils peuvent avoir doivent faire l'objet d'une surveillance appropriée afin que l'on puisse reconnaître à temps la présence de tout concentration excessive dans tel ou tel milieu.

1.12 Recommandations pour les recherches futures

Santé humaine

1. Etant donné que le contact cutané est la principale voie d'exposition humaine aux ASL, aux AOS et aux AS et que l'on ne dispose pas d'études à long terme suffisantes sur la toxicité cutanée ou la cancérogénicité de ces produits chez les animaux de laboratoire, il est recommandé de procéder à des études à long terme convenablement conçues au cours desquelles il sera procédé à l'application de ces composés sur la peau des animaux.

2. En raison de l'absence de données définitives sur la génotoxicité des AOS et des AS, il conviendrait de procéder à des études supplémentaires *in vivo* et *in vitro*.

3. En raison de l'insuffisance des études existantes concernant les effets toxiques de ces produits sur la reproduction et le développement, il conviendrait d'effectuer, sur des animaux de laboratoire, des études qui permettent d'obtenir des résultats définitifs sur la valeur des concentrations agissant ou au contraire, sans effets des ASL, des AOS et des AS.

4. Etant donné que l'on ne connaît pas de façon suffisamment précise l'exposition aux ASL, aux AOS et aux AS, il faudrait surveiller l'exposition de la population générale à ces produits, en particulier lorsque ces tensio-actifs sont utilisés pour le nettoyage et le lavage du linge à la main.

5. Etant donné que les ASL, les AOS et les AS peuvent favoriser le transport d'autres produits chimiques dans les différents milieux qui composent l'environnement et en faire varier la biodisponibilité et la toxicité dans les eaux de surface, les sédiments, les cours d'eau et les sols auxquels l'être humain pourrait se trouver exposé, il conviendrait d'étudier les interactions de ces produits avec d'autres substances présentes dans l'environnement et les conséquences qui en découlent pour la santé humaine.

Sûreté écologique

6. Des études supplémentaires devraient être effectuées afin d'élucider les mécanismes de l'adsorption et de la désorption des AOS et des AS. Elles devraient également porter sur le partage des ASL, des AOS et des AS entre les particules colloïdales en solution ou en suspension dans l'eau. Il faudrait effectuer une modélisation mathématique des coefficients de sorption et valider les modèles obtenus en fonction des paramètres physicochimiques.

7. En cas d'exposition à des sols amendés à l'aide de boues d'égout ou à des sédiments de rivière, il faudrait étudier la biodécomposition des AOS et des AS dans ces milieux. L'étude des sédiments (dans les zones d'aérobiose et d'anaérobiose) devrait s'effectuer en aval des points où sont rejetées des eaux traitées ou non traitées ou des émissaires d'évacuation.

8. Il faudrait surveiller au niveau régional et national les concentrations en ASL, AOS et AS dans l'environnement afin d'obtenir des données sur l'exposition. Il faudrait également mettre au point des méthodes d'analyse permettant de déceler la présence de faibles teneurs en AOS et en AS dans les compartiments appropriés de l'environnement.

9. Il faudrait établir des bases de données nationales sur la concentration des ASL, AOS et AS dans les eaux usées et les cours d'eau ainsi que sur les différents types de stations d'épuration, leur implantation et leur efficacité, afin de mieux étudier l'impact des décharges dans l'environnement.

10. Il faudrait effectuer des études à long terme sur la toxicité des AOS et des AS pour les poissons (espèces d'eau douce et espèces marines) et des invertébrés aquatiques, afin d'en établir la sensibilité relative.

ALKILSULFONATOS LINEALES Y SUSTANCIAS RELACIONADOS

1. RESUMEN GENERAL, EVALUACIÓN Y RECOMENDACIONES

1.1 Identidad y métodos analíticos

Los alkilsulfonatos lineales (ASL), los a-olefinsulfonatos (AOS) y los alkilsulfatos (AS) son sustancias tensioactivas aniónicas con moléculas que se caracterizan por tener un grupo hidrófobo y uno hidrófilo (polar). Las mezclas comerciales están formadas por isómeros y homólogos de compuestos relacionados entre sícon distintas propiedades fisicoquímicas, obteniéndose formulaciones con diversas aplicaciones.

Los ASL, los AOS y los AS se pueden analizar por métodos no específicos. El ensayo que se suele utilizar es el de las sustancias que reaccionan con el azul de metileno, es decir, todas las que contienen un grupo aniónico e hidrófobo. Por consiguiente, si se utiliza para muestras del medio ambiente se producen interferencias analíticas; por otra parte, la sensibilidad de este método es de unos 0,02 mg/litro. Aunque se han buscado alternativas no específicas a este método, su uso no es habitual. En el análisis del medio ambiente sólo hay métodos específicos para los ASL y los AS. Para el análisis de los AOS se dispone de un método mejorado basado en la reactividad del azul de metileno y la cromatografía líquida de alto rendimiento (HPLC).

Los ASL son sustancias no volátiles que se forman por la sulfonación del alkilbenceno lineal. Los productos comerciales son siempre mezclas de homólogos con la cadena alkilo de distintas longitudes (C_{10}–C_{13} o C_{14}) e isómeros que difieren en las posiciones del anillo de fenilo (2–5 fenil). En las muestras del medio ambiente y en otras matrices se pueden determinar todos los homólogos e isómeros de los ASL por medio de métodos analíticos específicos como la HPLC, la cromatografía de gases y la cromatografía de gases–espectrometría de masas.

Los AOS son sustancias no volátiles producidas por la sulfonación de las a-olefinas. Son mezclas de dos compuestos, el alkensulfonato de sodio y el sulfonato de hidroxialkano, con longitudes de la cadena alkilo de C_{14}–C_{18}.

Los AS son compuestos no volátiles producidos por la sulfatación de alcoholes oleoquímicos o petroquímicos. Son mezclas de homólogos con longitudes de la cadena alkilo de C_{10}–C_{18}. Se están perfeccionando métodos analíticos específicos para la vigilancia del medio ambiente.

1.2 Fuentes de exposición humana y ambiental

Los ASL, los AOS y los AS se utilizan como ingredientes activos en productos de uso doméstico y de aseo personal y en aplicaciones especializadas. Una vez utilizadas, dichas sustancias detergentes pasan al medio ambiente en las aguas residuales.

Se dan casos de exposición en el trabajo a estas sustancias. La exposición de la población humana general y de los organismos del medio ambiente depende de la aplicación de los ASL, los AOS y los AS (y de otras sustancias tensioactivas), de las prácticas de tratamiento de las aguas residuales y de las características del medio ambiente al que llegan.

En 1990, el consumo mundial fue de unos dos millones de toneladas de ASL, 86 000 toneladas de AOS y 289 000 toneladas de AS.

1.3 Concentraciones en el medio ambiente

1.3.1 Alkilsulfonatos lineales

Las concentraciones de ASL se han determinado cuantitativamente por medio de un método analítico sensible específico en casi todos los compartimentos del medio ambiente en los que pueden estar presentes. Las concentraciones disminuyen progresivamente en el siguiente orden: aguas residuales > efluente tratado > aguas superficiales > mar.

En las zonas donde los ASL son las sustancias tensioactivas predominantes utilizadas, las concentraciones suelen ser de 1–10 mg/litro en las aguas residuales, 0,05–0,1 mg/litro en los efluentes sometidos a un tratamiento biológico, 0,05–0,6 mg/litro en los efluentes tratados con un filtro de goteo, 0,005–0,05 mg/litro en las aguas superficiales por debajo de los desagües de aguas residuales (con concentraciones que disminuyen con rapidez a 0,01 mg/litro corriente abajo del desagüe), < 1–10 mg/kg en los sedimentos fluviales (≤ 100 mg/kg en los sedimentos muy contaminados cerca de las zonas de vertido), 1–10 g/kg en los fangos de alcantarillado y < 1–5 mg/kg en los suelos tratados con fangos (al principio 5–10 mg/kg; se ha registrado

una concentración de ≤ 50 mg/kg después de aplicaciones anormalmente elevadas de fangos). Las concentraciones de ASL en las aguas de estuario son de 0,001–0,01 mg/litro, aunque hay niveles más elevados en los lugares donde se vierten directamente aguas residuales. Las concentraciones en el agua marina cercana a la costa son < 0,001–0,002 mg/litro.

Hay que señalar que las concentraciones de ASL en el medio ambiente varían mucho. Esto se debe a diferencias en los métodos analíticos, las características de los lugares de muestreo (que van desde zonas muy contaminadas con un tratamiento inadecuado de las aguas residuales hasta zonas donde dichas aguas se someten a un tratamiento a fondo), la estación (los valores pueden ser en una el doble que en otra) y el consumo de ASL.

La vigilancia del medio ambiente pone de manifiesto que no se ha producido acumulación de ASL en sus compartimentos a lo largo del tiempo. Las concentraciones en el suelo no aumentan con el tiempo, sino que disminuyen debido a la mineralización. Los ASL no se degradan en condiciones estrictamente anaerobias (para formar metano), por lo que no se puede concluir que estén mineralizados en sedimentos anaerobios. Con la utilización presente, el ritmo de asimilación de ASL en todos los compartimentos del medio ambiente que los reciben es igual al ritmo de entrada, por lo que la situación es estable.

1.3.2 *α-Olefinsulfonatos y alkilsulfatos*

Los datos disponibles sobre las concentraciones de AOS en el medio ambiente son limitados debido a la dificultad para analizarlos en las muestras de dicho medio. Hay métodos colorimétricos no específicos (como el basado en el azul de metileno) que permiten detectar sustancias tensioactivas aniónicas en general, pero se ven afectados por interferencias analíticas y no son idóneos para determinar concentraciones determinadas de AOS. Se está preparando un método específico para medir los AS en muestras del medio ambiente.

En estudios de laboratorio se ha observado que los AOS y los AS se mineralizan con rapidez en todos los compartimentos del medio ambiente y prácticamente se eliminan del todo de las aguas residuales durante el tratamiento. Las concentraciones en el agua superficial, los sedimentos, el suelo, el agua de estuario y el medio marino son probablemente bajas. Se ha comprobado que la concentración de AOS en el agua fluvial es pequeña.

1.4 Transporte, distribución y transformación en el medio ambiente

A temperaturas por debajo de 5–10 °C, la cinética de la biodegradación de los ASL, los AOS y los AS se reduce debido a la disminución de la actividad microbiana.

1.4.1 *Alkilsulfonatos lineales*

Las vías de entrada de los ASL en el medio ambiente varían de un país a otro, pero la principal es el vertido de las depuradoras de aguas residuales. Cuando no hay depuradoras o son inadecuadas, las aguas residuales se pueden verter directamente en los ríos, los lagos o el mar. Otra vía de entrada de ASL en el medio ambiente es la dispersión de fangos de alcantarillado en las tierras cultivadas.

Durante su recorrido hasta llegar al medio ambiente, los ASL se eliminan mediante una combinación de adsorción y biodegradación primaria y final. Los ASL se adsorben sobre superficies coloidales y partículas en suspensión, con unos coeficientes medidos de adsorción de 40–5200 litros/kg, en función de los medios y de la estructura de los ASL. Se biodegradan en el agua superficial (semivida de 1–2 días), los sedimentos aerobios (1–3 días) y los sistemas marinos y de estuarios (5–10 días).

Durante el tratamiento primario de las aguas residuales se adsorbe alrededor del 25% (intervalo, 10–40%) de los ASL en los fangos residuales y se elimina con ellos. No se eliminan durante la digestión anaerobia de los fangos, sino durante su tratamiento aerobio, con una semivida de unos 10 días. Tras la aplicación de fangos al suelo, en general se degrada el 90% de los ASL en tres meses, con una semivida de 5–30 días.

Los factores de concentración en el organismo completo para los ASL oscilan entre 100 y 300 para la suma de los ASL-^{14}C y los metabolitos de ^{14}C. Los peces los absorben sobre todo por las branquias, distribuyéndose después al hígado y la vesículas biliar tras la biotransformación. Los ASL se excretan con rapidez, por lo que no hay pruebas de que se produzca bioampliación.

1.4.2 *α-Olefinsulfonatos*

Los datos disponibles sobre el transporte, distribución y transformación en el medio ambiente para los AOS son más escasos que

para los ASL. Cabe suponer que los AOS llegan al medio ambiente de manera análoga a la establecida para los ASL, los AS y otras sustancias tensioactivas detergentes, y su destino en él es semejante al de los ASL y los AS. En condiciones aerobias se biodegradan fácilmente y la biodegradación primaria se completa en 2–10 días, en función de la temperatura. Son limitados los datos disponibles sobre la bioacumulación de los AOS; en peces no se observó ninguna. No hay datos relativos a la degradación abiótica.

1.4.3 *Alkilsulfatos*

Los AS llegan al medio ambiente por mecanismos análogos a los de los ASL y los AOS.Son fácilmente biodegradables en condiciones aerobias y anaerobias en el laboratorio y en el medio ambiente; la biodegradación primaria se termina en 2–5 días. El factor de bioconcentración en el organismo entero oscila entre 2 y 73 y varía con la longitud de la cadena de los homólogos de los AS. Los peces absorben, distribuyen, biotransforman y excretan los AS de la misma manera que los ASL y no se produce bioconcentración en los organismos acuáticos.

1.5 Cinética

Los ASL, los AOS y los AS se absorben fácilmente en el aparato digestivo y se distribuyen ampliamente por todo el organismo, con una metabolización extensa. En los ASL se produce ω- y β-oxidación. Las sustancias originales y los metabolitos se excretan sobre todo a través de los riñones, aunque una parte de la cantidad absorbida se puede excretar en forma de metabolitos en las heces por excreción biliar. Parece que por la piel intacta solamente se absorben cantidades mínimas de ASL, AOS y AS, aunque el contacto prolongado puede poner en peligro la integridad de la barrera cutánea, permitiendo así una mayor absorción; las concentraciones elevadas pueden reducir el tiempo necesario para la penetración.

1.6. Efectos en los animales de laboratorio y en los sistemas de prueba *in vitro*

Los valores de la DL_{50} por vía oral para las sales sódicas de los ASL fueron de 404–1470 mg/kg de peso corporal en ratas y de 1259–2300 mg/kg en ratones, lo cual parece indicar que las ratas son más sensibles que los ratones a la toxicidad de los ASL. En ratones se midió para una sal sódica de AOS una DL_{50} por vía oral de

3000 mg/kg de peso corporal. Los valores de la DL_{50} de AS por vía oral en ratas fueron de 1000–4120 mg/kg de peso corporal. Los ASL, AOS y AS irritan la piel y los ojos.

Se han descrito efectos mínimos, entre ellos alteraciones bioquímicas y cambios histopatológicos en el hígado, en estudios de toxicidad subcrónica en los que se administraron ASL a ratas con los alimentos o el agua de beber en concentraciones equivalentes a dosis superiores a 120 mg/kg de peso corporal al día. Aunque en un estudio se observaron cambios estructurales de las células hepáticas a dosis menores, al parecer eran reversibles. En otros estudios no se detectaron efectos con dosis análogas, pero tal vez el examen de los órganos fuera más detenido en el primer estudio. Se han notificado efectos en la reproducción, por ejemplo menor tasa de gestación y pérdida de crías, en animales que recibieron dosis > 300 mg/kg al día. Se observaron cambios histopatológicos y bioquímicos tras la aplicación cutánea de larga duración a ratas de soluciones > 5% de ASL y después de la aplicación durante 30 días de 60 mg/kg de peso vivo en la piel de cobayas. La aplicación cutánea repetida de soluciones ≥ 0,3% de ASL indujo efectos fetotóxicos y en la reproducción, pero también se observó toxicidad materna.

Son escasos los datos disponibles de estudios en animales experimentales que permitan evaluar los posibles efectos de los AOS en el ser humano. No se observó ningún efecto en ratas que recibieron por vía oral dosis de 250 mg/kg de peso corporal al día en aplicación crónica, pero se apreció fetotoxicidad en conejas a las que se administró una dosis tóxica para la madre de 300 mg/kg de peso corporal al día. La aplicación de AOS a la piel y los ojos de animales experimentales indujo efectos locales.

Aunque se han investigado en varios estudios los efectos de la exposición de corta y larga duración de animales a los AS, en la mayoría de los casos el examen histopatológico fue inadecuado o el tamaño de los grupos pequeño; por otra parte, las dosis más altas utilizadas en los estudios de larga duración no produjeron ningún efecto tóxico, de manera que no se pudo establecer un NOAEL. Sin embargo, se han descrito habitualmente efectos en ratas que recibían AS en los alimentos o el agua de beber a concentraciones equivalentes a 200 mg/kg de peso corporal al día o más. Se han observado efectos locales en la piel y los ojos tras la aplicación tópica de concentraciones aproximadas del 0,5% de AS o más. A concentraciones más elevadas se han registrado efectos de toxicidad materna y fetotóxicos.

La mayoría de los estudios de larga duración son inadecuados para evaluar el potencial carcinogénico de los ASL, los AOS y los AS en animales experimentales, debido a factores como el pequeño número de animales, el número limitado de dosis, la ausencia de una dosis tolerada máxima y la limitación del examen histopatológico en la mayoría de los estudios. En los casos en que se describieron de manera apropiada los hallazgos patológicos no se utilizaron dosis toleradas máximas y las dosis no produjeron efectos tóxicos. Teniendo presentes estas limitaciones, sin embargo, en los estudios en los que se administraron por vía oral ASL, AOS y AS no se obtuvo ninguna prueba de carcinogenicidad; en estudios de larga duración de aplicación de AOS en la piel con un pincel no se observó ningún efecto.

Según los limitados datos disponibles, no parece que estas sustancias tengan efectos genotóxicos *in vivo* o *in vitro*.

1.7 Efectos en el ser humano

Los resultados obtenidos en pruebas de parche demuestran que la piel humana puede tolerar el contacto con soluciones de ASL, AOS o AS de hasta un 1% durante 24 horas con la única reacción de una irritación leve. Estas sustancias tensioactivas provocaron la pérdida de lípidos de la superficie de la piel, la elución del factor hidratante natural y la desnaturalización de las proteínas de la capa epidérmica externa y aumentaron la permeabilidad y la hinchazón de esta capa. Los ASL, los AOS y los AS no indujeron sensibilización cutánea en voluntarios y no se ha encontrado ninguna prueba definitiva de que induzcan la formación de eczemas. No se han comunicado lesiones graves ni muertes tras la ingestión accidental de estas sustancias tensioactivas por personas.

1.8 Efectos en el medio ambiente

1.8.1 Alkilsulfonatos lineales

1.8.1.1 Medio acuático

Los ASL han sido objeto de amplios estudios tanto en el laboratorio (estudios de corta y larga duración) como en condiciones más naturales (microcosmos y mesocosmos y estudios sobre el terreno). En general, la disminución de la cadena alkilo o la posición más interna del grupo fenilo van acompañadas de una menor toxicidad. Las observaciones en

peces y en *Daphnia* indican que al disminuir la longitud de la cadena en una unidad (por ejemplo de C_{12} a C_{11}) la toxicidad se reduce prácticamente a la mitad.

Los resultados de las pruebas de laboratorio han sido los siguientes:

— *Microorganismos*: Los resultados son muy variables debido al uso de diversos sistemas de prueba (Por ejemplo, inhibición de fango activado; cultivos mixtos y especies individuales). Los valores de la CE_{50} oscilan entre 0,5 mg/litro (especie única) y > 1000 mg/litro. Para los microorganismos no hay relación lineal entre la longitud de la cadena y la toxicidad.

— *Plantas acuáticas*: Los resultados dependen mucho de las especies. Para los organismos de agua dulce, los valores de la CE_{50} son de 10–235 mg/litro (C_{10}–C_{14}) en las algas verdes, de 5–56 mg/litro ($C_{11,1}$–C_{13}) en las algas cianofíceas, de 1,4–50 mg/litro ($C_{11,6}$–C_{13}) en las diatomeas y de 2,7–4,9 mg/litro ($C_{11,8}$) en las macrofitas; al parecer las algas marinas son aún más sensibles. En las algas es probable que no haya relación lineal entre la longitud de la cadena y la toxicidad.

— *Invertebrados*: Los valores de la $CL(E)_{50}$ aguda por lo menos en 22 especies de agua dulce son de 4,6–200 mg/litro (longitud de la cadena sin especificar; C_{13}) para los moluscos; 0,12–27 mg/litro (sin especificar; $C_{11,2}$–C_{18}) para los crustáceos; 1,7–16 mg/litro (sin especificar; $C_{11,8}$) para los gusanos; y 1,4–270 mg/litro (C_{10}–C_{15}) para los insectos. Los valores de la $CL(E)_{50}$ crónica son de 2,2 mg/litro ($C_{11,8}$) para los insectos y 1,1–2,3 mg/litro ($C_{11,8}$–C_{13}) para los crustáceos. La concentración crónica sin efectos observados (NOEC; basada en la letalidad o los efectos en la reproducción) es de 0,2–10 mg/litro (sin especificar; $C_{11,8}$) para los crustáceos. Parece que los invertebrados marinos son más sensibles, con valores de la CL_{50} de 1 a > 100 mg/litro (casi siempre C_{12}) para 13 especies y NOEC de 0,025–0,4 mg/litro (sin especificar en todas las pruebas) para siete especies ensayadas.

— *Peces*: Los valores de la CL_{50} aguda son de 0,1–125 mg/litro (C_{8}–C_{15}) para 21 especies de agua dulce; los valores de la $CL(E)_{50}$ crónica son de 2,4 y 11 mg/litro (sin especificar; $C_{11,7}$) para dos especies; y las NOEC de 0,11–8,4 y 1,8 mg/litro (sin especificar; $C_{11,2}$–C_{13}) para dos especies. También en este caso los peces marinos parecen ser más sensibles, con valores de la CL_{50} aguda de 0,05–7 mg/litro (sin especificar; $C_{11,7}$) para seis especies y de la CL_{50} crónica de 0,01–1 mg/litro (sin especificar) para dos especies. En la mayoría de los informes no se indicaba la longitud de la cadena. Para especies marinas se señaló una NOEC de < 0,02 mg/litro (C_{12}).

La longitud media de la cadena de los productos utilizados habitualmente en el comercio es C_{12}. Se han probado compuestos de numerosas longitudes de cadena en Daphnia magna y en peces, pero la longitud utilizada en otros organismos de agua dulce ha solido ser la de $C_{11,8}$. Los valores típicos de la $CL(E)_{50}$ para el ASL C_{12} son de 3–6 mg/litro en *Daphnia magna* y de 2–15 mg/litro en peces de agua dulce, y las NOEC crónicas típicas son de 1,2–3,2 mg/litro para Daphnia y de 0,48–0,9 mg/litro para los peces de agua dulce. Los valores típicos de la CL_{50} de los ASL con cadenas de esta longitud son en los peces marinos < 1–6,7 mg/litro.

Los organismos de agua salada, en particular los invertebrados, parecen ser más sensibles que los de agua dulce a los ASL. En los invertebrados, la acción inhibidora de los ASL sobre el calcio puede afectar a la disponibilidad de este ión para la morfogénesis. Los ASL tienen un efecto general sobre el transporte iónico. Los productos de la biodegradación y los subproductos de los ASL son 10–100 veces menos tóxicos que las sustancias de las que proceden.

Los resultados obtenidos en condiciones más reales son los siguientes: Se han utilizado ASL en todas las pruebas de agua dulce a varios niveles tróficos, como recintos cerrados en lagos (organismos inferiores), modelos de ecosistemas (sistemas de sedimentos y agua), ríos por debajo y por encima del desagüe de depuradoras de aguas residuales y corrientes experimentales. En casi todos los casos se emplearon ASL C_{12}. Al parecer las algas son más sensibles en verano que en invierno, puesto que los valores de la CE_{50} en tres horas fueron de 0,2–8,1 mg/litro después de la fotosíntesis, mientras que en los modelos de ecosistemas no se observó ningún efecto en la abundancia relativa de las comunidades de algas con 0,35 mg/litro. Los niveles sin efecto en estos estudios fueron de 0,24–5 mg/litro, en función del organismo y del parámetro ensayado. Estos resultados prácticamente coinciden con los de las pruebas de laboratorio.

1.8.1.2 *Medio terrestre*

Se dispone de información acerca de las plantas y las lombrices de tierra. Las NOEC para siete especies de plantas en pruebas realizadas con soluciones de nutrientes son < 10–20 mg/litro; la correspondiente a tres especies en suelo, mediante pruebas basadas en el crecimiento fue de 100 mg/litro (C_{10}–C_{13}). La CL_{50} en 14 días fue para las lombrices de tierra > 1000 mg/kg.

1.8.1.3 Aves

En un estudio con pollos tratados en la alimentación se obtuvo una NOEC (basada en la calidad de los huevos) > 200 mg/kg.

1.8.2 α-Olefinsulfonatos

Los datos acerca de los efectos de los AOS en los organismos acuáticos y terrestres son limitados.

1.8.2.1 Medio acuático

Solamente se dispone de datos de pruebas de laboratorio:

— *Algas*: Los valores de la CE_{50} que se han descrito para las algas verdes son > 20–65 mg/litro (C_{16}–C_{18}).

— *Invertebrados*: Para *Daphnia* se han notificado valores de la CL_{50} de 19 y 26 mg/litro (C_{16}–C_{18}).

— *Peces*: Los valores de la CL_{50} son de 0,3–6,8 mg/litro (C_{12}–C_{18}) para nueve especies de peces. De los estudios de corta duración realizados en la trucha común (*Salmo trutta*), *Idus melanotus* y *Rasbora heteromorpha*, cabe concluir que la toxicidad de los compuestos C_{14}–C_{16} es unas cinco veces inferior a la de los C_{16}–C_{18}, con valores de la CL_{50} (todas las concentraciones medidas) de 0,5–3,1 (C_{16}–C_{18}) y 2,5–5,0 mg/litro (C_{14}–C_{16}). En dos estudios de larga duración en la trucha arcoiris se comprobó que el crecimiento era el parámetro más sensible, con una CE_{50} de 0,35 mg/litro. En un pez marino, el pardete (*Mugal cephalus*), el valor de la CL_{50} en 96 horas fue de 0,70 mg/litro.

1.8.2.2 Medio terrestre

En un estudio de plantas con soluciones de nutrientes, la NOEC fue de 32–56 mg/litro. En un estudio con pollos tratados en la alimentación, se notificó una NOEC (basada en la calidad de los huevos) > 200 mg/kg.

1.8.3 Alkilsulfatos

1.8.3.1 Organismos acuáticos

Se han realizado estudios de los AS de corta y larga duración y uno en condiciones más reales. Su toxicidad también depende de la longitud

de la cadena alkilo; no se disponía de información sobre diferencias de toxicidad entre los AS lineales y ramificados.

Los resultados de las pruebas de laboratorio son los siguientes:

— *Microorganismos*: Los valores de la CE_{50} en un conjunto de microorganismos marinos fueron de 2,1–4,1 mg/litro (C_{12}). Las NOEC en *Pseudomonas putida* fueron de 35–550 mg/litro (C_{16}–C_{18}).

— *Plantas acuáticas*: Los valores de la CE_{50} fueron > 20–65 mg/litro (C_{12}–C_{13}) en algas verdes y de 18–43 mg/litro (C_{12}) en macrofitas. Las NOEC fueron de 14–26 mg/litro (C_{12}–C_{16}/C_{18}) en algas verdes.

— *Invertebrados*: Los valores de la $CL(E)_{50}$ fueron de 4–140 mg/litro (C_{12}/C_{15}–C_{16}/C_{18}) en especies de agua dulce y de 1,7–56 mg/litro (todos C_{12}) en especies marinas. La NOEC crónica en *Daphnia magna* fue de 16,5 mg/litro (C_{16}/C_{18}) y en especies marinas de 0,29–0,73 mg/litro (longitud de la cadena sin especificar).

— *Peces*: Los valores de la CL_{50} fueron de 0,5–5,1 mg/litro (longitud de la cadena sin especificar o C_{12}–C_{16}) en especies de agua dulce y de 6,4–16 mg/litro (todos C_{12}) en especies marinas. No había estudios de larga duración.

Hay que señalar que muchos de estos estudios se llevaron a cabo en condiciones estáticas. Los AS son fácilmente biodegradables, por lo que se puede haber infravalorado su toxicidad. En un estudio de 48 horas con Oryzias latipes, los valores de la CL_{50} fueron de 46, 2,5 y 0,61 mg/litro (concentraciones medidas) para los compuestos C_{12}, C_{14} y C_{16} respectivamente. Este y otros estudios indican que la toxicidad difiere en un factor de cinco por cada dos unidades de longitud de la cadena. En un estudio de biocenosis de paso de corriente con compuestos de C_{16}–C_{18} se observó una NOEC de 0,55 mg/litro.

1.8.3.2 *Organismos terrestres*

Se han notificado valores de la NOEC > 1000 mg/kg (C_{16}–C_{18}) en lombrices de tierra y en nabos.

1.9 Evaluación del riesgo para la salud humana

Los ASL son los agentes tensioactivos más utilizados en detergentes y productos de limpieza. También se utilizan AOS y AS en detergentes

y en productos de aseo personal. Por consiguiente, la principal vía de exposición humana es el contacto cutáneo. Pueden ingerirse pequeñas cantidades de ASL, AOS y AS con el agua potable y debido a la presencia de residuos en utensilios y alimentos. Aunque la información de que se dispone es limitada, la ingesta diaria de ASL por esos medios se puede estimar en unos 5 mg/persona. Puede producirse exposición en el trabajo a los ASL, AOS y AS durante la formulación de diversos productos, pero no hay datos acerca de los efectos de una exposición crónica a estas sustancias en el ser humano.

Los ASL, AOS y AS pueden irritar la piel después de un contacto cutáneo repetido o prolongado con concentraciones análogas a las presentes en los productos sin diluir. En los cobayas los AOS pueden inducir sensibilización cutánea cuando el nivel de sulfona g insaturada es superior a unas 10 ppm.

Los estudios disponibles de larga duración en animales experimentales son insuficientes para evaluar el potencial carcinogénico de los ASL, AOS y AS, debido a factores como el diseño del estudio, el uso de un número pequeño de animales, el ensayo de dosis insuficientes y lo limitado del examen histopatológico. En los escasos estudios en los que se administró ASL, AOS o AS a animales por vía oral no se observaron signos de carcinogenicidad; también fueron negativos los resultados de los estudios de larga duración en los que se administraron AOS mediante aplicación cutánea. Estas sustancias no parecen ser genotóxicas in vivo o in vitro, aunque se tienen noticias de pocos estudios.

Se han descrito efectos mínimos, entre ellos alteraciones bioquímicas y cambios histopatológicos hepáticos, en estudios subcrónicos en los que se administraron ASL a ratas en la alimentación o el agua de beber a concentraciones equivalentes a una dosis aproximada de 120 mg/kg de peso corporal al día, aunque no se observó ningún efecto en estudios en los que los animales estuvieron expuestos a dosis más elevadas durante períodos más largos. La aplicación cutánea de ASL ocasionó tanto toxicidad sistémica como efectos locales.

La ingesta diaria media de ASL de la población general, con arreglo a estimaciones limitadas de la exposición por medio del agua potable, utensilios y alimentos probablemente sea muy inferior (unas tres veces menor) a los niveles con los que se ha observado que inducen efectos leves en los animales experimentales.

Los efectos de los AOS observados en el ser humano en los escasos estudios disponibles son parecidos a los descritos en los animales expuestos a los ASL. Debido a que son insuficientes los datos para estimar la ingesta diaria media de AOS de la población general y los relativos a los niveles que inducen efectos en el ser humano y en los animales, no es posible evaluar con suficiente confianza si la exposición a los AOS en el medio ambiente representa un riesgo para la salud humana. Sin embargo, los niveles de AOS en medios a los que puede estar expuesto el ser humano probablemente sean inferiores a los de ASL, puesto que se utilizan menos.

Se han descrito repetidamente efectos en un pequeño número de estudios limitados en ratas que recibieron AS en la alimentación o en el agua de beber en concentraciones equivalentes a dosis de 200 mg/kg de peso corporal al día o más. Se han observado efectos locales en la piel y en los ojos tras una aplicación tópica repetida o prolongada. Los datos disponibles son insuficientes para estimar la ingesta diaria media de AS de la población general. Sin embargo, dado que no se utilizan tanto agentes tensioactivos con AS como los que contienen ASL, la ingesta de AS será probablemente como mínimo tres veces inferior a las dosis que se ha demostrado que inducen efectos en los animales.

1.10 Evaluación de los efectos en el medio ambiente

Los ASL, los AOS y los AS se utilizan en grandes cantidades y se liberan en el medio ambiente por medio de las aguas residuales. Para evaluar el riesgo es preciso comparar las concentraciones de exposición con las que no producen efectos adversos, y esto se puede hacer para varios compartimentos del medio ambiente. Para los agentes tensioactivos aniónicos en general, los compartimentos más importantes son las depuradoras de aguas residuales, las aguas superficiales, los suelos tratados con sedimentos y fangos y los medios estuarinos y marinos. Se produce tanto biodegradación (primaria y final) como adsorción, por lo que disminuyen las concentraciones y la biodisponibilidad en el medio ambiente. Con la reducción de la longitud de la cadena y la pérdida de la estructura original se forman sustancias menos tóxicas que la primera. Es importante tener en cuenta estos aspectos a la hora de interpretar los resultados de laboratorio con los posibles efectos en el medio ambiente. Por otra parte, al evaluar el riesgo asociado con la exposición del medio ambiente a estos tres compuestos aniónicos se debe establecer una comparación con los resultados de las pruebas de toxicidad de sustancias cuya cadena tenga la misma longitud.

Se han realizado abundantes pruebas de los efectos de los ASL en los organismos acuáticos. En las pruebas de laboratorio con agua dulce parece que los peces eran las especies más sensibles; la NOEC para Pimephales promelas fue de unos 0,5 mg/litro (C_{12}), y estos resultados se confirmaron en pruebas realizadas en condiciones más reales. en el fitoplancton se han observado diferencias: en ensayos de toxicidad agua de tres horas los valores de la CE_{50} fueron de 0,2–0,1 mg/litro (C_{12}–C_{13}), mientras que no se detectaron efectos en la abundancia relativa en otras pruebas con 0,24 mg/litro ($C_{11,8}$). Parece que las especies marinas eran ligeramente más sensibles que la mayoría de los otros grupos taxonómicos.

En el medio ambiente hay una amplia gama de concentraciones de las tres sustancias aniónicas, como se ha puesto de manifiesto en las numerosas mediciones de los ASL. Debido a esta amplia gama, no se puede hacer una evaluación del riesgo de estas sustancias para el medio ambiente de aplicación general. Para evaluar el riesgo se deben conocer de manera apropiada las concentraciones de exposición y las que tienen efectos en el ecosistema que interesa.

Se necesitan datos precisos sobre la exposición a los AS y los AOS si se quiere hacer una evaluación del riesgo para el medio ambiente. Por consiguiente se están utilizando modelos a fin de evaluar las concentraciones de exposición en los compartimentos del medio ambiente que los reciben. Los datos sobre la toxicidad de los AS y los AOS para los organismos acuáticos, especialmente después de una exposición crónica a concentraciones estables, son relativamente escasos. Los datos disponibles indican que la toxicidad de estos productos es análoga a la de otras sustancias tensioactivas aniónicas.

Los organismos de agua salada parecen ser más sensibles que los de agua dulce a estos compuestos; sin embargo, su concentración es menor en el agua del mar, excepto cerca de los desagües de alcantarillados. No se han investigado con detalle su destino y sus efectos en las aguas residuales vertidas en el mar.

Si se desea evaluar la inocuidad para el medio ambiente de agentes tensioactivos como los ASL, los AOS y los AS hay que comparar las concentraciones reales en el medio ambiente con las que no tienen ningún efecto. Las necesidades de investigación se determinan no sólo por las propiedades intrínsecas de un producto químico, sino también por sus características o la tendencia del consumo. Estos varían considerablemente entre las distintas zonas geográficas, por lo que la evaluación debe ser de ámbito regional.

1.11 Recomendaciones para la protección de la salud humana y el medio ambiente

1. Puesto que en el lugar de trabajo se puede producir exposición al polvo (durante la elaboración y formulación), deben utilizarse prácticas normalizadas de higiene del trabajo a fin de asegurar la protección de la salud de los trabajadores.

2. La composición de las formulaciones para uso privado e industrial se debe diseñar de manera que se evite el riesgo, especialmente en las formulaciones utilizadas para la limpieza o el lavado a mano.

3. La exposición y los efectos en el medio ambiente se deben vigilar de manera apropiada con objeto de tener una indicación pronta de cualquier acumulación excesiva en los compartimentos pertinentes del medio ambiente.

1.12 Recomendaciones de nuevas investigaciones

Salud humana

1. Debido a que la piel es la principal vía de exposición humana a los ASL, los AOS y los AS y a que no se dispone de estudios adecuados de larga duración acerca de la toxicidad cutánea o la carcinogenicidad en animales experimentales, se recomienda la realización de estudios de larga duración debidamente diseñados de aplicación cutánea de estas sustancias.

2. Ante la falta de datos definitivos sobre la genotoxicidad de los AOS y los AS, deben llevarse a cabo nuevos estudios in vivo e in vitro.

3. A la vista de la insuficiencia de los estudios disponibles sobre la toxicidad en la reproducción y el desarrollo, se han de realizar estudios definitivos en animales de laboratorio a fin de obtener datos relativos a los efectos de los ASL, los AOS y los AS y los niveles con efectos y sin ellos.

4. Puesto que la exposición a los ASL, los AOS y los AS no está debidamente definida, se debe vigilar la exposición de la población general, en particular cuando estas sustancias tensioactivas se utilizan en la limpieza y el lavado a mano.

5. Debido a que los ASL, los AOS y los AS pueden potenciar el transporte de otras sustancias químicas y regular su biodisponibilidad y toxicidad en las aguas superficiales, los sedimentos fluviales y los suelos a los que puede estar expuesto el ser humano, deben investigarse las interacciones con otras sustancias químicas del medio ambiente y las consecuencias para las personas.

Inocuidad para el medio ambiente

6. Deben realizarse nuevos estudios sobre los mecanismos de adsorción y desorción de los AOS y los AS. También se debe estudiar el reparto de los ASL, los AOS y los AS entre las partículas coloidales disueltas y suspendidas en el agua. Hay que elaborar modelos matemáticos de los coeficientes de sorción y validarlos con arreglo a parámetros fisicoquímicos.

7. Se han de realizar estudios de la biodegradación de los AOS y los AS en suelos tratados con fangos y en sedimentos fluviales (zonas aerobias y anaerobias) corriente abajo de los vertidos de aguas residuales tratadas y sin tratar.

8. Se deben vigilar en los ámbitos regional y nacional las concentraciones de ASL, AOS y AS en el medio ambiente, a fin de obtener información acerca de la exposición. Se han de preparar métodos analíticos para la detección de niveles bajos de AOS y AS en los compartimentos pertinentes del medio ambiente.

9. Hay que organizar bases de datos nacionales sobre las concentraciones de ASL, AOS y AS en las aguas residuales y en los ríos y sobre los tipos, la eficacia y el lugar de las depuradoras de aguas residuales, con objeto de facilitar la evaluación de los efectos de los vertidos de estas sustancias tensioactivas en el medio ambiente.

10. Deben realizarse estudios de la toxicidad de los AOS y los AS en los peces (de agua dulce y marinos) y los invertebrados acuáticos para establecer la sensibilidad relativa de estas especies.

Food additives and contaminants in food, principles for the safety assessment of (No. 70, 1987)
Formaldehyde (No. 89, 1989)
Genetic effects in human populations, guidelines for the study of (No. 46, 1985)
Glyphosate (No. 159, 1994)
Guidance values for human exposure limits (No. 170, 1994)
Heptachlor (No. 38, 1984)
Hexachlorobutadiene (No. 156, 1994)
Alpha- and beta-hexachlorocyclohexanes (No. 123, 1992)
Hexachlorocyclopentadiene (No. 120, 1991)
n-Hexane (No. 122, 1991)
Hydrazine (No. 68, 1987)
Hydrogen sulfide (No. 19, 1981)
Hydroquinone (No. 157, 1994)
Infancy and early childhood, principles for evaluating health risks from chemicals during (No. 59, 1986)
Inorganic lead (No. 165, 1995)
Isobenzan (No. 129, 1991)
Isophorone (No. 174, 1995)
Kelevan (No. 66, 1986)
Lasers and optical radiation (No. 23, 1982)
Lead (No. 3, 1977)[a]
Lead - environmental aspects (No. 85, 1989)
Lindane (No. 124, 1991)
Linear alkylbenzene sulfonates and related compounds (No. 169, 1996)
Magnetic fields (No. 69, 1987)
Man-made mineral fibres (No. 77, 1988)
Manganese (No. 17, 1981)
Mercury (No. 1, 1976)[a]
Mercury - environmental aspects (No. 86, 1989)
Mercury, inorganic (No. 118, 1991)
Methomyl (No. 178, 1996)
2-Methoxyethanol, 2-ethoxyethanol, and their acetates (No. 115, 1990)
Methyl bromide (No. 166, 1995)
Methylene chloride (No. 32, 1984, 1st edition)
Methyl ethyl ketone (No. 143, 1992)
Methyl isobutyl ketone (No. 117, 1990)
Methylmercury (No. 101, 1990)
Methyl parathion (No. 145, 1992)
Mirex (No. 44, 1984)
Morpholine (No. 179, 1996)
Mutagenic and carcinogenic chemicals, guide to short-term tests for detecting (No. 51, 1985)
Mycotoxins (No. 11, 1979)
Mycotoxins, selected: ochratoxins, trichothecenes, ergot (No. 105, 1990)
Nephrotoxicity associated with exposure to chemicals, principles and methods for the assessment of (No. 119, 1991)
Neurotoxicity associated with exposure to chemicals, principles and methods for the assessment of (No. 60, 1986)
Nickel (No. 108, 1991)
Nitrates, nitrites, and *N*-nitroso compounds (No. 5, 1978)[a]
Nitrogen, oxides of (No. 4, 1977)[a]
2-Nitropropane (No. 138, 1992)
Noise (No. 12, 1980)[a]
Organophosphorus insecticides: a general introduction (No. 63, 1986)
Paraquat and diquat (No. 39, 1984)
Pentachlorophenol (No. 71, 1987)
Permethrin (No. 94, 1990)
Pesticide residues in food, principles for the toxicological assessment of (No. 104, 1990)
Petroleum products, selected (No. 20, 1982)
Phenol (No. 161, 1994)
d-Phenothrin (No. 96, 1990)
Phosphine and selected metal phosphides (No. 73, 1988)
Photochemical oxidants (No. 7, 1978)
Platinum (No. 125, 1991)
Polybrominated biphenyls (No. 152, 1994)
Polychlorinated biphenyls and terphenyls (No. 2, 1976, 1st edition)[a] (No. 140, 1992, 2nd edition)
Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (No. 88, 1989)
Progeny, principles for evaluating health risks associated with exposure to chemicals during pregnancy (No. 30, 1984)
1-Propanol (No. 102, 1990)
2-Propanol (No. 103, 1990)
Propachlor (No. 147, 1993)
Propylene oxide (No. 56, 1985)
Pyrrolizidine alkaloids (No. 80, 1988)
Quintozene (No. 41, 1984)
Quality management for chemical safety testing (No. 141, 1992)

---

[a] Out of print

THE ENVIRONMEN ıL ı ALT CRITER'A SERIES (continued)

Radiofrequency and microwaves
(No. 16, 1981)
Radionuclides, selected (No. 25, 1983)
Resmethrins (No. 92, 1989)
Selected synthetic organic fibres
(No. 151, 1993)
Selenium (No. 58, 1986)
Styrene (No. 26, 1983)
Sulfur oxides and suspended particulate
matter (No. 8, 1979)
Tecnazene (No. 42, 1984)
Tetrabromobisphenol A and derivatives
(No. 172, 1995)
Tetrachloroethylene (No. 31, 1984)
Tetradifon (No. 67, 1986)
Tetramethrin (No. 98, 1990)
Thallium (No. 182, 1995)
Thiocarbamate pesticides: a general
introduction (No. 76, 1988)
Tin and organotin compounds (No. 15, 1980)
Titanium (No. 24, 1982)
Toluene (No. 52, 1986)
Toluene diisocyanates (No. 75, 1987)
Toxicity of chemicals (Part 1), principles
and methods for evaluating the (No. 6, 1978)
Toxicokinetic studies, principles of
(No. 57, 1986)
Tributyl phosphate (No. 112, 1991)
Tributyltin compounds (No. 116, 1990)
Trichlorfon (No. 132, 1992)
1,1,1-Trichloroethane (No. 136, 1992)
Trichloroethylene (No. 50, 1985)
Tricresyl phosphate (No. 110, 1990)
Triphenyl phosphate (No. 111, 1991)
Tris- and bis(2,3-dibromopropyl) phosphate
(No. 173, 1995)
Ultrasound (No. 22, 1982)
Ultraviolet radiation
(No. 14, 1979, 1st edition)
(No. 160, 1994, 2nd edition)
Vanadium (No. 81, 1988)
Vinylidene chloride (No. 100, 1990)

To obtain further copies of monographs in this series, please write to the Office of Distribution and Sales, World Health Organization, 1211 Geneva 27, Switzerland (Fax No. 41-22-7914857)

Price: Sw.fr. 47.–
Price in developing countries: Sw.fr. 32.90

ISBN 92 4 157169 1