Environment Canada Environnement Canada

Canada

SUPPORTING DOCUMENT FOR NONYLPHENOL AND ITS ETHOXYLATES, CEPA PSL 2

M.R. Servos, R.J. Maguire, D.T. Bennie, H.-B. Lee, P.M Cureton, N. Davidson, R. Stutcliffe and D.F.K. Rawn

NWRI Contribution No. 00-029

Canadian Environmental Protection Act

Priority Substances List

Supporting Document for

Nonylphenol and Its Ethoxylates

March, 2000

M.R. Servos[1], R.J. Maguire[1], D.T. Bennie[1], H-B. Lee[1], P.M. Cureton[2], N. Davidson[2], R. Sutcliffe[2] and D.F.K. Rawn[2].

[1]National Water Research Institute, Environment Canada, Canada Centre for Inland Waters, 867 Lakeshore Road, Burlington, Ontario, L7R 4A6, [2]Commercial Chemicals Evaluation Branch, Environment Canada, Place Vincent Massey, 351 St. Joseph Boulevard, Hull, Quebec, K1A 0H3.

Management Perspective

Nonylphenol and nonylphenol ethoxylates (NP/NPEs) are currently being evaluated as a Priority Substance under the Canadian Environmental Protection Act. The supporting document represents the background reviews and analysis of the exposure, hazard and risk characterization for the Canadian environment. It includes a comprehensive review and assessment of the methods available for quantifying alkylphenols and alkylphenol polyethoxylates in environmental samples, the dominate sources and releases to the environment, fate and behavior in the environment and in treatment systems, environment exposure, bioaccumulation and effects. A risk characterization includes a comparison of the environmental concentrations to the levels expected to cause a wariety of environmental effects. These detailed reviews and analysis formed the basis for the PSL-2 risk assessment on NP/NPEs.

Abstract

Comprehensive literature reviews were done on the environmental sources, fate, distribution, bioaccumulation and effects of nonylphenol and nonylphenol ethoxylates (NP/NPEs). Surveys were conducted to determine the predominate sources and releases into the environment. The available data in the literature or available through government laboratories was collated and summarized. Literature and other data available on effects was also collated and summarized for each of the individual NP/NPEs. Data available for other alkylphenols and alkylphenol polyethoxylates was also summarized although it was not used in the PSL-2 assessment. A risk characterization was conducted by comparing the environmental concentrations either measured or predicted and the expected no effect concentrations. No effect concentrations were determined in several ways ranging from using the most sensitive endpoint with a safety factor applied, the distribution of measured effects or consideration of potential endocrine responses. This analysis was conducted for NP, nonylphenol ethoxylates (NP1EO, NP2EO, and NP3-17EO), nonlypheno ethoxycaroboxylates (NP1EC, NP2EC) and for the combined effects. The analysis was done for predicted environmental concentrations associated with textile mill. Pulp mill and municipal effluents, as well as measured concentrations in surface waters and sediments. This data was used as the basis for the PSL-2 risk assessment of NP/NPEs under the Canadian Environmental Protection Act.

Sommaire à l'intention de la direction

On évalue actuellement l'ajout du nonylphénol et de ses éthoxylates à la liste des substances d'intérêt prioritaire de la *Loi canadienne sur la protection de l'environnement*. Le document justificatif représente les études et les analyses de base sur l'exposition, le danger et la caractérisation des risques pour l'environnement canadien. Il présente une étude et une évaluation générales des méthodes disponibles pour l'analyse quantitative des alkylphénols et des polyéthoxylates d'alkylphénol dans des échantillons de l'environnement, leurs principales sources et rejets dans l'environnement, leur devenir et leur comportement dans l'environnement et dans les réseaux d'épuration de l'eau, l'exposition à ces substances dans l'environnement, leur bioaccumulation et leurs effets. On y trouve une caractérisation des risques comparant les concentrations dans l'environnement aux teneurs susceptibles de causer divers effets environnementaux. Ces études et analyses détaillées formaient la base de l'évaluation des risques des nonylphénols et de leurs éthoxylates prévue pour la LSIP n? 2.

Résumé

On a effectué des examens exhaustifs de la littérature sur les sources dans l'environnement, le devenir, la distribution, la bioaccumulation et les effets du nonylphénol et de ses éthoxylates. On a compilé et résumé les données disponibles dans la littérature ou dans les laboratoires du gouvernement sur leurs principaux rejets et sources dans l'environnement, ainsi que celles de la littérature et d'autres sources sur les effets du nonylphénol et de chacun de ses éthoxylates. On a également résumé les données disponibles sur d'autres alkylphénols et leurs polyéthoxylates, bien qu'elles n'aient pas servi à l'évaluation prévue pour LDSIP n? 2. On a effectué une caractérisation des risques en comparant les concentrations dans l'environnement, mesurées ou calculées, aux concentrations sans effet prévues. On a déterminé ces dernières de plusieurs façons, par exemple par l'utilisation des résultats les plus sensibles après l'application d'un facteur de sécurité, par la répartition des effets mesurés et par l'examen des réponses endocriniennes possibles. On a effectué ces analyses avec le nonylphénol, les éthoxylates de nonylphénol (NP1EO, NP2EO et NP3-17EO) et les éthoxycarboxylates de nonylphénol (NP1EC, NP2EC), et on a également étudié leurs effets combinés. On a aussi effectué des analyses pour vérifier les concentrations à effets environnementaux prévisibles associées à des usines de textile. De plus, on a analysé les effluents des usines de pâtes et ceux des municipalités, et on a mesuré les concentrations de ces substances dans les eaux de surface et les sédiments. Ces données ont servi à fonder les évaluations des risques du nonylphénol et de ses éthoxylates prévues pour la LSIP n? 2 de la *Loi canadienne sur la protection de l'environnement*.

TABLE OF CONTENTS

List of Tables

List of Figures

List of Appendices

LIST OF ACRONYMS AND ABBREVIATIONS

AP	alkylphenol
APEC	alkylphenol carboxylate on ethoxylate chain (as a general class)
APnEC	alkylphenol carboxylate (on ethoxylate chain); where n = specific number of EO groups
APE	alkylphenol ethoxylate (as a general class)
APnEO	alkylphenol ethoxylate; where n = specific number of ethoxylate (EO) groups
BAF	bioaccumulation factor
BCF	bioconcentration factor
b.w.	body weight
CAPE	alkylphenol with alkyl chain carboxylated
CAPEC	alkylphenol with carboxylate groups on both the alkyl and ethoxylate chains
CAS	Chemical Abstracts Service
CEPA	*Canadian Environmental Protection Act*
CTV	Critical Toxicity Value
dw	dry weight
EC_{50}	median effective concentration
EEV	Estimated Exposure Value
ENEV	Estimated No-Effects Value
K_{oc}	organic carbon/water partition coefficient
K_{ow}	octanol/water partition coefficient
LC_{50}	median lethal concentration
LD_{50}	median lethal dose
LOEC	Lowest-Observed-Effect Concentration
LOEAL	Lowest-Observed-Adverse-Effect-Level
LOEL	Lowest-Observed-Effect Level
MWWTP	municipal wastewater treatment plant
NOEC	No-Observed-Effect Concentration
NOEL	No-Observed-Effect Level
NP	nonylphenol
NPEC	nonylphenol carboxylate on ethoxylate chain (as a general class)
NPnEC	nonylphenol carboxylate on ethoxylate chain; where n = specific number of EO groups
NPE	nonylphenol ethoxylate (as a general class)
NPnEO	nonylphenol ethoxylate; where n = specific number of EO groups
OECD	Organization for Economic Co-operation and Development
OP	octylphenol
OPEC	octylphenol carboxylate on ethoxylate chain (as a general class)

OPnEC	octylphenol carboxylate on ethoxylate chain; where n = specific number of EO groups
OPE	octylphenol ethoxylate (as a general class)
OPnEO	octylphenol ethoxylate; where n = specific number of EO groups
pKa	negative logarithm of the acid dissociation constant
PSL	Priority Substances List
TEQ	Toxic Equivalency Quotient

Synopsis

In this Supporting Document, a review of the uses of nonylphenol (NP) and its ethoxylates (NPEs) in Canada and elsewhere, their modes of entry to the environment, analytical methods for their determination, and their persistence, occurrence and effects is presented, primarily from an aquatic perspective. This information provides the basis for the Priority Substance List (PSL-2) Assessment Report for NP and NPEs. In addition, recommendations are made for further research on the environmental occurrence, persistence, fate and effects of NP and NPEs.

Nonylphenol ethoxylates (NPEs) are a class of the broader group of compounds known as alkylphenol ethoxylates (APEs). NPEs are high volume chemicals which have been used for more than forty years as detergents, emulsifiers, wetting agents and dispersing agents. Nonylphenol polyethoxylate-containing products are used in many sectors, including textile processing, pulp and paper processing, paints, resins and protective coatings, oil and gas recovery, steel manufacturing, pest control products and power generation. A variety of cleaning products, degreasers and detergents are also available for institutional and domestic use. These products have numerous applications, including controlling deposits on machinery, cleaning equipment, scouring fibres, wetting and de-wetting agents, in dyeing, and in machine felt cleaning and conditioning and in product finishing. They are also used in a wide range of consumer products, including cosmetics, cleaners, and paints, and in a variety of applications.

In 1996, an estimated 17,200 tonnes of NPEs were available for use in Canada. The primary use of NP itself is in the production of NPEs, although NP has been used in pesticide formulations in Canada, and as a marker in fuel oil for taxation purposes in other countries. In addition, NP is used to produce *tris*-4-nonylphenol phosphite, which in turn is used as an antioxidant for rubber, as a poly(vinyl chloride) stabilizer, and in the manufacture of lube oil additives.

NPEs and their degradation products (e.g., NP) are not produced naturally. Their presence in the environment is solely a consequence of anthropogenic activity. NP and NPEs enter the environment primarily *via* industrial and MWWTP effluents (liquid and sludge), but also by direct discharge, although it is not known how significant the latter pathway is in Canada. Once NPEs are released to sewage treatment systems, several transformations can occur. The mechanism of degradation is complex, but in general, there is an initial loss of ethoxylate (EO) groups from the original moiety. Under aerobic and anaerobic treatment conditions, biodegradation to more toxic (and estrogenic) metabolites occurs. These products are NP, nonylphenol ethoxylate (NP1EO), nonylphenol diethoxylate (NP2EO), nonylphenoxyacetic acid (NP1EC) and nonylphenoxyethoxyacetic acid (NP2EC). Discharges from municipal

wastewater treatment plants provide the two major routes for environmental release of NPEs and their degradation products. The first route is by discharge of the final treated effluent to nearby receiving waters. The final effluent composition is dependent on the treatment process(es) used in the facility. Where only primary treatment is used, the effluent composition reflects the short hydraulic retention time and ethoxylated products (*i.e.*, NPEs with 3-20 ethoxy groups) are dominant, with minor amounts of NP, NP1EO and NP2EO, and NP1EC and NP2EC. Secondary-treated effluent composition is substantially different from primary-treated effluent. NPEs with 3-20 ethoxy groups constitute a much smaller amount of the total nonylphenolic compounds in secondary-treated effluent while metabolites make up the majority with carboxylic acid metabolites (*i.e.*, NP1EC and NP2EC) present in higher concentrations than NP1EO, NP2EO, or NP. Due to its lower water solubility (and greater lipophilicity) than the other metabolites, NP tends to be associated with the sludge. Therefore, the second major environmental release route for nonylphenolics in sewage treatment processes is adsorption onto sludge. Most Canadian municipal wastewater treatment plants employing secondary or tertiary treatment utilize the activated sludge process, an anaerobic digestion process, which results in NP being the dominant nonylphenolic substance adsorbed onto the sludge. Up to 95% of the nonylphenolic composition of digested sludge may be attributed to NP. Sludge is disposed of in three ways, by incineration, by landfilling, and by spreading onto agricultural soils.

Earlier methods of chemical analysis of NPEs were developed for the parent compounds, not their low molecular weight (and much more toxic) degradation products. These earlier methods have been almost entirely supplanted by more modern methods of gas and liquid chromatography with much greater specificity and sensitivity, for example in the ng/L or µg/kg range for liquid and solid samples, respectively.

NPEs can be biodegraded through a mechanism of stepwise loss of ethoxy groups to form lower ethoxylated congeners, carboxylated products and NP. The intermediate and final products of metabolism are more persistent than the parent NPEs, but these intermediates are expected to be ultimately biodegraded. In aquatic environments, primary biodegradation of NPEs is fast, but the resultant products such as NP1EO, NP2EO, NP1EC, NP2EC and NP are moderately persistent, especially under anaerobic conditions. Microbial acclimation to such chemicals is required for optimal degradation efficiencies. Photodegradation of such products also is expected to be important. Based on the limited data available, NP and the lower ethoxylates and carboxylates are persistent in groundwater. NP can be moderately persistent in sediments. NP appears to be persistent in landfills under anaerobic conditions, but it does not appear to be persistent in soil under aerobic conditions.

NP and NPEs are present at low concentrations in ambient air, water, soil, sediments and biota. There are limited data on the occurrence of NP and NPEs, and their degradation

products, in the Canadian environment. Additionally, there are very few data available for NP/NPEs in Canadian soils, including those that have had sludge additions. Nevertheless, in Canada, these chemicals have been found in freshwater, sediment, fish and beluga whale tissue, textile mill effluents, pulp and paper mill effluents, MWWTP influents, effluents and sludges, and soil to which municipal sludges had been applied. For example, in freshwater, NP concentrations ranged from non-detectable to 4.25 µg/L. The highest freshwater concentrations of NP were observed in areas in close proximity to municipal wastewater treatment plant discharges, textile mill discharges, pulp mill discharges, large population centres, and heavy industry. NP has been found in almost all Canadian municipal wastewater treatment plant-related samples for which it was determined. Concentrations of NP in municipal wastewater treatment plant effluents in Canada are typically in the range of non-detectable to 10 µg/L. The concentration and relative composition of the NPE metabolites in the final effluent is dependent on many factors especially the degree of effluent treatment.

There are a large number of studies reporting acute and chronic effects of NP in aquatic biota. In contrast there are fewer studies reporting the toxicity of APEs and only a few studies that included the alkylphenol polyethoxycarboxylates (APECs). Although the data in the literature are scattered among many species, different test methods and chemicals, there is a consistent pattern in the toxicity reported. NP and octylphenol (OP) have acute toxicities in fish ranging from 17 to 1400 µg/L, 20 to 3000 µg/L in invertebrates and 27 to 2500 µg/L in algae. Chronic toxicity values (NOEC) are as low as 6 µg/L in fish and 3.7 µg/L in invertebrates. An acute to chronic toxicity ratio of 4:1 was determined based on the available literature. Ecological effects of NP were observed in mesocosm experiments at 30 µg/L. There is an increase in the toxicity of both NPEs and OPEs with decreasing EO chain length. NPECs and OPECs are less toxic than the corresponding APEs and have acute toxicities similar to APEs with 6-9 EO units. Since AP, APEs, and APECs occur as complex mixtures in effluents, their combined impact on the environment was considered.

APs and APEs have been reported to cause a number of estrogenic responses in a variety of aquatic organisms. Experiments in several different *in vitro* systems have indicated similar relative potencies among such compounds, with OP being the most potent, but only 27,000 times less potent than estradiol. NP was found to be approximately 25% less potent than OP, and NP2EO and NP1EC were only slightly less potent than NP in inducing vitellogenin in trout hepatocytes in one study. Addition of EO units to NPEs reduced the potency such that NP9EO was an order of magnitude less potent. APEs bind to the estrogen receptor, resulting in the expression of several responses both *in vitro* and *in vivo*, including the induction of vitellogenin. The threshold for vitellogenin induction in fish is 10 µg/L for NP and 3 µg/L for OP. The threshold for expression of intersex (ova-testes) in medaka was <50 µg/L for NP. The induction of mRNA in rainbow trout was recently reported to be 1 µg/L for NP. The estrogenic responses appear to be at least additive and NP and NPEs should, therefore,

be considered as a group. APs and APEs also affect the growth of testes, alter normal steroid metabolism and disrupt smoltification in fish.

APs, APEs and APECs are found as complex mixtures in effluents and if considered together may exceed the threshold for estrogenic effects. A critical consideration is the relative estrogenic potency of the APs and APEs and validation of the assumption of additivity. Estrogenic responses occur at concentrations similar to chronic toxicity, although biochemical and histological changes have been reported at concentrations a factor of ten lower. The relative importance and significance of estrogenic responses to the individual or population is not currently well understood.

The available literature suggests that the bioaccumulation of NP and NPEs in aquatic biota in the environment is low to moderate. BCFs and BAFs in biota, including algae, plants, invertebrates and fish range from 0.9 to 3400. There are relatively little data available for NPEs, but based on their structure they are not expected to bioaccumulate significantly. Very few data are available for the bioconcentration and bioaccumulation of OP and OPEs, but based on their structural similarities to NP and NPEs, they are predicted to have BCFs/BAFs slightly lower than those of NP and NPEs.

The major route of exposure of NP and NPEs to the Canadian environment is through discharge of effluents. The composition of the mixture can differ considerably among the various effluents depending on the source and the degree and type of treatment. Textile mill effluents represent a major source of NPEs to the environment. Untreated or partially treated textile mill effluents can have high concentrations of NP9EO, NP1EO and NP2EO. There appears to be a recent decrease in discharge of NPEs from pulp and paper mills but there are very few data available to validate this conclusion. Municipal effluents are a significant source of NPEs and are widespread across Canada. Untreated effluents can have high levels of NP, NP1EO and NP2EO which may exceed thresholds for chronic effects in the aquatic environment. Treated effluents have relatively low levels of longer EO chain length NPEs. NP1EO and NP2EO levels can remain at levels that may result in potential chronic toxicity in final effluents.

The potential for estrogenic responses in effluents is apparent, especially if the effects of the individual metabolites are considered to be additive. When dilution is considered, only a few sites have concentrations of NP that are of concern. However, if the additivity of the of the mixture of metabolites is considered, then the effluents have the potential to cause estrogenic responses in as many as 40% of receiving water in Canada (assuming a 10:1 dilution). Great caution must be used however, because the relative estrogenic potency is currently under scientific debate and the effects might not be expressed *in vivo* for metabolites such as NP1EC. The significance of estrogenic responses is also not fully understood. Emphasis was, therefore,

placed on the chronic toxicity endpoints in this assessment.

The concentration of NP is low in the dissolved fraction of treated effluents because it sorbs to sludge particles, however, NP sorbed to sediments may represent an alternative route of exposure that may result in potential chronic toxicity in sediment dwelling organisms. Despite a relatively low potential to bioaccumulate (BAF approximately 10), sediment dwelling organisms may be exposed to NP either directly through contact with water or sediment and/or through ingestion of sediment or food.

There is potential for chronic toxicity to occur in aquatic biota due to exposure to NPEs and their metabolites in a variety of effluents. This can be associated with different metabolites of NPEs depending on the source, degree and type of treatment. It is important that all of the NPE metabolites, not only NP, be considered together to assess the potential for impacts in the environment. Although this is a relatively well studied group of compounds and there are data available in the Canadian environment, it is recommended that the considerable knowledge gaps be filled, especially for the low chain length NPEs and NPECs.

Under current use patterns, NPEs in Canada can result in environmental concentrations that exceed the levels of concern. The degree of exceedance is normally small, except in untreated effluents (especially textile mills). However, the significance of the potential effects, and the widespread use and occurrence in NP and NPEs in effluents suggest that caution should be used and this group (including other APEs) should be managed together to ensure that their concentrations do not increase further above levels of concern for the Canadian environment in the future.

1.0 INTRODUCTION

The *Canadian Environmental Protection Act* (CEPA) requires the federal Ministers of Environment and Health to prepare and publish a Priority Substances List that identifies substances, including chemicals, groups of chemicals, effluents and wastes that should be given priority to determine whether they are harmful to the environment or constitute a danger to human health. The Act also requires both Ministers to assess these substances and determine whether they are "toxic" as defined in Section 11 of the Act, which states:

> "...a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions
>
> a) having or that may have an immediate or long-term harmful effect on the environment;
> b) constituting or that may constitute a danger to the environment on which human life depends; or
> c) constituting or that may constitute a danger in Canada to human life or health."

Substances that are assessed as "toxic" under Section 11 may be placed on Schedule 1 of the Act, and considered for possible risk management measures such as regulations, guidelines, or codes of practice to control any aspect of their life cycle, from the research and development stage through manufacture, use, storage, transport and ultimate disposal.

Based on an initial screening of readily accessible information, the rationale for assessing nonylphenol (NP) and its ethoxylates (NPEs) provided by the Ministers' Expert Advisory Panel on the Second Priority Substances List (Ministers' Expert Advisory Panel, Environment Canada 1995) was as follows:

> *"NPEs are discharged into the environment primarily from textile and pulp and paper production facilities. They are also used in coal processing, latex paints, grease and lubricating oils, pesticides and industrial detergents. Acute adverse effects have been reported in invertebrates, fish, mammals and algae. There are also concerns that these substances may interfere with endocrine function. An assessment is required to determine exposure levels and the risk they may pose to the environment and human health in Canada."*

The scientific literature databases which were searched for information on nonylphenol and its ethoxylates for the period 1960 to 1998 are listed in Appendix B. The assessment included data from research commissioned specifically for use in this assessment. These data were obtained following December 1998. Additional literature was considered for the assessment as the authors became aware of its existence. A separate PSL assessment of textile mill effluents was initiated concurrently and is presently ongoing. The approaches recommended for the environmental risk assessment are outlined in a Guidance Manual (Environment Canada, 1997a).

The Environmental Risk Assessment was developed by members of the Nonylphenol and its Ethoxylates Environmental Resource Group established by Environment Canada. Members were selected on the basis of their expertise, notably in the areas of analytical chemistry, environmental monitoring, environmental chemistry and environmental toxicology. Members of the Environmental Resource Group were:

D.T. Bennie, National Water Research Institute, Environment Canada
P. Cureton, Commercial Chemicals Evaluation Branch, Environment Canada
H.-B. Lee, National Water Research Institute, Environment Canada
K. Lloyd, Commercial Chemicals Evaluation Branch, Environment Canada, and
R.J. Maguire, National Water Research Institute, Environment Canada
M.R. Servos, National Water Research Institute, Environment Canada.

The environmental assessment was led by P. Cureton. Environmental sections of the Supporting Document and Assessment Report also were reviewed by internal reviewers of Environment Canada, namely:

K. Adare, Guidelines and Standards Division, Environment Canada
N. Davidson, Commercial Chemicals Evaluation Branch, Environment Canada
D. Dubé, Commercial Chemicals Evaluation Branch, Environment Canada
B. Ernst, Environmental Protection Service, Environment Canada
J. Haskill, National Office of Pollution Prevention, Environment Canada
B. Mander, Chemical Control Division, Environment Canada
K. Potter, Guidelines and Standards Division, Environment Canada
D. Rawn, Commercial Chemicals Evaluation Branch, Environment Canada
L. Rutherford, Environmental Protection Service, Environment Canada
R. Sutcliffe, Commercial Chemicals Evaluation Branch, Environment Canada
N. Tremblay, Chemical Control Division, Environment Canada.

This Supporting Document underwent an external scientific peer review, which was performed by:

C. Metcalfe, Environmental and Resources Studies, Trent University
K. Solomon, Centre for Toxicology, University of Guelph
G. Van Der Kraak, Department of Environmental Biology, University of Guelph
D. Moore, Cadmus Group.

A review of the uses of NP/NPEs in Canada and elsewhere, the analytical methods for their determination, occurrence, fate, persistence and effects is presented in this Supporting Document. This information provides the basis for the Assessment Report. Additionally, data for

related chemicals such as octylphenol (OP) and octylphenol ethoxylates (OPEs) are reviewed in this Supporting Document. These compounds have similar use patterns, environmental dynamics and effects, although there are fewer data on their environmental fate and effects.

Supporting documentation for the human health related sections are available upon request from:

Environmental Health Directorate
Health Canada
Tunney's Pasture
Ottawa, Ontario
K1A 0L2.

Copies of this Supporting Document are available upon request from:

Commercial Chemicals Evaluation Branch
Environment Canada
14th Floor, Place Vincent Massey
351 St. Joseph Boulevard
Hull, Québec
K1A 0H3.

Copies of the Assessment Report are available from:

Inquiry Centre
Environment Canada
1st Floor, Place Vincent Massey.

2.0 SUMMARY OF INFORMATION CRITICAL TO THE ASSESSMENT

2.1 IDENTITY AND CHEMICAL AND PHYSICAL PROPERTIES

2.1.1 Nomenclature

NP is a chemical intermediate composed of a phenol ring attached to a lipophilic straight or, more usually, branched nonyl group. NPEs belong to the larger group of compounds known as APEs and have the following general formula $C_{15}H_{24}O$+$(CH_2CH_2O)_n$. The predominant positional isomer of monoalkylphenols is the *para* isomer, which usually comprises ≥ 90% of industrial formulations, while the *ortho* isomer comprises ≤ 10%. In the United States, the U.S. Environmental Protection Agency and the Chemical Manufacturers Association Alkylphenols and Ethoxylates Panel have agreed that the commercial product that best represents "nonylphenol" is a chemical substance comprised of branched C_9-alkylphenols with Chemical Abstracts Service Registry Number 84852-15-3 (later called "4-NP, branched") (Hellyer, 1991). There may also be small amounts of 2,4-dinonylphenol in commercial nonylphenol preparations. Chemical Abstracts Service Registry numbers for a variety of alkylphenols, ethoxylates and other derivatives are given in Talmage (1994). Considering the branching of the C_9-chain, there may be scores, if not hundreds, of individual nonylphenol ethoxylate isomers in an industrial nonylphenol ethoxylate formulation. Each NPE is conventionally described by its average ethoxylate (EO) chain length, which ranges between 1 and 100 for different formulations. Halogenated (on the ring) derivatives of APEs and APECs have been found in the effluents of some municipal wastewater treatment plants that employ chlorine for disinfection (Maguire, 1999).

Structures of NPEs and associated degradation products are shown in Figure 1.

2.1.2 Physical and Chemical Properties

Physical and chemical properties that affect the environmental persistence of NP and OP are shown in Table 1, and will be referred to in the appropriate sections that follow. It should be noted, for example, that the pK_a (negative logarithm of the acid dissociation constant) of NP is 10.7 (Romano, 1991) which indicates that in most natural waters virtually all NP is present in the undissociated form.

NPECs are likely to be substantially, if not almost completely, ionized at the pH values of many natural waters (e.g., the pK_a of unsubstituted phenoxyacetic acid has been estimated as 5.12; U.S. National Toxicology Program, 1998), and their log K_{ow} values are expected to be much

lower than those of the corresponding ethoxylates (e.g., the log K_{ow} value of unsubstituted phenoxyacetic acid has been estimated as 1.34; Syracuse Research Corp., 1998).

No information was identified on the physical and chemical properties of higher chain parent NPEs, probably because of the complex nature of such mixtures. Because of their uses as surfactants, it is obvious that the parent chemicals will be water-soluble.

Ahel and Giger (1993a) determined aqueous solubilities (at 20.5 °C) for NP and NPnEO (n = 1-5) to be in the range 3-9 mg/L, and for OP and OPnEO (n = 1-4) to be in the range 8-25 mg/L. They also determined solubilities as a function of temperature between 2 °C and 25 °C; over this temperature range there was only a modest variation in solubilities. Ahel and Giger (1993b) determined the following log K_{ow} values (K_{ow} is the *n*-octanol-water partition coefficient) by a high performance liquid chromatographic (HPLC) method: OP (4.12), NP (4.48), NP1EO (4.17), NP2EO (4.21) and NP3EO (4.20). Estimates of log K_{ow} values for higher chain EOs also were determined by two different methods, but were not in agreement. McLeese *et al.* (1981) determined a value of 4.2 for the log K_{ow} of NP by HPLC. There are no experimentally determined log K_{ow} values for carboxylic acid derivatives such as NP1EC and NP2EC in the literature.

Bidleman and Renberg (1985) estimated the vapour pressure of NP using a gas chromatographic method. Six major resolvable components were estimated to have vapour pressures in the range 0.06 - 0.17 Pa at 25 °C. The Henry's law constant and vapour pressure of NP and especially NPEs are low; therefore, partitioning to air is extremely limited.

2.1.3 Analytical Methods

Early methods of surfactant analysis, including nonionic surfactants such as the NPEs, have been reviewed by Talmage (1994). These include the cobalt thiocyanate active substances (CTAS) method, the bismuth iodide active substances (BIAS) method and the Wickbold modification of the BIAS method. In the CTAS method, a blue complex is formed between NPEs with EO chain lengths > 5 and the ammonium cobaltothiocyanate reagent. In the BIAS method, the Dragendorff reagent, which is a preparation containing barium tetraiodobismuthate, forms a precipitate forms with polyoxyethylene compounds by interaction with the oxygen atoms of the EO groups. The Wickbold modification of the BIAS method involves removal of particulates by filtration, gas stripping into ethyl acetate using a stream of nitrogen presaturated with ethyl acetate, removal of cationic surfactants with cation exchange resin and precipitation of nonionic surfactants by barium bismuth iodide reagent. The measurement of the bismuth content of the precipitate is performed by potentiometric titration with pyrolidine dithiocarbamate solution. These methods were developed for parent surfactants and not their low molecular weight degradation products. These older techniques generally have been replaced by more

modern methods using gas and liquid chromatography, which result in greater specificity and sensitivity.

Extraction techniques for APs, APEs, and APECs from water and sludge samples have gradually changed from solvent sublation (gas stripping), steam distillation and liquid-liquid extraction to solid phase extraction. Soxhlet extraction remains a viable technique for most laboratories for solid samples, although supercritical fluid extraction is a more efficient alternative.

Liquid chromatography (LC) is frequently employed in the analysis of APEs and their metabolites. LC using gradient elution with normal phase columns and equipped with a fluorescence detector is very useful in the determination of oligomer distribution in environmental samples. Isocratic elution using a reversed phase column can provide quick separation of the octyl and nonyl homologues. This method is particularly suitable for liquid chromatography followed by mass spectrometry for detection of compounds (LC/MS). High resolution gas chromatography (GC) is very useful in the analysis of more volatile compounds (e.g., NP and OP) as well as the methylated lower chain APECs. Mass spectrometric detection coupled with LC or GC has become widely used because it is both a versatile and powerful tool for the analysis of nonylphenolics due to its sensitivity and specificity. Mass spectrometry also may be used in the absence of chromatographic separation if characteristic quasimolecular ions are formed.

2.1.3.1 Sample collection and storage

Water samples are generally collected in brown glass bottles and preserved with formaldehyde (1%, v/v). Sample are stored at 4 °C in the dark until extraction and analysis is performed. It has been shown that 24-hour composite samples are more representative of sewage and industrial effluents than grab samples (Lee, 1999). Few stability data are available for the nonylphenolics. In one study, however, NP, NP1EO, and NP2EO levels in a formaldehyde-preserved, biologically treated municipal wastewater samples remained constant over a storage period of 12 days at 4 °C (Ahel and Giger, 1985a). Chloroform, formaldehyde, copper (II) and mercury (II) salts have been tested as preservatives of water samples for the determination of nonionic surfactants, including Triton X-100 (OP9.4EO) (Szymanski *et al.*, 1995). Indirect tensametric measurements (a polarographic technique) were used in the analysis of surfactants. A 1% formaldehyde concentration was found to be sufficient to maintain stable surfactant concentrations for at least 20 days where refrigeration at 4 °C alone was ineffective.

Sediment and sludge samples, collected in wide-mouth glass bottles, generally are stored frozen, however, they can be stored at room temperature following air- or freeze-drying.

2.1.3.2 Extraction techniques

2.1.3.2.1 Wastewater and natural waters

A standardized procedure based on the original work of Wickbold has been developed for the extraction of APEs in water, including unfiltered samples such as raw sewage and industrial effluent (Ahel and Giger, 1985b; Scarlett *et al.*, 1994; Ibrahim and Wheals, 1996a). Typically, one litre samples adjusted to a pH of 7 to 8 were covered by 60 to 100 mL of ethyl acetate in a sublation apparatus and nitrogen was purged through the liquids for 5 to 10 minutes. The APEs enriched in the gas-liquid phase boundary were carried by the gas stream into the ethyl acetate layer. This procedure was repeated one to three times with fresh solvent for maximal recovery. Additionally, steam distillation and liquid-liquid extraction have been used in the extraction of APEs from water, wastewater and sewage samples (Naylor *et al.*, 1992; Stephanou 1984a; Ahel and Giger, 1985a; Clark *et al.*, 1992) (Table 2). Sodium chloride addition has been shown to increase extraction efficiency and reduce emulsions.

Steam distillation extraction of hydrophobic, semi-volatile organics developed by Veith and Kiwus (1977) has been applied to NP and NPEs in water (Giger *et al.*, 1981). After pH adjustment to 7.0-7.5 and addition of 20 g of sodium chloride, 2 L samples were refluxed for 3 hours using cyclohexane (1-2 mL) to concentrate the distillates. While this technique produced near quantitative results for NP, NP1EO and NP2EO, the recovery for NP3EO was poor (15%) (Ahel and Giger, 1985a). This procedure is unlikely to be applicable to the extraction of the higher ethoxylates in water samples due to their lower volatility and higher aqueous solubility (Ahel and Giger, 1993a,b).

Because of the polarity of APEs with higher EO chains, solvent extraction, either continuous or non-continuous liquid-liquid extraction of an aqueous sample, is usually limited to the less polar components such as NP/OP and lower chain APEs and APECs. Extraction recoveries of nonylphenolics in effluent using dichloromethane (DCM) have been reported to range between 87-98% for NP and 79% to 100% for NP1EO, NP2EO and NP3EO (Stephanou and Giger, 1982; Wahlberg *et al.*, 1990). NP1EC, NP2EC and NP3EC were successfully extracted from effluents using DCM (Stephanou, 1985) or chloroform (Ahel *et al.*, 1987) after acidification of the samples to pH 2. Similarly, OPECs and halogenated OPECs present in sewage were extracted by diethyl ether after the sample was acidified to pH 1.5 (Ball *et al.*,

1989). The extraction of NP and NPnEO (n = 1-3) also was demonstrated by means of a continuous extraction apparatus using a solvent heavier than water, such as DCM.

Lee and Peart (1995) developed a procedure which involved simultaneous liquid-liquid extraction of NP and OP and *in situ* acetylation of the phenols with acetic anhydride in the presence of a base, followed by the extraction of the acetyl derivatives with petroleum ether. Quantitative recoveries of the non-polar derivatives of the phenols in sewage effluent were obtained.

Solid phase extraction (SPE) cartridges also have been used in extraction of APs/APEs (Table 2). The advantages of SPE include lower solvent consumption and automated operation. Using SPE, extraction of polar and ionic species from large sample volumes is possible. Additionally, emulsion problems do not occur during SPE extraction of sewage and wastewater samples. The possibility of plugging the adsorption medium exists, therefore, samples are generally filtered through a glass fibre filter of pore size 1.2 μm, or smaller, prior to extraction.

Amberlite XAD-2, -4, and -8 resins have been applied to the extraction of APEs in water resulting in recoveries >80%. Resins require prepurified by washing them with organic solvent, prior to use and can be used in the extraction of variable sample sizes ranging from 1 to 2000 L. Following sorption of APEs onto individual resins, elutions have been performed using acetone/water (9:1, v/v) or sequentially with diethyl ether and methanol (Table 2). Desorption of the ethoxylates could also be performed using Soxhlet extraction of the resin with methanol (Jones and Nickless, 1978a,b).

Additionally, granular activated carbon has been used for the extraction of large volume (*e.g.*, 2000 L) water samples. NPEs sorbed onto the carbon (Ventura *et al.*, 1988; 1989) were successfully extracted using Soxhlet with DCM. The recently developed graphitized carbon black (GCB, 120-400 mesh) cartridges, however, are more efficient because they provide the simultaneous preconcentration of several classes of ionic as well as nonionic surfactants including NP, NPEs and NPECs in water samples (Di Corcia *et al.*, 1994; Crescenzi *et al.*, 1995).

Octadecylsilane (C_{18}) (ODS) cartridges have been used for the preconcentration of NP, OP, and NPEs, as well as NPECs, in effluent and water samples (Marcomini *et al.*, 1987, 1989b; 1990; 1993; Kubeck and Naylor, 1990; Blackburn and Waldock, 1995; Lee *et al.*, 1998). Prior to use, ODS cartridges require preconditioning, prior to addition of filtered samples (ranging from 10 mL to 1 L). ODS cartridges also may be used in the extraction of APECs if sample pHs have been adjusted to 2.

Strong anion exchange (SAX) solid phase extraction disks (25 mm diameter) have been used for the extraction of NP1EC-NP4EC, in paper mill effluents, sewage effluents and river waters (Field and Reed, 1996).

Kubeck and Naylor (1990) described a dual-column procedure for the extraction of APEs (n = 1-17) in natural waters. The first column containing a mixed-bed ion-exchange resin (Biorad 501 x 8 (D), 20-50 mesh) was used to remove ionic species from the sample. APEs adsorbed on the second column (0.7 g of ODS) were removed by warm (55 °C) methanol.

Solid-phase microextraction (SPME) using fibres coated with Carbowax/template resin and Carbowax/divinylbenzene coatings has been used in the extraction of OP9.4EO and other APEs from water samples. Extraction of the APEs using other coated phases was unsuccessful (Boyd-Boland and Pawliszyn, 1996).

2.1.3.2.2 Sludge, sediment and solid samples

Relatively few methods are available for the extraction of AP, APEs and APECs in solid matrices. Samples are usually air- or freeze-dried and pulverized prior to extraction. Soxhlet and steam distillation have generally been used for extraction of APs, APEs and APECs from solid samples, however, these methods are most appropriate for the more volatile, less polar components such as APs and the lower chain APEs. More recently, supercritical fluid extraction (SFE) using modified carbon dioxide has been applied to sediment and sewage sludge samples. Greater extraction efficiency has been observed with SFE than older techniques. Additionally, SFE can be used in the extraction of a wider range of surfactants, including APs, APEs, APECS and linear alkylsulfonates (LAS) (Field *et al.*, 1992; Lee and Peart, 1995; Lee *et al.*, 1997).

Soxhlet extraction of NP, NP1EO and NP2EO from solid samples such as river and lake sediments, sewage sludge, and detergent powder, has been successfully performed using a variety of individual solvents (Marcomini *et al.*, 1988c; 1990; 1991; Marcomini and Giger, 1987; Jungclaus *et al.*, 1978; Lee *et al.*, 1997) and solvent mixtures (Valls *et al.*, 1988; Lee *et al.*, 1997). More polar solvents are necessary for extraction of the higher chain APEs relative to the lower chain APEs and APs. Much greater extraction efficiencies of LAS, NP and NP1EO from dated sludge using Soxhlet extraction with methanol were observed if sodium hydroxide (20%, w/w) was added to dry sludge samples (Marcomini *et al.*, 1991). Additionally, steam distillation has been used in the extraction of NP, NP1EO and NP2EO from solid samples (Giger *et al.*, 1981, 1984; Ahel and Giger, 1985a; Ahel *et al.*, 1994b;c).

Although SFE is a relatively recent technique, its use has been reported in the extraction of NP, NPEs and NPECs from sewage sludge and sediment. Pure carbon dioxide has been used to extract APEs from homogenized air- or freeze-dried sediment or sewage sludge (1 g) using elevated temperature (80 °C) and pressure (*ca.* 345 atm). Extraction of NP and OP was performed using combined static and dynamic extractions, followed by a conversion to their acetyl derivatives *in situ* in the presence of acetic anhydride and triethylamine (Lee and Peart, 1995). Lee and Peart (1995) reported that addition of small volumes of water (2 mL) to samples prior to extraction increased extraction efficiency particularly for the higher chain NPEs. Extraction times are relatively short and (10 min. static and 15 min. dynamic) using the elevated temperature and pressure. NPEs sorbed onto ODS sorbent traps were eluted with methanol at 60 °C (Lee *et al.*, 1997). The lower chain carboxylates (NP1EC and NP2EC), were extracted and eluted under similar conditions, but were methylated off-line for later GC/MS analysis.

Recovery studies of NP, OPEs (Triton X-100), and NPEs (with an average of 13 ethoxy units) in spiked sediment samples using other SFE methods have been described in the literature. Addition of methanol prior to a 4-step, 45-min SFE (100 °C, 150 and 450 atm) with CO_2 resulted in recoveries of 85% for both NP and NPEs and 65% for OPEs. Recovery efficiencies were substantially reduced following a 4 month aging period (Kreisselmeier and Dürbeck, 1997).

2.1.3.3 Chromatographic separation and detection methods

Liquid chromatography (LC) is a versatile analytical technique suitable for compounds with a wide range of polarity, volatility, and molecular weights such as APEs. Some coelution problems do exist and lower resolution is attainable than if gas chromatography (GC) were employed. LC has been applied to the analysis of APs and APECs without derivatization. Recently reviews of LC used in the analysis of nonionic aliphatic surfactants and nonionic surfactants with EO chains have been published in the literature (Miszkiewicz and Szymanowski, 1996; Marcomini and Zanette, 1996). Because APE oligomers differ from each other by the length of the EO chain, they are best separated using normal phase LC where separation results from the different interactions (partition and/or adsorption) between EO chain and the polar stationary phase. The order of elution in a normal phase separation is relative to the number of ethoxy units. Retention times of a given oligomer increases with the length of its EO chain. A gradient elution is required for the separation of a multicomponent mixture because the polarity of APE oligomers vary widely. Isocratic elution is limited to the analysis of a few oligomers of similar polarity. The most popular packing materials used in normal phase LC separation of APE mixtures include, silica with particle size ranging from 3-5 μm modified by chemically bonded amino groups (Ahel and Giger, 1985b; Marcomini and

Giger, 1987; Ahel *et al.*, 1994b,c, 1996) and cyano groups (Pilc and Sermon, 1987; Kubeck and Naylor, 1990; Scarlett *et al.*, 1994). Jandera *et al.* (1990), however, observed that the amino-bonded phase offered better separation for the individual oligomers than the diol (Zhou *et al.*, 1990) and cyano bonded phases. A *p*-nitrophenyl-bonded silica phase also has been used in association with a ternary elution gradient (*n*-heptane/DCM/methanol) for the resolution of long chain NPEs such as Igepal CO 720, CO 890, and CO 990 (Desbène and Desmazieres, 1994). The separation of NPnEO (n = 1-17) oligomers in a standard and a sludge extract on a Hypersil APS column is shown in Figure 2 and Figure 3, respectively.

Separation of APEs with bare silica also has been reported. Anghel *et al.* (1994) reported baseline separation of APE oligomers with an average of 40 EO units using a silica column (Si-100, 5 μm). These authors also reported that the presence of a bulky alkyl group in the *ortho* position hindered the interaction between the hydroxyl group of silica and the EO chain of the surfactant. Consequently, for each oligomer, the *ortho*-isomer eluted prior of the *para*-isomer. In another study, a combination of a reversed phase precolumn (Hypersil ODS, 7.5 mm length) and a 3 μm silica column (150 mm length) was used for the separation of NPnEO (n = 3-50) in Tergitol surfactants. This LC method utilized a gradient elution with acetonitrile and water, which is usually used in reverse phase separations. This technique allowed for direct injections of aqueous samples rather than extracting into a solvent prior to LC separation (Kibbey *et al.*, 1996). Oligomers in Triton X-100 and Synperonic NP10/NP20 also have been separated on Spherisorb silica with eluents containing 20-50% acetonitrile in aqueous pH 3 phosphate buffer (Ibrahim and Wheals, 1996b).

Depending on the column and the mobile phase used, NP is either eluted as the first peak (Kubeck and Naylor, 1990), between NP1EO and NP2EO, or between NP2EO and NP3EO (Ahel and Giger, 1985a). OP coelutes with NP on aminosilica columns, and, at each level of ethoxylation, OPEs and NPEs either coelute, or they have very similar retention times. Only one example of normal phase LC (with a Lichrosorb-NH_2 column) separation of NPEC has been reported in the literature (Ahel *et al.*, 1987).

All NPE oligomers have the same hydrophobic moiety and, therefore, in reverse-phase LC using non-polar C_{18} and C_8 columns, they elute as a single peak. OPEs are more weakly hydrophobic than NPEs which lead to earlier elution using reverse phases columns. They also elute as a single peak. Although the C_1 (trimethylsilyl) column is classified as a reversed phase column, it is appreciably hydrophilic and behaves like a normal phase column (Wang and Fingas, 1993b). Separation of individual oligomers of OPEs with up to 40 EO units in Triton mixtures was possible with a C_1 column under isocratic conditions using a mixture of methanol and water as the mobile phase. Separation of other APEs and linear alkylbenzene sulfonates using a C_1 column has been reported in river water samples (Scullion *et al.*, 1996).

Coelution problems have been reported using a RP-C_8 column for OP and OPEs and NP and NPEs (Ahel and Giger, 1985a). Di Corcia *et al.*, 1994, however, were able to separate NP and NPEs using a C_8 column and resolve NPnEC (n = 1 to >3) peaks in a sewage effluent extract using this column (Di Corcia *et al.*, 1994).

The phenyl group in APEs is a chromophore, therefore, they can be readily detected by an ultraviolet (UV) or photodiode array (DAD) detector. For optimum selectivity, the wavelength of the detector is usually set at about 277 nm. At one time the UV molar absorptivity was assumed to be the same for all oligomers; however, it was later found that the absorptivity decreased with increasing length of the ethoxy chain (Ahel and Giger, 1985a; Zhou *et al.*, 1990). A more sensitive and selective method for the analysis of APEs can be provided by a fluorescence detector. The maximum excitation and emission spectra occur at *ca.* 230 and 300 nm, respectively for both OPEs and NPEs. Therefore, these wavelengths have been commonly used for the analysis of the APEs (*e.g.*, Kudoh *et al.*, 1984; Holt *et al.*, 1986). These wavelengths also are used in the UV and fluorescence detection of OP, NP and their carboxylates. Although chemical derivatization is rarely needed for the LC analysis of APEs, acetylation of APEs followed by fluorescence detection of the derivatives has been reported (Mackay *et al.*, 1997).

NPEs and OPEs and their carboxylates have been analyzed in drinking water samples using continuous liquid/liquid extraction followed by particle beam liquid chromatography using a reverse phase column with gradient elution (methanol and 0.01% ammonium acetate) followed by mass spectrometric determination (PB/LC/MS) (Clark *et al.*, 1992). Since PB/LC/MS was performed under electron impact conditions, the molecular ions of the ethoxylates were very weak. Although many fragmentation ions were observed, the $[M\text{-}C_6H_{13}]^+$ and $[M\text{-}C_5H_{11}]^+$ were the most intense for NPEs and OPEs and, therefore, were used as the characteristic ions. The $[M\text{-}C_5H_{11}]^+$ also was used to detect the presence of NPECs and OPECs. By using 500 L samples and concentration factors of 1,000,000, APnEO (n = 3-8) and APnEC (n = 2-7) have been reported in the ng/L levels in finished drinking water using this technique.

The atmospheric pressure ionization electrospray (API-ES) mass spectrometer has become very popular recently because of its wide application to large and small, polar and nonpolar, as well as singly and multiply charged species including APEs. It is classified as a true soft ionization technique because it results in minimal fragmentation of the molecular ion. In the API-ES process, ions are formed by ejection from shrinking charged droplets from the liquid phase. API-ES mass spectrometry has been used to identify the photocatalytic degradation products of nonionic surfactants such as the primary and secondary alcohol ethoxylates and NPEs used in wool scouring (Sherrard *et al.*, 1994). Surfactants that were diluted in a solution of water, methanol and acetic acid, were injected directly into the

spectrometer with nitrogen as both the drying gas and the nebulizer. The spectrum of NPEs showed a series of peaks corresponding to the $[M+H]^+$ ions (*e.g.*, *m/z* 397 for $[NP4EO+H]^+$). Each main peak was separated by 44 mass units, corresponding to individual EO units.

Methods also have been developed for the determination of nonionic surfactants in environmental waters and sewage effluents using ES/LC/MS (Crescenzi *et al.*, 1995; Mackay *et al.*, 1997). In these cases, major peaks of the Na^+ adduct, and to a much smaller extent, the H^+, K^+, and NH_4^+ adducts of the molecular ions, were observed. The base peak for NP9EO (molecular weight 616) using this technique was *m/z* 639 or $[NP9EO+Na]^+$. Despite all oligomers present as a single chromatographic peak, quantitative information on the oligomeric distribution in an environmental sample could readily be obtained by extracting chromatograms of selected ions (corresponding to each Na^+ adduct) from the total ion chromatogram (TIC) (Crescenzi *et al.*, 1995).

Atmospheric pressure chemical ionization mass spectrometry (APCI) is another soft ionization technique where ions are formed in the gaseous phase instead of the liquid phase as in the case of ES/MS used in APE analysis. Pattanaargson *et al.* (1995) have used APCI/MS to determine the oligomer distribution of Tergitol NP-4, NP-6, NP-10, NP-13, and NP-40. The surfactants which were prepared in an acetonitrile-water mixtures, were directly introduced into the spectrometer with no chromatographic separation. A series of protonated molecular ions $[MH]^+$ of NPEs (corresponding to *m/z* 221 + 44n where n is the degree of ethoxylation) were observed in the mass spectrum. A preliminary investigation by Scullion *et al.* (1996) indicated that the mobile phase used with a C_1 column was compatible with the APCI LC/MS analysis of OPEs. Only positive ions arising from OPEs were observed in that study and the dominant ones were $[M+Na]^+$ (*e.g.*, $[OP8EO+Na]^+$ at *m/z* 581) and $[M+2Na]^+$ (*e.g.*, $[OP8EO+2Na]^+$ at *m/z* 604).

Gas chromatography (GC) also has been used in the analysis of parent and derivatized APEs, NP and OP as well as derivatized APECs. The characteristic low volatility of the higher chain APEs and APECs, however, limit the use of this technique to the analysis of oligomers with six or less EO units. The majority of GC separation analysis of APs and APEs have been performed using capillary columns with low polarity stationary phases, such as OV-1 (Giger *et al.*, 1981), DB-5 (Stephanou, 1984a), and SE-54 (Ahel *et al.*, 1987), although packed columns have been used. Separation of NP isomers with differences on the alkyl side chain is possible using high resolution-GC which employs the use of capillary columns. Similarly, at each level of ethoxylation, a group of peaks are observed for the free or derivatized NPEs and derivatized NPECs. Only one peak is observed for OP since it is a single isomer, *i.e.*, 4-(1,1,3,3-tetramethylbutyl)phenol.

Gas chromatographic separation of NP and Marlophen 83 (NPnEO with an average n of 3.15) components was possible using a 15 m OV-73 column using flame ionization detection (FID) (Stephanou and Giger, 1982). Five series of peaks, representative of NP and NPnEO (n = 1-4) isomers were observed with an elution profile based on increasing molecular weight. Separation and characterization of a technical mixture of NP has been performed using a cross-linked methyl silicone capillary column (PONA) (Bhatt *et al.*, 1992). Tentative structures of 14 isomers of NP were assigned using GC/MS, GC with a fourier transform infrared detector (FTIR), and ^{13}C nuclear magnetic resonance (NMR) results. Isomers of 2-NP, octadecene, OP, and dinonylphenol also were identified in this mixture. Chee *et al.* (1996) also have described the optimization of GC analysis of NP using an orthogonal array design and electronic pressure programming using a 25 m HP-1 column.

APEs and their metabolites: AP and APECs, do not possess electron-capturing groups and, therefore, attachment of a halogen-containing group by chemical derivatization is required for their analysis with an electron capture detector (ECD). Pentafluorobenzoyl (PFB) ether derivatives of NP have been analyzed using electron capture detection following separation with a 30 m DB-5 column (Chalaux *et al.*, 1994). Nine major peaks were observed and the method detection limit was estimated to be 20 pg using this technique. Heptafluorobutyryl and PFB derivatives also have been used for the determination of NP and NPnEO (n = 1-6) in water, sludge, and biota samples. PFB derivatives were found to have better responses by electron capture detection than the heptafluorobutyryl derivatives although the retention times were longer (Wahlberg *et al.*, 1990).

Gas chromatography - electron impact mass spectrometry (GC-EI/MS) has been most widely used in the analysis of free or derivatized AP, APEs, and APECs. Mass spectra of the different isomers for the free NP were first reported by Giger *et al.* (1981) and were later described in detail by Bhatt *et al.* (1992) and Wheeler *et al.* (1997). Major fragmentation ions were observed at *m/z* 107, 121, 135, 149, 163, and 177 due to the loss of fragments of the alkyl chain. Additionally, it was observed that most of the major NP isomers have at least one alkyl substituent (*e.g.*, methyl, ethyl, dimethyl, *etc.*) on the α- carbon of the side chain and the NP molecular ion (*m/z* 220) was weak for all isomers.

Fragmentation patterns were similar in mass spectra of acetylated NP, although the M^+ was very weak (*m/z* 262) (Lee and Peart, 1995). With a base peak at *m/z* 135 and M^+ at *m/z* 248, the mass spectrum of the acetylated OP was consistent with its α,α-dimethyl structure (Lee and Peart, 1995). TICs of an acetylated NP/OP standard relative to the extract of a primary sewage effluent are shown in Figure 4 and Figure 5, respectively.

A weak M^+ ion of the PFB derivative was observed in the mass spectrum of NP. The two major fragments of the PFB derivative were observed at *m/z* 315 and 181 (Chalaux *et al.*, 1994).

Determination of NPEs using GC-EI/MS has been reported, this technique has only been applied to the analysis of the more volatile oligomers, *i.e.*, ethoxylates with four or less EO units (Giger *et al.*, 1981; Stephanou and Giger, 1982). The major fragmentation ions observed were *m/z* 179, 193, and 235 for NP1EO and *m/z* 223, 237 and 279 for NP2EO. The M^+ for both NP1,2EO (*m/z* 264 and 308) were weak. Due to the presence of longer chain oligomers in environmental samples, GC/MS is not suitable for the analysis of total APEs. Halogenated and non-halogenated OPE residues, however, have been identified using EI GC/MS (Stephanou *et al.*, 1988). The base peaks of the mass spectra for OPnEO (n = 1-3) were $[M-71]^+$ or $[M-C_5H_{11}]^+$ fragments. To enhance chromatography, APECs are usually derivatized to their methyl esters prior to analysis (Figure 6). Different isomers of the nonyl group, result in a group of peaks which represent the methyl esters of NP1EC and NP2EC in the chromatogram of a sewage effluent extract (Figure 7).

Although the volatility of APECs in environmental samples is a limitation of GC/MS analysis, it is not considered as serious as observed in the analysis of APEs because the major APECs generally have only a few EO units. The molecular ions of the methyl esters of AP1EC and AP2EC are weak and thus are not suitable for selected ion monitoring work using EI. Although detailed descriptions of the mass spectra of these derivatives have been reported by Ahel *et al.* (1987) and Stephanou *et al.* (1988).

Positive ion chemical ionization (PCI) has been applied in the analysis of OPnEO (n = 1-5) and methyl ester derivatives of NPnECs (n = 1-4) (Stephanou, 1984a,b; Stephanou *et al.*, 1988) in surfactants and effluent samples. Use of methane as the reagent gas in the analysis of the OPEs, resulted in ion rich spectra. The characteristic ions of varying intensities, were derived from the proton and ethyl adducts ($[M+H]^+$, $[M+C_2H_5]^+$) of the substrate. However, when ammonia was used as the reagent gas, the major OPE ions were for the $[M+NH_4]^+$ species and represented the base peaks (Stephanou, 1984b). Although PCI GC/MS using methane has been documented in the analysis of methyl esters of both halogenated and non-halogenated OPnECs (n = 1-3) (Stephanou *et al.*, 1988), ammonia resulted in intense $[M+NH_4]^+$ adducts for the NPnEC (n = 1-4) derivatives with little or no secondary fragmentation (Field and Reed, 1996). Therefore, multiple ion monitoring at *m/z* 310, 354, 398, and 442 has been successfully applied to the selective detection of NP1EC, NP2EC, NP3EC, and NP4EC, respectively, in paper mill and sewage effluents as well as river waters (Field and Reed, 1996).

There are only a few reports of the analysis of alkylphenolics by negative ion chemical ionization (NCI) GC/MS. The PFB derivatives of NP and NPEs have been demonstrated to produce intense molecular ions under NCI conditions using methane as reagent gas (Wahlberg *et al.*, 1990). Therefore, ions at *m/z* 414, 458, 502, and 546 were used for the selective detection of NP, NP1EO, NP2EO, and NP3EO, respectively, in sewage effluents. The detection limit of NP (2 pg absolute) as PFB ether derivatives by methane-NCI was reported to be five times lower than that by the EI/GC/MS procedure (Chalaux *et al.*, 1994). The higher sensitivity under NCI conditions was attributed to the absence of further fragmentation for the characteristic ion $[M\text{-}PFB]^-$ (*m/z* 219) used for quantitative work.

Field desorption mass spectrometry (FDMS) was used in the confirmation of OPEs and NPEs in wastewater extracts in some of the older literature (Otsuki and Shiraishi, 1979; Yasuhara *et al.*, 1981; Crathorne *et al.*, 1984). Major ions corresponding to the molecular masses of all APE oligomers were observed in one case (Otsuki and Shiraishi, 1979), although quasimolecular ions such as $[M+Na]^+$, $[M+K]^+$, and $[M+H]^+$ were more commonly found in FD mass spectra (Yasuhara *et al.*, 1981; Crathorne *et al.*, 1984). The latter phenomenon often occurred in surface water extracts where there were sufficient amounts of coextracted alkali salts to form these ions.

Fast atom bombardment (FAB) mass spectrometry and FAB-MS/MS have been used in the identification of NPEs and NPECs in raw and drinking water extracts (Rivera *et al.*, 1987; Ventura *et al.*, 1988; 1989; 1991; 1992). Thioglycerol saturated with NaCl was used as a matrix for FABMS of the surfactants. Methyl ester derivatives of NPE and NPEC oligomers can readily be distinguished in FAB mass spectra by their $[M+Na]^+$ ions, which are separated by 44 mass units. Thus, presence of NP3EC and NP4EC in a sample is evidenced by the observation of ions at *m/z* 447 and 491, respectively (Ventura *et al.*, 1991). The formation of $[M+Na]^+$ ions instead of $[M+H]^+$ ions is enhanced by the addition of salt to the matrix. The intensity of the quasimolecular ions decrease with molecular weight, therefore, this technique is not suitable for quantitative analysis unless isotopically labeled internal standards are used. Field desorption MS and desorption chemical ionization MS methods generate abundant quasimolecular ions $[M+H]^+$, therefore, they are well suited for molecular weight determinations of APEs.

The use of supercritical fluid chromatography (SFC) for the quantitative analysis of nonionic surfactants has been investigated (Wang and Fingas, 1993a,c). Separation of Triton (OPEs) and Igepal (NPEs) mixtures was achieved by a 5 m x 50 μm i.d. SB-Phenyl-30 (30% biphenyl, 70% methylpolysiloxane) capillary column followed by flame ionization detection. A linear pressure program of the mobile phase, supercritical carbon dioxide, from 2000 to 5500 psi, and column oven temperatures of 100, 150, and 180 °C were used. The retention times of APE oligomers were a function of their polarity and molecular masses. The less polar, shorter

chain oligomers were eluted first by carbon dioxide of lower solvating power (pressure and density). Under optimized conditions, APE oligomers of up to 25 ethoxy units were satisfactorily separated. While SFC and C_1 reversed phase HPLC produced similar quantitative results for the oligomeric distribution of Triton and Igepal mixtures, SFC offered a better resolution of the oligomers. Since SFC is not yet widely used, there has been no reported method for its application in environmental analysis involving APEs (Lee, 1999).

Capillary electrophoretic (CE) separation of APEs was reported by Heinig *et al.* (1996). The addition of sodium dodecyl sulfate (SDS) is necessary for the analysis of nonionic surfactants using this technique, to provide a charged pseudophase. Additionally, high contents of an organic modifier (≥ 20% acetonitrile) are needed. Separation was achieved by using a 50 cm x 75 µm i.d. fused-silica capillary column followed by UV detection at 200 or 214 nm. The migration order for APEs in CE is the reverse of the elution order observed in HPLC, *i.e.*, NPEs and OPEs are detected in order of decreasing number of EO units. In another study by He and Lee (1996), the addition of cyclodextrin to the SDS buffer was shown to improve the separation of NP isomers by micellar electrokinetic chromatography. While CE provides a faster and higher resolution separation than LC for many ionic and nonionic surfactants, this emerging technique has not yet been utilized in environmental analysis of APEs.

2.2 ENTRY CHARACTERIZATION

2.2.1 Natural sources of nonylphenol and its ethoxylates

There are no known natural sources of AP and APEs. Their presence in the environment, therefore is solely a consequence of anthropogenic activity.

2.2.2 Uses, production and market trends - general

Earlier reviews of the production and use of nonylphenol and its ethoxylates are provided by Reed (1978), Cahn and Lynn (1983), Beak Consultants Ltd. (1987), the Proceedings of the Seminar on Nonylphenol Ethoxylates and Nonylphenol (1991), Holt *et al.* (1992), U.K. Department of the Environment (1993), Aboul-Kassim and Simoneit (1993), Talmage (1994), Balson and Felix (1995), Nimrod and Benson (1996a), Metcalfe *et al.* (1996), Dickey (1997), U.K. Environment Agency (1997; 1998) and World Wildlife Fund Canada (1997).

APs of commercial importance are manufactured almost exclusively by a catalyzed reaction of olefins with phenols, cresols or xylenols (Reed, 1978). The olefins used all are readily available from petrochemical operations. NP is manufactured industrially by alkylating phenol with isomeric nonenes (propylene trimer) in the presence of an acid catalyst. The product, consisting largely of a mixture of 4-substituted alkylphenols with various isomeric, branched-chain nonyl groups, is recovered by fractional distillation under reduced pressure. It is a viscous liquid possessing a slight phenolic odour. At low temperatures, NP sets to a clear glass-like solid without crystal formation (Reed, 1978). OP is made by alkylating phenol with diisobutylene followed by vacuum distillation.

By far the most important industrial reaction of NP is etherification, whereby condensation with ethylene oxide using a basic catalyst yields NPEs. Under variable reaction conditions such as differences in reaction time or ratio of NP to ethylene oxide, ethoxylation of NP or other APs results in the formation of a series of oligomers with from one to up to 100 EO units. APE products usually consist of a mixture of these oligomers. Their relative abundance follows a Poisson distribution and the product is characterized by the average number of EO units. Low condensates with 4-5 ethylene oxide units per molecule of nonylphenol are used as oil-soluble detergents and emulsifiers and can be sulfonated or phosphorylated to give anionic detergents, lubricants and antistatic agents. Although the 8 and 9 ethoxylated compounds form the basis of high performance detergents, especially for textile scouring, in some countries they have been replaced by straight-chain C_{12}-C_{14} alcohol ethoxylates in some household detergents because of concerns about their persistence. APEs

with 13 to 15 EO units, in conjunction with an oil-soluble anionic surfactant, are excellent emulsifiers for a wide range of solvents and agricultural pesticides (Reed, 1978). APEs have been used in cleaning products and industrial processing for more than 40 years (Talmage, 1994). Ethylene oxide (another PSL-2 chemical) may be present in NPE formulations at concentrations below 10 mg/L (Talmage, 1994). Table 3 lists a few examples of some commercial preparations of APEs and their approximate compositions.

The production of *tris*-4-nonylphenyl phosphite (TNPP) is used as an antioxidant for rubber and the manufacture of lube oil additives (Reed, 1978). NP has been found in extracts of food grade poly(vinyl chloride) (PVC). Its presence is thought to be derived from the use of TNPP as a pre-stabilizer during polymer drying (Gilbert *et al.*, 1982). Junk *et al.* (1974) detected NP in PVC tubing used by the U.S. Food and Drug Administration-U.S. Department of Agriculture and PVC tubing prepared for use in processed milk storage. Other significant uses of NP in the European Union include: the production of phenol/formaldehyde resins, the production of TNPP antioxidant as a catalyst in the curing of epoxy resins and in the manufacture of phenolic oximes which are used in the extraction of copper from ores (U.K. Environment Agency, 1998).

Major uses of NPEs in the European Union include industrial and institutional cleaning; as textile auxiliaries in the textile industry (*e.g.*, in scouring, fibre lubrication and dye leveling); as leather auxiliaries (wet degreasing of hides); in pesticide formulations as wetting agents; as emulsion polymers (in specialist coatings, adhesives and fibre bonding); in paints (preparation of paint resin and as a paint mixture stabilizer); in the pulp and paper industry in the wetting of pulp fibres; and in metal cleaning and cutting.

Although it is not known if NP has been used for marking fuel oil for taxation purposes in Canada, it has been used for this purpose in the United States (Reed, 1978). It is apparently still used for this purpose in some countries (Müller *et al.*, 1998), but amounts used are unknown.

OP can be condensed with formaldehyde to give oil-soluble phenol resins, which are used in the manufacture of surface coating compositions, brake and clutch linings and in the production of special printing inks. OP also is used for the manufacture of nonionic surfactants by condensation with ethylene oxide using a basic catalyst. In rubber, OP is a useful antiflex cracking agent, fungistat and plasticizer. 4-Octylphenol sulfide is used in vulcanizing synthetic rubbers (Reed, 1978).

NP1EO and NP2EO are minor constituents of NPE surfactants found in a variety of cleaning products, lubricants, defoaming agents, dyeing assists, emulsifiers, *etc.* Marcomini *et al.* (1988a) found that parent NPEs contributed between 3.3% - 22.8% to commercial liquid

cleaning products. Higher chain NPEs ranged from below detection to 6.0% in laundry detergent samples and comprised between 7.0% - 25.1% of surface cleaners. Marcomini *et al.* (1988a) also detected OPEs in some cleaning products.

Although APEs are used as "inert" adjuvants in pesticide formulations, NPEs with an average of 5 EO units were found to be effective against apple powdery mildew when sprayed at 3.5% in the United Kingdom (Bent *et al.*, 1977). NPE use as "inerts" in insecticidal formulations was recently reviewed by Narayanan *et al.* (1997).

NPEs also are used as spermicides in contraceptive foams, jellies and creams. Although this use is small, it is important because of direct human contact. For example, Nonoxynol-9 (NP9EO) is a nonionic surfactant commonly used in commercial vaginal spermicidal formulations (Abrutyn *et al.*, 1982).

Detailed historical information on NPE (and other APE) production and demand is not available. However it is known that 2.18×10^6 tonnes of all types of surfactants were produced in the United States in 1980 (68% anionic, 25% nonionic, 6% cationic and 0.5% amphoteric) (Cahn and Lynn, 1983). 1.42×10^5 tonnes of APEs were produced, which represented 26% of all the nonionic surfactants. NPEs accounted for 73.6% production of APEs in 1980 (Cahn and Lynn, 1983).

Total consumption of APEs in the Federal Republic of Germany was about 18,500 tonnes in 1984, and about 4,900 tonnes in 1990 (Poremski, 1991). Concerns about the persistence and toxicity of degradation products of NPEs led to voluntary elimination of NPE surfactants from many uses in Europe and agreements by the Oslo and Paris Commissions (OSPAR) that NPE would be phased out of domestic cleaning products by 1995 and out of industrial cleaning products by 2000 (PARCOM, 1992). In 1997, 73,500 tonnes of NP were produced in the European Union, 3,500 tonnes were exported and 8,500 tonnes were imported. Of the total of 78,500 tonnes used in the European Union in 1997, 47,000 tonnes were used in the production of NPEs, 29,000 tonnes were used in the production of resins, plastics, stabilizers, *etc.*, and 2,500 tonnes were used in the production of phenolic oximes (UK Environment Agency, 1998). The total amount of NP used in the European Union were identical in 1994 and 1997.

NP demand was estimated to be 1.03×10^5 tonnes, 1.04×10^5 tonnes and 1.12×10^5 tonnes in the United States for 1997, 1998 and 2002, respectively (Anonymous, 1998). Historical growth in demand for NP in the United States was 2% per year in the period 1988-1997, and was forecast at 1-2% per year from 1998 through 2002 (Anonymous, 1998).

2.2.3 Uses, production and market trends in Canada

Over 4500 companies operating in Canada were surveyed in 1997 under authority of Section 16 of the *Canadian Environmental Protection Act* to determine the uses and releases of priority chemicals to the environment. Data were collected on the amount of NP and NPEs produced, imported, exported, shipped, acquired, used and released in Canada.

A total of 190 companies responded that they were involved with NPEs above the trigger quantity of 1 tonne per year. The amounts available for use (amount produced plus amount imported less the amount exported) were 21,500 and 17,200 tonnes in 1995 and 1996, respectively (Table 4). The survey did not distinguish between NP and NPEs, therefore, these data are taken to be the total of NP plus NPEs.

NPEs were manufactured by three companies in Canada in 1995 and 1996 and imported by 27 and 29 companies in 1995 and 1996, respectively. NPEs were used in descending order as feedstock, formulation, articles, chemical aid, manufacturing aid and containers based on the 1997 survey[1].

---

[1] Glossary of terms:

Article - incorporated into a consumer product or 'manufactured article'.

Chemical Aid - a substance that is added to a reaction mixtures to aid in the manufacture, synthesis or purification of a chemical or process stream (*e.g.*, process solvents, catalysts, inhibitors, buffers, flocculation agent, *etc.*).

Container - Manufacture of bottles, pails and other containers.

Feedstock - used as feedstock or chemical intermediate and becomes chemically transformed into another chemical.

Formulation - incorporated into a formulated product or packaged as a product, other than a consumer product or manufactured article for re-sale.

Manufacturing Aid - a substance that aids manufacturing process (*e.g.*, lubricants, metalworking fluids, coolants, hydraulic fluids, degreasers)

Manufactured Article - a consumer product or an article for which its final use depends in whole or in part on the physical shape or design of the article. For instance, vinyl film or tubing containing a listed substance would be considered a manufactured article whereas plastic granules which are intended for extrusion would not. Except for consumer products for the retail market, fluid formulations would not be considered to be manufactured articles. Although companies were required to report the quantities of substances they consumed making manufactured articles, they were not required to report actual quantities of the actual 'manufactured articles'.

Source for glossary terms: Environment Canada (1997b).

NPE-containing products are used in many sectors, including textile processing, pulp and paper processing, paints, resins and protective coatings, oil and gas recovery, steel manufacturing, pest control products and power generation. A variety of cleaning products, degreasers and detergents also are available for institutional and domestic use. These products have numerous applications, including controlling deposits on machinery, cleaning equipment, scouring fibres, wetting and de-wetting agents, in dyeing, and in machine felt cleaning and conditioning and in product finishing.

Estimates of domestic production of NP in 1989 was 3700 tonnes, while 1800 tonnes were imported (Camford Information Services, 1990). Estimated exports were 1000 tonnes and domestic demand was estimated to be 4500 tonnes, 1000 tonnes of which were in ethoxylated textile specialities, 1600 tonnes in ethoxylated pulp mill specialities, 500 tonnes of miscellaneous ethoxylates, 500 tonnes for trinonylphenyl phosphite and 900 tonnes miscellaneous, including pesticides and lube oil.

Based on more recent estimates, an increase in NPE use in Canada has been observed, (7,000 tonnes) (Metcalfe *et al.*, 1996). Eleven sectors reported use of NPEs as detergents, emulsifiers, wetting agents and dispersing agents. Five sectors were identified as major contributors to aquatic discharges of NPEs which included: pulp and paper manufacturing, petroleum production, household/industrial/institutional cleaning, textile manufacturing and leather manufacturing (IEC International Environmental Consultants Ltd., 1982; Chen, 1989). Minor uses of NPEs occur in the building and construction, paint and protective coating, metal processing, plastic and elastomer manufacturing, and food and beverage sectors. NPEs also are used as spermicides in contraceptive foams, jellies and creams.

Releases of NPEs to the environment can occur at various points in the product life cycle including: primary production, manufacture of NPE-containing products, product use, and disposal of the product to wastewater treatment, septic system or landfill.

NPE release estimates from 65 companies involved in primary production, manufacture of NPE-containing products or industrial use of NPEs in Canada were based on the 1997 survey results (Table 5). These release estimates are totals of NP and NPEs. Formulators and distributors of surfactants and industrial users of cleaning products, degreasers and detergents each released between 25 to 60 tonnes NP and NPEs in 1996. These two groups of industries accounted for the majority of total releases from industrial sources. Producers of paints, protective coatings, resins and adhesives released between 5.0-9.999 tonnes/yr. Formulators of industrial, institutional and domestic cleaning products, degreasers and detergents, pulp and paper mills, oil and gas recovery, producers of wastewater treatment products, formulators and distributors of products for the pulp and paper industry, and miscellaneous industries each

released between 0.100-4.999 tonnes NP and NPEs in 1996. Data in Table 5 does not include total NP/NPE releases to the environment via individual households, institutions and municipal wastewater treatment plants (MWWTPs). NP/NPE releases from textile mills are underestimated in Table 5.

NP and NPEs were not on the National Pollutant Release Inventory list of reportable substances for reporting years 1993-1999.

NPEs have been prohibited in Canada since 1997 as an active ingredient in soil supplements which are regulated under the Fertilizers Act (Webster, 1998).

NP and/or NPES are present in 211 pesticides currently registered for use in Canada. Forty percent of these products contain less than one percent NP or NPEs, 85% of these products contain less than 10% NP or NPEs and 95% of these products contain less than 20% NP or NPEs (Figure 8). NP and NPEs appear only as formulants in these pesticides, primarily as emulsifiers, surfactants, wetting agents, *etc.* The pesticide products containing NP/NPEs include acaracides, adjuvants, air sanitizers, disinfectants, antifouling paints, antisapstain fungicides, herbicides, insect repellents, insecticides, joinery and remedial wood preservatives, laundry additives, material preservatives, plant growth regulators, sanitizers, slimicides and swimming pool bactericides. The application rate of these products varies considerably, and is dependent on their use (Moore, 1999). In past, NP was used in aminocarb (4-dimethylamino-3-methylphenyl-N-methylcarbamate) insecticide sprays which were used against the spruce budworm (*Choristoneura fumiferana* Clem.) in eastern Canada (National Research Council of Canada, 1982). A typical formulation ("1.8D oil soluble concentrate") consisted of 19.5% aminocarb, 30% diluent 585 (the fraction of No. 2 fuel oil that distils at or below 585 °F), and 50.5% NP (by weight). In the field, additional diluent 585 was usually added to make a spray formulation containing 70 g of aminocarb and 182 g of NP in 1-1.5 L, and the formulation was typically sprayed at 1-1.5 L/ha (Maguire, 1999). In 1981, this formulation was replaced by an aminocarb formulation that did not contain NP.

2.2.4 Summary of entry of NP and NPEs to the environment

21,500 and 17,200 tonnes of NP and NPEs were available for use in Canada, in 1995 and 1996, respectively (Table 4). NP and NPEs have many industrial, commercial, institutional and household uses in Canada. They are used in textile manufacturing, pulp and paper manufacturing, petroleum refining, leather processing, various cleaning products, metal processing, paint and protective coatings, food and beverage processing, plastics manufacture, the building and construction industry, pesticide formulations and spermicidal preparations. Consequently, there are many routes for these substances into the environment during the

course of their production and use.

2.3 EXPOSURE CHARACTERIZATION

2.3.1 Environmental Fate

The majority of the literature on environmental persistence of NP and NPEs refer to the aquatic environment. The persistence of NP and NPEs in laboratory tests, municipal wastewater treatment plants and the natural environment is reviewed in this Supporting Document. Earlier reviews of environmental aspects of alkylphenols and alkylphenol ethoxylates have been performed by Beak Consultants Ltd. (1987), the Proceedings of the Seminar on Nonylphenol Ethoxylates and Nonylphenol (1991), Holt *et al.* (1992), U.K. Department of the Environment (1993), Talmage (1994), Balson and Felix (1995), Nimrod and Benson (1996a), Metcalfe *et al.* (1996), Dickey (1997), U.K. Environment Agency (1997), World Wildlife Fund Canada (1997), Thiele *et al.* (1997) and Staples *et al.* (1998).

The environmental persistence of a chemical will be affected by its physical and chemical properties in addition to ecosystem-specific properties such as (for aquatic ecosystems), the nature and concentration of microbial populations, the nature and concentration of dissolved and suspended material, temperature, degree of insolation, *etc.* In general, the important physical, chemical and biological removal mechanisms for chemicals in aquatic ecosystems are (i) volatilization and adsorption to suspended solids and sediment, (ii) chemical and photochemical degradation or transformation, and (iii) uptake and transformation by micro-organisms, respectively. The importance of such pathways for specific chemicals is dependent upon the chemical and the ecosystem. A detailed description of the way in which both physical-chemical properties and ecosystem-specific properties determine the fate of chemicals has been performed by Howard (1989).

2.3.1.1 Air

There is very little published data on NP or NPEs in air. Dachs *et al.*, (1999) detected NP (sum of 11 isomers) in all air samples collected from the urban and coastal atmosphere of the Lower Hudson Estuary. Concentrations of NPE in air of the New York-New Jersey Bight ranged from 2.2 to 70 $ng \cdot m^{-3}$. Dachs *et al.* (1999) predicted that NP may volatilize out of water into the air in areas where NPE concentrations are elevated in surface waters although the Henry's Law Constant for NP is low. The U.K. Environment Agency (1998) has estimated a half-life of 0.3 days for the reaction of hydroxyl radicals with NP in the atmosphere, indicating that it would be unlikely for any NP in air to be transported to remote regions. NPEs are far less volatile than NP

and, thus, it is expected that they would not partition to the atmosphere. Because of the presence of NPEs in aerially applied pesticide formulations, however, there is a need to determine their atmospheric chemistry, photochemistry and fate.

2.3.1.2 Water and Sediment

2.3.1.2.1 Abiotic Degradation

Based on the stability of NP during storage of control samples in biodegradation studies, the U.K. Environmental Protection Agency (1998) reported that hydrolysis and photolysis were negligible removal processes in the aquatic environment (Corti *et al.*, 1995; Trocmé *et al.*, 1988).

Under continuous sunny conditions, photolysis could be a significant degradation pathway for NP in shallow waters. The photolytic degradation rates for NP and OP were found to range between 10 and 15 hours in surface layers of natural water (Ahel *et al.*, 1994d). The rate of photolysis was found to be approximately 1.5 times slower at depths of 20-25 cm. A longer half-life was observed for NP1EO and higher chain NPEs were found to have half-lives exceeding 25 hours (Ahel *et al.*, 1994d). Sherrard *et al.* (1996) studied the degradation of NP(8-9)EO using heterogeneous photocatalysis with TiO_2. The ethylene oxide chain was found to be more susceptible to degradation than aliphatic or aromatic moieties. After irradiation for 96 hours of, a shift in the most abundant oligomer was observed to be n = 6-7 from n = 9. Within each homologue group, isomer peak patterns remained the same, suggesting little degradation of the aliphatic or aromatic moiety. It was, therefore, inferred that cleavage of ethylene oxide units, particularly the terminal units, had occurred. Recently, Brand *et al.* (1998) reported that OP (Igepal 520) was readily degraded using radiation at 365 nm or sunlight (90% degradation in 24 hours) in water, in the presence of Fe(III) (*e.g.*, 3×10^{-4} M). In the absence of Fe(III), no significant photodegradation was observed.

2.3.1.2.2 Biodegradation in Water in Laboratory Tests

The persistence of any intermediate or final product of both biodegradation and abiotic degradation should be considered during the examination toxicology and ecotoxicology of a given compound. Some chemicals which are produced during NPE degradation are more persistent than the parent compounds.

Although there are some conflicting reports in the literature, in general, NPEs and NP are not readily biodegradable using standard test methods. Substantial biodegradation, however, will occur after a period of acclimation. NPEs are, therefore, inherently biodegradable, and the

mechanism involves stepwise loss of ethoxy groups to lower NPE congeners, followed by the production of NPEC and NP, depending upon experimental conditions (Maki *et al.*, 1994; Rudling and Solyom, 1974). Branching of the nonyl group in NP and NPEs also retards biodegradation. APs and APEs are more persistent than alkylbenzene sulfonates and alcohol ethoxylates (*e.g.*, Patterson *et al.*, 1967, 1968, 1970; Davis and Gloyna, 1969; Sturm, 1973; Kravetz *et al.*, 1984, 1991; Dorn *et al.*, 1993; Strujis and Stoltenkamp, 1994; Ahel *et al.*, 1994b; Salanitro *et al.*, 1995; Salanitro and Diaz, 1995). Results from some representative studies that illustrate the above statements are described below. High chemical concentrations in biodegradation studies may result in artificially high persistence, if the chemical poisons the test organisms (U.K. Environment Agency, 1997).

NP9EO had a half-life of 6 weeks at an initial concentration of 5 mg/L in laboratory biodegradation tests (with dried activated sludge medium reconstituted) at neutral pH (Patterson *et al.*, 1968).

A branched chain NP9.5EO was 37% degraded in 12 days in a river die-away test, while a straight chain alkylbenzene sulfonate was 95% degraded in 12 days (Davis and Gloyna, 1969). One hundred percent degradation of a linear alkane sulfonate was observed in 5 days in a river die-away test, however, only 54% of the NPE was degraded in 30 days. Davis and Gloyna (1969) observed that algae and associated bacteria contributed to the degradation of surface active agents (including NPE 9.5EO) commonly found in household detergents to a small extent. Most of the degradation was attributed to bacteria and other microorganisms found in wastewater environments.

Quiroga *et al.* (1996) studied the biodegradation of NP15EO in river water using the "river die-away test" at concentrations of 1.5, 3 and 7 mg/L. Primary biodegradation of 85-90% of initial NP15EO levels was observed and disappearance of nearly all the homologues with >2 EO groups occurred by day 6 of 17 days. NPEC concentrations were not determined in this study. In a further study by this group (Manzano *et al.*, 1998), formation of NP2EC and NP1EC was reported. Primary biodegradation of 85% of the NP15EO at 2.5, 5 and 10 mg/L were observed by day 5, although subsequent degradation over the next 25 days was very limited.

Sturm (1973) developed a screening test consisting of the Thompson-Duthie CO_2 test (scaled down from 20 L to 6 L) and a biochemical oxygen demand test, performed with acclimated sewage-derived micro-organisms. In this study, the application of CO_2 production was used exclusively to examine nonionic surfactant degradation. Very little % theoretical CO_2 production was observed using branched NP8EO.

Rudling and Solyom (1974) determined biodegradation of branched chain NPnEO, where n = 8, 10, 14, 16 and 30, according to a screening procedure recommended by the Organization

for Economic Co-operation and Development (OECD), and in a laboratory-scale activated sludge system study operated with presettled sewage under treatment plant conditions. Typical test concentrations were 5 mg/L, the presettled sewage contained approximately 0.5 mg/L of nonionic surfactants. Gas chromatographic analysis of NP8EO, NP10EO and NP14EO samples incubated at 20 °C for 4 days showed that primary degradation was > 90%. The major product was NP2EO. Analysis of the NP8EO, NP10EO and NP14EO samples incubated for 28 days at 20 °C in the OECD screening test indicated that approximately 50% of the NP2EO had degraded, while at 15 °C no degradation was observed. In the activated sludge test, > 80% degradation was observed for all NPE compounds tested in 10 days. Chromatographic analyses of these test systems indicated that disappearance of the starting material was not due to adsorption to activated sludge, and no NP2EO was found.

Kravetz *et al.* (1984) studied both primary and ultimate biodegradation of an alcohol ethoxylate ("AE 25-9") and NP9EO under conditions that simulated a MWWTP under winter conditions. The rate of primary biodegradation of NP9EO decreased with a decline in temperature, but biodegradation of AE 25-9 was less temperature dependent. At low temperatures, NP9EO effluents foamed considerably while AE 25-9 effluents did not. Ultimate biodegradation of NP9EO to CO_2 was much less extensive than AE 25-9 at all temperatures studied.

Neufahrt *et al.* (1987) reported that > 95% primary degradation of NP10EO occurred in "biosimulators" with an activated sludge feature. Influent concentrations were 15 mg/L and 1-1.5, 6-7, and 9-12 µg/L, respectively, for NP10EO, NP, NP1EO and NP2EO. Concentrations of NP10EO, NP, NP1EO and NP2EO in the outflow were 460, 0.5, 5 and 20 µg/L on average, respectively.

Kravetz *et al.* (1991) observed that branching of the alkyl chain retarded biodegradation in continuous flow-through activated sludge tests that simulated waste treatment systems. NP9EO was not readily biodegradable by the OECD biological oxygen demand (BOD) test. Low biodegradability of NP9EO also was observed using the OECD modification of the Sturm test for ultimate biodegradation via CO_2 evolution (30% over 28 days) at 25 °C. At 8 °C biodegradation was further reduced.

Maki *et al.* (1994) isolated an APE-degrading bacterium from activated sludge in a Japanese MWWTP by enrichment culture and tested it on a NP9.5EO formulation. NP2EO was the predominant biodegradation product, although intermediates with a high degree of ethoxylation were observed. NP2EC also was found in the culture broth although relative amounts were not stated. Degradation of the nonyl moiety was not observed.

Struijs and Stoltenkamp (1994) reviewed the development of screening biodegradability tests for surfactants and concluded that a positive result in a "ready biodegradability test" could safely be extrapolated to aerobic environments in regions where domestic wastewater was processed by MWWTPs. Their results indicated that NP10EO and OP10EO was not readily biodegraded in dissolved organic carbon (DOC) die-away, manometric respirometry and closed bottle tests.

Ahel *et al.* (1994a) studied the aerobic transformation of NPnEO (n = 1-3) by mixed bacterial cultures using a shake-flask technique. Initial concentrations ranged between 0.5-2.5 mg/L. Transformation was almost complete in 6-23 days, but was slower in a mineral medium where the NPEs were the only carbon source. NPECs were the major metabolites of degradation. Autochthonous bacterial cultures from river water and a secondary sewage effluent also transformed NPnEO (n = 1-3) fairly efficiently, however, the transformation rate was highly dependent on temperature. Faster biodegradation rates were observed when microbes acclimated to NPEs were used. Bacteria from a relatively pristine source (a forest soil) also could effect biotransformation, albeit more slowly. Ahel *et al.* (1994a) concluded that NPEs could not be regarded as truly persistent compounds under aerobic conditions. NPnEO (n = 1-3) transformations were strongly dependent upon temperature, suggesting that their degradation in the aquatic environment during winter may be significantly retarded. Biotransformation of APEs was thought to occur via oxidation of the alkyl chain, however, no metabolites were found containing carboxylate groups in the alkyl side chain, nor was transformation of the aromatic ring found in the study. Ahel *et al.* (1994a) found that short-chain NPEs can be readily biotransformed in aerobic environments such as secondary sewage treatment and natural waters that are chronically polluted with surfactants and that NPECs are more persistent than NPEs with 1-3 EO units.

Salanitro *et al.* (1995) developed an automated pressure transducer system to evaluate the ready and ultimate biodegradability of surfactants in 28 days at low concentrations. NP9EO was not considered readily biodegradable because it did not result in production of >60% theoretical CO_2 (Balson and Felix 1995; Salanitro *et al.*, 1995).

Kveštak and Ahel (1995) studied the biotransformation of NPEs with 1-16 EO groups under laboratory conditions using a static die-away method under aerobic conditions. Mixed bacterial cultures from the brackish water layer of the Krka River estuary, Croatia, exhibited a significantly greater ability to transform NPEs than those from the saline water layer. Assuming first order kinetics, the biodegradation rates showed a strong temperature dependence. The elimination of higher NPEs was followed by a significant formation of the short-chain NPnEOs (n = 1-4). The main intermediate formed during the experiments was NP2EO, which accumulated quickly in the medium and was degraded slowly. Because the experiment was

performed under aerobic conditions, NP was not observed. The authors suggested that likely end-products were NPECs.

Anaerobic degradation studies over periods extending to 50 days were performed with NP9EO at 35 °C (Salanitro and Diaz; 1995). Mineralization of NP9EO to 32-43% of the theoretical methane (TM) was measured at the 50 mg C/g solids/L. It was, however, readily metabolized (70%) to methane at 10 mg C/g solids/L medium, indicating that higher concentrations may be inhibitory to methanogenesis. Surfactant concentrations found in activated sewage generally range between 0.5 - 10 mg/ g sewage solids, therefore, Salanitro and Diaz (1995) recommended that test concentrations for screening anaerobic metabolism should be performed at concentrations ranging from 0.5 to 10 mg surfactant/g solids/L or 0.3 - 5 mg C/g/L.

Frassinetti *et al.* (1996) isolated and characterized three different Gram negative bacteria which are primary degraders of NP9EO, i.e., use it as the sole energy and carbon source in axenic cultures. The source was activated sludge from a tannery wastewater treatment plant. The isolates were identified as a strain of *Pseudomonas putida*, strain Fus1B1, *Pseudomonas* sp. strain SscB2, and *Xanthomonas* sp. strain SscB3.

Cady (1996) studied the inherent biodegradability of ^{14}C-NPnEO (n = 1-14) (uniformly ring-labelled) by activated sludge in a laboratory test similar to the U.S. EPA modified semi-continuous activated sludge (SCAS) test. Mineralization of the ^{14}C-NPE to $^{14}CO_2$ was about 9% of the applied dose during a 30-day steady-state phase. A further 23% of sludge-incorporated ^{14}C activity was released as $^{14}CO_2$ during the die-away phase. Extensive degradation to highly polar intermediates was observed, although specific intermediates were not identified. A large portion of the radioactivity in the supernatant was too polar to be extracted from the acidic aqueous phase with dichloromethane, although 60% was still recovered. No NPE biodegradation products were identified in the remaining supernatant.

Williams *et al.* (1996) demonstrated that the degradation of NP1EC and NP2EC resulted in greater than 60% theoretical CO_2 production after 28 days of incubation using the OECD method 301B, which is a modification of the Sturm method. More than 10 days were required for the degradation to proceed from 10% to 60% theoretical CO_2. For classification as 'readily biodegradable' using the OECD method, however, degradation must proceed from 10% to 60% theoretical CO_2 in less than 10 days. Therefore, NP1,2EC were not considered to be readily biodegradable, but OP1,2EC were classified as readily biodegradable (Williams *et al.*, 1996).

Serak and Zhixing (1997) determined the persistence of ^{14}C-NPE in an aerobic river die-away test in the dark at 20 °C for 128 days (patterned after ASTM Standard Method E1279-89). After 128 days, 31-53% of the applied test substance was mineralized to $^{14}CO_2$. The organic-extractable radioactivity decreased from 74% at day 28 to 33% at day 128 and non-extractable

radioactivity increased from 12% at day 28 to 46% at day 128. Based on these results, Serak and Zhixing (1997) reported that NPEs could be depleted from the environment due to microbially-mediated mineralization.

Corti *et al.* (1995) studied the aerobic biodegradation of (pure) 4-(1-nonyl)phenol by a yeast (*Candida maltosa*) strain isolated from an aerobic sludge sample taken from a treatment plant that received textile plant effluents. The yeast was able to utilize 4-NP as a sole carbon and energy source. Degradation resulted in production of 4-acetylphenol, which indicated attack on the NP alkyl chain. Results of nuclear magnetic resonance (NMR) analyses coupled with comparisons between gas chromatography data from an extract of the incubation mixture and results of a 4-acetylphenol sample confirmed 4-acetylphenol presence in the extract.

Tanghe *et al.* (1998) reported that NP degradation is strongly temperature-dependent based on studies with laboratory scale activated sludge. Degradation was very efficient at 28 °C, although efficiency was dramatically reduced at temperatures between 10 and 15 °C.

In a laboratory-scale OECD screening test, Di Corcia *et al.* (1998) found that MWWTP microorganisms were capable of transforming NPEs to a variety of compounds that were carboxylated on both the ethoxy and alkyl side chain (CAPECs). Production of lower ethoxylates with carboxyl groups on C_8 and C_6 side chains, rather than on the ethoxylate moiety (CAPES) also was observed. HPLC was used in these analyses and had low resolution efficiency, therefore, individual peaks likely contained several isomers. The positions of each carboxylate group on the alkyl side chains were not determined. NP2EC was a found to be a major metabolite over the first 20 days. The maximum NP2EC concentration was observed at approximately 12 days, followed by a decline in concentration by about 50%. The reduced NP2EC levels remained stable over a 2-3 month period prior to declining to low levels. CAPEs and CAPECs were detected approximately 12 days into the experiment. The concentration of CAPEs peaked at about 30 days, followed by a decline in concentration to undetectable levels at 175 days. CAPEC concentrations increased until about 30 days and remained at a constant concentration until the end of the experiment at 180 days.

Degradation of the ethoxylate moiety of NPEs results in the production of polyethylene glycol (PEG). A few studies have shown that the PEG residues are more persistent than the parent NPEs (Patterson *et al.*, 1967; Tobin *et al.*, 1976; Maki *et al.*, 1994). Tobin *et al.* (1976) found that the polyethoxylate moiety of an alkyl ethoxylate surfactant (Dobanol 25-9) remained essentially undegraded long after the Wickbold analysis indicated removal of 95% of the parent compound.

Although NPEs are not readily biodegradable, they are inherently biodegradable and the mechanism involves stepwise loss of EO groups to lower NPE congeners, followed by the

production of NPECs and NP, depending upon experimental conditions. The degradation pathway is shown in Figure 9. This pathway is an over-simplification because it does not include NPnEC where n > 2, or CAPECs or CAPEs. The intermediate and final products of metabolism are more persistent than the parent NPEs, although these compounds also will ultimately be biodegraded. Branching of the nonyl group in NP and NPEs retards biodegradation, as does increase in length of the EO chain. APs and APEs are more persistent than alkylbenzene sulfonates and alcohol ethoxylates (Kravetz *et al.*, 1991; Maguire, 1999). The use of high concentrations of chemicals in biodegradability tests may result in artificially high persistence data if the chemical poisons the test organisms. This possibility has been suggested to account for some differences in results for the biodegradability of NPEs (e.g., U.K. Environment Agency, 1997).

There are much fewer biodegradation data for APEs other than NPEs, consequently, general conclusions for all APEs can not be made. Ball *et al.* (1989), however, studied the biotransformation of halogenated and nonhalogenated OPE residues under both aerobic and anaerobic conditions. Under aerobic conditions, the ethoxy chain of longer chain OPEs and OPECs was transformed to relatively stable OP2EO and OPnEC ($2 \leq n \leq 4$). Further transformation to unidentified products was possible after long adaptation times. Under anaerobic conditions, transformation was incomplete despite long adaptation periods (190 days). Biotransformation of halogenated OPEs and OPECs having greater than two EO chains resulted predominantly in the formation of the halogenated aromatic compound XOP2EC, which was resistant to biotransformation under both the aerobic and anaerobic conditions under investigation. Williams *et al.* (1996) reported that OP1EC and OP2EC would be classified as readily biodegradable based on the OECD method 301B.

2.3.1.2.3 Biodegradation in municipal wastewater treatment plants

Primary biological degradation of NPEs is the major route of degradation. Degradation is faster in MWWTPs than in natural environments because of the higher concentration of microorganisms in MWWTPs relative to natural environments. Most municipalities in Canada have some type of sewage treatment system. MWWTPs play a significant role in the transformation and degradation of NP and NPEs before their entry into the environment. It has been estimated that MWWTPs discharge 60 - 65% of all nonylphenolics that enter the plant to the environment either as effluents or sludges (Ahel *et al.*, 1994a),.

Once APEs, including NPEs are released to sewage treatment systems, several transformations can occur. Initially, there is a loss of EO groups from the original moiety. Under aerobic and anaerobic treatment conditions, biodegradation to more toxic and estrogenic metabolites occurs. NPE biodegradation products, include NP, NP1EO, NP2EO, NP1EC and

NP2EC. Similar metabolites are formed by the degradation of OPEs and other APEs in MWWTPs.

Discharges from MWWTPs provide the two major routes for environmental release of NPEs and their degradation products. The first route is by discharge of the final treated effluent to nearby receiving waters. The final effluent composition is dependent on the treatment process(es) used in the facility. If primary treatment is used exclusively, the effluent composition reflects the short hydraulic retention time and longer chain ethoxylated products (*i.e.*, NPnEO, where n = 3-20) are dominant (82%) with minor components of NP (3%), NP1EO and NP2EO (12%), and NP1EC and NP2EC (3%). Effluent composition from secondary-treated MWWTPs is substantially different from primary-treated effluent (Figure 10). Longer chain NPEs comprise only 28% of the nonylphenolic compounds in secondary-treated effluent and metabolites make up the rest. Carboxylic acid metabolites (*i.e.*, NP1EC and NP2EC) account for about 46% of the effluent composition, NP1EO and NP2EO represent 22% and NP accounts for only 4% of total nonylphenolic compounds in the effluent (Ahel *et al.*, 1994a).

NP and OP tend to be associated with the second major environmental release route for alkylphenolics in sewage treatment processes, the sludge and sediments because of their low water solubility relative to the other metabolites. Most Canadian MWWTPs that employ secondary or tertiary treatment utilize the activated sludge process which is an anaerobic digestion process. NP is the dominant nonylphenolic substance adsorbed onto the sludge in the activated sludge process. Up to 95% of the nonylphenolic composition of digested sludge may be attributed to NP. Sludge is disposed of in three ways, by incineration, by landfilling and by spreading on agricultural soils. Little research has been done on the fate of nonylphenolics in sludge disposed by any of these three techniques.

Full-scale MWWTPs can provide greater removal efficiencies of NPEs than bench-scale systems, which may be due to a greater variety of microbial populations and nutrients in the former (Holt *et al.*, 1992). In general, primary biodegradation of NPEs in MWWTPs is readily achievable, but ultimate biodegradation is not. Substantial differences in treatment efficiencies for NPEs and their degradation products exist among MWWTPs. These differences have been attributed to the load of NPEs in influent streams and MWWTP design and operating conditions, including temperature of treatment. In some locations, more persistent products such as NP and lower-chain NPEs have been observed in MWWTP final effluents and receiving waters. In addition, substantial concentrations of NP and lower-chain NPEs are found in sludges from MWWTPs. The application of NP-containing sludges to agricultural land may result in potential exposure in terrestrial environments.

Mann and Reid (1971) studied biodegradation of OPEs at a trickling filter MWWTP where organisms were acclimated to inputs of the various detergents in Preston, England. To avoid a lag phase in biodegradation, acclimated bacteria were used for this study. The entire village was supplied with detergents during acclimation trials. During winter, 20% of the OPEs were biodegraded, however, in summer biodegradation increased to 80%, indicating that biodegradation of OPEs was temperature dependent. Despite the relatively high rate of biodegradation, effluents had a considerable tendency to foam even in summer, therefore, Mann and Reid (1971) concluded that in general, OPEs were not biodegradable.

Stiff *et al.* (1973) studied the biodegradation of an OPE (Shell Nonidet 40, 8-9 EO groups per mole, predominantly 1,2,3,3-tetramethylbutylphenol) and two alcohol ethoxylates using small-scale "porous pot" activated sludge MWWTPs. At 15 °C, 95% of the OPE was degraded, however, at lower temperatures biodegradation rates were dependent upon concentration. At 5 mg/L, greater than 90% removal was achieved, but at 20 mg/L the degree of removal fluctuated between 40 and 95% at 11 °C. At 8 °C, biodegradation ranged between 20 and 80%. These results confirmed the effect of temperature on OPE biodegradation observed by Mann and Reid (1971).

In another early study, Jones and Nickless (1978b) determined nonionic detergents concentrations in MWWTP influents and effluents downstream from Bath, England. Low levels of polyethoxylated material were expected because regional domestic consumption of detergents based on secondary alcohol ethoxylates was fairly light. Concentrations of the nonionic detergent components at the influent were estimated to be 700 µg/L, while effluent concentration estimates were an order of magnitude lower (70 µg/L). Concentrations in the receiving river water in the Avon River were 8 µg/L downstream from the MWWTP. APEs were found to be more persistent than aliphatic alcohol ethoxylates.

Giger *et al.* (1981) and Stephanou and Giger (1982) detected NP, NP1EO, NP2EO and NP3EO in the River Glatt which flows through a densely populated area east of Zurich and in secondary sewage effluents in Switzerland. Higher ethoxylated NPEs were not observed in effluent or river samples in these studies, indicating that the lower ethoxylated NPEs and NP are more persistent than the parent NPEs.

During the chlorination stage of wastewater treatment, bromination of APEs to a slight extent was observed by Reinhard *et al.* (1982). Production of carboxylates corresponding to APEs also was observed during sewage treatment processes (Reinhard *et al.*, 1982). Stephanou (1985) also reported production of brominated and chlorinated AP1EO and AP2EO and AP1EC from APEs during water chlorination treatment. These metabolites were found in secondary effluent samples from Swiss MWWTPs which utilize conventional mechanical-biological activated sludge systems.

Surfactant biodegradation has been studied extensively in the Glatt River in Switzerland (Giger *et al.*, 1984; Giger *et al.*, 1986, Ahel and Giger 1985a; Ahel and Giger 1985b; Giger *et al.*, 1987). This river system has low flow [3-8 m^3/s] and, therefore, MWWTP effluent dilution is low relative to many other research sites.

In some of the early research with NPEs, Giger *et al.* (1984) concluded that the large amount of NP they found in anaerobically treated sewage sludge (mean 1 g/kg dw) originated from NPEs. NP concentrations in anaerobically stabilized sludge were much higher than in aerobically stabilized sludge. Giger *et al.* (1984) proposed that during aerobic treatment of wastewater, NPE ethoxylate chains were shortened by microbial transformation during aerobic treatment of wastewater, resulting in NP1EO and NP2EO which were less biodegradable. These less water-soluble metabolites were partially removed from the water column via adsorption onto lipophilic components of sludge. When the sludge was stabilized, NP1EO and NP2EO degraded further to NP, which accumulated in the digested sludge.

Ahel and Giger (1985a) also reported NP in anaerobically digested sludge (1000 mg/kg) and effluent (467 µg/L) from the anaerobic sludge digester from the Glatt MWWTP, Zürich, Switzerland. In some of the earlier laboratory biodegradation experiments with NPEs, NP was not detected, however, the results of the work by Giger *et al.* (1984) and Ahel and Giger (1985a) indicated the formation of NP by anaerobic processes during wastewater and sludge treatment.

Ahel and Giger (1985b) studied the biodegradation of Marlophen 810 NP surfactant in a laboratory-scale, continuously operated biodegradation apparatus used for simulating aerobic sewage treatment. NPEs with 1-17 EO units were observed in the influent to the apparatus, however, NPEs with <4 EO groups were present in effluent samples. Presence of NPECs also was suspected in effluent samples. Higher ethoxylated NPEs were found in municipal wastewater samples, while NP1,2EO were dominant in treated effluents from both the Dubendorf and the Zürich-Glatt MWWTPs. NPEs with 1 to 5 EO groups were present in river water samples collected a short distance downstream from the point where MWWTP effluents entered the river system.

Giger *et al.* (1986) reported NPnEOs (n = 3-20) in raw and primary sewage effluents of MWWTPs in Switzerland at concentrations ranging between 400 and 2200 µg/L, corresponding to 3-10% of the total dissolved organic carbon. Using the activated sludge treatment, >90% of the NPEs were eliminated. Biologically treated sewage effluents contained NP and the lower ethoxylated NPEs, NP1EO and NP2EO at concentrations of 10-100 nmol/L. An increase in NPEC concentrations were observed in samples collected following activated sludge treatment (200-3000 nmol/L).

Brown *et al.* (1986) applied an optimized bismuth iodide active substance (BIAS) procedure to follow the removal of nonionic surfactants, including NP9.5EO in an activated sludge plant. They reported typical NP9.5EO concentrations of 1-2 mg/L in Hochdahl (Düsseldorf, Germany) raw/settled sewage. Greater than 90% of NP9.5EO was removed during the activated sludge treatment. Using the same methodology, Brown *et al.* (1987) studied the removal of nonionic surfactants in a trickling filter plant (at Hösel-Dickelsbach, Germany) under winter (March) and summer (September) conditions. In both March and September, 88-89% removal was observed for cationic surfactants, indicating no temperature affect. However, removal of the nonionic surfactants was lower (81±3%) in March than in September 88±1%.

Giger *et al.* (1987) analyzed APEs in effluents and sludges from 13 Swiss sewage treatment plants. Parent NPEs were efficiently removed by the activated sludge treatment. The majority of APEs were present as NPEs although OPEs and decyl polyethoxylates also were detected. Total APnEO (n = 3-20) concentrations in influents ranged from 400 to 2200 µg/L. APEs in Swiss laundry detergents generally followed Poisson distributions, with the majority having 9-10 EO units. APE with 1-2 EO chains were very minor constituents (<1%) and NP was not present in these laundry detergents. Raw wastewaters and primary effluents, however, typically contained bimodal NPE oligomer distributions, with one maximum occurring at 1 or 2 EO units and the second at about 7 EO units. NP also was present in substantial concentrations. These results reflected aerobic and anaerobic biotransformation which occurred in the sewers and during mechanical treatment (i.e., settling). The majority of NPEs were present as 1 or 2 ethoxylates in effluent from secondary treatment which was operated under low-loading, nitrifying conditions or high-loading non-nitrifying conditions. Giger *et al.* (1987) compared NPE concentrations in primary and secondary effluents of four MWWTPs. NPEs with 3-20 EO groups were substantially removed in the secondary effluents, NP1EO and NP2EO concentrations either declined or increased, NP concentrations decreased and higher NP1EC and NP2EC concentrations were observed in secondary effluents relative to primary effluents. Similar observations were found in the Uster MWWTP, which had tertiary treatment. Considerable build-up of NP also was observed in activated and digested sludge. NP1EO and NP2EO also was produced in digested sludge.

Ahel *et al.* (1987) measured NP1EC and NP2EC levels in influents and effluents of mechanical-biological MWWTPs and in the River Glatt in Switzerland. High concentrations of NP1EC and NP2EC were observed in secondary sewage effluents (71-330 µg/L), whereas untreated sewage and primary effluents contained much lower levels (<1-17 µg/L). NPECs (2-116 µg/L) also were observed in the Glatt River. Total NP1EC and NP2EC concentrations were higher in the Glatt River than total NP, NP1EO and NP2EO concentrations. NP2EC concentrations were higher than NP1EC levels in the Glatt River and MWWTP effluents. NPEC concentrations represented up to 1.9% of the DOC in the river. Although earlier research had suggested that carboxylation of branched chains may occur, this was not observed in any samples.

NPEs were more abundant in untreated sewage and primary effluents than NPECs, but the reverse was observed in secondary effluent. Therefore, it was concluded that typical NPE metabolites under aerobic-activated sludge treatment in the MWWTPs were NPECs and that NP1EO and NP2EO levels were reduced in secondary effluents relative to untreated sewage and primary treatment effluents. Biotransformation of NP1EO and NP2EO to the less lipophilic NPECs was suspected during secondary treatment.

Marcomini *et al.* (1988a) measured NP, NP1EO and NP2EO in both the dissolved and particulate phases of raw sewage, primary effluent, secondary effluent, and primary and secondary sludge from the Glatt MWWTP, Zürich, Switzerland on two consecutive days in May 1986. Substantial accumulation of NP (1100 mg/kg) was observed in anaerobically-digested sludge, while NP1EO (230 mg/kg) and NP2EO and (30 mg/kg) concentrations were much lower. Poor removal of NP1EO and NP2EO was observed in the secondary effluent.

Brunner *et al.* (1988) determined NP, NP1EO and NP2EO fluxes through sewage and sludge treatment at 29 Swiss MWWTPs. Sludges stabilized by aerobic treatment contained less NP than those treated anaerobically. About 50% of the NPEs in sewage was transformed to NP and accumulated in the digested sludge. Both NP1EO and NP2EO, which are precursors for NP, were partially degraded during aerobic and anaerobic sewage and sludge treatment. Because NP1EO and NP2EO loadings in the raw sludge were higher than in raw sewage implied that higher ethoxylated NPEs were thought to produce NP1EO and NP2EO during aerobic wastewater treatment.

Chen (1989) found that secondary (biological) treatment was up to 90% effective in reducing parent nonionic surfactant concentrations using the CTAS analysis during an environmental assessment of the Canadian textile industry in 1985-1986. This assessment included a detailed examination of the effluents of 10 mills that produced a variety of fabrics. The analysis of NPE degradation products was not performed in this study.

Kubeck and Naylor (1990) reported that removal efficiency of NPEs with 1 to 18 EO chains exceeded 90%. They also found that exposure of NPEs to oxygen during the course of extraction could lead to degradation, with production of abnormally high levels of lower ethoxylated NPE oligomers. This may have affected experimental results in studies where proper preservation procedures were not employed.

Based on results from the Zürich-Glatt MWWTP, Giger and Ahel (1991) estimated that 60-65% of all nonylphenolic compounds (i.e., compounds still having the alkylbenzene moiety) that enter sewage treatment plants are released into the environment, 38-42% are discharged via secondary effluents and 21-23% via digested sewage sludge.

Clark *et al.* (1991) analyzed effluents from three publicly owned treatment works (POTWs) in New Jersey. POTW-A was located in a rural area with no known industrial contributor of waste. POTW-B had 73% (by volume) domestic waste, and 27% industrial waste (pharmaceutical, yeast, paper processing and chemical manufacturing). POTW-C had 82% (by volume) domestic waste and 18% industrial waste (from 300 industries, mostly textile and dye manufacturers). POTW-B was about three times larger than POTW-A, and POTW-C was about three times larger than POTW-B. NP, NP2EO, NP3EO, NP4EO and NP5EO concentrations were generally < 15 µg/L in effluents from each POTW. The maximum NP2EO concentration (123 µg/L) was observed in POTW-C effluent. Elevated levels of NP5EO (25 µg/L) also were observed in effluent from POTW-C.

Birch (1991) studied the effect of temperature and sludge retention time on degradation efficiency of NPEs using a porous pot activated sludge reactor. Primary degradation was extensive at 15 °C and 11 °C, but at 7 °C, high levels of the parent NPEs were found in plants operating at sludge retention times (SRT) of 2, 4 and 6 days. The results of the study suggested a critical SRT of about 6 days at 11-15 °C which was consistent with other biodegradation studies performed during winter months.

Because of the high use of NPEs in Israel, Zoller (1992) studied removal efficiencies of nonionic surfactants in Israeli MWWTPs. He reported approximately 69-81% surfactant removal (Zoller, 1992) and in later work, reported 97% removal if combined activated sludge/soil aquifer treatment were used (Zoller, 1994).

In a survey of wastewater treatment effluents across the U.S., Naylor (1992) found high removal efficiencies of NPEs from plants that employed biological treatment. Greater than 92% removal was observed in all but one plant which had a removal efficiency of 84%. In effluents from two North Carolina plants, NPE oligomer distribution was not skewed, nor was enhancement of NP or NP1EO observed. In a Midwest wastewater plant, however, the oligomer distribution was skewed to the lower ethoxylated NPEs (NP1EO and NP2EO). Although NP accumulation was anticipated in sludge from the plant based on results from MWWTP effluents in Switzerland, NP levels in dry sludge remained ≤ 10 mg/kg (Naylor, 1992). This work and later work (Naylor, 1995) indicated that digested sludge in some U.S. MWWTPs is not a major sink for NP.

Ahel *et al.* (1994b) studied the behaviour of NPEs in several full-scale mechanical-biological MWWTPs in the Glatt Valley, Switzerland. Untreated sewage and primary effluents contained considerable amounts of surfactant-derived nonylphenolic compounds (3.0-9.6% of the DOC). The average elimination efficiency of parent NPEs having 3 to 20 EO groups was 72%. Biodegradation was limited due to the formation of biorefractory metabolites, including NP, NP1EO and NP2EO, and NPECs. As expected, the highest elimination rates were achieved in

the MWWTPs characterized by low sludge-loading rates and nitrifying conditions. Ahel *et al.* (1994b) estimated that 60-65% of all nonylphenol compounds (i.e., compounds that retained an alkylbenzene moiety) that entered sewage treatment were released into the environment either in secondary effluent or sludge. Approximately 19% of all nonylphenolic compounds introduced to MWWTPs was released to the environment in the form of NPEC, 11% in the form of NP1EO and NP2EO, 25% in the form of NP, and 8% as higher ethoxylated NPEs. Almost all of the released NPEs and NPECs, as well as most of the NP1EO and NP2EO, were discharged into natural receiving waters via secondary effluents. Secondary effluents contributed 60% of all nonylphenolic compound inputs into the environment. In contrast, most NP (>90%) was emitted to the environment via digested sewage sludge, which is representative of approximately 40% of the total load. Only a minor fraction of NP reached receiving waters directly via secondary effluents. The sludge-bound NP, which is often disposed via application of sludge onto soil, may enter natural waters through run-off and leaching.

Di Corcia *et al.* (1994) provided evidence for the production of NPnEC, where n = 3-10, in effluents from an Italian secondary treatment MWWTP. The maximum total NPEC (NP1-3EC and NP>3EC) concentrations reported in effluents was 145 µg/L.

Field and Reed (1996) found that NP2EC was the dominant oligomer in 14 paper mill effluents. The average proportions of NPECs in paper mill effluents were NP1EC (16%), NP2EC (72%), NP3EC (10%), and NP4EC (2%). Industrial wastewater treatment is generally characterized by higher temperatures, increased hydraulic residence times and greater degrees of acclimation than that of MWWTPs. Because MWWTPs operate at ambient temperatures, more seasonal variation in effluent composition would be expected from MWWTPs than from industrial effluents. Field and Reed (1996) reported NPEC concentrations in the Fox River and other U.S. rivers and observed that concentrations were approximately a factor of 10 lower than reported in the Glatt River in Switzerland. This difference was related to the lower dilution observed in the Glatt River (3-8 m^3/s) relative to U.S. systems (e.g., Fox River 41 m^3/s).

Paxéus and Schröder (1996) reported removal efficiencies of >90% via primary degradation of NP9EO in two small Swedish MWWTPs. NP removal was < 10% in one plant and 38% in another. Sludges contained 20-26 mg NP/kg of sludge (dw).

Weeks *et al.* (1996) reported that levels of NP1EC and NP2EC were comparable to NP, NP1EO and NP2EO in U.S. rivers. These results were in contrast with the reports of high NPEC concentrations in MWWTP effluents reported by Ahel *et al.* (1994c) in Switzerland. Weeks *et al.* (1996) also reported studies where efficient NPE treatment in wastewater was observed and little residue remained in sludge.

NPE and OPE concentrations as high as 450 µg/L in the influent to two Australian tertiary MWWTPs were reported by Mackay *et al.* (1997). Parent NPEs and OPEs were reduced to below detection limits (5 µg/L) in the effluents, although no details of the tertiary treatment processes were provided.

Fytianos *et al.* (1997) studied biodegradation of NPEs in a MWWTP of Thessaloniki, Greece. They reported the removal efficiency from the secondary treatment, which was defined in this work as biodegradation plus adsorption to sludge, to be 92-97% over a six-month period.

Lee *et al.* (1998) measured NP1EC and OP1EC concentrations in influents, primary effluents and final effluents of some Canadian MWWTPs. NP1EC concentrations in influents ranged from 0.9 to 8.3 µg/L (n = 5, mean = 3.6 ± 3.3 µg/L , median = 1.6 µg/L), concentrations in primary effluents were between 2.4 and 17.7 µg/L (n = 10, mean = 6.2 ± 4.7 µg/L , median = 4.2 µg/L) and concentrations in final effluents ranged between 3.2 and 703 µg/L (n = 10, mean = 82.9 ± 218.1 µg/L, median = 13.9 µg/L). Although NP2EC concentrations were not measured, Lee *et al.* (1998) anticipated that similar trends for production of NP2EC and NP1EC would occur in the MWWTPs. Similarly, OP1EC and OP2EC production trends were similar.

Lee and Peart (1998) measured NPEs and their metabolites in municipal wastewater samples and effluents from a Canadian MWWTP that used primary and secondary treatment. Composite samples of raw sewage, primary effluent and final effluent were collected over 24 hour periods on a monthly basis over the course of one year between 1997 and 1998. Concentrations of NPEs with 1 to17 EO groups, NP1EC and NP2EC were determined. Lee and Peart (1998) reported that approximately 85% of the total nonylphenolic compounds in raw sewage were present as ethoxylates, however, the major component (nearly 80%) in the final effluent was in the form of carboxylic acids. During the study period, the median total alkylphenolic compound concentrations in raw sewage and final effluent were 526 and 248 nmol/L, respectively, which represented an overall elimination rate of 53%. The estimated median daily discharge of nonylphenolic compounds to the aquatic environment was 20 moles. These data suggested that conventional sewage treatment was ineffective in the removal of NPE metabolites. The overall elimination rate of 53% was similar to the mean elimination rate of nonylphenolic compounds reported by Ahel *et al.* (1994b) for 11 MWWTPs in Switzerland (59%).

Degradation studies with NPEs containing 3-17 EO groups were performed in two Canadian MWWTPs during 1997 by the Water Technology International Corporation (1998b). MWWTP A was monitored for two weeks, but MWWTP B was only monitored for two days, however, it was the same MWWTP studied by Lee and Peart (1998). MWWTP A was a conventional activated sludge secondary treatment plant where nitrification was performed, followed by tertiary treatment and UV disinfection. MWWTP B employed a conventional non-

nitrifying activated sludge process where disinfection by chlorination was only performed between May and October. Because of significant textile industry discharges, MWWTP A received a high load of NPEs. Total APs represented the sum of NP, NP1EO, NP2EO, NP(3-17)EO, NP1EC, NP2EC, OP, OP1EC and OP2EC concentrations. The total alkylphenolic mass loading rate in the treated effluent plus sludge releases from MWWTP A was substantially lower than the average influent mass loading of 44 kg/d. The average discharge loadings in the final effluent and digested sludge were 1.2 kg/d and 6.4 kg/d, respectively. A net destruction efficiency of 83% was reported. The majority (84%) of the alkylphenolic compounds were present in the sludge rather than in the final effluent from MWWTP A (Figure 11). In the sludge, 75% of the total alkylphenolic compounds were present as NP, 22% as APEs and 2% as APECs. However, in the final effluent, 75% was present APECs, 21% as APEs and 3% was as NP. The total alkylphenolic compound mass loading rate in the treated effluent plus sludge releases from MWWTP B also was substantially lower than the influent mass loading of 40.4 kg/d. Discharge loadings of 5.6 kg/d and 5.9 kg/d were reported in the final effluent and digested sludge, respectively. A net destruction efficiency of 72% was reported for the second MWWTP under investigation. Only 51% of the total alkylphenolic compounds in the discharge (sum of effluent plus sludge) were present in the sludge fraction. Fifty-six percent of the alkyphenolic compounds were present as NP, 37% as APEs and 4% as APECs. In the final effluent, 69% of the alkylphenolic compounds were present as APECs, 29% as APEs and 2% as NP.

Differences observed in treatment efficiency of NPEs and their degradation products between MWWTPs, have been attributed to differences in NPE loads in influent streams, MWWTP design and operating conditions (e.g., temperature of treatment). NPE treatment in trickling filter MWWTPs is significantly reduced in the winter relative to summer. Birch (1991) and Watkinson and Holt (1991) also have reported that the sludge retention time (SRT) is a critical control parameter for the treatment of NPEs in MWWTPs utilizing the activated sludge process. The growth rate for competent organisms within the total microbial population is dictated by the SRT. When the growth rate of the organisms is less than the SRT, the competent organisms are washed out of the system and little treatment of the specific substance occurs. The growth rate of organisms is influenced by temperature and, thus, decreasing both the SRT and temperature will result in a less effective biodegradation system. Watkinson and Holt (1991) reported the normal range of SRTs for activated sludge plants to be between 6 and 20 days. Ahel *et al.* (1994b) observed that the highest NPE elimination rates were achieved in MWWTPs characterized by low sludge-loading rates and nitrifying conditions. This was confirmed in a limited study of two Canadian MWWTPs (Water Technology International Corporation, 1998b). Substantial differences in NPE treatment efficiencies have been observed between treatment facilities dedicated to the treatment of industrial wastewater and relative to MWWTPs. Field and Reed (1996) reported that industrial wastewater treatment may be characterized by higher temperatures, increased hydraulic residence times and greater degrees of acclimation than observed in MWWTPs. Because MWWTPs operate at ambient temperatures, more seasonal

variation in effluent composition would be anticipated from MWWTPs than from industrial effluents (Figures 12 and 13).

2.3.1.2.3.1 Summary

In general, a wide range of treatment efficiencies have been observed for NPEs during wastewater treatment. Primary biodegradation, as measured by the loss of surfactant properties or determination of parent compound is facile. The biodegradation mechanism initially involves the loss of EO groups, followed by the production of NP1EO and NP2EO, their carboxylate derivatives (NP1EC and NP2EC NPnECs [where n >2] and CAPECs and CAPEs) and the final product, NP. NPE biodegradation results in some production of more persistent, lipophilic compounds that are not completely degraded during sewage treatment (e.g., NP and NP1EO and NP2EO). These compounds tend to accumulate in sludges, while the more water-soluble NPECs are generally ionized completely at the pH of most natural waters. The majority of NPECs are found in the final effluents, sometimes at much higher concentrations than other nonylphenolic compounds. Although the majority of NP, NP1EO and NP2EO are associated with sludge, they also may be found in effluents, and have been detected in receiving waters. Halogenation of some of the NPE metabolites has been observed in MWWTPs that use chlorine for disinfection.

2.3.1.2.4 Biodegradation in water and sediment

There are far fewer persistence and fate studies for NPEs and NP in aquatic ecosystems relative to the number of biodegradation studies performed in MWWTPs and their effluents. Some of the persistence and fate studies are, however, described below.

Schöberl and Mann (1976) reported a degradation efficiency of 33-36% for NP9EO in freshwater at 20-23 °C over 50 days, although 95% degradation was observed in sea water over 25 days. Approximately 37% of the NP9EO was degraded at 3-4 °C in freshwater over 50 days, but only 15% degradation was observed in sea water over 50 days. Only primary degradation was determined in this study due to limitations in the methodology.

Yoshimura (1986) studied biological degradation of NP9EO in river water and sediment mixtures. Greater than 98% degradation of NP9EO was observed in a stirred water phase within 5 days but, in standing water it required 10 days. NP1EO, NP2EO and NP3EO were detected at low concentrations (< 0.4 mg/L) in samples collected at both 5 and 10 days. NP1EC and NP2EC were both identified as intermediates in the NP9EO degradation process.

Ekelund *et al.* (1993) studied NP biodegradation in sea water and sediment, using ^{14}C ring-labelled NP at a concentration of 11 µg/L. NP degradation was very slow during the first 3-4 weeks in the absence of sediment, but increased after 28 days, which indicated the need for microbial acclimation. After 58 days, 50% degradation of NP was observed in this study. In the presence of sediment, no lag time prior to the commencement of NP degradation was observed. Within 58 days 40% degradation was observed under aerobic conditions and 20% degradation was reported for anaerobic conditions, indicating some persistence in both water and sediment.

Kveštak *et al.* (1994) studied NPE inputs via untreated wastewater effluents and distribution of NPEs in the stratified Krka River estuary of Croatia. Partitioning of the lower ethoxylated NPEs to suspended solids was observed in both sewage and estuarine water samples. NPE oligomers were distributed around a maximum of NP10EO, which was consistent with heavy duty detergents used regionally (Kveštak *et al.*,1994). Biodegradation in the estuary appeared to be slower than observed in freshwater systems, although no kinetics were derived.

Ahel *et al.* (1994c) studied the behaviour of NPEs and their metabolites in the Glatt River where treated wastewater constitutes 20% of the lower reaches of the river. Biodegradation was reported to be the predominant mechanism controlling the fate of nonylphenolic compounds in this river system. The majority of the nonylphenolic compounds in the Glatt River were present as persistent metabolites. NPECs were the most abundant NPE metabolites observed in the Glatt River, with less frequent detection of NP1EO and NP2EO. NP was significantly less abundant and the higher ethoxylated NPEs were minor contributors to total nonylphenolic compounds. NPE degradation products in the river water were present in similar ratios to those observed in MWWTP secondary effluents in the valley. Nonylphenolic compounds contributed between 0.4 and 1% to the total DOC in the most polluted part of the river. The elimination efficiency of nonylphenolic compounds in the 35 km reach of the river under investigation was estimated to be 24%, although a net accumulation of NP1EC and NP2EC was observed. In a survey of Swiss rivers, NP was detected at concentrations up to 100 µg/L and was a predominant species in river sediments (Ahel *et al.* 1994c).

Ahel *et al.* (1996) studied the behaviour of various relatively persistent NPE metabolites during infiltration of river water to groundwater in the Glatt and Sitter rivers, in Switzerland. NP1EO, NP2EO and NP were generally removed during infiltration, although NP1EC and NP2EC were not. NP adsorption to soil matter in the aquifer was reduced corresponding to a decrease in temperature. The elimination efficiency was found to be greatest for NP2EO, followed in descending order by: NP1EO > NP > NP1EC = NP2EC.

Maki *et al.* (1996) studied the biodegradation of NP9.5EO by bacteria from river water in Japan, and concluded that NP1EC was the most probable ultimate biodegradation product of NPE under aerobic conditions.

Heinis *et al.* (1999) studied the persistence and distribution of NP in littoral enclosures following repeated applications in a mesotrophic pond in Minnesota. Partitioning of NP to macrophytes, sediment and the enclosure walls, which were composed of polyolefin plastic, was observed within 2 days of subsurface application to the water. Nominal aqueous concentrations ranged from 0 to 300 µg/L in this study and measurements of actual concentrations were performed. Sediment was found to be the primary sink for NP. NP levels in sediment from the 300 µg/L treatment enclosure 440 days after the initial application, was approximately 2 mg/kg. Dissipation times for 50% and 95% of the NP (DT_{50} and DT_{95}) in sediment were reported to be 66 (range 28-104) and 401 (range 354-448) days, respectively. Maximum NP concentrations were accumulated in macrophytes within 2 days of application in the 30 and 300 µg/L treatment levels (11.5 and 139 mg/kg, respectively). Mean DT_{50} and DT_{95} estimates in/on the macrophytes were 10 and 189 days, respectively.

NP dissipated rapidly from the water column (half-life <1 d) in large littoral enclosure studies, although it was more persistent in the sediments (Liber *et al.*, 1999a). The estimated DT_{50}s in sediment, based on results from the 30µg/L and 300 µg/L enclosures were 28 and 104 days, respectively. NP residues were still detectable more than one year after treatment in the 300 µg/L enclosure (Liber *et al.*, 1999a).

NP was used in aminocarb insecticide sprays between the mid-1970s and 1981 to control spruce budworm in eastern Canada. A few studies of NP distribution, dissipation and persistence after aerial sprays have been performed. Ernst *et al.* (1980) monitored NP in surface water from Britt Brook Lake, New Brunswick, which is located in a forest sprayed with an aminocarb formulation containing NP. Although a 400 m buffer zone was established between the sprayed area and the lake, NP concentrations as high as 12 µg/L were observed in the surface water 1 hour after the spray. Concentrations decreased to < 1 µg/L between 10 and 66 hours after the spray. In laboratory experiments, substantial loss of NP was observed in exposure tanks over 24-48 hour periods. These losses may have been due to volatilization, aided by aeration and/or adsorption to container walls. Holmes and Kingsbury (1980) studied NP persistence by simulating a forest spray and spraying NP onto water at a rate of 0.47 L/ha. No NP residues were observed in flowing or standing water after 3 days. Sundaram *et al.* (1980) measured NP residues in spruce foliage, forest soil, stream water and sediment after aerial application at 0.47 L/ha. Residues in white spruce foliage were the highest 1 hour after spraying (18.9 mg/kg) and decreased by 40% within two hours. Only 3% of the NP remained after 30 days and levels were below detection limits (< 0.20 mg/kg) after 62 days. NP was not detected in any forest soil sample collected after spraying and residues in stream water 1 hour after application were approximately 9 µg/L although, one sample was contaminated by a surface slick which yielded a concentration of 1100 µg/L. Concentrations declined by 50% in the next two hours. Only trace amounts (< 2 µg/L) of NP were detected after 5 days. Trace levels (< 0.1 mg/kg) of NP were detected in one sediment

sample taken 4 hours post spray. Sundaram and Szeto (1981) studied NP persistence in natural water and sediment. In water-only experiments at 16 °C, the dissipation for 50% of the NP from open containers was 2.5 d. The loss of NP was solely attributed to volatilization and the possibility of adsorption to container walls was not discussed. NP was adsorbed to sediments in test containers and its half-life in closed containers was estimated to be 16 days (Sundaram and Szeto, 1981). Microbial degradation was found to be important, given that NP concentrations in sediment were reduced to 20% after 70 days, but persisted in autoclaved sediments for more than 70 days.

Although rigorous mass balances were not attempted in test systems, studies have indicated that NP is not persistent in water or sediment. It is possible that some NP losses may be attributed to physical redistribution rather than degradation. Although volatilization losses of NP were considered to be the route of dissipation in some studies, this contradicts what would be expected based on its low Henry's Law constant (11.02 Pa m^3/mol; U.K. Environment Agency, 1997). It is possible, however, that NP sprayed on the water surface may volatilize more rapidly than NP in subsurface water. This phenomenon has been observed for some aerially-applied pesticides such as fenitrothion and deltamethrin, where volatilization from the sprayed water surface is much faster than volatilization losses from subsurface water (Maguire 1991). In general, NP enters the environment via effluents from MWWTP and industrial effluents, therefore, entry occurs mid-water column rather than to the water surface.

Approximately 75% of the nonionic surfactants including APEs contain hydrophilic poly(ethylene glycol) (PEG) groups. PEGs attached to alkylphenol moieties in APEs have been studied in surface water samples (Leenheer *et al.*,1991). Leenheer *et al.* (1991) reported PEG residues in the Mississippi River ranging from below detection levels to 145 µg/L. In a more recent study, Crescenzi *et al.* (1997) reported widespread occurrence of PEG residues in MWWTP influents and effluents in Rome, Italy. Although they were degraded fairly efficiently in MWWTPs, some PEG residues remained in effluents. PEG residues were detected at ng/L levels in sea water 16 nautical miles from the Italian coast and in five groundwater samples collected at depths of 60-208 m.

2.3.1.2.4.1 Summary

Degradation of NPEs in water and sediment follows the same pathway as biodegradation in MWWTPs. Primary biodegradation of NPEs occurs more rapidly than ultimate degradation of the transformation products (e.g., NP1EO, NP2EO, NP1EC, NP2EC and NP). Microbial acclimation is required for optimal degradation efficiencies of APEs. Photodegradation also is expected to be important for NPEs. Although the higher chain NPEs are not persistent, some degradation products have moderate persistence, especially under anaerobic conditions. The half-

life for NP resulting from biodegradation studies was estimated as approximately 150 days (U.K. Environment Agency, 1997). NP and the lower ethoxylates and carboxylates are considered to be persistent in groundwater, although this is based on a limited dataset. Heinis *et al.* (1999) observed moderate persistence of NP in sediments. It is expected that the more water-soluble (and ionized) carboxylate derivatives (NP1EC and NP2EC) will largely partition from out of the sediments into the water.

2.3.1.3 Soil

Relative to NPE/NP persistence in aquatic compartments, there are very few studies focused on the persistence of NPEs or NP in soil, under laboratory or natural conditions.

During a study of NP toxicity, Trocmé *et al.* (1988) determined the persistence of NP in a compost-sandstone mixture throughout the incubation period. NP disappeared readily upon incubation at 100 mg/kg following a 5 day lag phase at 25 °C. The half-life for NP was reported to be 15 days. In the 1000 mg/kg trial, 50% degradation was observed by day 10 of the study, although no subsequent degradation was observed through the remainder of the 40 day test. Persistence was shown to increase under aseptic conditions and volatilization was reported to be insignificant.

Marcomini *et al.* (1989a) studied the fate of NPEs and NP in sludge-amended soil and sludge-only landfills. In sludge-amended soil, the initial concentrations of NP, NP1EO and NP2EO were 4.7, 1.1 and 0.1 mg/kg dw, respectively. Concentrations fell to approximately 20% of initial concentrations within a 3 week period, followed by a slower rate of decline and during the final 130 days of the study, concentrations remained constant. NP, NP1EO and NP2EO concentrations in sludge-amended soil after 320 days were 0.5, 0.1 and 0.01 mg/kg dw, respectively. These data indicated that there was some measure of persistence of these compounds and that carry-over from year to year was possible (Marcomini et al, 1989a). Biodegradation of NP and NP1EO in two sludge-only landfills was much faster under aerobic than under anaerobic conditions, although minimal biodegradation was observed over a 15 year period (Marcomini *et al.*, 1991). Marcomini (1991) reported that the persistence of NP, NP1EO and NP2EO is greatly increased in landfills under anaerobic conditions.

Hughes *et al.* (1996) studied the biodegradation of NP9EO in soil laboratory biometer experiments and observed that 57% of the NP9EO was degraded in 64 days at room temperature. Evidence of aromatic ring destruction was observed, which indicated ultimate biodegradability. The test used for these experiments was based on a U.S. Food and Drug Administration test which required observation of 50% degradation for that compound to be considered biodegradable in soil. The results of this study indeed indicated that NP9EO was biodegradable in soil.

The Water Technology International Corporation (1998a) applied de-watered sewage biosolids from the Guelph, Ontario, MWWTP at rates of 0, 8 (the maximum allowed under Ontario legislation), 20 and 40 tonnes/ha to test plots and monitored concentrations of APEs and their degradation products. Approximate initial concentrations in the sludge were 450 mg/kg NP, 15 mg/kg OP, 10 mg/kg NP2EO, 350 mg/kg NP(3-17)EO, and ≤ 15 mg/kg APEC. NP1EO was not detected. No biodegradation products were identified after 121 days between July and October, 1997, in the 8 tonne/ha test plots.

Jones and Westmoreland (1998) reported a 96-98% decline in NPE concentrations within a 14 week period, in a compost mixture. NPEs were known to be present in the compost which contained sludge from the scouring of wool and municipal "greenwaste" (shredded municipal prunings). Initial NPE concentrations were 14 g/kg compost (dw) and the half-life for NP from this compost was approximately 35 days. NP degradation products were not detected, which suggested that degradation of NP transformation products was rapid.

NP persistence studies in soils indicated that NP can be rapidly degraded to carbon dioxide by soil microorganisms (Topp, 1999). Soil organisms have been shown to rapidly mineralize NP present at concentrations up to 250 mg/kg in cultivated agricultural soils at 4°C, temperate, non-cultivated soils, and arctic soils. No lag phase in the mineralization was observed, which indicated that the soil contained active microflora, already conditioned to mineralize other natural phenols. Rapid disappearance of initial concentrations of NP (5.5 mg /kg soil) was observed in sludge treated soil plots, in a study conducted at the Guelph Turfgrass Institute by Bennie *et al.* (1998). NP was undetectable after 90 days. NPEs are degraded to NP and as a result, a continuing source of NP will be present in the soil as NPEs are degraded. This step-wise degradation should, therefore, result in a nonlinear disappearance of NP after sludge application (Figure 14). Bokern *et al.* (1998) concluded that the NP uptake from soil was slow and that it was quickly mineralized by soil microorganisms. Bokern *et al.* (1998) reported that NP accumulation in several species of plants was minimal and metabolism of NP to hydroxylated and conjugated derivatives was observed. Terrestrial plants appeared to be relatively insensitive to the effects of NP and NPEs (Bokern *et al.*, 1998).

Based on the limited data available, NP appears to be persistent in landfills under anaerobic conditions, but not under aerobic conditions. The U.K. Environment Agency (1997) estimated a half-life for primary biodegradation in soil of about 30 days and for ultimate mineralization, the half-life was estimated to be 300 days. Mass balance studies which factor in biodegradation, photodegradation, adsorption to suspended solids and bed sediment under both aerobic and anaerobic conditions using different temperature conditions would contribute greatly to the understanding of NPEs in the environment. Because NPEs are used in aerially-applied

pesticide formulations, there is a need to determine their atmospheric chemistry, photochemistry and fate.

2.3.2 *Concentrations in the Canadian environment*

NP and NPE concentrations have been reported in Canada in a number of studies. Additionally unpublished environmental concentration data in effluents, sludges, surface waters and aquatic sediments are reported in Appendix A and summarized in Table 6 and 7. NP and NPE concentrations in Canadian sediments, effluents and sludges are similar to levels observed in other countries. NP and NPEs are released into the environment primarily through industrial (e.g., textile, pulp mill) and municipal effluents as well as agricultural practices such as municipal sludge additions. Pesticide formulations containing NPEs also represent a source to the environment.

2.3.2.1 Air

Although NPEs and their degradation products have been observed in air samples elsewhere, there have been no reports of NP, NPEs or other related compounds in Canadian air. Based on the physical/chemical properties of NP and NPEs, they are not expected to readily volatilize into air. The Henry's Law Constant for NP has been reported to be 11.0 Pa m^3/mol. Based on the fact that NPEs are far less volatile than NP, it is expected that they would not partition to the atmosphere and these compounds are expected to degrade rapidly in the atmosphere.

2.3.2.2 Water and effluents

In general, NPEs are found at high concentrations (maximum concentration 8810 µg/L) in untreated or partially treated industrial (e.g., textile mills) and municipal effluents in Canada. Untreated effluents typically have elevated NP3–17EO concentrations and relatively high levels of NP and NP1,2EO. Treatment significantly reduces the concentration of NP3–17EO in final effluents (Figures 10 and 11). The levels of NP3–17EO, NP1,2EO and NP in final effluents can, therefore, vary dramatically, depending on the type and degree of treatment. Well-treated effluents typically have very low levels of NP3–17EO. As higher-chain-length NPEs move through the treatment system, they are degraded to lower-chain-length NPEs and NPECs and ultimately to NP, which itself can be further degraded or sorbed to particles or sludges. Although NP1EO and NP2EO are created during treatment, concentrations of these

transformation products are generally reduced in well-treated effluents. In contrast, NP1EC and NP2EC can increase in concentration with increased degree of treatment (Figures 10 and 11). The nature of the inputs and type and degree of treatment strongly influence the concentrations and relative proportions of NPEs released in final effluents. The relative distribution and concentrations of NPEs in influent, final effluent and sludges are, therefore, very different (Figure 11).

OP has only a slightly lower octanol-water partition coefficient than NP, therefore, it is expected to behave very similarly to NP in the environment. Daily and seasonal variability of APs and APEs releases was examined by Water Technology International Corporation (1998b) at a site in Southern Ontario. Only minor variations in 24-h composite sample concentrations were observed over a two week period (n=10) for all of the APs and APEs under investigation (Figure 12). Seasonal variability at this site also was minimal, although there did appear to be an increase in concentrations during mid-winter (Figure 13).

Nonylphenol

NP concentrations in Canadian freshwater ranged from non-detectable (<0.02 µg/L) to 4.25 µg/L (Table 7) (mean 0.20 µg/L; median <0.02 µg/L) based on sampling at 42 sites (n=126) (Bennie 1998; Bennie *et al.* 1997a). The highest freshwater concentrations of NP were observed in areas in close proximity to MWWTP discharges, pulp mill discharges, large population centres or regions of heavy industry (Appendix A). The different types of sites sampled included rivers, lakes (primarily Great Lakes) and harbours. In rivers across Canada, NP concentrations ranged from non-detectable (<0.02 µg/L) to 4.25 µg/L, although Carey *et al.* (1981) reported values up to 2600 µg/L in Canagagigue Creek in Elmira, Ontario. These latter values were not considered representative because they were associated with an industrial spill into this small creek. NP concentrations were below detection limits at reference locations upstream from the point source. NP concentrations in lakes and harbours ranged from <0.02 to 0.06 µg/L and <0.02 to 0.98 µg/L, respectively.

MWWTPs equipped with primary, secondary and tertiary treatment systems have been sampled across Canada (Bennie, 1998; Lee *et al.*, 1998; Water Technology International Corporation, 1998b). Final effluents contained concentrations of NP that ranged from <0.02 to 62.1 µg/L (mean of 8.49 µg/L and median of 1.83 µg/L, n=21), from 0.12 to 4.79 µg/L (mean of 1.14 µg/L and median of 0.18 µg/L, n=54 at 21 sites) and from <0.02 to 3.20 µg/L (mean of 0.87 µg/L and median of 0.75 µg/L, n=37) for primary, secondary and tertiary treatment systems, respectively (Table 7) (Lee *et al.*, 1998; Bennie, 1998; Wastewater Technology International Corporation 1998b). MWWTPs that use a lagoon system had NP effluent concentrations ranging from 0.75 to 2.15 µg/L (mean of 1.03 µg/L and median of 0.75 µg/L)

(Bennie, 1998; Water Technology International Corporation, 1998b).

NP concentrations entering MWWTP influents are substantially higher than observed in final effluents. Bennie *et al.* (1998) measured 4-NP concentrations in raw sewage from nine communities in eastern Canada (0.69–156 µg/L). The highest concentrations (>100 µg/L) were associated with two cities where textile mill inputs to the municipal treatment system were significant. The mean concentration was 43.1 µg/L (n = 9) and the median concentration was 12 µg/L. Primary effluent concentrations of 4-NP entering five MWWTPs in the Toronto area ranged from 2.8 to 30 µg/L (median and mean values were 4.4 µg/L and 10.8 µg/L, respectively, n = 5). All raw and primary effluent samples contained detectable levels of 4-NP.

NP concentrations were determined in a study of APE degradation at two MWWTPs in 1997 (Water Technology International Corporation, 1998b). MWWTP A was a state-of-the-art tertiary facility utilizing conventional activated sludge secondary treatment with nitrification and was comprehensively monitored for two weeks while MWWTP B, a conventional, non-nitrifying, activated sludge secondary treatment plant, was monitored for two days. MWWTP A received a significant loading of surfactants from textile processing plants. Raw influent concentrations of 4-NP at MWWTP A ranged from 13 to 21 µg/L and discharged effluent with 4-NP concentrations ranging from 0.99 to 1.85 µg/L. The raw influent concentration of 4-NP at MWWTP B during the two day sampling period was 6.0 µg/L. 4-NP concentrations in final effluent from MWWTP B were 1.6 µg/L and the digested sludge contained 290 µg/g (dw) 4-NP.

NP also has been detected in a variety of industrial effluents in Canada. NP concentrations in pulp mill effluent samples taken between 1990–1993 were quite variable (Bennie, 1998). Due to recent changes to reduce the use of NPEs in Canadian pulp and paper mill processes, final effluent concentrations from pulp and paper mills were divided into those values obtained prior to 1998 and those obtained more recently (Bennie, 1998a; Lee and Peart, 1999) for this assessment. Of the fourteen pulp mills sampled between 1990 and 1993, 4-NP was detected in four mill effluents and concentrations ranged from < 0.02 to 26.2 µg/L (mean of 3.71 µg/L and median of 0.02 µg/L, n=33 at 14 sites) (Bennie, 1998). One mill, where consistently high NP concentrations were observed, had a combined effluent treatment system which treated effluent from the pulp mill, in addition to sewage from the entire surrounding community. Consequently, it was difficult to ascertain the NP contribution from each individual waste stream. Concentrations of NP in pulp and paper mill effluents after 1998 ranged from 0.10 to 4.30 µg/L (mean of 0.56 µg/L and median of 0.10 µg/L, n=19 at 19 sites) (Lee and Peart, 1999).

Three categories of textile mill effluents were studied including: untreated, those with on-site treatment and those which flow into a MWWTP. Concentrations of NP in untreated

textile mill effluents ranged from 2.68-13.3 µg/L (mean of 8.21µg/L and median of 10.4 µg/L, n=5 at 2 sites). NP concentrations in on-site treated textile mill effluents ranged from 0.09 to 3.56 µg/L (mean of 1.23 µg/L and median of 0.64 µg/L, n=4 at 2 sites) and in textile mill effluents which discharge through a municipal treatment facility, NP levels ranged from 0.23 to 25.6 µg/L (mean of 4.83 µg/L and median of 2.48 µg/L, n=14 at 9 sites) (Bennie, 1998).

4-NP was below method detection limits (<0.02 µg/L) in two effluent samples from a major Canadian oil refinery (Bennie, 1998).

Nonylphenol Ethoxylate, Diethoxylate and Polyethoxylates

NP1EO and NP2EO are present as minor constituents of NPE surfactants (Bennie, 1999) and also may be produced by anaerobic degradation of the higher ethoxylated NPEs during wastewater treatment processes. Most of the NP1,2EO concentration data reported in the literature are based on MWWTP effluent studies.

Low levels of NP1EO and NP2EO were observed in freshwater samples collected in the Great Lakes basin and the upper St. Lawrence River (Bennie, 1998; Bennie *et al.*, 1997a). NP1EO concentrations ranged from below detection levels (< 0.02 µg/L) to 10.3 µg/L (mean of 0.60 µg/L; median 0.12 µg/L where n = 81 from 28 different sites and 25 samples with concentrations below detection). NP1EO concentrations in rivers ranged from <0.02 to 2.30 µg/L. Concentrations of NP1EO in lakes were between <0.02 and 5.07 µg/L and levels in harbours ranged from <0.02 to 10.3 µg/L. NP2EO concentrations in freshwater ranged from below the level of detection (<0.02 µg/L) to 10.4 µg/L (mean of 0.33 µg/L and median of 0.06 µg/L, n=81 at 28 sites, 32 samples had levels below detection limits). NP2EO concentrations in all the samples collected from lakes were <0.02 µg/L and levels in river samples ranged from <0.02 to 2.45 µg/L. In samples collected from Canadian harbours, NP2EO levels ranged from <0.02 to 10.4 µg/L. The higher ethoxylated NP3-17EO concentrations ranged from 0.11 to 17.6 µg/L in two Southern Ontario rivers (mean of 1.41 µg/L and a median of 0.39 µg/L, n=27 at 3 sites).

NP1EO, NP2EO and NP3-17EO concentration data in textile mill effluents are relatively limited. NP1EO concentrations in untreated effluents ranged from 37.2-257 µg/L (mean of 98.7 µg/L and median of 57.9 µg/L, n=5 at 2 sites) (Bennie, 1998). In effluents which undergo onsite treatment, NP1EO levels ranged from 1.12 to 4.10 µg/L (mean and median of 2.61 µg/L, n=2 at one site) and concentrations of 0.74 - 69.2 µg/L (mean of 26.27 µg/L and median of 16.2 µg/L, n=14 at 10 sites) were observed in effluents which flow into MWWTPs (Bennie, 1998). Slightly higher concentrations of NP2EO were determined in untreated effluents, in onsite treated effluent and in effluents destined for municipal sewage treatment,

106-592 µg/L (mean of 362 µg/L and median of 489 µg/L, n=5 at 2 sites), 0.93-3.92 µg/L (mean and median of 2.43 µg/L n=2 at one site) and 0.64-285 µg/L (mean of 113 µg/L and a median of 35.4, n=14 at 10 sites), respectively. NP3-17EO concentrations in textile mill effluents were still higher, ranging from 798 to 8810 µg/L (mean of 6044 µg/L and median of 7905 µg/L, n=5 at 2 sites) in untreated effluents, 2.07 - 315 µg/L (mean of 175 µg/L and a median of 208 µg/L, n=3 at 2 sites) in onsite treated effluents and 50.2 - 5768 µg/L (mean of 2166 µg/L and median of 1644 µg/L, n=14 at 10 sites) in effluents which required further treatment in MWWTPs.

NP1EO and NP2EO concentrations ranged from <0.02 - 3780 µg/L (mean of 206 and median of 3.94 µg/L, n=33 at 14 sites) and <0.02-67.8 (mean of 6.41 µg/L and median of <0.02 µg/L, n=33 at 14 sites), respectively, in pulp and paper mill effluent samples collected between 1991 and 1993 (Bennie,1998). Concentrations of NP3-17EO were not determined in these samples. In a more recent survey, Lee and Peart (1999) reported NP1EO concentrations ranging from <0.10 - 6.90 µg/L (mean of 2.77 µg/L and a mean of 1.30 µg/L, n=3 at 3 sites), NP2EO concentrations between 0.10 and 35.6 µg/L (mean of 13.6 µg/L and a median of 5.10 µg/L, n=3 at 3 sites) and NP3-17EO levels which ranged between 5.90 and 28.8 µg/L (mean of 14.8 µg/L and a median of 9.80 µg/L, n=3 at 3 sites) in pulp and paper mill effluents. Sithole and Allen (1989) reported NPE concentrations (310 µg/L) in unbleached whitewater from a Canadian pulp mill.

MWWTPs equipped with primary, secondary, tertiary and lagoon treatment systems have been sampled across Canada (Bennie, 1998, Bennie *et al.*, 1998, Water Technology International Corporation, 1998b, and Lee *et al.*, 1998). NP1EO concentrations in primary MWWTP effluents ranged from 0.07 to 56.1 µg/L (mean of 11.2 µg/L and a median of 2.75 µg/L, n=26 at 10 sites), in secondary effluents ranged from below detection (<0.02) to 43.4 µg/L (mean of 3.08 µg/L and a median of 1.61 µg/L, n=46 at 20 sites), in tertiary effluents from 0.30 to 26.4 µg/L (mean of 2.85 µg/L and median of 1.90 µg/L, n=37 at 7 sites) and in lagoon facilities from 0.34 to 0.90 µg/L (mean of 0.79 µg/L and median of 0.90 µg/L, n=5 at 5 sites). NP2EO concentrations were similar, with values ranging from 0.34 to 36.3 µg/L (mean of 8.67 µg/L and median of 4.25 µg/L, n=26 at 10 sites), from <0.02 to 32.6 µg/L (mean of 2.60 µg/L and median of 1.44 µg/L), from 0.25 to 12.5 µg/L (mean of 2.37 µg/L and median of 1.80 µg/L, n=46 at 20 sites) and from 0.03 to 0.90 µg/L (mean of 0.73 µg/L and median of 0.90 µg/L, n=5 at 5 sites) in primary, secondary, tertiary and lagoon effluents, respectively. In primary MWWTPs, NP3-17EO concentrations in final effluents ranged from 4.81 to 735 µg/L (mean of 173 µg/L and median of 50.0 µg/L, n=22 at 8 sites). In secondary treated effluents, NP3-17EO concentrations ranged from 1.0 to 52.8 µg/L (mean of 10.8 µg/L and median of 6.34 µg/L, n=36 at 16 sites), whereas NP3-17EO levels in tertiary and lagoon treated effluents were between 0.40 and 18 µg/L (mean of 4.58 µg/L and median of 3.70 µg/L, n=37 at 7 sites) and

1.0-2.10 µg/L (mean of 1.38 µg/L and median of 1.20 µg/L, n=4 at 4 sites), respectively.

NPE loadings were determined in a comparison between two MWWTPs, one of which used tertiary nitrifying conditions (MWWTP A) and one which employed secondary non-nitrifying conditions (MWWTP B) (Water Technology International Corporation, 1998b). At MWWTP A, mean NP1EO levels of 40 µg/L and 1.9 µg/L were observed in the influent and final effluent, respectively. Mean NP2EO levels at MWWTP A were 38 µg/L and 1.5 µg/L in the influent and final effluent, respectively. Mean concentrations of NP3-17EO also were measured at MWWTP A (1300 µg/L and 4.4 µg/L for the influent and the final effluent, respectively). MWWTP B concentrations in raw sewage were 12.3 µg/L, 10.8 µg/L and 534 µg/L for NP1EO, NP2EO and NP3-17EO, respectively. In the final effluent at MWWTP B NP1EO, NP2EO and NP3-17EO concentrations of 10 µg/L, 8.9 µg/L and 3.9 µg/L were observed. Bennie *et al.* (1998) reported that concentrations of NP1,2EO in primary treated final effluents were not significantly different than their corresponding influent. However, some advanced tertiary plants discharged more NP1EO and NP2EO in final effluent than was found in their influent, suggesting that these plants were very efficient at the biotransformation of the higher ethoxylated NPEs (Bennie *et al.*, 1998).

Nonylphenoxyacetic Acid and Nonylphenoxyethoxyacetic Acid

NP1EC and NP2EC are produced during aerobic wastewater treatment of NPEs. NP1EC concentrations measured in a Southern Ontario river, 1 to 4 km downstream of the outfall of a MWWTP, ranged from 0.44 to 3.17 µg/L (mean of 2.15 µg/L and median of 2.24 µg/L, n=37 at one site) (Bennie, 1998). NP2EC concentrations, at the same site, ranged from 0.65 to 5.9 µg/L (mean of 2.57 µg/L and a median of 2.30 µg/L, n=37 at one site).

Bennie (1998) measured NP1EC and NP2EC levels in effluents from several textile mill effluents. NP1EC and NP2EC concentrations were below the level of detection (<0.45 µg/L) (n=2) in effluent from an untreated textile mill. At two separate textile mills with onsite treatment, concentrations of NP1EC and NP2EC ranged from 0.74-5.2 µg/L (mean of 2.68 µg/L and median of 2.39, n=4) and <0.45-55.13 µg/L (mean of 16.06 µg/L and median of 4.33µg/L, n=4), respectively. NP1EC and NP2EC concentrations in textile mill effluents which feed into municipal treatment facilities ranged from <0.45-1.90 µg/L (mean of 0.70 µg/L and median of <0.45 µg/L) and <0.45-2.80 µg/L (mean of 0.83 µg/L and median of <0.45 µg/L), respectively.

NP1EC and NP2EC concentrations in effluent from 15 different pulp and paper mills were reported by Lee and Peart (1999). Concentrations ranged from 1.00-10.13 µg/L (mean of 2.43 µg/L and median of 1.00 µg/L, n=15) for NP1EC and 1.00-32.32 µg/L (mean of 4.47 µg/L

and median of 1.00 µg/L, n=15) for NP2EC.

NP1EC and NP2EC concentrations were measured in final effluents from three primary, 14 secondary, six tertiary, and four lagoon facilities (Water Technology International Corporation, 1998b; Bennie, 1998; Lee *et al.*, 1998). NP1EC concentrations in primary treated effluents, ranged from 1.17 to 11.0 µg/L (mean of 3.72 µg/L and median of 2.15 µg/L, n=7), in secondary treated effluent concentrations ranged from 2.15 to 75.0 µg/L (mean of 13.86µg/L and median of 7.75 µg/L, n=34), in tertiary treated effluents levels ranged from 2.15 to 48.58 µg/L (mean of 11.1 µg/L and median of 7.20 µg/L, n=34) and in lagoon facilities NP1EC levels were between 2.15 and 2.6 µg/L (mean of 2.26 µg/L and median of 2.15 µg/L, n=4). NP2EC concentrations were between 1.01 and 5.20 µg/L (mean of 2.66 µg/L and median of 2.15 µg/L, n=7), 2.15-45.4 µg/L (mean of 16.1 µg/L and median of 12.6 µg/L, n=34), 2.15-59.5 µg/L (mean of 27.6 µg/L and median of 23.8 µg/L, n=34) and 2.15-3.00 µg/L (mean of 2.73 µg/L and median of 2.90 µg/L, n=4) in effluents from primary, secondary, tertiary and lagoon facilities, respectively.

Lee *et al.* (1997, 1998) determined NP1EC in raw sewage, primary effluent and final effluent from eight facilities in Ontario and Québec which ranged from 0.9 to 8.3 µg/L, 2.4 to 18 µg/L, and 3.2 to 700 µg/L, respectively. Levels of NP2EC in the same plants were in the 1.7-20 µg/L, 3.5-39 µg/L and 11-565 µg/L for raw sewage, primary effluent and final effluent, respectively. In the study of two Canadian MWWTPs NP1EC and NP2EC concentrations were measured in raw sewage and final effluents (Water Technology International Corporation, 1998b). Concentrations of NP1EC in raw sewage from these plants ranged from 2.0 to 15 µg/L and were between 1.9 and 22 µg/L in final effluents. Concentrations of NP2EC in MWWTPs A and B ranged from 1.9 to 35 µg/L and from 11 to 32 µg/L in raw sewage and final effluent, respectively.

Octylphenol

Biodegradation of OPEs results in the production of the refractory metabolite 4-*tert*-octylphenol (OP). OP is also found as a contaminant in NP solutions manufactured for industrial and agricultural use. There are much fewer OP concentration data in the literature than reports of nonylphenolic compounds. The only published data for OP in Canada were reported by Lee and Peart (1995) and Bennie *et al.* (1997a, 1998). However, additional work with OP in Canadian water and effluents was performed Bennie (1998). In freshwater samples from various rivers, lakes and harbours, OP concentrations ranged from < 0.003 to 0.61 µg/L (mean of 0.023 µg/L and median of < 0.003 µg/L, n=126 at 42 sites).

OP concentrations in textile mill effluents ranged from 1.85 to 9.01 µg/L (mean of 5.41

µg/L and median of 3.85 µg/L, n=5) at two untreated effluent sites, from <0.02 to 0.22 µg/L (mean of 0.077 µg/L and median of 0.037µg/L, n=4) at two secondary treated effluent sites and <0.02 to 0.65 µg/L (mean of 0.25 µg/L and median of 0.15 µg/L, n=14) at 9 different textile mills where the effluent flows into municipal wastewater treatment facilities (Bennie, 1998).

OP levels in 13 pulp and paper mill effluent samples collected between 1991 and 1993 were below detection limits (< 0.005 µg/L) based on results from 29 samples. Lee and Peart (1999) reported OP levels in pulp and paper mills sampled after 1998 ranging from <0.005 to 0.06 µg/L in 19 samples collected at 19 sites.

Concentrations of OP in final MWWTP effluents from 9 primary treatment facilities ranged from <0.005 to 1.23 µg/L (n=21). OP levels in effluent from 21 secondary treatment facilities, 7 tertiary treatment facilities and 5 lagoon treatment facilities were <0.005-0.57 µg/L (n=5421), <0.005-0.28 µg/L (n=37) and 0.08-0.12 µg/L (n=5), respectively (Lee and Peart, 1995; Bennie *et al.*, 1997a, 1998; Bennie, 1998; Water Technology International Corporation 1998a; Lee *et al.*, 1998).

Concentrations of OP in MWWTP influents and final effluents ranged from <0.005 to 21 µg/L and <0.005 to 1.7 µg/L, respectively (Lee and Peart, 1995; Bennie *et al.*, 1997a; 1998). Elevated levels of OP were observed in MWWTP influents that received significant amounts of textile processing effluent. The highest final effluent concentrations were associated with a secondary treatment facility in Toronto that treats a highly diverse mixture of industrial, commercial and domestic effluent. In a comparison of two Canadian MWWTPs, OP was detected in the various waste streams of both plants (Water Technology International Corporation; 1998b). Influent concentrations ranged from 0.61 to 1.22 µg/L, and final effluent levels ranged from 0.016 to 0.13 µg/L.

Octylphenol ethoxylate, diethoxylate and polyethoxylates

The only Canadian data for OPEs in effluents are those reported by Rutherford *et al.* (1992), obtained in a survey of the chemical characteristics and toxicity of effluent discharges from two textile processing plants in Nova Scotia and one plant in New Brunswick. These textile mill effluents contained OPEs at concentrations ranging from 5400 to 50000 µg/L.

Octylphenoxyacetic Acid and Octylphenoxyethoxyacetic Acid

Canadian data on OPECs in the Canadian environment are very limited. Bennie (1998) determined concentrations of OP1EC and OP2EC in textile mill effluents. At one mill with untreated effluents, mean concentrations of OP1EC and OP2EC were 0.76 µg/L (n=2) and 1.90 µg/L (n=2), respectively. In two separate textile mills with onsite treatment processes,

concentrations of OP1EC and OP2EC ranged from <0.05-0.43 µg/L (mean of 0.19 µg/L and median of 0.14 µg/L, n=4) and <0.05-11.81 µg/L (mean of 2.99 µg/L and median of <0.05 µg/L, n=4), respectively.

Lee and Peart (1999), reported final effluent concentrations of OP1EC and OP2EC from 15 pulp and paper mills ranging from <0.10 to 3.95 µg/L (mean of 0.37µg/L and median of 0.10 µg/L, n=15) and 0.10-3.22 µg/L (mean of 0.34 µg/L and median of 0.10 µg/L, n=15), respectively.

Concentrations of OP1EC and OP2EC also were determined in final MWWTP effluents (Lee *et al.*, 1998; Bennie, 1998; Water Technology International Corporation 1998a). In three primary MWWTPs, effluent concentrations of OP1EC and OP2EC ranged from 0.19-0.46 µg/L (mean of 0.26 µg/L and median of 0.19 µg/L, n=7) and 0.19-0.37 µg/L (mean of 0.25 µg/L and median of 0.20µg/L, n=7), respectively. OP1EC and OP2EC levels in effluents from 14 secondary MWWTP ranged from 0.19-9.98 µg/L (mean of 3.14 µg/L and median of 1.48 µg/L, n=34) and 0.19-13.1 µg/L (mean of 2.96 µg/L and median of 0.88 µg/L, n=34), respectively. OP1EC concentrations in tertiary and lagoon treatment facilities ranged from <0.05 to 29.3 µg/L (mean of 1.66 µg/L and median of 0.46 µg/L, n=34 at 6 sites) and a mean and median concentration of 0.19 µg/L (n=4 at 4 sites), respectively. OP2EC concentrations in tertiary MWWTPs effluents ranged from 0.12 to 9.04 µg/L (mean of 2.64 µg/L and 0.66µg/L, n=34 at 6 sites). The mean OP2EC concentration in lagoon MWWTP effluents was 0.19 µg/L (n=4 at 4 sites).

Lee *et al.* (1998) reported OP1EC and OP2EC concentrations in four MWWTP influent samples ranging from 0.7 to 1.1 µg/L and from 0.14 to 1.62 µg/L, respectively. OP1EC and OP2EC were detected in all samples from both primary and final effluent from 10 MWWTPs. Final effluent concentrations in these plants ranged from 0.29 to 4.6 µg/L (mean 2.2 µg/L, median 1.2 µg/L) for OP1EC and from 0.54 to 7.7 µg/L (mean 3.1 µg/L, median 2.4 µg/L) for OP2EC. OP1EC concentrations in primary effluent ranged from 0.18 to 1.2 µg/L with a mean concentration of 0.51 µg/L and median of 0.43 µg/L while OP2EC levels ranged from 0.12 to 1.6 µg/L and the mean and median concentrations were 0.48 µg/L and 0.37 µg/L, respectively. The Water Technology International Corporation (1998b) also reported OP1EC and OP2EC levels in MWWTP waste streams from two MWWTPs. OP1EC concentrations ranged from 0.56 to 1.33 µg/L for raw sewage, and from <0.05 to 7.37 µg/L in the final effluent. Levels of OP2EC in raw sewage varied from 0.24 to 2.3 µg/L and in final effluent from 0.12 to 4.0 µg/L.

2.3.2.3 Sediments

Most of the APE concentration data in Canadian sediments are restricted to reports of NP and OP. Shang *et al.* (1999), however, recently studied NPE distributions in marine sediments from the Straight of Georgia. Generally, in commercial NPE formulations (with 0-100 EO groups), NP8-10EO would be the dominant oligomeric species, however, NP and NP1EO were the dominant nonylphenolic compounds observed in these sediments. Shang *et al.* (1999) reported that in addition to NP and NP1EO, sometimes NP2EO and NP3EO contributed significant amounts to the surface sediment. Greater than 50% of the NPE oligomers had EO chain lengths >2. In sediment cores, very little shift in the oligomer pattern from commercial formulations was observed, which suggested very little degradation in deeper sediments.

Nonylphenol

NP concentrations in sediments from the Great Lakes basin and the upper St. Lawrence River ranged from below detection limits (<0.002 µg/g dw) to 72.20 µg/g dw (Lee and Peart, 1995; Bennie *et al.*, 1997a; Bennie, 1998; Bennett and Metcalfe, 1998). Brewer *et al.* (1998) reported NP concentrations in sediment from the upper and lower reaches of the Fraser River, as well as the Thompson River sub-basin, which ranged from < 0.005 µg/g to 0.570µg/g. The highest NP concentrations in the Great Lakes dataset were associated with Hamilton Harbour samples taken near the discharge of the Burlington MWWTP. The mean concentration of NP in sediment from all sites across Canada was 4.46 µg/g with a median value of 0.21 µg/g (n=58 at 23 sites).

Nonylphenol Ethoxylate, Diethoxylate and Polyethoxylates

Bennie *et al.* (1997a), and Bennie (1998) observed NP1EO in sediments ranging from <0.01 to 38.12 µg/g dw (mean concentrations: 3.13 µg/g dw.; median <0.03 µg/g dw.; n=14 at 6 sites) (Appendix A). NP2EO also was observed in sediment at concentrations ranging between <0.01 and 6.02 µg/g dw., with a mean of 0.51 µg/g dw. and a median of <0.02 µg/g dw. (n=14 at 6 sites). NP3-17EO concentrations in sediment were measured at one site in Ontario (Bennie, 1998) . NP3-17EO ranged between <0.01 and 0.17 (mean of 0.05 µg/g dw. and a median of 0.02 µg/g dw., n=4).

Nonylphenoxyacetic Acid and Nonylphenoxyethoxyacetic Acid

No data were identified on the concentrations of these substances in sediments in Canada.

Octylphenol

Lee and Peart (1995), Bennie *et al.* (1997a, 1998) Bennie (1998), Bennett and Metcalfe

(1998) measured OP concentrations in sediments from Eastern Canadian lakes and rivers which ranged from < 0.01 to 23.7 μg/g dw. (mean of 0.62 μg/g dw. and median of <0.01 μg/g dw., n=52 at 20 sites). The highest OP concentrations observed, were present in sediment collected from industrial harbours.

Octylphenol ethoxylate, diethoxylate and polyethoxylates

No data were identified on the concentrations of these substances in sediments in Canada.

Octylphenoxyacetic Acid and Octylphenoxyethoxyacetic Acid

No data were identified on the concentrations of these substances in sediments in Canada.

2.3.2.4 Sludges

Nonylphenol

Lee and Peart (1995), Lee *et al.*, (1998), Bennie *et al.* (1998), Bennie (1998) and Water Technology International Corporation (1998a) measured 4-NP concentrations in sludge samples from MWWTPs across Canada. Levels ranged from 0.74 to 1260 μg/g dw., with a mean concentration of 299 μg/g dw.(n = 107 at 30 sites) and the median value was 217 μg/g dw. Maximum NP concentrations were associated with MWWTPs that utilize anaerobic secondary sludge digestion processes.

Nonylphenol Ethoxylate, Diethoxylate and Polyethoxylates

Concentrations of NPEs in sludge from Canadian MWWTPs were determined by Bennie (1998), Water Technology International Corporation (1998a), Lee *et al.* (1997), Lee *et al.* (1998) and Lee and Peart (1995). NP1EO and NP2EO were reported in sludge at concentrations ranging from 2.9 μg/g dw to 1830 μg/g dw and from 2.1 to 297 μg/g dw., respectively. NP2EO in sludge is usually present at much lower concentrations than NP1EO, but in some samples, concentrations of NP2EO were slightly higher. NP3-17EO concentrations ranged from 0.43 to 215 μg/g dw (mean of 49.58 μg/g dw. and median of 47.6 μg/g dw., n=90 at 28 sites).

Nonylphenoxyacetic Acid and Nonylphenoxyethoxyacetic Acid

Concentrations of NP1EC and NP2EC in sludge from Canadian MWWTPs were determined by Bennie (1998), Water Technology International Corporation (1998a), Lee *et al.* (1997), Lee *et al.* (1998), and Lee and Peart (1995). NP1EC concentrations in sludge ranged from below detection levels (<0.30 µg/g dw.) to 8.70 µg/g dw. (mean of 2.53 µg/g dw. and median of 2.26 µg/g dw., n=66 at 17 sites) while NP2EC concentrations were reported at levels ranging from <0.30 µg/g dw. to 26.0 µg/g dw. (mean of 9.27 µg/g dw. and median of 9.56 µg/g dw., n=66 at 17 sites).

Lee *et al.* (1997; 1998) reported concentrations in digested sludge from 9 MWWTPs ranging from <0.5 to 25 µg/g dw. for NP1EC and from <0.5 to 38 µg/g for NP2EC. Detectable levels of NP1EC (2.8-6.6 µg/g dw) and NP2EC (7.1-23 µg/g) were found in sludge from two Canadian MWWTPs (Water Technology International Corporation, 1998b).

Octylphenol

OP concentrations ranging from < 0.04 to 20.0 µg/g dw. (mean of 0.62 µg/g dw. and a median of <0.01 µg/g dw.) were detected in sludge samples from Canadian MWWTPs (Water Technology International Corporation, 1998b; Bennie, 1998; and Lee *et al.*, 1998). Highest concentrations were observed in sludges from MWWTPs that employ an activated sludge treatment, particularly those with significant inputs from textile processing or other industrial activities (Lee and Peart, 1995; Bennie *et al.*, 1997a, 1998).

Octylphenol ethoxylate, diethoxylate and polyethoxylates

No data were identified on the concentrations of these substances in sludges in Canada.

Octylphenoxyacetic Acid and Octylphenoxyethoxyacetic Acid

OP1EC and OP2EC levels in digested sludge from MWWTPs ranged from below detection (<0.03) to 1.2 µg/g dw. (mean of 0.14 µg/g dw. and median of 0.06 µg/g dw., n=64 at 15 sites) and <0.03 to 2.33 µg/g dw.(mean of 0.42 µg/g dw. and median of 0.96 µg/g dw., n=64 at 15 sites), respectively (Water Technology International Corporation,1998b); Bennie, 1998; Lee *et al.*,1998).

2.3.2.5 Soil

There are essentially no data available for APE concentrations in Canadian soils. A reference sample collected during a sludge addition study was found to have concentrations below detection limits (<0.03 µg/g dw.) (Water Technologies International Corporation; 1998a).

Bennie (1998) reported a concentration of NP of 2.72 mg/kg and traces of NPEs in sludge-amended soil. Following the aerial application of 0.47 L NP/ha in a pesticide formulation to 40 ha of forest, concentrations in all soil samples collected for up to 62 days were below the limit of detection (0.1 ppm) (Sundaram *et al.*, 1980)

2.3.2.6 Biota

There are no published data on NP in fish or other aquatic biota in Canada; however, 4-NP levels in a limited number of specimens have been determined in an unpublished study (Bennie, 1998). Two carp (*Cyprinus carpio*) samples from Hamilton Harbour had non-detectable levels (< 0.02 μg/g) of 4-NP, while a third carp sample contained 0.02 μg/g (whole tissue wet weight). Nine rainbow trout (*Oncorhynchus mykiss*) taken from western Lake Ontario had non-detectable levels of 4-NP (< 0.02 μg/g), but a tenth fish contained 0.043 μg/g (whole tissue wet weight). Liver and fat samples from five different beluga whales (*Delphinapterus leucas*) collected on the St. Lawrence River shore were analyzed for 4-NP. NP levels in all five liver samples were below detection (<0.02 μg/g), but three of the five fat samples contained detectable NP concentrations (0.02-0.12 μg/g wet weight) (Bennie, 1998). OP concentrations were below the detection limit (0.01 μg/g) in the carp, rainbow trout and beluga whale samples (Bennie, 1998).

2.3.3 Concentrations in the environment outside Canada

Most environmental measurements of NP/NPEs have been conducted in Europe, particularly Switzerland, where there has been much more interest in the significance of these compounds to the environment. In general, there have been few studies where NP/NPEs have been measured outside of Europe and North America. However, an early survey of three Israeli MWWTPs (Zoller and Romano, 1983) was performed and a more recent survey of two tertiary Australian MWWTPs (Mackay *et al.*, 1997) have been reported. The environmental occurrence data for NP/NPEs outside Canada are summarized in Table 8. The majority of AP/APEs detected in the environment are *para*- or 4-substituted isomers, but no differentiation between NP isomers is found in most of the literature. It is assumed in this section that the compounds in question are *para*- or 4-substituted isomers, unless otherwise noted.

2.3.3.1 Air

Dachs *et al.* (1999) detected NP (11 isomers) in all samples of ambient air from urban and coastal areas of the Lower Hudson Estuary. Concentrations of NP in air of the New York-New Jersey Bight ranged from 2.2 to 70 $ng \cdot m^{-3}$. No data on the levels of NPEs in ambient air were identified. Volatilization of NP was predicted using fugacity calculations based on concentration results obtained in the Hudson River Estuary (Dachs *et al.*,1999).

2.3.3.2 Water and effluents

Nonylphenol

Although NP has been detected in drinking water, freshwater, groundwater, landfill leachates, industrial effluents and influents to, and effluents from, MWWTPs, most observations of NP are in connection with MWWTPs and their receiving waters.

Sheldon and Hites (1979) detected NP (0.02 µg/L) in source water for a Philadelphia, PA drinking water plant, although it was not detected in the finished drinking water. Guardiola *et al.* (1991) sampled tap water weekly for a period of seven months in Barcelona, Spain and found NP concentrations which ranged from below detection limits to 0.14 µg/L. Analyses were performed using full scan GC-MS analysis. One of the river sources of this tap water was also sampled during the same period and contained levels of NP ranging from below detection to 11.7 µg/L, with a median concentration of 5.4 µg/L (Guardiola *et al.*, 1991).

Ahel *et al.* (1985a, 1994a, 1996), Naylor *et al.* (1992), Blackburn and Waldock (1995) and Stephanou (1985) studied NP levels in numerous European and North American lakes and rivers. NP and NPEs were measured in 30 U.S. rivers, which resulted in NP concentrations ranging from <0.11 to 0.64 µg/L with an average concentration of 0.12µg/L (Naylor *et al.*, 1992). Ahel and Giger (1985a) and Ahel *et al.* (1994a, 1996) studied nonylphenolic compounds in both the Glatt River, which is a heavily contaminated river with input from ten municipal MWWTPs along its course, and the Sitter River, a moderately contaminated river, in Switzerland. Over the course of the different sampling periods, NP concentrations in the Glatt River water ranged from <0.3 to 45 µg/L, although concentrations greater than 10 µg/L were observed at one site exclusively (Ahel *et al.*, 1994a). Blackburn and Waldock (1995) measured NP in six rivers, six estuaries and one harbour in England and Wales in 1993 and 1994. River concentrations of dissolved NP and total extractable NP ranged from <0.2 to 53 µg/L and from < 0.4 to 180 µg/L, respectively. The highest NP concentrations were present in samples from the River Aire, which is known to have high surfactant loadings from textile mills as well as from other industrial effluents. Estuarine and harbour concentrations of NP ranged from <0.08 µg/L to 3.1 µg/L for dissolved NP and from <0.08 to 5.2 µg/L for total extractable NP. These

values are low relative to riverine concentrations and reflect dilution and dispersion effects in these larger bodies of water.

Ahel and Giger (1985a) and Ahel *et al.* (1994a, 1996) also have measured nonylphenolics in groundwater in Switzerland. NP concentrations in groundwater from the Glattenfelden field site ranged from <0.1 to 33 µg/L in samples collected from the aquifer at distances of 2.5 m, 5 m, 7 m and 13 m from the Glatt River bed. Groundwater concentrations in samples taken from nearby Engelberg were found to contain a mean NP concentration of 0.09 µg/L.

NP was detected in groundwater (0.79 µg/L) at a site near Falmouth, MA (Barber *et al.*,1988). In this case, the groundwater had been infiltrated by secondary-treated sewage effluent over a long period of time.

Öman and Hynning (1993) detected NP in leachates from a municipal landfill site in Vasteras, Sweden at concentrations ranging from < 10 to 107 µg/L.

NP was found at high concentrations (600 µg/L) in effluent from a chemical plant in the Delaware River near Philadelphia, PA, in some of the earliest studies of the environmental occurrence of NP (Sheldon and Hites, 1978; 1979).

Influent concentrations of NP were reported to be between 0.69 and 280 µg/L in a number of studies involving MWWTPs from six countries. Final effluent concentrations in these studies ranged from below detection limits to 330 µg/L and concentrations in sludge ranged from 20 to 4000 µg/g (Table 8). NP concentrations in raw sewage, primary effluent and final effluent of Swiss MWWTPs were 14-280 µg/L, 14-150 µg/L, and N.D.-69 µg/L, respectively (Stephanou and Giger, 1982; Giger *et al.*, 1984; Ahel and Giger, 1985a; Stephanou, 1985; Ahel *et al.*, 1987; Marcomini *et al.*, 1987; Brunner *et al.*, 1988; Marcomini *et al.*, 1988a).

NP concentrations observed in final effluent from MWWTPs in the U.K. were similar to observations in Swiss plants, with final effluent concentrations ranging from below detection limits to 330 µg/L (Waldock and Thain, 1986; Sweetman, 1994; Blackburn and Waldock, 1995). Waldock and Thain (1986) studied NP in MWWTP effluents and Thames River (U.K.) water after dumping of NP-containing sludge in the Thames Estuary, concentrations ranged from <0.002 to 0.021 µg/L. Water column samples were analyzed after an "atypical" sludge material was dumped into the Estuary, NP concentrations in the water at a depth of 1 m were 15 µg/L but, decreased to <0.5 µg/L within 30 minutes.

Di Corcia *et al.* (1994) measured NP concentrations in raw sewage and final effluents from Italian MWWTPs which ranged from 2.7 to 7.5 µg/L and 0.7 to 2.6 µg/L, respectively. Final effluent concentrations of NP in three Swedish MWWTPs varied from 0.5 to 3.0 µg/L (Paxéus, 1996). NP concentrations in final effluent from a North Carolina MWWTP ranged from 0.8 to 2.5 µg/L (Kubeck and Naylor, 1990).

Nonylphenol Ethoxylate, Diethoxylate and Polyethoxylates

NP1EO and NP2EO which may be minor constituents of NPE surfactants and also can be produced by the anaerobic degradation of longer ethoxylate chain NPEs during wastewater treatment processes (Bennie, 1999). NPEs have been detected in drinking water, freshwater, sea water, groundwater and influents to, and effluents from MWWTPs although most data is related to MWWTPs and their receiving waters.

Clark *et al.* (1992) estimated NP1EO, NP2EO and NP3-7EO concentrations in a single 500 L drinking water sample from New Jersey. The NP1EO concentration was reported to be 0.077 µg/L, while NP2EO was 0.147 µg/L. The concentration of NP(3-7)EO was estimated to be 0.501 µg/L. By employing GC-MS analysis on the extract of such a large sample, it was possible to identify specific isomers of both the NP1EO and NP2EO. Because the analytes were quantified using internal standards rather than identical external standard solutions, the reported concentrations were considered estimates. Guardiola *et al.* (1991) identified NP1EO and NP2EO in tap water of Barcelona, Spain at concentrations ranging from below detection to 1.10 µg/L and below detection to 0.25 µg/L, respectively. NP3EO was present at trace levels (< 0.05 µg/L) in the tap water.

NP1EO and NP2EO have been reported in rivers and lakes in Switzerland (Ahel and Giger, 1985a; Stephanou, 1985; Naylor *et al.*, 1992; Ahel *et al.*, 1994a; Ahel *et al.*, 1996). NP1EO and NP2EO levels in Swiss rivers ranged from below detection levels to 69 µg/L and from below detection levels to 30 µg/L, respectively. Stephanou (1985) found concentrations of NP1EO in Lake Geneva ranging from 1.1 to 4.1 µg/L while NP2EO concentrations ranged from 1.3 to 5.8 µg/L. In Barcelona, Spain, NP1EO and NP2EO concentrations in river water ranged from below the level of detection to 4.80 µg/L and 1.90 µg/L, respectively (Guardiola *et al.*,1991). Trace levels (< 0.05 µg/L) of NP3EO also were found in Spanish river water.

Naylor *et al.* (1992) determined NP1EO, NP2EO and NP3-17EO concentrations in river water during their study of 30 U.S. rivers. NP1EO concentrations ranged from <0.06 to 0.60 µg/L, NP2EO concentrations ranged from <0.07 to 1.2 µg/L and NP3-17EO concentrations ranged from <1.6 to 14.9 µg/L. Non-detectable levels of NP1EO, NP2EO and NP3-17EO were observed in 17, 12 and 19 rivers, respectively.

NPE concentrations in seawater samples from the Venice lagoon in Italy were between 0.5 and 4.5 µg/L. NPE concentrations in an earlier sample from the Venice lagoon were 19.55 µg/L (Marcomini *et al.*, 1989b). Total NPE concentrations were 0.84 µg/L in the Mediterranean Sea near Barcelona (Valls *et al.*,1988).

NP1EO and NP2EO concentrations in groundwater from two groundwater infiltration sites in Switzerland during 1996 were between <0.1 and 4.9 µg/L and <0.1 and 23 µg/L, respectively (Ahel *et al.*, 1996).

NPEs frequently have been detected in MWWTP influents and effluents. In a study of three Israeli MWWTPs, Zoller and Romano (1983) reported NP10EO concentrations in final effluents ranging from 3000 to 4000 µg/L. Maximum NP1EO concentrations of 140 µg/L have been reported in raw sewage and up to 189 µg/L have been detected in final effluents from MWWTPs in Switzerland. Concentrations of NP2EO in Swiss MWWTP raw sewage and final effluent ranged between 16 and 67 µg/L and from below detection levels to 198 µg/L, respectively. NPEs have been observed in raw sewage and final effluents at concentrations ranging from 844 to 2250 µg/L and 230 to 1067 µg/L, respectively (Stephanou and Giger, 1982; Ahel and Giger, 1985a;b; Stephanou, 1985; Ahel *et al.*, 1987; Marcomini *et al.*, 1987; Marcomini and Giger, 1987; Brunner *et al.*, 1988; Marcomini *et al.*, 1988a). Valls *et al.* (1988) observed NPEs in raw sewage in Barcelona, Spain, at concentrations from 37 to 123 µ g/L.

NP1EO concentrations were between 2.6 and 3.8 µg/L and NP1-18EO (n = 1-18) concentrations of 56-102 µg/L were observed in the final effluent of MWWTPs in High Point, North Carolina, (Kubeck and Naylor,1990). NP1EO and NP1-18EO concentrations were 36-69 µg/L and 1600-2520 µg/L, respectively in the raw sewage entering the MWWTPs,.

Di Corcia *et al.* (1994) reported NPE concentrations in raw sewage between 64 and 115 µg/L, while concentrations in final effluent from an Italian MWWTP ranged from 4.7 to 9.7 µg/L. NPE removal efficiency in the MWWTP was found to be 84.6-98.3%. NP1EO and NP2EO levels in final effluents from three Swedish MWWTPS were reported to be 1-11 µg/L and 0.5-6 µg/L, respectively (Paxéus, 1996). Mackay *et al.* (1997) analyzed samples from primary, secondary and tertiary treatment facilities in Australia for various APEs and reported NPE concentrations of 86 to 450 µg/L in the influent to two tertiary treatment plants, while effluent concentrations were < 5 µg/L.

Nonylphenoxyacetic Acid and Nonylphenoxyethoxyacetic Acid

NP1EC and NP2EC have been detected in drinking water, freshwater, groundwater, industrial effluents, and influents to, and effluents from MWWTPs. As has been observed for other nonylphenolic compounds, most NPEC observations are related to MWWTPs and their receiving waters. NPECs are produced during aerobic wastewater treatment from NPEs.

Clark *et al.* (1992) estimated NP2EC concentrations to be 0.164 µg/L and NP3-7EC levels to be 0.062 µg/L in New Jersey drinking water.

NP1EC concentrations in freshwater outside Canada ranged from below the level of detection to 45 µg/L in freshwater (Table 8). Ahel *et al.* (1994a, 1996) reported NP1EC and NP2EC concentrations in the Glatt River, Switzerland ranging from <1 to 45 µg/L and 2 to 71 µg/L, respectively. Several NPEC oligomers have been observed in water samples from the Fox River in Wisconsin and other rivers in the eastern United States (Field and Reed, 1996). The Fox River was the focus of study because it is known to be heavily impacted by paper mill and MWWTP effluents. The other rivers where NPEC concentrations were determined were part of an investigation of 30 rivers reported by Naylor *et al.* (1992). Levels of NP1EC and NP2EC in these rivers ranged from below the level of detection to 2.0 µg/L and 11.8 µg/L, respectively. NP3EC and NP4EC concentrations were below limits of detection in all samples.

At their Glattfelden field site in Switzerland, Ahel *et al.* (1996) measured NP1EC and NP2EC concentrations in groundwater from an aquifer supplied by the Glatt River. NP1EC and NP2EC levels ranged from <0.1 to 13 µg/L and from <0.1 to 23 µg/L, respectively. In general, the concentrations of these two oligomers decreased as the distance of the aquifer from the river bed increased.

Field and Reed (1996) reported NP1EC, NP2EC, NP3EC and NP4EC concentrations of 0.2 µg/L to 140 µg/L, 0.4 µg/L to 931 µg/L, 2.0 µg/L to 172 µg/L and 2.0 µg/L to 26.7 µg/L in U.S. paper mill effluents.

Ahel *et al.* (1987) measured NP1EC and NP2EC concentrations in various wastewater streams of six Swiss MWWTPs. In the raw sewage, NP1EC was not detected, while NP2EC concentrations ranged from below detection levels to 14 µg/L. Final effluent concentrations ranged from 71 to 233 µg/L for NP1EC and from 6 to 17 µg/L for NP2EC. During a study of 11 Swiss MWWTPs, Ahel *et al.* (1994b) reported that 46% of the nonylphenolic compounds in the secondary treated effluent were NP1EC and NP2EC. NPEC concentrations in the secondary effluent were substantially higher than those in primary effluents, leading to the conclusion that aerobic biological treatment causes significant formation of NP1EC and NP2EC. Di Corcia *et al.* (1994) measured NP1EC and NP2EC levels in raw sewage and final effluents from an Italian MWWTP. NP1EC levels were reported to be below detection levels in the raw sewage and between 1.5 and 3.9 µg/L in the final effluent. NP2EC concentrations also

were below detection in raw sewage and ranged from 5.1 to 9.4 µg/L in the final effluent. NP1EC, NP2EC, NP3EC and NP4EC concentrations in U.S. MWWTP effluents were found to be 7.6-29.4 µg/L, 64-144 µg/L, 25-105 µg/L, and 9.7-29 µg/L, respectively (Field and Reed, 1996).

There are limited data on dicarboxylic acid derivatives of NPEs, carboxylated on both the ethoxy and alkyl side chain (CAPECs). Ding *et al.* (1996), however, reported 10 CAPECs in tertiary MWWTP effluents in California, at concentrations between 1 and 11 µg/L using granular activated carbon filtration. Five brominated CAPEC residues which resulted from the chlorination process, were detected at concentrations of 0.8 to 2.0 µg/L (Ding *et al.*, 1996). More recently, Ding and Tzing (1998) reported dicarboxylic acid derivatives of NPEs, carboxylated on both the ethoxy and alkyl side chain, in river water and sewage effluent samples in Taiwan, at concentrations ranging from 67 to 138 µg/L and 15 to 67 µg/L, respectively. Di Corcia *et al.* (1998) reported CAPECs, as well as lower ethoxylates with carboxyl groups on C_8 and C_6 side chains, not on the ethoxylate moiety (CAPEs; specifically: $CA_8$1EO, $CA_8$2EO, $CA_6$1EO and $CA_6$2EO) in effluent from the Cobis MWWTP in Rome, Italy in a 24 hour composite sample collected in November 1997. CAPECs represented most of the APE degradation products identified in the study. The total concentration of APE degradation products was 92 µg/L, which consisted of 12 µg/L NP2EO (13%), 21 µg/L NP2EC (23%), 58 µg/L CAPECs (63%), and 0.68 µg/L CAPEs (0.7%) (Di Corcia *et al.*, 1998). The three most abundant CAPECs were tentatively identified as CA_8NP2EC, CA_8NP1EC and CA_6NP2EC. Because the HPLC method used in the analyses had low resolution efficiency, the identified peaks likely contained several isomers and the positions of the carboxylate groups on the alkyl side chains were not determined in this study (Di Corcia *et al.*,1998).

Octylphenol

OP is found as a contaminant in NP solutions manufactured for industrial and agricultural use. OP is more estrogenic to fish than NP but, it is usually found at levels of about one order of magnitude lower in the environment. The frequency of reporting of this analyte in the literature is substantially lower than for nonylphenolic compounds. OP, however, has been detected in drinking water, freshwater, groundwater, industrial effluents and influents to, and effluents from MWWTPs.

Sheldon and Hites (1978; 1979) detected OP in drinking water in Philadelphia, PA. at a concentration of 0.01 µg/L while the raw water entering the plant had an OP concentration of 0.4 µg/L.

Sheldon and Hites (1978; 1979) reported OP in freshwater from the Delaware River, in the U.S. at concentrations in the ranging from below the level of detection to 3 µg/L.

OP in groundwater in Falmouth, MA was reported to be 0.36 µg/L (Barber *et al.*, 1988).

Sheldon and Hites (1978; 1979) observed OP in effluent from a chemical plant in the Philadelphia, PA. area at a concentration of approximately 5000 µg/L.

OP was observed in raw sewage and final effluent of a Philadelphia, PA. area MWWTP at concentrations of 400 µg/L and 200 µg/L, respectively (Sheldon and Hites, 1978; 1979). Paxéus (1996) analyzed effluents from the three largest MWWTPs in Sweden and found detectable OP levels in the effluent of just one of these plants at an estimated concentration of 1 µg/L.

Octylphenol ethoxylate, diethoxylate and polyethoxylates

No data were identified on the concentrations of these substances in natural waters outside Canada, however, some data exist on their concentrations in drinking water and in influents to, and effluents from MWWTPs.

Clark *et al.* (1992) reported OP2EO and higher EO chain OPEs at concentrations of 0.002 µg/L and 0.124 µg/L, respectively in New Jersey drinking water.

OP1EO and OP2EO levels ranged from <0.5 to 0.9 µg/L and from 1.5 to 6.0 µg/L in dechlorinated final effluents of a California MWWTP, respectively (Ball and Reinhard, 1985). In a study of the three largest Swedish MWWTPs, Paxéus (1996) detected OP1EO (1.5 µg/L) and OP2EO (0.5 µg/L) in the final effluent from only one plant. Mackay *et al.* (1997) measured OPE concentrations in raw sewage (< 5 to 23 µg/L) and final effluent (<5 µg/L) in two tertiary Australian MWWTPs.

Octylphenoxyacetic Acid and Octylphenoxyethoxyacetic Acid

No data were identified on the concentrations of these substances in natural waters outside Canada, but some concentrations were reported in drinking water, and in effluents from MWWTPs. Chemical standards for these substances are not yet commercially available.

Using particle beam LC-MS, Clark *et al.* (1992) estimated concentrations of 0.04 µg/L for OP2EC and 0.012 µg/L for OP(3-4)EC in a drinking water sample from New Jersey.

Ball and Reinhard (1985) reported OP1EC and OP2EC in samples of dechlorinated final effluent from a Palo Alto wastewater treatment plant at concentrations ranging from 4.9 to 11 µg/L and 24 to 84 µg/L, respectively.

2.3.3.3 Sediments, soils, sludges and biota

Nonylphenol

There are numerous data for NP in sediments, soils, sludges and biota from locations outside of Canada (Table 8).

Ahel and Giger (1985a) and Ahel *et al.* (1994a, 1996) studied NP in sediment from both the Glatt River which is heavily contaminated with input from 10 MWWTPs along its course, and the Sitter River which is a moderately contaminated river in Switzerland. Sediment concentrations of NP in the Glatt River ranged from 0.5 to 13.1 µg/g dw. Marcomini and Giger (1987) reported NP concentrations in Swiss river sediment to be 0.9 µg/g dw. Naylor *et al.* (1992) measured NP and NPEs in sediments from 30 rivers in the United States. NP concentrations ranged from <0.003 to 2.96 µg/g dw and the average concentration was 162 µg/g dw. The highest NP concentration was observed in sediment from the Grand Calumet River in Gary, Indiana which is a highly industrialized area. Chalaux *et al.* (1994) measured NP in Nile estuary sediments at concentrations ranging from 19 to 44 µg/g and concentrations in marine sediments from the Mediterranean Sea ranging from 6 to 69 µg/g.

Marcomini and Giger (1987) reported the NP concentration in a sludge-amended soil of 1.6 µg/g dw. The residual mean soil concentration of NP found in a sludge-amended soil 330 days after sludge application in Liebefeld, Switzerland was 0.5 µg/g (Häni, 1990).

NP in digested sludge from Swiss MWWTPs has been reported to be between 890 and 1570 µg/g dw (Stephanou and Giger, 1982; Giger *et al.*, 1984; Ahel and Giger, 1985a; Stephanou, 1985; Ahel *et al.*, 1987; Marcomini *et al.*, 1987; Brunner *et al.*, 1988; Marcomini *et al.*, 1988a). Marcomini and Giger (1987) reported that NP was present in digested sewage sludge at a concentration of 1200 µg/g dw. NP levels in U.K. MWWTP sludges were comparable to levels observed in Swiss plants. Digested sludge concentrations ranged from 30 to 4000 µg/g dw in U.K. studies. (Waldock and Thain, 1986; Sweetman, 1994; Blackburn and Waldock, 1995). In Germany, NP concentrations in sludge from MWWTPs were determined in two studies (Jobst, 1995; Kujawa *et al.*, 1997) and concentrations ranged from 22 to 1200 µg/g dw. Sludge from a MWWTP in Barcelona, Spain contained 20 to 350 µg/g of NP compared to 370 µg/g in a MWWTP sludge from Los Angeles, CA (Chalaux *et al.*, 1994).

Ahel *et al.* (1993) measured NP in aquatic plants, freshwater fish and ducks in receiving waters downstream from a Swiss MWWTP. NP concentrations ranged from 2.5-38 μg/g dw. in the macrophytes: *Cladophera glomerata*, *Fontinalis antipyretica* and *Potamogeton crispus*. Bioconcentration factors for NP in *Cladophera glomerata* were estimated to be between 6600 and approximately 9000. NP also was determined in chub (*Leuciscus* [*Squalius*] *cephalus*), barbel (*Barbus barbus*) and rainbow trout (*Oncorhynchus mykiss*) from Chriesbach Creek. Various tissues were analyzed individually with NP concentrations ranging from < 0.03 to 1.6 μg/g dw. NP concentrations in the edible portion (*i.e.*, muscle tissue) of the fish ranged from 0.15 to 0.38 μg/g. Bioconcentration factors for muscle tissue in the fish were <100. Concentrations of NP in mallard duck (*Anas boscas*) tissue ranged from < 0.03 μg/g dw. in heart tissue to 1.20 μg/g in muscle.

Nonylphenol Ethoxylate, Diethoxylate and Polyethoxylates

NP1EO, NP2EO and higher ethoxylated NPEs have been reported in sediment, soil, sludge and biota in the literature (Table 8)

Naylor *et al.* (1992) reported NP1EO concentrations (<2.3-175 μg/g dw.) in sediment as part of the assessment of NPEs in river systems. NPEs also have been reported in sediments from the Glatt River (Ahel *et al.*, 1994a), Rhine River (Marcomini and Giger, 1987) and the lagoons of Venice and Italy (Marcomini *et al.*, 1990). Concentrations of NP1EO and NP2EO in Glatt River sediment were found to range from 0.10 to 8.85 μg/g dw., and from below detection levels to 2.72 μg/g, respectively. Concentrations of these contaminants in a sample from the Village Neuf on the Rhine River were reported as 0.80 μg/g dw. for NP1EO and 0.70 μg/g for NP2EO. Marine sediment from the lagoons of Venice were found to contain 0.2 to 6.6 μg/g dw. of NP1EO and 0.003 to 0.020 μg/g of NP2EO.

Marcomini and Giger (1987) reported that NP1EO and NP2EO concentrations in a single sludge-amended soil sample in Switzerland were 0.40 μg/g and 0.07 μg/g dw., respectively. Winther-Nielsen *et al.*(1997) reported total combined concentrations of NP plus NP1EO and NP2EO in Danish sludge to be 150-370 μg/g and 1.5-4.9 μg/g, respectively. Current regulations in Denmark allow sludge to be used as an agricultural soil amendment if the combined concentrations of nonylphenolics are less than 50 μg/g dw. Concentrations of NP1EO and NP2EO in sludge from MWWTPs in Switzerland have been reported to range between 60 and 680 μg/g dw., and from below the limit of detection to 280 μg/g dw. (Stephanou and Giger, 1982; Ahel and Giger, 1985a,b; Stephanou, 1985; Ahel *et al.*, 1987; Marcomini *et al.*, 1987; Marcomini and Giger, 1987; Brunner *et al.*, 1988; Marcomini *et al.*, 1988a).

Ahel *et al.* (1993) determined NP1EO and NP2EO in aquatic plants, freshwater fish and ducks in receiving waters downstream from a Swiss MWWTP. Concentrations ranged from 0.9 to 80 µg/g dw. for NP1EO and from 0.6 to 29 µg/g for NP2EO in the macrophytes *Cladophera glomerata, Fontinalis antipyretica* and *Potamogeton crispus*. Bioconcentration factors calculated for the plant species ranged from 3500 to 5000 and 1000 to 1800 for NP1EO and NP2EO, respectively. NP1EO and NP2EO also were measured in chub (*Leuciscus* [*Squalius*] *cephalus*), barbel (*Barbus barbus*) and rainbow trout (*Oncorhynchus mykiss*) from Chriesbach Creek. Various tissues from the three fish species were analyzed individually with NP1EO concentrations ranging from 0.06 to 7.0 µg/g dw., and NP2EO concentrations ranging from < 0.03 to 3.0 µg/g. Bioconcentration factors in the fish ranged from 3 to 300 for NP1EO and from 3 to 326 for NP2EO. Concentrations of NP1EO and NP2EO in mallard duck (*Anas boscas*) tissues ranged from <0.03 µg/g in heart tissue to 2.10 µg/g in muscle for NP1EO and from <0.03 µg/g in all tissue except muscle to 0.35 µg/g in the muscle tissue for NP2EO.

Nonylphenoxyacetic Acid and Nonylphenoxyethoxyacetic Acid

No data were identified for these substances in sediments, soils, sludges or biota outside Canada.

Octylphenol

No data were identified for OP in sediments, soils, sludges or biota outside Canada.

Octylphenol ethoxylate, diethoxylate and polyethoxylates

No data were identified for these substances in sediments, soils, sludges or biota outside Canada.

Octylphenoxyacetic Acid and Octylphenoxyethoxyacetic Acid

No data were identified for these substances in sediments, soils, sludges or biota outside Canada.

2.4 EFFECTS CHARACTERIZATION

2.4.1 Ecotoxicology

Although the data in the literature are scattered among many species, different test methods and chemicals, there is a consistent pattern in the toxicity reported for APs and APEs. NP and OP are both acutely toxic to fish (LC_{50} values 17-1400 µg/L), invertebrates (LC_{50} values 20-3000 µg/L) and algae (LC_{50} values 27-2500 µg/L) (Tables 9 and 10). Chronic toxicity values (NOEC) as low as 6 µg/L in fish and 3.9 µg/L in invertebrates have been reported. There is an increase in the toxicity of both NPEs and OPEs with decreasing EO chain length (Figure 15). NPECs and OPECs are less toxic than their corresponding APEs and have acute toxicities similar to those of APEs with 6-9 EO units. APs and APEs have been reported to cause a number of estrogenic responses in a variety of aquatic organisms. The relative estrogenic potency determined in several different *in vitro* systems in decreasing order is OP>NP>NP1EO=NP2EO>NP1EC=NP2EC>NP9EO. APEs bind to the estrogen receptor, resulting in the expression of several responses both *in vitro* and *in vivo*, including the induction of vitellogenin. The threshold for vitellogenin induction in fish is 10 µg/L for NP and 3 µg/L for OP. The estrogenic responses appear to be at least additive and should, therefore, be considered as a group. APEs also affect the growth of testes, alter normal steroid metabolism, disrupt smoltification and cause intersex (ova-testes) in fish.

Literature reports indicate that the bioaccumulation potential of APs and APEs in aquatic and terrestrial biota in the environment is low to moderate. Bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) in biota, including algae, plants, invertebrates and fish range from 0.9 to 3400. Although there are relatively fewer data available for OP and OPEs, based on their similarity in structure to NP and NPEs, bioaccumulation of OP is expected to be similar to NP (*i.e.*, low to moderate) and OPEs are not expected to biomagnify in biota.

2.4.1.1 Toxicity *via* atmospheric exposure

No data were identified on the toxicity of NP, NPEs or other APs and APEs, to aquatic organisms *via* atmospheric exposure.

2.4.1.2 Toxicity *via* aquatic exposure

Most of the data reported in the literature have been focused on the effects of NP, although there are some data on the toxicity of NPEs, NPECs, OP, OPEs and OPECs to freshwater organisms. There are, however, relatively few toxicity data for marine organisms.

Recently, several detailed reviews of AP/APE toxicity have been published by Servos, (1999a), Staples *et al.* (1998), Talmage (1994) and Lewis (1990). Data from published reports in addition to the extensive studies conducted by the Chemical Manufacturers Association (CMA) APE Panel were included in the reviews. Additionally, much of the toxicity and bioaccumulation data for APEs measured by the CMA have been summarized by Weeks *et al.* (1996) and Naylor (1995). Tables 9 and 10 provide a summary of the toxicity data in both freshwater and marine organisms, respectively. Some of the limitations of the data in the literature include, the lack of validation of dose concentrations in many studies. Additionally many of the studies were performed using static or static renewal systems, rather than flow through systems. Loss or degradation of APs/APEs during the toxicity tests may have resulted in lower compound concentrations, which would have subsequently lead to an underestimation of toxicity. A level of confidence (I-III) was applied to individual studies, based on the methodologies used and reported, the availability of supporting information, such as measured concentrations, water quality, *etc.* and the availability of the original reports. In this assessment, higher emphasis was placed on more recent studies, which used flow-through systems, where nominal concentrations were validated and the experimental procedures and conditions were well documented. Results from all studies were considered in this assessment as part of a comprehensive review of toxicity. Many data were available in preliminary reports or presentations and although they were considered to be of high quality, the data were not available to assess the validity of the results, which resulted in a lower confidence rating. Additionally, emphasis was placed on studies that measured toxicity of individual compounds rather than mixtures or commercial preparations.

Although early toxicity studies were performed using commercial products which contained numerous APEs, more recently NP, OP or individual ethoxylates, e.g., NP9EO or OP10EO have been studied. In general, it has been observed that toxicity of APEs in most aquatic organisms increases as the length of the EO chain decreases (Figure 15; Tables 9 and 10). Yoshimura (1986) reported that the 48-h LC_{50} in Japanese killifish (*Oryzias latipes*) increased from 1400 µg/L for NP (Figure 16) to 110 000 µg/L for NP16.6EO. Macek and Krzeminski (1975) tested NPEs and OPEs with EO chain lengths from 4 to 30 and observed similar responses in bluegill sunfish (*Lepomis macrochirus*). The 96-h LC_{50} in bluegill sunfish increased from 209 µg/L for NP (Brooke, 1993a) to 1300 µg/L for NP4EO to 7900 µg/L for NP9EO and >100000 µg/L for NP30EO. The 96-h LC_{50} data for OPEs in bluegill sunfish followed a similar trend; 2800 µg/L for OP5EO, 12000 for OP10EO and 531000 for OP30EO

(Macek and Krzeminski; 1975). Janicke *et al.* (1969) observed similar results for the toxic threshold in *Daphnia magna* with NPEs ranging in EO chain length from 4 to 50 units. Hall *et al.* (1989) reported a decrease in toxicity with increasing chain length for both NPEs and OPEs (1.5-50) in mysid shrimp (*Mysidopsis bahia*). The 96-h LC_{50} for NP was 43 µg/L (Weeks *et al.*, 1996), while the 48-h LC_{50} for NP1.5EO was 110 µg/L and ranged from 900 to 2000 µg/L for NP9EO (Hall *et al.*, 1989). Hall *et al.* (1989) also reported a 48-h LC_{50} for NP40EO to be >100000 µg/L. Maxwell and Piper (1968) reported EC_{50}s (emergence) in mosquito (*Culex pipiens*) for a range of NPEs from NP1EO to NP50EO which showed a dramatic decrease in toxicity (6000 to >400000 µg/L) as EO chain length increased. Toxicity of NPEs to algae also increased with decreasing chain length (Ukeles, 1965; Janicke *et al.*, 1969; Nyberg, 1988). A decrease in EO chain length results in an increased K_{ow}. This property has been used to predict toxicity with structure activity relationships (QSAR) (Schuurmann, 1990; Schuurmann, 1991; Roberts, 1991).

2.4.1.2.1 Nonylphenol

Freshwater

Although 96-h LC_{50}s for NP ranged from 128 to 1400 µg/L in 13 different species of freshwater fish, the majority were between 100 and 300 µg/L (Table 9; Figure 16). Fathead minnow LC_{50}s (96-h) for NP were reported in several validated studies ranged from 128 to 300 µg/L (Figure 17) (Holcombe *et al.*, 1984; Brooke, 1993a; Ward and Boeri, 1991b; Naylor, 1995). The 96-h LC_{50}s for NP in rainbow trout were similar 190-920 µg/L (Ernst *et al.*, 1980; Brooke, 1993b; Naylor, 1995; Dwyer *et al.*, 1995).

Weeks *et al.* (1996) reported a 96-h EC_{50} for *Ceriodaphnia dubia* for NP of 69 µg/L and a 7 d NOEC of 134 µg/L based on reproduction. *Daphnia magna* 48-h LC_{50} values for NP range from 85 to 470 µg/L (Naylor, 1995; Comber *et al.*, 1993; Brooke, 1993b; Ankley *et al.*, 1990; Table 9; Figure 17). Comber *et al.* (1993) reported a 21-d NOEC based on reproduction in *Daphnia magna* of 24 µg/L, although Brooke (1993b) reported a 21-d NOEC of 116 µg/L. The 96-h LC_{50} values for NP in the freshwater amphipod (*Hyalella azteca*) were reported to be 20 µg/L (Brooke, 1993b) and 170 µg/L (England and Bussard, 1994). In dragonflies (*Ophiogomphus* sp.) and snails (*Physella virgata*), however, LC_{50}s for NP were much higher >768 µg/L and 774 µg/L, respectively (Brooke, 1993b).

NP toxicity to algae is within the range reported for other organisms (Figure 17). Ward and Boeri (1990b) reported a 96-h EC_{50} (growth) for NP of 410 µg/L in *Selenastrum capricornutum* while the 72-h EC_{10} value in *Scenedesmus subspicatus* was found to be 500 µ

g/L (Hüls, 1996). Weinberger and Rea (1981) determined a 24-h LC_{50} for *Chlorella pyrenoidosa* was 1500 µg/L NP, although effects on growth were observed at concentrations of 25 µg/L. Moody *et al.*, (1983) reported inhibition of growth for *Chlamydomonas reinhardtii* at 750 µg/L NP, cell membrane disruption was observed at 500 µg/L (Weinberger and Rea, 1981). Prasad (1989) reported inhibition of frond production in pond weed (*Lemna minor*) within 2 days of exposure to NP at concentrations ranging from 500 - 5000 µg/L. A reduction in growth was observed at concentrations of 125 - 500 µg/L and between 500 and 2500 µg/L, effects on photosynthetic activity were reported. Reduced growth, frond production and bleaching of fronds were observed in *Salvinia molesta* exposed to NP at concentrations ranging from 2500 - 25000 µg/L (Prasad, 1989).

Freshwater mesocosms

Large littoral enclosure studies were conducted with NP and described by Liber *et al.* (1999a; b), O'Halloran *et al.* (1998) and Schmude *et al.* (1998). Each treatment was conducted in triplicate with repeated (11) dosings over 20 d, at nominal concentrations between 3 and 300 µg/L (measured concentrations of 5±4, 23±11, 76±21 and 241±41 µg/L, respectively). No effects to zooplankton were observed in the enclosures with the lowest NP exposure (5 µg/L) (O'Halloran *et al.*, 1998). Several sensitive taxa were affected at the 76 and 23 µg/L treatments. In the 241 µg/L treatment, all cladoceran and copepod taxa were significantly reduced. The abundance of cladocerans was most greatly reduced between days 7 and 28 with reduction ranging from 48% to 99%, depending on the species. In the 76 µg/L treatment, *Ceriodaphnia* and *Acroperus* abundances were reduced between 94% and 99%. The greatest reduction in copepod abundance occurred between 21 and 34 days. In the 23 µg/L treatment enclosure, a 90% and 91% reduction in abundance was observed in *Calanoida* and *Paracyclops*, respectively. Although a reduction in abundance was observed in several species (e.g., *Trichocerca*, was reduced by 73% on day 21 in the 76 µg/L treatment), many rotifer taxa were unaffected by NP, even in the 241 µg/L treatment enclosure. Effects seen on individual zooplankton abundance usually occurred within the first week of treatment and recovery usually occurred within 7 to 28 d. In the 23 µg/L and 76 µg/L treatment enclosures, two taxa (*Acroperus* and *Calanoida*) did not recover before the termination of the study (83 d). Taxonomic richness was reduced in all enclosures with treatments ≥ 23 µg/L, although there was only a slight change in the overall zooplankton community in the 23 µg/L treatment. A maximum acceptable toxicant concentration (MATC) of 8-12 µg/L was estimated for the protection of all zooplankton species, although overall community diversity was not affected in the 23 µg/L treatment (O'Halloran *et al.*, 1998).

Periphyton growth was not affected at any treatment level (O'Halloran *et al.*, 1998). Snails and clams (*Pisidium*) were the most affected macroinvertebrates in exposed enclosures, significantly reduced abundances (up to 100 %) were observed in the 241 µg/L treatment during the two year duration of the study (Schmude *et al.*, 1998). Oligochaetes and chironomid midges also were reduced in the 241 µg/L treatment but recovered within 6 weeks. Only minor effects on snails and oligochaetes were observed in the 76 µg/L treatments and no effects were seen on macroinvertebrates in the 5 or 23 µg/L treatments (Schmude *et al.*, 1998).

Reduced survival of juvenile bluegill sunfish (*Lepomis macrochirus*) added to the highest treatment (241 µg/L) enclosure was observed. A similar reduction in survival was observed in only one of the three replicates at the next lower treatment level (mean 76±21 µg/L) (Liber *et al.*, 1999b). No bluegill growth was observed in either the control or treatment enclosures throughout the study. Bluegills accumulated NP and a non-equilibrium BAF of 87± 124 was determined in this study. Concentrations of NP in fish were reduced by 61% within 6 d and were below the level of detection 28-34 d after the last treatment (23 µg/L treatment) (Liber *et al.*, 1999b). There was some evidence that the major endemic fish species in the enclosures (ninespine stickleback (*Pungitus pungitus*)), also were affected in the 241 µg/L enclosure, although it was not found to be statistically significant. In two of the three 241 µg/L enclosures, stickleback could not be trapped and mean capture success was 65% lower than in control enclosures (Liber *et al.*, 1999b).

Sea Water

The 96-h LC_{50} values in marine species were generally within the range reported for freshwater fish species. Ward and Boeri (1990d) reported a 96-h LC_{50} for NP in sheepshead minnow (*Cyprinodon variegatus*) of 310 µg/L. The 96-h LC_{50} values for winter flounder, (*Pleuronectes americanus*) and cod (*Gadus morrhua*) were (17 µg/L) (Lussier *et al.*, 1996) and 370 µg/L (Swedmark, 1968), respectively (Table 10).

A 96-h LC_{50} of 43 µg/L was reported for the marine mysid (*Mysidopsis bahia*) (Ward and Boeri, 1991c). Twenty-eight day NOEC values of 3.9 µg/L and 6.7 µg/L were reported for growth and reproduction, respectively in the marine mysid, *M. bahia* (Ward and Boeri, 1991c; Weeks *et al.*, 1996). McLeese *et al.* (1981) reported 96-h LC_{50}s for NP ranging from 170 µg/L in lobster (*Homarus americanus*) to 300 µg/L in shrimp (*Crangon septemspinosa*). A 144-h LC_{50} of >700µg/L for soft shell clam (*Mya arenaria*) was reported by McLeese *et al.* (1980b). Waldock and Thain (1991) reported a 96-h LC_{50} in brown shrimp *(C. crangon)* of 420 µg/L. The 96-h LC_{50}s for grass shrimp (*Paleomonetes pugio*), mud crabs (*Dyspanopeus sayi*) and coot clams (*Mulinia lateralis*) were reported to be 59 µg/L, >195 µg/L and 38 µg/L,

respectively (Lussier *et al.*,1996), and for marine algae (*Skeletonema costatum*), the LC_{50} was determined to be 27 µg/L (Ward and Boeri, 1990a).

Sediment-Water

In sediment-water exposure systems, 14-d LC_{50} values were determined for the midge (*Chironomus tentans*) based on interstitial water concentrations (75 µg/L), dosed water concentrations (119 µg/L) and dosed sediment (>34 mg/kg) (England and Bussard, 1993; Table 11). Additionally, England and Bussard (1993) reported a 14-d NOEC based on growth and survival of 20 mg/kg (dosed sediment) for the midge (*C. tentans*). A NOEC of 42 µg/L and LOEC of 91 µg/L were established for the midge during life-cycle tests, which evaluated survival, growth, emergence and fecundity (Kahl *et al.*,1997). Tadpoles (*Rana catesbiana*) had a 30-d LC_{50} of 260 mg/kg and NOEC of 155 mg/kg in sediment (Ward and Boeri, 1992; Weeks *et al.*, 1996). The LC_{50} was similar based on exposure periods of 10, 20 and 30 days.

2.4.1.2.2 Nonylphenol Ethoxylate, Diethoxylate and Polyethoxylates

LC_{50}s and EC_{50}s for NP9EO are much higher than those reported for NP in fish, invertebrates and algae (Figures 16, 17 and 18). In fathead minnows (*P. promelas*), a 96-h LC_{50} value of 4600 µg/L (Naylor, 1995; Dorn *et al.*, 1993) was reported for NP9EO. Much higher 96-h LC_{50} values for NP8EO, NP9.5EO and NP10EO were reported in rainbow trout (4700 µg/L, 7500-12500 µg/L and 2500-6200 µg/L, respectively) than observed for NP (Calamari and Marchetti, 1973; Unilever Research Laboratories, 1977; Marchetti, 1965).

NPE toxicity to invertebrates decreases with increasing EO chain length, similar to observations in fish. Dorn *et al.* (1993) reported the 48-h EC_{50} for NP9EO in *D. magna* of 14000 µg/L. The 48-h LC_{50} values for the marine amphipod (*M. bahia*) were 900-2000 µg/L for NP9EO (Patoczka and Pulliam, 1990; Hall *et al.*, 1989), 2570 µg/L for NP15EO, >100000 µg/L for NP40EO and >411000 µg/L NP50EO (Hall *et al.*, 1989). The 96-h LC_{50}s for NP10EO were determined in a number of crustaceans and clams (Swedmark *et al.*, 1971; 1976) and were generally >10000 µg/L. NP12EO toxicity was low relative to NP in shrimp, crabs and molluscs (19300->100000 µg/L) (Portmann and Wilson, 1971; Waldock and Thain, 1991). Eggs and larvae of the mussel (*Mytilus edulis*) were more sensitive to NP10EO than adults (Swedmark *et al.*, 1971). Swedmark *et al.* (1971) also reported that the larvae of the spider crab (*Hanas araneus*) and the barnacle *(Balanus balanoides*) were more sensitive than the adults to NP10EO. Collyard *et al.* (1994) also demonstrated that a 2-3 fold decrease in toxicity of *Hyalella azteca* exposed to NPEs occurred with increased age of the organism.

The toxicity of NPEs to algae (*S. capricornutum*) is much lower than NP. The reported 96-h EC_{50} of NP9EO to *S. capricornutum* ranged from 12000 to 50000 µg/L (Dorn *et al.*, 1993; Lewis, 1986). Twelve species of marine algae were tested using branched NPEs (Igepal) and either complete or some growth inhibition was observed at high concentrations (>100000 µg/L) (Ukeles, 1965).

2.4.1.2.3 Nonylphenoxyacetic Acid and Nonylphenoxyethoxyacetic Acid

Very few studies of NPEC toxicity have been performed. Yoshimura (1986) reported 48-h LC_{50}s in Japanese killifish for NP1EC and NP2EC of 9600 (Figure 18) and 8900 µg/L respectively. These LC_{50} values are slightly lower than reported for NP8.4EO/NP8.9EO (11200-14000 µg/L), but much higher than for NP (1400 µg/L). Williams *et al.* (1996) reported similar results in fathead minnows. The 96-h LC_{50} values determined for NP1EC (2000 µg/L) and OP1EC (5000 µg/L) were lower than both NP9EO (6600 µg/L) and OP10EO (8900 µg/L) (Williams *et al.*, 1996).

A similar trend was observed in recent studies with *D. magna, M. bahia* and *C. dubia* for both NPEC and OPEC (Naylor *et al.*, 1997) (Figure 18). Maki *et al.* (1998) determined the 48-h LC_{50}s for NP2EO in *Daphnia magna* to be 148 (115-198) µg/L compared to 990 (770-1295) µg/L for NP2EC. These data suggest that the NPECs are much less toxic than the corresponding NPEs.

2.4.1.2.4 Octylphenol

OP toxicity to rainbow trout (96-h LC_{50}: 450 µg/L) is similar to NP (Analytical Bio Chemistry, 1984b). The 14-d LC_{50} for rainbow trout was reported to be 84 µg/L and the NOEC was 120µg/L for OP. The 96-h LC_{50} for OP in fathead minnows was reported to be 290 µg/L, (Figure 18; Analytical Bio Chemistry, 1984a). Chronic early life stages with rainbow trout resulted in a 90 d NOEC of 6.1 µg/L for OP (McAllister *et al.*, 1986).

The acute toxicity for OP (48-h LC_{50}: 260 µg/L) was similar to NP in *D. magna* (Analytical Bio Chemistry, 1984c). Forbis (1988) reported a chronic 21-d EC_{50} (survival) of 340 µg/L and a 21-d NOEC (reproduction and growth) of 37 µg/L for OP. The 96-h LC_{50} value reported was 55-113 µg/L in the marine amphipod, *M. bahia*, which is similar to results reported for NP (Cripe *et al.*, 1989). The 96-h EC_{50} for OP in the algae (*S. capricornutum*) was reported to be 1900 µg/L (Forbis *et al.*, 1984).

2.4.1.2.5 Octylphenol Ethoxylate, Diethoxylate and Polyethoxylates

The 96-h LC_{50} for OP10EO in fathead minnow (8900 µg/L) was much higher than for OP (290 µg/L) (Williams *et al.*, 1996; Naylor *et al.*, 1997; Analytical Bio Chemistry, 1984a), similar to the pattern observed for NP and higher chain NPEs. OPEs were considerably less toxic in *Mysidopsis bahia* than OP with OP1.5EO and OP5EO having 48-h LC_{50}s of 7070 µg/L and 1830 µg/L, respectively (Hall *et al.*, 1989). Nyberg (1988) also observed low toxicity for both NP9EO and OP9.5EO in 21-d growth studies with *S. capricornutum* (>300000 µg/L).

2.4.1.2.6 Octylphenoxyacetic Acid and Octylphenoxyethoxyacetic Acid

The acute toxicity of OP1EC is similar to that of NP1EC in fathead minnows, based on the 96-h LC_{50} results, 5000 µg/L and 2000 µg/L, respectively (Williams *et al.*, 1996). This pattern also was observed in *D. magna, M. bahia* and *C. dubia* (Figure 18). Naylor *et al.* (1997) reported acute 96-h LC_{50} toxicity values of <48000 µg/L and 10000 µg/L for *C. dubia* and *M. bahia*, respectively and 48-h LC_{50} of 20500µg/L for *D. magna*.

2.4.1.3 Toxicity to terrestrial plants and animals

Limited data are available on the toxicity of NP to plants and no data in the published literature exists for other APs and APEs. The NP concentration causing 50% growth reduction in cell suspension cultures of 14 species ranged from 0.05 to more than 1.00 mM (220 mg/L) (Bokern and Harms, 1997). NP also was reported to be toxic to plant roots. *Lupinus hartwegii* showed a 50% growth reduction at 0.1 mM (Bokern *et al.*, 1998). The growth *of L. polphyllus* root cultures also was inhibited, although it did not result in 50% growth reduction at 1 mM NP. NP uptake from soil was slow and was quickly mineralized by soil micro-organisms. NP was accumulated in several species of plants and was metabolized to hydroxylated and conjugated derivatives.

A few studies also have shown effects of NP and NPEs on bacteria. The 30-min EC_{10} for oxygen consumption by the bacterium *Pseudomonas putida* was >10000 µg/L for NP (Knie *et al.*, 1983). Similar to results from other organisms, soil microorganisms show an increase in APE toxicity with decreasing EO chain length (Cserhati *et al.*, 1991). Dorn *et al.* (1993) reported an EC_{50} for NP to *Photobacterium phosphoreum* (Microtox) of 60600 µg/L. *P. phosphoreum* toxicity (EC_{50}) decreased with increasing EO chain length for both NPEs and OPEs (Ribosa *et al.*, 1993). Cserhati *et al.* (1991) reported that NPEs inhibited growth of several species of soil bacteria in agar cultures at high concentrations, but at low concentrations

NPEs stimulated growth of some bacteria. Bacteria appear to be less responsive than other biota to APs and APEs.

There are very little data available in the literature on the toxicity of NP and NPEs to soil-dwelling animals. The earthworm (*Apporectodea calignosa)* tested by Krogh *et al.* (1996) had a 21-d EC_{10} (reproduction) of 3.4 µg/g in soil. In the same study, an EC_{10} (survival of adults) of > 40 mg/kg and an EC_{50} (growth) of 23.9 mg/kg also was determined. There are no toxicity data available for soil-dwelling organisms for the other NPE metabolites.

2.4.1.4 Effects of Alkylphenols and Alkylphenol Polyethoxylates on Endocrine Function

One of the functions of endogenous estrogens in fish is to stimulate the liver to produce vitellogenin, a large phospholipoprotein (Chen, 1983). It is released into the blood stream and sequestered by developing oocytes for production of egg yolk (Tyler, 1991; Tyler *et al.*, 1988a;b; Wallace, 1985). In maturing female fish, vitellogenin is a major constituent of the blood proteins, while in male fish it is not normally present in appreciable amounts. If male fish are exposed to estrogens, however, vitellogenin can be produced at similar levels to that found in maturing females. Although the implications of the induction of vitellogenin on fish reproductive functions are not fully understood, it has been used as a very sensitive indicator of exposure of fish to exogenous estrogens.

Environmental contaminants can affect the reproduction and development of fish and wildlife through a wide variety of mechanisms. Recently, a number of chemicals have been identified that can bind to the estrogen receptor and regulate the activity of estrogen responsive genes. Estrogens play numerous roles in the normal physiology and development of fish including sexual differentiation and maturation (Hunter and Donaldson, 1983; Piferrer and Donaldson, 1989; Bye and Lincoln, 1986). Estrogens are involved in metabolism, including lipid deposition (Haux and Norberg, 1985; Washburn *et al.*, 1993) and oocyte growth and development (Tyler *et al.*, 1988a;b; Hyllner *et al.*, 1991; Specker and Sullivan, 1993). Concern has been raised about the potential estrogenic effects of APs and APEs because of a number of recent studies that have demonstrated estrogenic responses in aquatic biota, including fish and invertebrates, both *in vitro and in vivo* (Nimrod and Benson, 1996a).

Soto *et al.* (1991) accidentally found that NP released from polystyrene centrifuge tubes caused proliferation of human estrogen sensitive MCF-7 breast tumor cells (the "E-screen" assay). NP also was shown to trigger mitotic activity in rat endometrium (Soto *et al.*, 1991). OP, however, was found to be more potent than NP in the E-screen assay (Soto *et al.*, 1995). Colerangle and Roy (1996) demonstrated that exposure of Noble rats to NP increased the proliferation activity and altered cell-cycle kinetics of epithelial cells of the mammary gland.

APs are known to compete for the estradiol (E_2) binding site (receptor) (Mueller and Kim, 1978; Soto *et al.*, 1995; Routledge and Sumpter, 1997). In the presence of NP, a reduction in the binding of: E_2 to the human estrogen receptor, 5α-dihydrotestoterone (DHT) to the rat androgen-binding protein and human sex hormone-binding globulin has been reported (Danzo, 1997). Lee and Lee (1996) reported that NP mimics the effects of E_2 in rats in its uterotrophic action, but is 2000 to 10000 times less potent. The relative binding affinity of OP and NP to the estrogen receptors within intact breast cancer cells (MCF-7 cells) was determined to be 0.072% and 0.026% of the estradiol affinity, respectively (Nagel *et al.*, 1997). Abraham and Frawley (1997) demonstrated that OP stimulates prolactin gene expression, which plays an important role in the regulation of lactation as well as neonatal development in mammals. The degree of stimulation caused by OP is similar to E_2, although the concentration of OP must be 1000-fold greater (trigger concentration) for the responses to be observed. Tamoxifen, an estrogen antagonist, caused reduction of the estrogenic effect induced by OP, suggesting that the response to OP is indeed an E_2-receptor mediated. NP has been shown to induce estrogen receptor gene expression in fish (Ren *et al.*, 1996) and MCF-7 cells (Ren *et al.*, 1997).

Jobling and Sumpter (1993) demonstrated that APEs caused synthesis of vitellogenin in rainbow trout (*Oncorhynchus mykiss*) hepatocytes. *Para*-substituted compounds were estrogenic, Tergitol-NP-9 and 4-nonylphenoxycarboxylic acid were weakly estrogenic and 2-*tert*-butylphenol and 3-*tert*-butylphenol were not estrogenic. A decrease in estrogenic effects of NPEs was observed with increasing EO chain length. The highly ethoxylated Tergitol NP40EO did not stimulate synthesis of vitellogenin. Tamoxifen caused inhibition of the estrogenic effect incurred by the nonylphenolic compounds, suggesting further that the action of NP/NPEs is mediated through the estrogen receptor. Nimrod and Benson (1996b) showed an increase in vitellogenin production in channel catfish (*Ictalurus punctatus*) after an *i.p.* (intraperitoneal) dose of 237 mg/L 4-NP. Hewitt *et al.* (1998) demonstrated that NP does compete for the E_2 binding site of the estrogen receptor. Tremblay and van der Kraak (1995) reported that NP bound to the estrogen receptor of rainbow trout and goldfish and that NP acted as an estrogen agonist by affecting estrogen dependent processes including induction of vitellogenin synthesis.

OP, NP, NP2EO and NP1EC all stimulated vitellogenin gene expression in trout hepatocytes and effects were detectable at concentrations of 10^{-7} M for OP and 10^{-6} M for the nonylphenolic compounds (White *et al.*, 1994). Although NP1EC stimulated human breast cancer cell (MCF-5 and ZR-7) growth, OP stimulated cell growth at lower concentrations. In this assay, NP and NP2EO were the least estrogenic alkylphenolic compounds. By studying the reporter gene, EREBLCAT, in transiently transfected cells, White *et al.* (1994) demonstrated that OP also stimulated transcription of the reporter gene in a similar way as 17β-E_2. Higher concentrations of NP1EC, NP and NP2EO (10^{-5} - 10^{-6} M) were required to stimulate transcription relative to OP (10^{-7} M)(White *et al.*, 1994). Addition of estrogen antagonists reduced or eliminated the responses, further indicating that the effect of OP is mediated through

the estrogen receptor. Hydroxytamoxifen reduced the transcriptional activity of both estradiol and OP, while ICI 182780, an estrogen antagonist, was completely inhibitory. Stimulation of EREBLCAT transcription in CEFs (chicken embryo fibroblasts) by OP was observed only if the CEFs were cotransfected with mouse estrogen receptor (White *et al.*, 1994). The theory that alkylphenolic compounds are mediated by the estrogen receptor was confirmed because the presence of a cotransfected receptor was required to stimulate transcription. Additionally, White *et al.* (1994) compared the ability of various OPEs, with side-chains ranging from 2-12 EO groups, to stimulate EREBLCAT transcription in MCF-7 cells. OP was the most potent, a reduction in estrogenic activity was observed when OP2EO was tested and OP3EO and higher ethoxylated compounds produced a negligible effect on transcription.

Alkylphenolic compounds compete with 17β-estradiol for binding to the rainbow trout estrogen receptors (White *et al.*, 1994). OP was the most potent, with a K_d of 1.1×10^{-5} M, the K_d for NP was 5×10^{-5} M, for NP1EC was 2×10^{-4} M and NP2EO did not impair binding. Tremblay and Van Der Kraak (1998) reported the NP potency for vitellogenin production in primatry cultures of rainbow trout hepatocytes to be 1.6×10^{-3} which was higher than the estimates reported other studies. Although the concentrations required to induce the vitellogenin response *in vivo* (Jobling *et al.*, 1996) are two orders of magnitude higher than *in vitro* (Jobling and Sumpter, 1993) the relative potency of the chemicals is similar. The threshold concentration for vitellogenin synthesis induction by NP in rainbow trout was estimated to be approximately 10 µg/L, similar potencies were estimated for both NP1EC and NP2EO, but the threshold for OP was estimated to be approximately 3 µg/L (Jobling *et al.*, 1996). Routledge *et al.* (1998) reported the threshold for induction of plasma vitellogenin with OP in rainbow trout to be between 1 and 10 µg/L, while the threshold in roach (*Rutilus rutilus)* was between 10 and 100 µg/L. Winter flounder (*Phatichys flesus*) exposed to 30 µg/L NP did not result in plasma vitellogenin induction (Allen *et al.*, 1998). The effects of these chemicals also may be expected to be additive (Soto *et al.*, 1994; Sumpter and Jobling, 1995).

Routledge and Sumpter (1997) used a recombinant yeast screen to compare the relative potency of APs and APEs. The DNA sequence of the human estrogen receptor was integrated into the yeast (*Saccharomyces cerevisiae*) genome which also contained expression plasmids carrying an estrogen-responsive sequence that controls a reporter gene (β-galactosidase). Potencies relative to estradiol were: OP (1.5×10^{-3}), NP (7×10^{-3}), NP1EC and NP2EC (2.5×10^{-4}) and NP2EO (5×10^{-5}). Burnison (1998) measured a relative potency for NP of 1.9×10^{-3} in a similar human (HR) yeast system. The presence of EO groups markedly reduced the estrogenic activity and the highly ethoxylated NP12EO was found not to be estrogenic. This suggests that all alkylphenolic compounds with a high degree of ethoxylation are not estrogenic. APs, APEs with a low degree of ethoxylation and their carboxylic acids derivatives however, all were found to be active in the assay. These results are consistent with previous estimates of the relative estrogenic potency of APs and APEs. A study by Andersen *et al.*

(1999) compared the relative potency of numerous chemicals including 4-NP, 4-OP and NP12EO in competitive binding to estradiol receptors, YES (rainbow trout) assay and vitellogenin induction in rainbow trout after *i.p.* injections. Neither NP nor OP caused a vitellogenin induction after 9 d with a dose of 50 mg/kg. The relative potency in recombinant human estrogen receptor (hER) was similar relative to estradiol at $3.6x10^{-4}$ and $3.4x10^{-4}$ respectively. In contrast, OP was more potent than NP at $>1.0x10^{-5}$ and $1.8x10^{-6}$, respectively. In the E-screen assay, OP was generally more potent than NP but it varied considerably among three different labs.

During a three week exposure to APs, reduced testicular growth was observed in rainbow trout, which was correlated to the estrogenic potency (Jobling *et al.*, 1996). Jobling *et al.* (1996) also reported that the degree of testicular growth inhibition by OP or NP was dose-dependent and was affected by the timing of the exposure although, the mechanism of inhibition of testicular growth is not known. Inhibition of the synthesis of androgens which are required for spermatogenesis is one possible mechanism (Trudeau *et al.*, 1991). Inhibition of spermatogenesis may be the result of an effect on one or more hormone levels involved in the regulation of testes development (Jobling *et al.*, 1996). Estrogens may inhibit gonadotropin-releasing hormone (GnRH) or gonadotropin synthesis in the hypothalamus or pituitary, respectively. The GnRH and gonadotropin genes of fish contain estrogen-responsive elements (Klungland *et al.*, 1993; Xiong *et al.*, 1994a;b) suggesting that their synthesis could be controlled at least in part by estrogens. 17β-estradiol in the diet can cause complete inhibition of gonadal development in salmonids (Billard *et al.*, 1981).

During the first month after hatch, Ashfield *et al.* (1998) exposed female rainbow trout to NP, OP, NP2EO, and NP1EC and followed the growth and ovarian weight for up to 431 d. A decrease in fish weight was observed at the termination of the first experiment for all four chemical treatments. Fish were exposed to the individual compounds for a period of 22 days and sampling was performed at day 108. In the second experiment where the exposure period was 35 d and sampling was performed over 431 d, an inconsistent response was observed with slight increases or decreases in weight. These differences may have been due to the limited sample sizes, Ashfield *et al.* (1998) concluded that these alkylphenolic compounds can affect growth in fish at environmentally relevant concentrations both positively and negatively depending on chemical and time of exposure. Additionally, slight changes in ovosomatic index after 466 d was observed, with NP having elevated values at 30 µg/L and NPIEC having reduced ovosomatic indices at 1 and 10 µg/L but not 30 µg/L. Neither OP nor NP2EO had significant changes in ovosomatic indices at the end of the study (Ashfield *et al.*, 1998).

Christiansen *et al.* (1998) reported that exposure of male eelpout (*Zoarces viviparus*) to repeated *i.p.* injections of NP at 10 or 100 µg/g/week caused significant induction of plasma vitellogenin and a reduction in gonadosomatic index after 21-25 d. Histological examination of

testes showed changes in the testicular structure of exposed fish, including degeneration of the seminiferous lobules as well as increased number of spermatozoa in the seminiferous lobules and squamous appearance of Sertoli cells. Electron microscopy revealed a greater number of phagocytized spermatozoa in Sertoli cells. Reduced activity of γ-glutamyl transpeptidase suggested impaired Sertoli cell function in exposed fish.

Fish growth may be affected by endocrine disrupting substances. In rainbow trout, the period of sensitivity to estrogen exposure appears to be at sexual differentiation, which coincides with yolk sac re-absorption and onset of exogenous feeding (van der Hurk and Slof, 1981). In one study, a reduction in weight and length was observed in rainbow trout exposed to 17β-estradiol (Johnstone *et al.*, 1978). Growth was reduced in channel catfish (*Ictalurus punctatus*) exposed to a potent synthetic estrogen, diethylstilbestrol (Bulkley, 1972). Blazquez *et al.* (1998) suggested that exposure of sea bass (*Dicentrarchus labrax*) to 17β-estradiol or 17 α-ethynylestradiol in food resulted in the mobilization of fat reserves from viscera to muscle which lead to reduced growth.

Numerous mechanisms are involved in organism growth. Sex steroid hormones can modify the synthesis and secretion of growth hormone (GH) which play an important role in growth (Sumpter, 1992). Bovine GH administered to coho salmon (*Oncorhynchus kisutch*) results in enhanced growth (Markert *et al.*, 1977). Decreased growth also may be due to toxicity or energy being diverted for the production or metabolism of vitellogenin or other compounds. A reduction in growth may lead to reduced competitive ability of exposed fish.

Gimeno *et al.* (1997) exposed male common carp (*Cyprinus carpio*) to 0.14 mg/L 4-*tert*-pentylphenol for 3 days during the embryo-larval period and observed no effect on sexual differentiation or proliferation of primordial germ cells. However, longer exposures, starting before and including the period of sexual differentiation, induced the formation of an oviduct, which remained after 59 days in clean water. The numbers of primordial germ cells were reduced in a dose-related manner in this study.

The synthesis of fish zona radiata (vitelline envelope) proteins is induced *in vivo* by NP at levels lower than exposures causing vitellogenic responses in fish (Arukwe *et al.*, 1997b). Zona radiata proteins are synthesized in the liver in response to treatment with 17β-estradiol (Oppen-Bertsen *et al.*, 1992; Hyllner *et al.*, 1991). Arukwe *et al.* (1997a) showed that NP, which is known to mimic the actions of endogenous estrogens, causes variations in isoforms of hepatic cytochrome P450-dependent steroid and xenobiotic-metabolizing enzymes. It is known that steroid hormones such as estradiol, progesterone and androstendione modify the response of cytochrome P450 isozymes in the tissues of fish and mammals (Stegeman and Hahn, 1994; Zimniak and Waxman, 1993). NP treatment of juvenile salmon caused a decrease in ethoxy resorufin -*o*-deethylase (EROD) activity (Arukwe *et al.*, 1997a). Low exposures to NP resulted

in reduced plasma estradiol levels, although no effect was observed at higher doses. Jobling *et al.* (1996) suggested that NP also may affect the estradiol feedback system or the pituitary gland. Arukwe *et al.* (1997a) suggested that NP is involved in the regulation of hepatic cytochrome P450-dependent steroid metabolism in fish, and these changes occur at doses much lower than needed to alter vitellogenin production. Exposure of the invertebrate *Daphnia magna* to NP altered the uptake and metabolism of testosterone (Baldwin *et al.*, 1997).

Gray and Metcalfe (1997) exposed Japanese medaka (*Oryzias latipes*) to aqueous solutions of NP at nominal concentrations of 10, 50, and 100 μg/L, from hatch for 3 months. Fifty per cent of the male fish in the 50 μg/L treatment and 86% of males in the 100 μg/L treatment developed testes-ova (intersex). The sex ratio in the highest treatment also was shifted in favour of females relative to the controls. Miles-Richardson *et al.* (1999) exposed adult fathead minnow (*Pimephales promelas*) for 42 d to NP and observed a dose-dependent change in histologic lesion in testes at 0.33 to 2.4 μg/L. There was an apparent relative or absolute increase in the number of Sertoli cells, necrotic spermatozoa and germ cell syncytia. A similar response was not seen in fish exposed to 17β-estradiol. There was no difference in the secondary sex characteristics or size of gonads. Similar effects were not observed with exposure to NPEs (Solfonic N-95) and neither NP or NPE caused histological effects on the ovary.

Endogenous sex steroids are normally low during smoltification and salt water migration; however, exposure of fish to estrogenic compounds during this sensitive parr-smolt transformation could have adverse effects on their performance and survival. Weekly *i.p.* injections (30 d) of both 17β-estradiol (50 μg) and 4-NP (3 mg) in 23 g Atlantic salmon (*Salmo salar*) activated the vitellogenic system and had an inhibitory effect on smoltification and hypoosmoregulatory physiology (Madsen *et al.*, 1997a). There was a reduction in gill Na^+, K^+-ATPase activity, relative α-subunit gill Na^+,K^+-ATPase mRNA expression, gill chloride cell density and a poorer hypoosmoregulatory performance. Impaired salt water tolerance was strongly correlated with decreased gill Na^+,K^+-ATPase activity in these studies. The potential impacts of AP and APEs on smoltification and metamorphosis in aquatic organisms has not adequately been studied. A recent report by Fairchild *et al.* (1999) suggested a linkage between the exposure of Atlantic salmon in the 1970s to 4-nonylphenol (in pesticide formulations (Matacil 1.8D) used to control spruce budworm in eastern Canada) and the numbers of fish returning to the exposed streams in subsequent years. NP was shown to impair the parr-smolt transformation in Atlantic salmon and cause increased mortality and decreased growth in salt water (Brown *et al.* 1998).

Purdom *et al.* (1994) performed experiments on the estrogenic effects of sewage treatment works (STW) in response to casual observations by anglers of hermaphrodite fish in sewage treatment works (STW) lagoons in England. Levels of plasma vitellogenin were highly

elevated in both male and female fish caged for three weeks in the STW lagoons (Purdom *et al.*, 1994). The responses were seen in fish exposed to sewage effluents regardless of the characteristics or size of the catchment basin. Vitellogenin induction in fish several kilometres downstream of many sewage outfalls in England have been reported in additional studies (Harries *et al.*, 1997). Relatively high concentrations of APs and APEs have been reported in sewage treatment systems. Although these compounds have been shown to cause estrogenic responses in fish, there is strong evidence suggesting that other chemicals are responsible for the estrogenic responses observed in fish exposed to sewage effluents. Natural and synthetic estrogens (estradiol, estrone, ethynylestradiol) have been identified in sewage effluents at concentrations that can cause vitellogenin induction in fish (Desbrow *et al.*, 1996; 1998; Routledge *et al.*, 1998). Isolation of estrogen-responsive chemicals into a fraction which contained these natural and synthetic estrogens was performed by fractionation of the effluents using SPE and HPLC. Municipal effluents are complex mixtures that may contain a number of estrogenic compounds. Although AP and APE concentrations are generally lower than the threshold for estrogenic responses in these effluents, treatment conditions or contributions from municipal or industrial effluents (*e.g.*, textiles) may result in higher concentrations and exposure of biota.

2.4.1.4.1 Summary of Estrogenic Responses

APs and APEs have been reported to cause a number of estrogenic responses in a variety of aquatic organisms. These responses occur at concentrations similar to those at which chronic effects are reported in aquatic biota. Experiments in several different *in vitro* systems have indicated similar relative potencies among APEs, OP was the most potent, although only 27000 times less potent than estradiol. NP was approximately 25% less potent than OP, and NP2EO and NP1EC were only slightly less potent than NP in inducing vitellogenin in trout hepatocytes. Addition of EO units to NPEs reduced the potency, such that NP9EO was an order of magnitude less potent *in vitro* (Jobling and Sumpter, 1993; Routledge and Sumpter, 1997) (Table 12).

APEs bind to the estrogen receptor, resulting in the expression of several responses, including the induction of vitellogenin in both *in vitro* and *in vivo* systems. The threshold for vitellogenin induction in fish is 10 µg/L for NP and 3 µg/L for OP (Jobling *et al.*, 1996). The threshold for expression of intersex (ova-testes) in medaka was <50 µg/L for NP (Gray and Metcalfe, 1997). The induction of vitellogenin mRNA in rainbow trout was recently reported at 1 µg/L of NP (Fent *et al.*, 1999). The estrogenic responses appear to be at least additive (Soto *et al.*, 1994; Sumpter and Jobling, 1995) and, therefore, the sum of effects should be considered. APEs also affect the growth of testes in fish, alter normal steroid metabolism and disrupt smoltification.

Levels of individual APEs may be low in the environment, but APs, APEs and APECs are found as complex mixtures in effluents, and if considered together, may exceed the threshold for effects. A critical consideration is the relative estrogenic potency of the APs and APEs. Currently, there is considerable debate which has resulted from inconsistencies in relative potencies reported based on data from estradiol receptor binding, YES assay and vitellogenin induction in trout hepatocytes. Additional research is required to fully understand the potential estrogenic effects of APs and APEs on the environment. The significance of estrogenic responses to the individual or population also is not known.

2.4.1.5 Bioaccumulation in the environment

Bioaccumulation of AP and APEs has been studied in a number of algae, plants, invertebrates and fish species, both in the laboratory and under field conditions. Bioconcentration factors (BCFs) of APs and APEs determined in the laboratory and bioaccumulation factors (BAFs) measured in the field are similar and represent a low to moderate tendency to bioaccumulate (Table 13).

2.4.1.5.1 Nonylphenol

BCFs for NP in several fish species have been found to range from 0.9 to 3400 (Table 13). Brooke (1993a) measured a BCF in fathead minnows (*Pimephales promelas*) of 741 in 28 d exposures. Ward and Boeri (1991a) measured BCFs of 271 to 344 in fathead minnows (*Pimephales promelas*) in an intermittent flow-through system after 20 d exposure and 7 d depuration. Clearance of NP was very rapid, with half-lives of 1.2-1.4 d. McLeese *et al.* (1981) measured BCFs of 75 to 280 during 4 d exposures in Atlantic salmon (*Salmo salar*), with a clearance half-life of 4 d. Brooke (1993a) reported a BCF for bluegill sunfish (*Lepomis macrochirus)* of 220 after 28 d. In large littoral enclosures, bluegill sunfish had a BAF of 87± 124 (Liber *et al.*, 1999b). Japanese studies determined BCFs for NP ranging from 0.9 to 3.3 after an 8 week exposure in carp (*Cyprinus carpio)* and 12-469 for OP (CITI, 1992). In rainbow trout (*Oncorhynchus mykiss*), Lewis and Lech (1996) measured BCF values for ^{14}C-NP (ring labeled) of 24 and 98 in carcass and viscera, respectively. Uptake was very rapid and depuration half-lives were 19.8 h for fat and 18.6 h in muscle. They detected glucuronic acid and oxidative metabolites in bile. Meldahl *et al.* (1996) also identified several hydroxylated and glucuronic acid conjugates in rainbow trout both *in vivo* and *in vitro*. The NP used in these studies was not typical of commercial preparations (Staples *et al.*, 1998).

Coldham *et al.* (1998) examined the biotransformation, tissue distribution and persistence of tritiated nonylphenol (^{3}H-NP) residues in juvenile (mean weight: 122 g) rainbow trout (*Oncorhynchus mykiss*) after a single injection of 375 µg into the caudal vein. Total ^{3}H-

NP residues in the tissues after 144 h were greatest in bile>>faeces>>liver>pyloric caecae, kidney > brain, gonad, heart, plasma, skeletal muscle, and skin. They determined that clearances of NP from tissues was biphasic with the first phase (α) being relatively rapid with half-lives ranging from 0.17 h (plasma, gonad, spleen) to 5.8 h (muscle). The second phase (β) of clearance was much longer, especially in muscle, liver and skin, with half lives of 99 h. HPLC analysis of the bile, liver, pyloric caecae and faeces indicated that the ^{3}H was mostly glucuronide conjugates, while the residues in muscle were the parent material after 144 h.

In laboratory exposures to ^{14}C-NP, Ekelund *et al.* (1990) estimated BCFs of 90-110 for shrimp (*Crangon crangon),* 2740-3430 for common mussel (*Mytilus edulis*) and 1200-1300 for stickleback (*Gasterosteus aculeatus*). Lipid-normalized BCF values were reported as 5500-7500, 169300-216600 and 16700-17800 in shrimp, mussel and sticklebacks, respectively. The clearance of NP from fish was relatively rapid, but was much slower for the mussels. Because more than 80% of the ^{14}C-NP was found as metabolites in the tissue, the BCF values are overestimated.

McLeese *et al.* (1980a) reported a BCF of 1.4 to 7.9 for NP in mussels based on measured concentrations in tissue and water and a BCF of 10, based on uptake and depuration rates. The clearance of NP in mussels was very rapid with a half-life of only 0.3 d (McLeese *et al.,* 1981). As with fish, the bioaccumulation of NP in mussels under laboratory conditions is low to moderate, and clearance is very rapid. Granmo *et al.* (1991) studied caged mussels (*Mytilus edulis*) near an outfall of an industrial facility producing surfactants, and reported a BAF of 340 (wet weight) for NP.

Algae, plants, fish and ducks were collected by Ahel *et al.* (1993) to measure the BAFs of APEs in the Glatt River and one of its tributaries, the Chiesbach, in Switzerland. NP concentrations of 38, 4.2 and 2.5 mg/kg dw were measured in the macrophytes; *Cladophora glomerata, Fontinalis antipyretica* and *Potamogeton crispus,* respectively, although the average concentration of NP was 3.9 µg/L in the river water. The concentration of NP in *C. glomerata* was observed to vary both temporally and spatially in relation to the sewage outfall. The concentrations of NP in fish muscle were lower than those observed in algae. NP concentrations were 0.18 mg/kg dw in *Squalius cephalus*, 0.38 mg/kg in *Barbus barbus*, 0.15 in *Onchorhynchus mykiss* and 1.20 mg/kg in the muscle of ducks (*Anas boscas*). The concentrations varied among the tissues with liver, generally having a slightly higher concentrations. BAFs for fish were based on dw, therefore, values ranged between 13 and 408. Staples *et al.* (1998) corrected these values to wet weight, based on the assumption of a 95% water concentration in tissue and derived BAFs of 487, 54, 32, 7, 15 and 6 for NP in *C. glomerata, F. antipyretica, P. crispus, S. cephalus, B. barbus* and *O. mykiss*, respectively.

The BAFs measured in the field are similar to those in the laboratory and represent a low to moderate tendency to bioaccumulate in biota. This is expected based on a measured log K_{ow} of 4.48 (Ahel and Giger, 1993b) for NP; moreover, OECD (1997) predicted a theoretical BCF of 1,280. Metabolism and excretion could alter this predicted value considerably from the theoretical value

2.4.1.5.2 Nonylphenol polyethoxylates

The BAFs for NP1EO and NP2EO were lower in *C. glomerata* (10, 23), *F. antipyretica* (2, 3), and *P. crispus* (2, 10) relative to NP (Staples *et al.*, 1998; Ahel *et al.*, 1993). The BAFs for NP1EO and NP2EO in fish were slightly higher than NP in *B. barbus* (19, 37) but lower than NP in *S. cephalus* (1, 2) and *O. mykiss* (3, 0.8) (Staples *et al.*, 1998; Ahel *et al.*, 1993). The uptake and depuration in cod (*G. morhua*) were very rapid, with radioactive NP10EO being eliminated in 24 h (Granmo and Kollberg, 1976). Wahlberg *et al.* (1990) measured BAFs of 60 for NP3EO, 100 for NP2EO, 170 for NP1EO and 340 for NP in caged mussels placed in the effluent of a NPE production plant.

2.4.1.5.3 Octylphenol and octylphenol polyethoxylates

Japanese studies determined BCFs between 12 and 469 for OP after an 8 week exposure in carp (*Cyprinus carpio)* (CITI, 1992). Based on a slightly lower K_{ow} of 4.12 (Ahel and Giger, 1993b), BCFs for OP are expected to be slightly lower than those of NP.

2.4.1.5.4 Bioaccumulation Summary

The ability of NP and NPEs to bioaccumulate in aquatic biota in the environment is low to moderate. BCFs and BAFs in biota, including algae, plants, invertebrates and fish range from 0.9 to 3430 for NP. There are relatively few bioaccumulation data for NPEs, but based on their structure, they are not expected to bioaccumulate. Fewer bioconcentration and bioaccumulation data are available for OP and OPEs, but based on their structural similarities to NP and NPEs, they are predicted to have BCFs/BAFs slightly lower than those of NP and NPEs.

2.4.2 Effects on experimental mammals

Available from Health Canada.

2.4.3 *Human toxicology*

Available from Health Canada.

2.4.4 *Abiotic atmospheric effects*

None identified.

2.5 ENVIRONMENTAL QUALITY STANDARDS, CRITERIA AND GUIDELINES AND ACTIONS

APs and APEs, in particular NP and NPEs, are high volume industrial chemicals that are discharged to the environment in significant quantities. Some of the degradation products of APEs are more lipophilic than the parent compounds and demonstrate toxic effects to aquatic organisms. Consequently, there have been regulatory and voluntary initiatives to phase out NPE use from various products in North America and Europe. For example, concerns about the persistence and toxicity of NPE degradation products lead to voluntary elimination of NPE surfactants from many uses in Europe, and agreements by the Oslo and Paris Commissions (OSPAR) that NPEs would be phased out of domestic cleaning products by 1995 and out of industrial cleaning products by 2000 (PARCOM Recommendation 92/8, 1992). The Oslo and Paris Conventions for the Prevention of Marine Pollution Working Group on Diffuse Sources (1996) noted that a preliminary environmental risk assessment for NP, based on measured environmental concentrations (PEC) and a calculated predicted no effect concentration (PNEC), revealed that the PEC/PNEC quotient in river water and coastal sea water generally lies around or above 1, indicating the potential for ecotoxicological effects. That organization is currently performing a full risk assessment and it noted that the significance of estrogenic effects of NP, OP and their derivatives on aquatic biota and wildlife remains to be determined. Nonylphenol is not currently classified in the European Union. For the environment, nonylphenol has been provisionally classified by industry as "N R50-53 - dangerous for the environment" (U.K. Environment Agency, 1998), and the following symbols, risk phrases and safety phrase have been provisionally assigned: "R50-53 - very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment; S61-61 - avoid release to the environment - refer to special instructions/safety data sheets".

In Switzerland the use of NPEs and OPEs in domestic washing agents was banned in 1987. In Germany, in 1986, some industry associations voluntarily agreed to phase out the use of APEs in domestic and commercial laundry detergents, which lead to an estimated 85% reduction

in the use of these chemicals for such purposes in the period 1986-1997. In the United Kingdom the British Association for Cleaning Specialities and the Soap and Detergent Industry Association agreed to remove all APEs from industrial and institutional detergents in 1998 (U.K. Environment Agency, 1998). Other industrial uses of NPEs, however, are still allowed in Europe. The Danish "cut-off" values for the sums of concentrations of NP, NP1EO and NP2EO in sludge, according to Danish Ministerial Order No. 823, 16 Sept. 1996, are 50 mg/kg dw from 1 July 1997, and 10 mg/kg dw from 1 July 2000 (Madsen *et al.*, 1997b). The Swedish "target value" for NP in sludge from 1997 is 50 mg/kg dw (Tideström, 1997). Australia decided that there was insufficient data to derive a full or interim guideline for NP in 1997, however, a freshwater environmental concern level (ECL) of 0.2 µg/L and a marine ECL of 1.2 µg/L were recommended (Australian and New Zealand Guidelines for Fresh and Marine Water Quality, July, 1999).

In the United States, the U.S. Environmental Protection Agency (EPA) Testing Priority Committee in 1987 formally nominated NP as a candidate for rule-making under the Toxic Substances Control Act (TSCA). In 1988, the U.S. EPA proceeded with delisting 2-nonylphenol from the TSCA Public Inventory since it was no longer manufactured (Hellyer, 1991). The U.S. EPA has required testing of NPEs and NP for persistence, fate and toxicity by the U.S. Chemical Manufacturers Association Alkylphenols and Ethoxylates Panel. The CMA APE panel also has undertaken some voluntary studies, notably of occurrence of NPEs and NP in thirty U.S. rivers.

3.0 ENVIRONMENTAL RISK CHARACTERIZATION

The environmental risk assessment of a PSL substance is based on the procedures outlined in Environment Canada (1997a). Analysis of exposure pathways and subsequent identification of sensitive receptors are used to select environmental assessment endpoints (e.g., adverse reproductive effects on sensitive fish species in a community). For each endpoint, a conservative Estimated Exposure Value (EEV) is selected and an Estimated No-Effects Value (ENEV) is determined by dividing a Critical Toxicity Value (CTV) by an application factor (AF). A conservative (or hyperconservative) quotient (EEV/ENEV) is calculated for each of the assessment endpoints in order to determine whether there is potential ecological risk in Canada. If these quotients are less than one, it can be concluded that the substance poses no significant risk to the environment, and the risk assessment is completed. If, however, the quotient is greater than one for a particular assessment endpoint, then the risk assessment for that endpoint proceeds to an analysis where more realistic assumptions are used and the probability and magnitude of effects are considered. This latter approach involves a more thorough consideration of sources of variability and uncertainty in the risk analysis.

3.1 Assessment endpoints

In Canada, the releases of NP and NPEs occur solely via anthropogenic activity. The largest source of this group of compounds appears to be effluent from textile mills, pulp and paper mills and MWWTPs. A second major source is sludge from MWWTPs, to which the compounds are adsorbed. Environmental effects are expected to be greatest in the aquatic environment in regions near effluent release points. NP is expected to partition into sediments in aquatic environments and exist in the sludge fraction of treated effluents.

3.1.1 Aquatic

Assessment endpoints include abundance, growth and survival of fish, invertebrates and algae. Additional endpoints include reproductive effects on invertebrates and fish. Although NPEs are associated with endocrine disrupting properties, the outcome of this assessment is based primarily on chronic toxicity. A discussion of the endocrine effects and a comparison between these two endpoints, however, have been included in this assessment. Both marine and freshwater species were used in aquatic risk characterizations, depending on species sensitivity.

3.1.2 Terrestrial

Assessment endpoints in the terrestrial environment are based on chronic toxicity data from one earthworm study. These were the only data available for terrestrial organisms.

3.1.3 Atmospheric

Air is not considered to be a compartment of concern; therefore, a risk characterization in this compartment was not performed.

3.2 Environmental risk characterization

Significant releases of NPEs occur in industrial and municipal effluents in Canada. The emphasis of this risk characterization is, therefore, on the exposure from effluents. NPEs are released as complex mixtures whose composition may differ considerably, depending on the type of effluent and environmental compartment. NPEs are released in relatively constant concentrations as effluents (e.g., municipal effluents), with only minor seasonal variability; this, therefore, results in a relatively continuous exposure of aquatic organisms. However, changes in effluent dilution in the receiving environment will alter the exposure both spatially and temporally at most sites. Unfortunately, there are limited data available with which to confirm dilution at most sites, and there are considerably fewer data available for ambient surface waters, especially for the major metabolites. The available environmental data were

used to validate the exposure predicted from effluent concentrations.

Sediment toxicity data are available only for NP and not for the other metabolites. Therefore, the relative aquatic toxicity factors for NP were used as a surrogate for NPEs. Although the application of sludges from MWWTPs to agricultural soils represents a major route of exposure, soil toxicity data again were available only for NP. The relative toxicity, therefore, was estimated from the available literature on NP.

3.2.1 Overview of approach for determination of risk quotients

Because NPEs occur as complex mixtures in the environment and have different toxicities and estrogenic potencies, the approach used in this assessment was to first assess each chemical separately, then assess the complex mixtures found in the environment.

In the hyperconservative assessment, the EEV was taken to be the maximum effluent or environmental concentration. The ENEV for the hyperconservative assessment was determined by taking the most sensitive endpoint (e.g., LC_{50}, NOEC, *etc.*) and applying an AF (Tables 14 and 15) to account for uncertainty in extrapolating from laboratory to field conditions and interspecies and intraspecies variations in sensitivity, or other identified uncertainties. Effluents directly from textile mills from all sites were considered, even though many of these effluents received subsequent treatment in municipal treatment systems.

In aquatic environments, the conservative EEV was the maximum concentration of the final effluent (textile mills, municipal treatment systems and pulp and paper mills) that is discharged from each site directly to the environment (no additional treatment), diluted by a factor of 10. Although many sites have dilution factors greater than 10 outside of the immediate mixing zone, especially in ocean or lake sites, there have been instances of lesser dilution. Therefore, a dilution factor of 10 was considered appropriate for this assessment. The case for lesser dilution was observed at the Galt Treatment Plant in the Grand River, studied by Bennie *et al.* (1998) (Figures 19 and 20). In this case, the effluent remained on one side of the river for up to 8 km downstream of the outfall. There was very little degradation of the NPEs and NPECs in the river water over the 8 km, and the decreased concentrations downstream were attributed to physical dilution, not chemical degradation. The ENEV for the conservative approach was based on a more realistic CTV together with an AF (Table 14). In the conservative risk characterization for sediment, concentration data for all locations were used, rather than the worst-case scenario data. Concentration data in sludge-applied soil were used as the EEV in the conservative risk characterization for the terrestrial environment for NP, whereas the concentrations in the sludge alone were considered in hyperconservative approach.

A distributional assessment was performed only for the aquatic system, because it was

the environmental compartment of main concern. In this assessment, rather than using one of the most sensitive studies to determine the ENEV, an assessment of all of the available literature was used to determine an ENEV. The ENEV was based on an assessment of the distribution of acute and chronic effects reported in the literature. The EEV used was the mean diluted effluent concentration for each site, rather than the maximum value observed. As a means of graphically displaying the relationship between the EEVs and ENEVs, the distribution of the exposure data was plotted as a cumulative rank percent (Figure 21).

Although there is considerable information on the acute toxicity of NP, there are few data available on its chronic toxicity, and there are relatively few data available for the other APs/APEs. Based on an assessment of the data, giving weight to the studies with highest confidence and for which the same species were tested, an acute to chronic ratio (ACR) of 4:1 was determined. This value was applied to the acute toxicity curves to determine a chronic toxicity endpoint (Figure 21).

To generate the chronic toxicity curve, the acute toxicity data available for NP were first plotted as a cumulative rank percent. The acute data were then converted by applying an ACR of 4:1. Using the slope of these data after a log probit transformation, a value of 10 µg/L was determined as the concentration at which 95% of species are protected: i.e., the EC_{50} is exceeded in less than 5% of species. An AF of 10 was applied to take into consideration uncertainty associated with species differences and reported sublethal responses. The resulting ENEV for NP was 1 µg/L. The limited data available suggest that a similar ACR for the other APs/APEs may be appropriate; therefore, an ACR of 4:1 was assumed to apply to all metabolites.

The ENEVs for the metabolites, other than NP, were determined by estimating the relative toxicity of the metabolites to NP, considering all of the available literature in the assessment, using the distributional approach. Greater weight was placed on stronger studies and those that compared the toxicity of various metabolites directly. The relative toxicity was then applied to the ENEV of NP, which was considered to have a much larger and more reliable data set for toxicity. This approach allows for a more consistent assessment of the relative influence of the NPE metabolites.

The detection limits for environmental concentrations vary considerably among studies, over time and over matrices for the various metabolites. In the hyperconservative assessment, values less than the detection limit were taken as the reported detection limit for each sample. This was considered very conservative and in line with the preliminary screening philosophy of hyperconservative assessment. The detection limit for NP in some samples was higher than the ENEV, but it was felt that the uncertainty of the real environmental values warranted this conservative approach at this stage. In the conservative and distributional approaches, one-half

of the reported detection limit was used to calculate mean concentrations, because this was considered to be a more realistic estimate.

3.2.2 Additivity of effects

Since NP and NPEs exist together in mixtures in environmental samples, the combined impact of the mixture was examined. Although there is no direct evidence in the literature, it has been assumed that the lower-chain-length NPEs (NP1EO, NP2EO) and NPECs (NP1EC, NP2EC) have a mode of action similar to that of NP and that their effects are additive. The longer-chain-length NPEs (e.g., NP9EO) may differ from NP, because the mechanism of action is likely a physical surfactant effect. In addition to examining the exposure and toxicity of each metabolite individually, a toxic equivalency approach was applied, which factored in contributions from NP as well as the lower-chain-length (1,2) NPEs and NPECs to determine the overall potential risk of the group. The toxicity of each metabolite relative to NP was determined from the available literature, as indicated in Table 12. The EEV_{TEQ} was calculated by multiplying the exposure concentration (C_x) of each compound by its relative potency (RP_x). The sum of values for each compound was determined to be the total EEV_{TEQ}. NP was used as a reference compound, because there were considerably more data available on the toxicity of this compound, which provided a better reference point.

$$\text{Total } EEV_{TEQ} = \Sigma (C_x \times RP_x)$$

The Toxic Equivalency Quotient (TEQ) was, therefore, calculated as follows:

$$TEQ = \frac{EEV_{TEQ}}{ENEV_{NP}}$$

The report is structured to evaluate the risk associated with each metabolite individually, followed by an assessment of the mixture.

3.2.3 Aquatic environment

There is a relatively large database on the occurrence of NPEs in the Canadian environment and in effluents, although the majority of the data are for NP. Occurrence data have been reported in surface waters and sediments from streams, lakes and harbours, and considerable data also are available for municipal, textile, pulp mill and refinery effluents. Although NPEs with an average of 9–10 EO units are the major product used, they degrade to NPEs or NPECs with fewer EO units and ultimately to NP. The composition of these chemicals, therefore, varies considerably in effluents and in the environment.

There are many studies reporting acute and chronic effects of NP, fewer studies reporting the toxicity of NPEs and only a few studies that included the NPECs (see Section 2.4; Servos, 1999a). Unfortunately, few of these studies compared all of the NP, NPEs and NPECs together in a consistent way in the test organism species. Although studies reported in the literature have used many species, different test methods and different chemicals, there is a consistent pattern in the toxicity reported. The relative toxicity of the various metabolites was determined using comparisons of the published toxicity data. Emphasis was placed on high-quality studies that reported acute and chronic data in a single species using standard methodologies.

NP is relatively toxic to fish (17–1400 µg/L), invertebrates (20–3000 µg/L) and algae (27–2500 µg/L). There is an increase in the toxicity of NPEs with decreasing EO chain length, such that NP is 200 times more toxic than NP9EO. NPECs are less toxic than the corresponding NPEs and have acute toxicities similar to those of NPEs with 6–9 EO units. The relative toxicities differ from the relative estrogenicities (Table 12). NPEC is much less acutely toxic relative to NP but has only a slightly lower relative estrogenic potency. Because the relative potency of the various metabolites is based on *in vitro* or trout hepatocyte studies, not whole-organism studies, caution in interpretation is necessary (Table 12).

3.2.3.1 Risk characterization for nonylphenol (NP)

The toxicity data set for NP is relatively large, particularly for freshwater fish, for which 19 acute studies are available. The most sensitive acute and chronic effects reported for freshwater and marine species of fish, invertebrates and algae are summarized in Table 14.

Hyperconservative approach

In the hyperconservative approach, the 96-hour LC_{50} of 17 µg/L in winter flounder (*Pleuronectes americanus*) (Lussier *et al.*, 1996) was the CTV; the CTV was divided by an AF of 100 because it was an acute LC_{50} value, which resulted in an ENEV of 0.17 µg/L (summarized in Table 16). The EEV was the maximum concentration, without dilution, at each type of site.

The hyperconservative quotient was calculated as follows for a primary MWWTP effluent:

$$\text{Quotient} = \frac{\text{EEV}}{\text{ENEV}}$$

$$= \underline{62.08\ \mu g/L}$$

$$= \frac{0.17\mu g/L}{365}$$

Similar calculations were performed for all other types of sites. When the EEV was compared with the ENEV for each effluent, the resulting EEV/ENEV quotient exceeded one for most of the undiluted effluents (Table 17). All textile mill effluents had quotients greater than one. Four of 14 pulp mills sampled prior to 1998 and 5 of 19 sampled more recently had quotients greater than one for NP. Most (38 of 41) MWWTP effluents also exceeded a quotient of one. Since there were quotients greater than one for each type of site, a conservative assessment of the environmental concentrations of NP relative to chronic toxicity endpoints was considered necessary for textile mills, MWWTPs and pulp and paper mills.

Conservative approach

The winter flounder (a marine species) is more sensitive to NP than 18 other fish species reported in the literature by almost a factor of 10 (Figure 16). The consistency of the other acute toxicity data in fish suggested that this value was hyperconservative. In the conservative assessment, the CTV of the 28-day NOEC (length) for the mysid shrimp, *Mysidopsis bahia* (Ward and Boeri, 1991c), was divided by an AF of 10 to derive an ENEV of 0.39 µg/L (Table 18). An AF of 10 was considered appropriate because the ENEV was based on a NOEC and because there were considerable supporting data for at least three trophic levels.

The EEV was taken as the maximum concentration of effluent from each type of site, divided by a dilution factor of 10. The consideration of dilution, even using a conservative value of 10:1, dramatically decreased the number of effluents that resulted in an EEV/ENEV quotient of greater than one (Table 18). However, the conservative risk quotient exceeded a value of one in receiving water concentrations associated with several textile, municipal wastewater and pulp and paper mill effluents based on predicted concentrations. Three of 8 primary-treated municipal wastewater effluents, one of 21 secondary-treated municipal wastewater effluents, none of the tertiary- or lagoon-treated municipal wastewater effluents, one of 19 pulp and paper mill sites after 1998 and three of 14 pulp and paper mill sites prior to 1998 exceeded a quotient of one for NP. One of the two untreated textile mill sites exceeded a quotient of one, but neither of the two textile mills with on-site treatment had quotients greater than one for NP (Table 18). Several surface waters, including rivers that receive municipal or pulp mill effluents and harbours, also had risk quotients greater than one. Therefore, a distributional assessment was conducted.

Distributional approach

The ENEV for NP was taken to be 1 µg/L for protection of aquatic biota in the distributional approach (Figure 21). A further check on the appropriateness of the value 1 µg NP/L showed that NOECs for chronic effects in fish and invertebrates are reported in the range of 3–10 µg/L. Histological and biochemical responses also have been seen in the same concentration range. The induction of vitellogenin mRNA in rainbow trout has been reported at 1 µg/L. Therefore, the ENEV of 1 µg NP/L takes into account the uncertainty associated with species differences and extrapolation between laboratory and field studies, as well as taking into account reported molecular and histological responses.

Approximately 40% of the MWWTPs have mean concentrations of NP above 1 µg/L, although these are predominately effluents from primary (minimal) MWWTPs. However, if a dilution factor of 10 is applied, only five sites exceeded 1 µg/L for all sectors (Figure 22). The exceedances are usually less than a factor of 2 and included an untreated textile mill, three primary treated MWWTPs, a pulp mill effluent sampled prior to 1998 and a receiving water sample collected immediately downstream of a large MWWTP (Table 19). Based on recent data used in this assessment, NP alone may be at levels of concern to aquatic biota in areas immediately adjacent to industrial or municipal effluents (Figure 23). However, it is important to recognize that NP is only a single component of the APs/APEs released into the environment (Figure 23).

3.2.3.2 Risk characterization for nonylphenol polyethoxylates (NP1EO, NP2EO)

The acute 48-hour toxicity value of NP1EO for the mysid shrimp, *Mysidopsis bahia*, of 110 µg/L (Hall *et al.*, 1989) was taken to be the hyperconservative CTV. This value was divided by an AF of 100, resulting in a hyperconservative and conservative ENEV of 1.1 µg/L (Table 16). An AF of 100 was used because acute toxicity data rather than chronic data were used and because there were poor supporting data (Table 16). The ENEV value was considered appropriate for both NP1EO and NP2EO. An overview of available data suggests that the relative toxicity of both of these metabolites is similar and approximately half that of NP (e.g., Japanese medaka; Yoshimura, 1986). In the distributional approach, based on the more robust data on NP and assuming that the toxicity of NP1,2EO is about half that of NP, the ENEV for NP1,2EO was determined to be 2 µg/L.

Among the major degradation products of the higher-chain-length NPEs are NP1EO and NP2EO. Treatment systems, including MWWTPs, can, therefore, contain high levels of these metabolites, depending on the sources and the efficiency of degradation. Despite being less toxic than NP, these metabolites are found in higher concentrations, resulting in frequent exceedances of hyperconservative and conservative risk quotients, particularly for NP1EO (Tables 16 and 17). Almost all (9 of 10) primary-treated and more than half of both secondary- and tertiary-treated MWWTPs had conservative risk quotients greater than one, even after a

dilution of 10:1 in the environment (Figure 24). In contrast, none of the wastewater lagoon systems (0 of 5) had exceedances. Textile mill effluents that are not treated can also exceed the ENEV by a considerable amount (Figure 25). Pulp and paper mill effluents seldom (less than 20%) exceed the ENEV for these metabolites and appear to have been reduced in recent years, although the recent data available for these compounds are limited (Figures 26 and 27). A variety of surface water concentrations frequently (65%) exceed the conservative ENEV. The presence of NP1EO and NP2EO in effluents, especially textile mill effluents and poorly treated municipal wastewater, represents a potential risk to the environment and was, therefore, considered further. When compared with a distributional ENEV of 2 µg/L, no pulp mills sampled after 1998 (n = 3) and only 3 of 42 (2 primary, 1 secondary) MWWTPs have mean concentrations of NP1EO greater than the ENEV. There was one pulp mill sampled after 1998 with elevated NP2EO and only one primary-treated MWWTP at levels above the ENEV of 2 µg/L (Table 19).

3.2.3.3 Risk characterization for nonylphenol polyethoxylates (NP3–17EO)

NPEs with greater than 3 EO units were grouped together and treated as if they had the same toxicity as NP9EO. There are considerable acute and chronic toxicity data available for NP9EO and NP10EO, but few data for many of the other polyethoxylates. There are toxicity data available for several trophic levels, including fish, invertebrates and algae in freshwater and marine environments. The most sensitive reliable value is a 48-hour LC_{50} value in the mysid shrimp, *Mysidopsis bahia*, of 900 µg/L (Hall *et al.*, 1989) (Table 15). An AF of 100 was applied to that CTV, resulting in an ENEV of 9.0 µg/L. This value was applied to both the hyperconservative and conservative assessments (Table 16). The conservative approach considers dilution of effluents and is, therefore, more realistic.

Untreated or even on-site treated textile mill effluents had very high concentrations of NP9EO and consequently had high hyperconservative and conservative risk quotients (Tables 16 and 17). Untreated (primary) municipal wastewater effluents also contain high levels of NP9EO, and five of eight sites have concentrations that are predicted to exceed a conservative risk quotient of one. MWWTPs are effective at reducing the concentrations of NP9EO in the final effluent, and none of the secondary- or tertiary-treated effluent or lagoons had predicted concentrations that would exceed the conservative risk quotient of one. The dilution of most of the primary-treated effluents is likely underestimated and would further reduce the predicted risk of these chemicals. Unfortunately, there are relatively few data available for pulp and paper mill effluents, but the three effluents for which data are available all have conservative risk quotients less than one. NP9EO concentrations in two of the three surface water sites sampled were greater than 9.0 µg/L. Only untreated textile effluents had mean NP3–17EO concentrations above the distributional ENEV (200 µg/L) (Table 19). This value (200 µg/L) was based on the ENEV of NP divided by the relative toxicity factor of 0.005 (Tables 12 and

15). The concentrations of NP9EO in treated effluents are not likely to pose a risk to the environment. However, untreated or partially treated effluents, particularly textile mill effluents, may pose a potential risk, especially if effluent dilution is low.

3.2.3.4 Risk characterization for nonylphenoxyacetic acid and nonylphenoxyethoxyacetic acid

A NOEC of 1000 µg/L for NP1EC in fathead minnow was reported by Williams (1997), and an AF of 10 was applied, resulting in an ENEV of 100 µg/L (Table 16). This value was used for both the hyperconservative and conservative approaches. Maki *et al.* (1998) reported an LC_{50} in *Daphnia magna* for NP2EC of 990 µg/L. Applying an AF of 100 resulted in an ENEV of 9.9 µg/L, and this value was used in the hyperconservative assessment (Table 16). The study on NP1EC is more robust and better documented than the study by Maki *et al.* (1998). Since it is expected that NP1EC and NP2EC will have similar toxicities based on an overall assessment of the toxicity data, the values of Williams (1997) were also used as a conservative ENEV assessment for NP2EC.

NPECs can be created during effluent treatment, and concentrations in final effluent can be considerably higher than those in influent. Even in untreated effluents, the concentration of NP1EC does not appear to be high enough to result in chronic toxicity. The hyperconservative assessment resulted in some exceedances for NP2EC in wastewater effluents, particularly secondary and tertiary treatment (Table 17). When dilution is considered, no effluents in any sector have NPEC values that result in conservative risk quotients above one. Unfortunately, there are very few actual receiving water concentrations reported for these compounds. Despite the elevated concentrations of NPECs in treated final effluents, they have considerably lower toxicities; therefore, when considered alone, they do not represent a significant risk based on chronic toxicity.

3.2.3.5 Risk characterization for the combined effects of nonylphenol ethoxylates

As observed in field measurements, NP and NPEs occur as complex mixtures, and the toxicities of the metabolites are expected to be additive. When NP is considered alone, only three sites have predicted concentrations in receiving waters that exceed a value of 1 µg/L. When NP1EO and NP2EO are considered in addition to NP, an additional four sites exceed the ENEV. The chronic toxicity of NPE mixtures in municipal effluents and receiving waters is dominated by the effects associated with NP1EO and NP2EO and, to a lesser extent, NP. In situations where the concentrations of NP9EO are high, the concentrations of NP1EO and NP2EO also are elevated and contribute significantly to the overall acute and chronic toxicity. Although the concentrations of NP1EC and NP2EC are often higher in municipal wastewater effluents, they contribute very little to the toxicity of the mixture because of their relatively low

toxicity. Consideration of the additivity of NPEs slightly increased the number of sites that had conservative risk quotients greater than one (Table 18). Limited data indicate that untreated or partially treated textile mill effluents are likely to cause adverse effects on at least the most sensitive species in the receiving water because of the extremely high concentrations of NP3–17EO or its metabolites (NP1,2EO). NPEs also have the potential to exceed conservative ENEVs at a limited number of pulp mill and municipal effluents, particularly if effluent dilution is low or if there is little or no treatment.

In the distributional assessment, there was greater confidence in using the NP toxicity data together with the relative potencies for other metabolites to derive the TEQ than in using toxicity data for the metabolites alone. In the distributional assessment, the average effluent concentration at each site together with a dilution factor of 10 was used as the EEV. The combined TEQ results of the distributional assessment indicated levels of concern at 21 sites (Figures 28 and 29) (2/2 untreated textile mills; 4/14 pulp mills prior to 1998; 1/19 pulp mills post-1998; 5/10 primary MWWTPs; 1/21 secondary MWWTPs; 2/25 river sites; 2/5 lake sites; and 4/12 harbour sites) (Table 19). The river, lake and harbour sites that exceeded the TEQ were typically in water bodies adjacent to industrial sites or MWWTPs. The exceedance is usually due to the contribution of NP1EO.

3.2.3.6 Endocrine disruption in aquatic biota

Numerous studies have demonstrated the ability of APs/APEs to disrupt the normal function of the endocrine system of various organisms. These effects occur at a range of concentrations similar to those at which chronic effects occur in fish and invertebrates. Histological or biochemical responses have been reported at levels even lower than the NOEC for chronic toxicity. For instance, the threshold of NP for induction of vitellogenin in rainbow trout was reported as 10 µg/L, while the induction of mRNA for vitellogenin was reported at concentrations as low as 1 µg/L.

Recent reports by Miles-Richardson *et al.* (1999) suggest that in fathead minnows, histological and biochemical effects can occur at concentrations approaching or below 1 µg/L. However, the significance of these responses is not fully understood, and the effects on the organism or population have not been determined. Recent work by Brown *et al.* (1998) has demonstrated that NP can affect smoltification, resulting in reduced growth and survival in Atlantic salmon (*Salmo salar*) after very short term (24-hour) exposure to concentrations as low as 20 µg/L (nominal). Intersex in Japanese medaka has been demonstrated at 50 µg/L (Gray and Metcalfe, 1997). Vitellogenin induction is a biological response in fish that is mediated through binding of a chemical to the estrogen receptor. The threshold for this response is very similar to the LOEC for early life stage tests with rainbow trout and only slightly below the thresholds for *in vivo* responses such as intersex and impaired smoltification. Although

potential effects mediated through the estrogen receptor have been identified both *in vitro* and *in vivo* for NP in fish, this is only one mechanism by which a chemical such as NP can potentially interact with endocrine systems. The application of a factor of 10 to the whole-organism vitellogenin induction in rainbow trout results in a hyperconservative ENEV of 1 µg/L, which would be similar to the threshold for induction of mRNA of vitellogenin in rainbow trout (Fent *et al.*, 1999). A value of 1 µg/L seems justified for application to the EEV data for NP to determine if there is potential for endocrine-mediated responses in biota.

In the hyperconservative assessment, NP concentrations alone in final effluents are high enough in municipal effluents to cause concern for endocrine responses in 40% of the effluents. However, in the conservative assessment, when a dilution factor of 10 is applied, there are only three municipal effluent sites, one textile mill site and one pulp mill site that exceed a predicted aqueous concentration of 1 µg NP/L, and none that exceed a value of 10 µg/L. NP values were above 1 µg/L only in surface water immediately adjacent to industrial or municipal treatment sites. NP alone is unlikely to result in widespread effects mediated through the estrogen receptor in Canadian surface waters.

Similar to toxicity, the relative estrogenicity (RE) was used to determine a total estrogenic equivalency (EEV_{EEQ}) for the combination of NP and NPEs found as mixtures in the environment using NP as the reference, as follows:

$$\text{Total } EEV_{EEQ} = \Sigma(C_x \times RE_x)$$

where:

- total EEV_{EEQ} = total estrogenic equivalency of a mixture based on estrogenicity of NP
- C_x = concentration of metabolite x in the mixture
- RE_x = relative estrogenicity of metabolite x compared with NP

As with acute and chronic toxicity, there are few data available on the relative estrogenicity of the other metabolites, and there is considerable discrepancy among the few existing studies. The relative estrogenicity of the metabolites differs considerably from their relative acute toxicity (Table 12). The data of Jobling and Sumpter (1993), based on vitellogenin induction in trout hepatocytes, were considered for relative potencies. Based on these data, both NP1,2EO and NP1,2EC are expected to be only slightly less estrogenic than NP. This contrasts with acute toxicity, where NP1,2EC is much less toxic than NP. Because of the prevalence of NP1,2EC in treated effluents, the estrogenic responses may be a concern. However, considerable debate has emerged on the relative estrogenicity of these compounds. NP1EC was slightly less potent than NP in rainbow trout estradiol receptor assays, while the potency of NP1EO was much lower than that of NP (Servos, 1999b; Van Der Kraak, 1999). In transfected yeast cell assays (YES, with hER), there is little or no binding of NP1,2EC to the estrogen receptor, suggesting a very low or zero potency. Mixtures of NP1,2EC did not cause

ova-testes in Japanese medaka at concentrations similar to those that resulted in this response for NP (Metcalfe, 1999). Although this difference may be due to the characteristics of the *in vitro* assays, it does raise some uncertainty regarding the relative estrogenicity of these compounds. The discrepancies between potency estimates for NP1,2EC are particularly problematic. Although there remains debate, the trout hepatocyte assay results of Jobling and Sumpter (1993) are considered the best data currently available in the literature and were applied in this risk characterization. Caution must be used in interpretation of these results until the relative potency is validated in *in vivo* systems. NP1,2EO may exceed the threshold for endocrine responses in a few textile mill and municipal treatment effluents and receiving waters. NP1,2EC has the potential to cause estrogenic effects in most municipal and some pulp mill effluents. As many as 40% of receiving waters associated with municipal effluents (Figure 30) and a few pulp mill effluent exposed sites (Figure 31) may have the potential for endocrine responses.

When considered alone, concentrations of NP would not exceed the threshold for estrogenic response, except in environments receiving primary treated municipal effluents; however, the dilution at these sites is also likely underestimated. If the effects of the NPEs are added to the effect of NP, then about 15% of the sites are expected to exceed the threshold of 1 µg/L. When the NPECs are also added, almost 60% of the municipal sites and a few of the pulp mill sites exceed the value of 1 µg/L. These effluents would be a cause for concern, as histological and biochemical responses may be expected. Many municipal effluents would be expected to cause vitellogenin induction, but they are not expected to exceed the threshold (10 µg NP/L for rainbow trout) in receiving waters after a 10:1 dilution, even when considered as a group.

There is considerable uncertainty associated with predicting estrogenic responses, particularly for NP1,2EC and NP1,2EO. If the relative estrogenic potency of NP1,2EC is much less than that reported by Jobling and Sumpter (1993), as is indicated by some of the *in vitro* data, there would be very few sites where the threshold for estrogenic-mediated responses would be exceeded.

3.2.4 Risk characterization for sediment

NP, NP1EO and NP2EO have a tendency to adsorb to sediments, whereas NPECs are more water soluble and tend to remain in the overlying water (final effluent). NP is also moderately persistent in sediment; therefore, the effects of NP and NPEs are of primary interest for sediment-dwelling organisms. Unfortunately, there are no data available on the effects on benthic organisms for compounds other than NP. The CTV is the 14-day NOEC (growth and survival) for the midge, *Chironomus tentans*, of 20 µg/g (England and Bussard, 1993); dividing by an AF of 10 results in an ENEV of 2.0 µg/g (Table 14). The chironomid data were selected

because they represented the sediment-exposed biota. There are many factors that can affect the bioavailability of NP in sediments, including sorption to organic matter, which will alter the expression of toxicity. Variability in the sediment characteristics makes it difficult to extrapolate directly from laboratory results to toxicity in the environment. NP can also bioaccumulate in the environment, with a BAF of approximately 10. This could result in slightly higher exposure of selected organisms. The toxicity in sediments for NPEs was estimated relative to NP using the aquatic toxicity factors and dividing the ENEV by the relative toxicity for NP (2.0 μg/g). This resulted in an ENEV for NP1,2EO of 4.0 μg/g.

The concentrations of NP in Canadian sediments are generally low (<1 μg/g), with the exception of industrial harbours and sites near the outfalls of MWWTPs. A comparison of the sediment concentrations with the ENEV of 2.0 μg/g shows risk quotients greater than one in about one-fifth of the sites (5 out of 24), generally in areas immediately adjacent to MWWTP outfalls (Table 18). There are fewer data available for NP1EO and NP2EO in sediments, but their concentrations are usually less than those of NP. At one of six sites, the concentrations of NP1EO and NP2EO exceeded the ENEV. Limited data on NP9EO suggest that they are found at very low concentrations in sediments. No sediment data were available for NP1EC or NP2EC, but, based on their high water solubility and much lower toxicity, they are not expected to pose a risk for chronic toxicity in sediments.

Exposure to NP and NP1,2EO in sediments downstream of industrial or municipal effluents may result in potential risk to aquatic biota. The pattern of NP and NPEs in sediment is very different from that of the aqueous phase. The more hydrophobic chemicals are more prevalent in sediment, which is in contrast to higher levels of NP1,2EC in final effluents. The distribution and fate in river, harbour or lake sediments also may be very different from the distribution in water. For example, at Galt, Ontario, on the Grand River, the concentrations of NP decline rapidly with dilution downstream to 8 km. In contrast, the concentrations of NP in sediment increased at the 8-km site relative to locations close to the outfall (0 and 0.1 km). This is likely due to changes in the sediment composition, which shifts from hard rock and cobble at the outfall to higher organic content silts farther downstream. The potential effects of NP and NPEs in sediment can, therefore, be remote from the outfall, where dilution of the effluent is relatively high.

3.2.5 Risk characterization for the terrestrial environment

The earthworm, *Apporectodea calignosa*, tested by Krogh *et al.* (1996), had a 21-day EC_{10} (reproduction) of 3.4 μg NP/g in soil, which was taken to be the CTV. An AF of 10 was applied to the CTV to obtain an ENEV of 0.34 μg/g (Table 14). This ENEV was used in both the hyperconservative and conservative approaches. The difference between the approaches was that the concentration in raw sludge was used as the EEV in the hyperconservative

assessment, while the concentration in sludge-applied soil was used as the EEV in the conservative assessment. There are no toxicity data available for soil-dwelling organisms for the other NPE metabolites.

NP is found in relatively high concentrations in sludge relative to other NPE metabolites. In general, concentrations of NP1EO are usually lower than concentrations of NP, although in a limited number of cases NP1EO concentrations were higher than those of NP. NP9EO and NP1,2EC concentrations are consistently low in sludge. NP is more hydrophobic than the other metabolites and is expected to be the most persistent in soils. The focus of the sludge application was therefore on NP. In the conservative assessment, the predicted soil concentrations after sludge application were estimated based on the sludge concentrations measured in MWWTPs and the application rates recommended by the Ontario Ministry of Environment and Energy and Ontario Ministry of Agriculture, Food and Rural Affairs (1996) (maximum 8 tonnes per hectare over 5 years). Sludge NP concentrations ranged from 10 to 1260 μg/g. The sludge is assumed to be applied and tilled to a depth of 15 cm, and 1 ha is assumed to weigh 2000 tonnes. When applied to the soil and tilled, the sludge concentrations are, therefore, diluted by a factor of 0.004.

In the conservative analysis, the risk quotients for NP in soils based on predicted concentrations immediately after application are usually greater than one (18 of 30 sludge sites), indicating a potential for chronic effects (Table 18). The predicted concentrations in soil are higher than the ENEV by less than a factor of 2 and are rarely in exceedance by a factor of 10. During risk management of sludge application to agricultural soils, consideration must be given to application rates, the concentration of NP and NPEs in the sludge and the duration of the potential exposure (i.e., degradation).

A distributional assessment for NP and NPEs in soil was not undertaken, since actual concentrations in soil in Canada were not available. Further evaluation on a qualitative basis suggests that NP alone degrades rapidly in soil. However, when sludges that contain both NP and NPEs are considered, the degradation of the NPEs to NP may contribute to maintaining higher levels of NP for longer periods than would be expected if only NP existed in the sludge. However, NP is not expected to persist in soils for extended periods of time (<90 days), and sludge applications are normally followed by a period of fallow.

3.2.6 Summary of risk by specific sector

3.2.6.1 Textile mill effluents

Textile mills are a major source of APEs and a concern for the environment. Raw textile mill effluents have very high levels of NP and NPEs (especially those with high EO chain lengths).

At one of the monitored Canadian textile mills, in past, effluents were discharged directly into the environment. At another, on-site treatment is performed, although the system performs poorly. Very high levels of NP3–17EO (798–8811 µg/L), NP1EO and NP2EO (37–592 µg/L) and NP (2.7–13.3 µg/L) in receiving waters were measured at these two sites. Effluent concentrations measured at the point of release into water bodies were divided by a dilution factor (10) to estimate environmental concentrations. The distributional ENEV values were exceeded in all samples from these two sites. Two additional textile mill sites that have on-site secondary treatment systems were monitored. Results showed that these mills were not completely effective at eliminating the NP or NPEs. The concentrations of NP1,2EC in untreated textile mill effluents are very low. In most cases, the textile mills discharge into municipal treatment systems. Although these systems are very efficient at removing NPEs, significant amounts of NP and NPEs can potentially be released into the environment, even after treatment. The fate of NPE in textile mill effluents needs to be determined in both treatment systems and the environment, particularly at sites where there is little or no treatment.

3.2.6.2 Pulp and paper mill effluents

There has been a move within the pulp and paper industry to reduce or eliminate the use of NPEs from effluents, and there appears to have been a reduction in exposure over recent years. Pulp and paper mill effluents were, therefore, separated into samples collected prior to 1998 and those collected as part of a more recent study. Samples collected prior to 1998 commonly had detectable levels of NPEs and in one case had effluent concentrations in excess of 100 µg/L (Figure 26). Although concentrations of NPEs were low in samples collected after 1998 (Figure 27), there were only three sites sampled for these compounds, making it difficult to draw a conclusion. However, only a very few of the pulp mills studied had NP or NPEs in their effluents at concentrations high enough to be a concern for the environment.

3.2.6.3 Municipal treatment plant effluents

NP would not be expected to currently cause significant effects in the aquatic environment through exposure to treated effluents via water, although effluents from MWWTPs subject to primary treatment may be of some concern (Figure 10). However, NP is relatively insoluble in water and, therefore, bound to particles and sludges, which may result in exposure through alternative routes. Exposure of biota in sediment may occur at different locations compared with exposure of aquatic organisms (e.g., Galt–Grand River situation). NP was shown to represent a potential risk to sediment biota downstream of MWWTP outfalls.

Sludges that are applied to soils result in exposure of terrestrial biota. Considering the levels, which are only slightly above the ENEV, the rapid degradation in soil and low accumulation in plants, it is unlikely that land application of sludges under best management

practices is a major concern. However, there are very few data available, and the potential roles of NP1EO and NP2EO need to be considered in terms of both their toxicity and their potential to prolong the exposure to NP in soils.

The higher-chain-length NPEs degrade not only to NP, but to a variety of lower-chain-length NPEs and NPECs in municipal treatment systems. These compounds can occur at relatively high concentrations and represent a risk for both chronic toxicity and estrogenic effects in the aquatic environment. Depending on the inputs and the degree and type of treatment at the plant, NP1EO and NP2EO can be found at levels that are expected to cause chronic toxicity. This is based on an assumption of a dilution of 10:1, which may be an underestimate, especially for primary-treated effluents or discharges to marine environments. However, during low-flow periods, many treated effluents discharge into aquatic environments with dilutions considerably less than 10, even approaching a factor as low as 2. Although instantaneous dilution estimates may be much higher, many environments may have much lower dilutions for significant distances from the outfall because of entrapment or poor mixing of the effluent. The assessment of receiving waters, including a case study on the Grand River, confirms there is potential for significant exposure to both NP1EO and NP2EO considerable distances downstream of the outfall.

The NPECs are also formed as a result of the degradation of higher-chain-length NPEs and are considerably more water soluble. They can, therefore, increase in concentration during the course of treatment and can reach levels considerably higher than those of NP or NPEs in final effluent. Secondary- and tertiary-treated effluents can have relatively high concentrations of NP1EC and NP2EC, despite having extensive and efficient treatment systems. The toxicities of the NPECs are much lower than those of the corresponding NPEs, and, therefore, despite their higher concentrations, the NPECs do not represent a concern for chronic toxicity in municipal effluents (no municipal effluent had a conservative risk quotient of greater than one for NP1EC or NP2EC). In contrast, because the NPECs may be only slightly less estrogenic than NP, they could contribute to potential estrogenic responses, even in well-treated effluents. This is based on extrapolation of cell culture results, as there are few published *in vivo* data available on these compounds. Additionally, the significance of estrogenic responses at the whole-organism or population level has not been determined. Caution must, therefore, be used in interpreting the significance of these observations, and further work is needed to determine the significance for the Canadian environment.

3.2.7 Summary of risk characterization

The major route of entry of NP and NPEs into the Canadian environment is through discharge of effluents. The composition of the mixture can differ considerably among the various effluents, depending on the source and the degree and type of treatment. Textile mill effluents

represent a major source of release of NPEs to the environment. Untreated or partially treated textile mill effluents can have high NP9EO, NP1EO and NP2EO concentrations, which exceed the chronic effect level (ENEV). Although pulp and paper mill effluents currently have low levels of NP, there is evidence that, at least in the past, some sites had very high concentrations of NPEs in their final effluent. There appears to be a recent decrease in discharge of NPEs from pulp and paper mills, but there are very few data available with which to validate this conclusion (Figures 11 and 12). Municipal effluents are a significant source of NPEs and are widespread across the country. Untreated effluents can have high levels of NP, NP1EO and NP2EO, which may exceed thresholds for chronic effects (ENEV) in the aquatic environment, resulting in risk quotients exceeding one (Tables 16–18). Treated effluents have relatively low levels of high EO chain length NPEs. NP1EO and NP2EO may be present in final effluents at concentrations that may result in potential chronic toxicity. Although the dilution may be greater than predicted, it is likely that significant areas near outfalls are potentially impacted, and this is supported by measurement of NPEs in surface waters. Treated effluents can elevate the concentration of NPECs in final effluents, but, due to their low relative toxicity, NPECs are not expected to cause chronic toxicity in the environment. The potential for estrogenic responses in effluents is apparent, especially if the effects of the individual metabolites are considered to be additive (Figure 14). However, the significance of estrogenic responses is not fully understood. The concentration of NP is low in treated effluents, as it degrades and sorbs to sludge particles; however, the concentration of NP sorbed to sediments is expected to be higher. Despite a relatively low potential to bioaccumulate (Table 13), sediment-dwelling organisms may be exposed to NP directly, either through contact with water or sediment or through ingestion of sediment or food.

There is potential for chronic toxicity to occur in aquatic biota due to exposure to NPEs and their metabolites in a variety of effluents. This can be associated with different metabolites of NPEs, depending on the source and degree and type of treatment. It is important that all of the NPE metabolites, not only NP, be considered together to assess the potential for impacts in the environment.

Under current use patterns, NP/NPE releases in Canada can result in environmental concentrations that exceed the levels of concern. The significance of the potential effects of NP and NPEs and their widespread use and occurrence in effluents suggest that caution should be used with this group of compounds. Although other APEs, such as OPEs, have physical/chemical properties similar to those of NPEs, which make them attractive as replacements for NPEs, they also have similar toxicological properties and greater estrogenic properties. Therefore, the potential impact of replacement of NPEs with other APEs should be considered in risk management activities.

3.3 UNCERTAINTIES IN THE ENVIRONMENTAL RISK CHARACTERIZATION

There are a number of uncertainties associated with the environmental risk characterization that remain because of knowledge and data gaps in the current literature.

- The dilution of effluent by ambient river, harbour or lake water was assumed to be a factor of 10 and this is likely to vary considerably. Actual dilution factors may be much greater, or considerably less than 10 and most almost certainly vary with seasonal flow conditions. The dilution factor can play a large role in the calculation of risk quotients for NP and NPEs. However, it is believed that a dilution factor of 10 is an appropriate value to represent the areas near outfalls.

- This assessment compared the exposure levels with the best information available on chronic and acute toxicity, as well as the potential impacts of NP and NPEs on reproductive and developmental toxicity. However, much of the information, especially on the endocrine-mediated mechanisms is still evolving and much of it still needs to be validated. Endocrine-mediated mechanisms of toxicity were deemed to be important to consider, but the lack of confidence, and in some cases uncertainty on the interpretation of data, was seen as a limitation in applying this data to determine environmental no-effect concentrations. The ENEVs in the distributional approach, at least for NP, based on chronic toxicity, were considered adequate to protect against endocrine-mediated effects. The problems associated with determination of relative estrogenicity of the other metabolites, especially NP1,2EC, raise a number of uncertainties. Several key areas of research on the effects of NP and NPE are required, which may have significance for the evaluation of whether adverse effects are occurring through endocrine-mediated mechanisms.

- Because NP and NPEs occur together in effluents and in the environment, and because their mode of action is likely the same, their combined impact or potential impact is important in determining the risk associated with complex effluents or environmental samples. Although the assumption of additivity seems appropriate (for both chronic and endocrine mediated effects) it has not been validated for NP and NPEs.

- It is not known whether biological responses such as vitellogenin induction translate to an adverse effect in the organism or the population. However, vitellogenin induction and other endocrine-mediated responses do occur at concentrations that are likely to result in whole organism responses, such as intersex and impaired smoltification. They also occur in the range of LOECs reported for early life stage tests in fish.

- Some metabolites of NPEs (e.g. NP) tend to partition to sediment. However, the effect of NP on sediment-dwelling organisms is poorly characterized. For instance, few studies exist

on NP and no studies show the relative effect of NP on benthic organisms compared with the NPEs. Estimates of relative potency in sediment organisms were based on the relative potency of NP and NPEs in aquatic water column organisms.

- The lack of data on the concentration of NP and particularly NPEs in sediments associated with outfalls is a limitation of the current datasets. Fate, exposure and bioavailability of NP and NPEs associated with sediments are not well characterized, even though sediments could be a major route of exposure.

- Lack of data on the extent, persistence and effects of NP and NPEs in soils that receive sludge amendments is a limitation on evaluating the potential level of concern for this activity.

- NP and NPEs do not occur in the environment alone but usually in complex mixtures with other types of substances. The influence of these mixtures on the overall toxicity has not been well assessed. For example, it is known that MWWTP effluents in Canada and elsewhere contain natural and synthetic estrogens, which are expected to have similar estrogenic effects in the environment.

4.0 RESEARCH AND INFORMATION NEEDS

A number of research needs were identified during the development of the risk assessment for NP and NPEs. Addressing these knowledge or data gaps would reduce the uncertainties identified in this assessment and increase the understanding of NP/NPEs in the environment, which may be beneficial in risk management activities.

4.1 TREATABILITY AND DEGRADATION

An improved understanding of the treatment and persistence (in the natural environment) of NP/NPEs would be greatly beneficial. Specific areas of additional research include:

- determination of the degradation efficiency of NP/NPEs in MWWTPs that employ various treatment processes including the more advanced systems *e.g.*, ultraviolet oxidation, ozonation, adsorption "polishing", *etc.*
- study of the production, treatability, persistence and environmental significance of halogenated derivatives of NPE degradation products
- mass balance studies that take into account the relative importance of biodegradation, photodegradation, adsorption to suspended solids and bed sediment, formation of unextractable residues, aerobic and anaerobic conditions and temperature effects in aquatic

and terrestrial environments
- study of the atmospheric chemistry and fate of NPEs both in the atmosphere and terrestrial ecosystems because their use in aerially-applied pesticide formulations

4.2 FATE AND OCCURRENCE

Although there is a large amount of data available on the distribution of APs, there are clearly some knowledge gaps on the fate and occurrence of NPEs in the Canadian environment. These include:

- additional work to determine the presence of the short chain NPEs and NPECs in effluents (particularly from textile mills) and in harbour sediments
- additional study to determine the NP/NPE concentrations in pulp and paper mill effluents to confirm that levels have decreased recently
- additional study of the fate of NPEs in textile mill effluents and their receiving environments in treatment systems and the environment, particularly at sites where there is little or no treatment

4.3 BIOLOGICAL EFFECTS

The emphasis of biological effects studies has been on NP and longer chain length NPEs with relatively few quality data available on the chronic toxicity of lower chain length NPEs and NPECs. Additional research is needed in the following areas:

- studies comparing toxicity of APs, APEs and APECs using standardized methodology
- bioaccumulation estimated for NPEs and NPECs, OP and OPEs.
- measurement of the partitioning properties (*e.g.*, K_{oc}) and bioavailability of APs, APEs and APECs
- extensive study of the relative estrogenic potency of the APs and APEs using validated standards for testing and performing tests both *in vitro* and *in vivo* to aid in the elimination of debate resulting from the inconsistency in relative potency reported for E_2 receptor binding, YES assay and Vg induction in trout hepatocytes
- potential endocrine-mediated effects of APs and APEs needs to be studied for mechanisms other than estrogenicity
- validation that the assumption of additivity of AP, APE and APEC estrogenic responses in the environment is critical for interpretation of potential risk together in complex mixtures
- determination of the significance of estrogenic responses at the individual or population levels so that an assessment of the relative risk of this mode of action relative to other

endpoints would be possible

- validation of the predicted responses in aquatic field studies, especially at textile and municipal effluent sites
- study of the fate and effects of APs and APEs during sludge additions to the agricultural fields
- determination of the relative contribution of APs and APEs to the toxicity and/or estrogenicity of complex environmental mixtures and effluents

5.0 REFERENCES

Aboul-Kassim, T.A. and B.R.T. Simoneit. 1993. Detergents: a review of the nature, chemistry, and behavior in the aquatic environment. Part I. Chemical composition and analytical techniques. Crit. Rev. Environ. Sci. Technol. 23:325-376.

Abraham, E.J., and L.S. Frawley. 1997. Octylphenol (OP), an environmental estrogen, stimulates prolactin (PRL) gene expression. Life Sci. 60:1457-1465.

Abrutyn, D., B.E. McKenzie and N. Nadaskay. 1982. Teratology study of intravaginally administered nonoxynol-9-containing contraceptive cream in rats. Fertil. Steril. 37:113-117.

Ahel, M., T. Conrad and W. Giger. 1987. Persistent organic chemicals in sewage effluents. 3. Determinations of nonylphenoxy carboxylic acids by high-resolution gas chromatography/mass spectrometry and high-performance liquid chromatography. Environ. Sci. Technol. 21:697-703.

Ahel, M. and W. Giger. 1985a. Determination of alkylphenols and alkylphenol mono- and diethoxylates in environmental samples by high-performance liquid chromatography. Anal. Chem. 57:1577-1583.

Ahel, M. and W. Giger. 1985b. Determination of nonionic surfactants of the alkylphenol polyethoxylate type by high-performance liquid chromatography. Anal. Chem. 57:2584-2590.

Ahel, M. and W. Giger. 1993a. Aqueous solubility of alkylphenols and alkylphenol polyethoxylates. Chemosphere 26:1461-1470.

Ahel, M. and W. Giger. 1993b. Partitioning of alkylphenols and alkylphenol polyethoxylates between water and organic solvents. Chemosphere 26:1471-1478.

Ahel, M., W. Giger and M. Koch. 1994b. Behaviour of alkylphenol polyethoxylate surfactants in the aquatic environment - I. Occurrence and transformation in sewage treatment. Water Res. 28:1131-1142.

Ahel, M., W. Giger and C. Schaffner. 1994c. Behaviour of alkylphenol polyethoxylate surfactants in the aquatic environment - II. Occurrence and transformation in rivers. Water Res. 28:1143-1152.

Ahel, M., D. Hršak and W. Giger. 1994a. Aerobic transformation of short-chain alkylphenol polyethoxylates by mixed bacterial cultures. Arch. Environ. Contam. Toxicol. 26:540-548.

Ahel, M., J. McEvoy and W. Giger. 1993. Bioaccumulation of the lipophilic metabolites of

nonionic surfactants in freshwater organisms. Environ. Poll. 79:243-248.

Ahel, M., C. Schaffner and W. Giger. 1996. Behaviour of alkylphenol polyethoxylate surfactants in the aquatic environment - III. Occurrence and elimination of their persistent metabolites during infiltration of river water to groundwater. Water Res. 30:37-46.

Ahel, M., F.E. Scully, Jr., J. Hoigne and W. Giger. 1994d. Photochemical degradation of nonylphenol and nonylphenol polyethoxylates in natural waters. Chemosphere 28:1361-1368.

Allen, Y., A.P. Scott, P. Matthiessen, S. Haworth, J.E. Thain and S. Feist. 1998. A survey of estrogenic activity in UK estuarine and coastal waters and its effects on gonadal development of the flounder (*Platichthys flesus*). Environ. Toxicol. Chem., in press.

Amato, J.R. and D.D. Wayment. 1998. Surfactant toxicity identification with a municipal wastewater. *In* Environmental Toxicology and Risk Assessment: 7th Vol., ASTM STP 1333, pp. 272-283, E.E. Little, A.J. DeLonay and B.M. Greenberg, eds., American Society for Testing and Materials, West Conshohocken, PA, U.S.A. 19428-2959.

Analytical Bio Chemistry. 1984a. Dynamic 96-hour acute toxicity of octylphenol to fathead minnows (*Pimephales promelas*). Report No. 31910, Analytical Bio-Chemistry Laboratories, Columbia, MO, USA.

Analytical Bio Chemistry. 1984b. Dynamic 14-day acute toxicity of octylphenol to rainbow trout (*Salmo gairdneri*). Report No. 31911, Analytical Bio-Chemistry Laboratories, Columbia, MO, USA.

Analytical Bio Chemistry. 1984c. Dynamic 48-hour acute toxicity of octylphenol to *Daphnia magna*. Report No. 31912, Analytical Bio Chemistry Laboratories, Columbia, MO, USA.

Analytical Bio Chemistry. 1981. Acute toxicity of nonyl phenol to Fathead Minnows (*Pimephales promelas*). Report # 26822, Analytical Bio Chemistry Laboratories, Columbia, MO, USA.

Andersen, H.R., A-M. Andersson, S.F. Arnold, H. Autrup, M. Barfoed, N.A. Beresford, P. Bjerregaard, L.B. Christiansen, B. Gissel, R. Hummel, E.B. Jorgensen, B. Korsgaard, R.L. Guevel, H. Leffers, J. McLachlan, A. Moller, J.B. Nielsen, N. Olea, A. Oles-karasko, F. Pakdel, K.L. Pedersen, P. Perez, N.E. Skakkeboek, C. Sonnenschein, A.M. Soto, J.P. Sumpter, S.M. Thorpe and P. Grandjean. 1999. Comparison of short-term estrogenicity tests for identification of hormone disrupting chemicals. Environ. Health Perspect. 107:89-108.

Anghel, D.F., M. Balcan, A. Voicu and M. Elian. 1994. Analysis of alkylphenol-based non-ionic surfactants by high performance liquid chromatography. J. Chromatogr. A 668:375-383.

Ankley, G.T., G.S. Peterson, M.T. Lukasewycz and D.A. Jenner. 1990. Characteristics of

surfactants in toxicity identification evaluations. Chemosphere 21:3-12.

Anonymous. 1998. Chemical profile for nonylphenol. Chemical Market Reporter 254 (31), Sept. 28, 1998, p. 37.

Argese, E., A. Marcomini, P. Miana, C. Bettiol and G. Perin. 1994. Submitochondrial particle response to linear alkylbenzene sulfonate, nonylphenol polyethoxylates and their biodegradation derivatives. Environ. Toxicol. Chem. 13:737-742.

Armstrong, J.A., and P.D. Kingsbury. 1979. Interim Progress Report, Forest Pesticide Management Institute, Canadian Forestry Service, New Brunswick, Canada (as cited in Staples *et al.* (1998)).

Arukwe, A., L. Förlin and A. Goksøyr. 1997a. Xenobiotic and steroid biotransformation enzymes in Atlantic salmon (*Salmo salar*) liver treated with an estrogenic compound, 4-nonylphenol. Environ. Toxicol. Chem. 16:2576-2583.

Arukwe, A., F.R. Knudsen and A. Goksøyr. 1997b. Fish zona radiata (eggshell) protein: a sensitive biomarker for environmental estrogens. Environ. Health Perspect. 105:418-422.

Ashfield, L.A., T.G. Pottinger and J.P. Sumpter. 1998. Exposure of female juvenile rainbow trout to alkylphenolic compounds results in modification to growth and ovosomatic index. Environ. Toxicol. Chem. 17:679-686.

Australian and New Zealand Guidelines for Fresh and Marine Water Quality, July, 1999. Http://www.environment.gov.au/science/water/index.html.

Baldwin, W.S., S.E. Graham, D. Shea and G.A. LeBlanc. 1997. Metabolic and androgenization of female *Daphnia magna* by the xenoestrogen 4-nonylphenol. Environ. Toxicol. Chem. 16:1905-1911.

Ball, H.A. and M. Reinhard. 1985. Discharge of halogenated octylphenol polyethoxylate residues in a chlorinated secondary effluent. *In* Jolley, R. L. *et al.* (ed.), Water chlorination: chemistry, environmental impact and health effects, Vol. 5, pp. 1505-1514, Lewis Publishers, Chelsea, MI, U.S.A.

Ball, H.A., M. Reinhard and P.L. McCarty. 1989. Biotransformation of halogenated and nonhalogenated octylphenol polyethoxylate residues under aerobic and anaerobic conditions. Environ. Sci. Technol. 23:951-961.

Balson, T. and M.S.B. Felix. 1995. Biodegradability of non-ionic surfactants. In D.R. Karsa and M.R. Porter (eds.), Biodegradability of surfactants. Blackie Academic and Professional Publ., New York, NY, U.S.A., ISBN 0-7514-0206-0, pp. 204-230.

Barber, L.B., II, E.M. Thurman, M.P. Schroeder and D.R. LeBlanc. 1988. Long-term fate of organic micropollutants in sewage-contaminated groundwater. Environ. Sci. Technol. 22:205-211.

Beak Consultants Ltd. 1987. Development of methods to assess the fate and impacts of nonionic surfactants in the aquatic environment. Report IP-73 for Industrial Programs Branch, Environment Canada, Ottawa, Ontario K1A 0H3.

Bennett, E.R. and C.D. Metcalfe. 1998. Distribution of alkylphenol compounds in Great Lakes sediments, United States and Canada. Environ. Toxicol. Chem. 17:1230-1235.

Bennie, D.T. 1999. Review of the environmental occurrence of alkylphenols and alkylphenol ethoxylates. Water Qual. Res. J. Canada. 34:79-122.

Bennie, D.T. 1998. Personal communication. National Water Research Institute, Environment Canada, Burlington, Ontario L7R 4A6. (Data presented in Appendix A1, A3, A5, A7 and A9).

Bennie, D.T., C.A. Sullivan, H.-B. Lee, T.E. Peart and R.J. Maguire. 1997a. Occurrence of alkylphenols and alkylphenol mono- and diethoxylates in natural waters of the Laurentian Great Lakes basin and the upper St. Lawrence River. Sci. Total Environ. 193:263-275.

Bennie, D.T., C.A. Sullivan, H.-B. Lee and R.J. Maguire. 1997b. Alkylphenol polyethoxylate metabolites in Canadian sewage treatment plant waste streams. Environment Canada, National Water Research Institute Contribution No. 97-219, Burlington, Ontario L7R 4A6.

Bennie, D.T., Sullivan, C.A., Lee, H.-B., and Maguire, R.J. 1998. Alkylphenol polyethoxylate metabolites in Canadian sewage treatment plant effluent streams. Water Qual. Res. J. Can. 33:231-252.

Bent, K.J., P.D. Scott and J.A.W. Turner. 1977. Control of apple powdery mildew by dormant-season sprays: prospects for practical use. Proc. 1977 British Crop Protection Conference - Pests and Diseases, Brighton, England, Nov. 21-24, 1977, publ. British Crop Protection Council, London, England, pp. 331-339.

Bergström, B. 1984. Unpublished data cited in Wahlberg *et al.* (1990).

Bhatt, B.D., J.V. Prasad, G. Kalpana and S. Ali. 1992. Separation and characterization of isomers of *p*-nonylphenols by capillary GC/GC-MS/GC-FTIR techniques. J. Chromatogr. Sci. 30:203-210.

Bidleman, T.F. and L. Renberg. 1985. Determination of vapor pressures for chloroguaiacols, chloroveratroles, and nonylphenol by gas chromatography. Chemosphere 14:1475-1481.

Billard, R., B. Breton and M. Richard. 1981. On the inhibitory effect of some steroids on spermatogenesis in adult rainbow trout (*Salmo gairdneri*). Can. J. Zool. 59:1479-1487.

Birch, R.R. 1991. Prediction of the fate of detergent chemicals during sewage treatment. J. Chem. Tech. Biotechnol. 50:411-422.

Blackburn, M.A. and M.J. Waldock. 1995. Concentrations of alkylphenols in rivers and

estuaries in England and Wales. Water Res. 29:1623-1629.

Blazquez, M., S. Zanuy, M. Carrillo and F. Piferrer. 1998. Persistent effects of early exposure to estrogens estradiol-17b and 17a-ethynylestradiol on the gonads of the teleost *Dicentrarchus labrax*. Gen. Comp. Endocrinol., in press.

Bokern, M. and H.H. Harms. 1997. Toxicity and metabolism of 4-*n*-nonylphenol in cell suspension cultures of different species. Environ. Sci. Technol. 31:1849-1854.

Bokern, M., M. Nimtz and H.H. Harms. 1996. Metabolites of 4-*n*-nonylphenol in wheat cell suspension cultures. J. Agric. Food Chem. 44:1123-1127.

Bokern, M., P. Raid and H.H. Harms. 1998. Toxicity, uptake and metabolism of 4-*n*-nonylphenol in root cultures and intact plants under septic and aseptic conditions. Environ. Sci. Pollut. Res. 5:21-27.

Boyd-Boland, A.A. and J.B. Pawliszyn. 1996. Solid-phase microextraction coupled with high-performance liquid chromatography for the determination of alkylphenol ethoxylate surfactants in water. Anal. Chem. 68:1521-1529.

Brand, N., G. Mailhot and M. Bolte. 1998. Degradation photoinduced by Fe(III): method of alkylphenol ethoxylates removal in water. Environ. Sci. Technol. 32:2715-2720.

Brewer, R., Sekela, M., Moyle, G., Baldazzi, C. and Tuominen, T. 1998. Contaminants in bed sediments from 14 reaches of the Fraser River basin. Report of the Aquatic and Atmospheric Sciences Division, Environmental Conservation Branch, Pacific and Yukon Region, Environment Canada, Vancouver, B.C.

Bringmann, G., and R. Kühn. 1982. Results of toxic action of water pollutants on *Daphnia magna* Straus tested by an improved standardized procedure. Z. Wasser Abwasser Forsch. 15:1-6.

Brooke, L.T. 1993a. Accumulation and lethality for two freshwater fishes (fatehead minnow and bluegill) to nonylphenol. Report to the U.S. Environmental Protection Agency, Duluth, MN (contract No. 68-C1-0034). Lake Superior Research Institute, University of Wisconsin-Superior, Superior, WI, U.S.A.

Brooke, L.T. 1993b. Acute and chronic toxicity of nonylphenol to ten species of aquatic organisms. Report to the U.S. Environmental Protection Agency, Duluth, MN (contract No. 68-C1-0034). Lake Superior Research Institute, University of Wisconsin-Superior, Superior, WI, U.S.A.

Brown, D., H. de Henau, J.T. Garrigan, P. Gerike, M. Holt, E. Keck, E. Kunkel, E. Matthijs, J. Waters and R.J. Watkinson. 1986. Removal of nonionics in sewage treatment plants. Removal of domestic detergent nonionic surfactants in an activated sludge sewage treatment plant. Tenside Detergents 23:190-195.

Brown, D., H. de Henau, J.T. Garrigan, P. Gerike, M. Holt, E. Kunkel, E. Matthijs, J. Waters and R.J. Watkinson. 1987. Removal of nonionics in sewage treatment plants. II. Removal of domestic detergent nonionic surfactants in a trickling filter sewage treatment plant. Tenside Surfactants Detergents 24:14-19.

Brown, S.B., K. Haya, L.E. Burridge, E.O. Swansburg, J.T.Arsenault, J. Sherry, D. Bennie, J.G. Eales and W. L. Fairchild. 1998. Effects of water-borne 4-nonylphenol on Atlantic salmon (*Salmo salar)* smolts. Poster presented at 25th Annual Toxicity Workshop, Quebec City, QC, October 18-21, 1998.

Brunner, P.H., S. Capri, A. Marcomini and W. Giger W. 1988. Occurrence and behaviour of linear alkylbenzenesulphonates, nonylphenol, nonylphenol mono- and nonylphenol diethoxylates in sewage and sewage sludge treatment. Water Res. 22:1465-1472.

Bulkley, R.V. 1972. Diethylstilbestrol in catfish feed. Trans. Amer. Fisher. Soc. 101:537-599.

Burlington Research Inc. 1985. Acute and chronic bioassays of industrial surfactants. Final Report, Burlington, NC, USA.

Burnison, B.K. 1998. Personal communication, National Water Research Institute, Environment Canada, Burlington, Ontario L7R 4A6 Canada.

Bye, V.J., and R.F. Lincoln. 1986. Commercial methods for the control of sexual maturation rainbow trout (*Salmo gairdneri* R.). Aquacult. 57:299-309.

Cady, C. 1996. Activated sludge biodegradation test with ^{14}C-nonylphenol ethoxylate. Final report No. 42221 to Chemical Manufacturers Association. Analytical Bio Chemistry Laboratories, Inc., 7200 East ABC Lane, Columbia, MO 65202, U.S.A., 195 pp.

Cahn, A. and J.L. Lynn, Jr. 1983. Surfactants and detersive systems. *In* Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed., Vol. 22, ISBN 0-471-02075-3, John Wiley and Sons, Inc., New York, NY, U.S.A., pp. 332-432.

Calamari, D., and R. Marchetti. 1973. The toxicity of metals and surfactants to rainbow trout (*Salmo gairdneri* Rich.). Water Res. 7:1453-1464.

Camford Information Services Inc. 1990. CPI product profile - nonylphenol. 801 York Mills Road, Suite 201, Don Mills, Ontario M3B 1X7, 3 pp.

Carey, J.H., M.E. Fox, B.G. Brownlee, J.L. Metcalfe, R.J. Tkacz, L. Coletta and G. MacInnis. 1981. Occurrence of nonyl phenol surface slicks in Canagagigue Creek - 1981. National Water Research Institute Report, Environment Canada, Burlington, Ontario L7R 4A6 Canada.

Chalaux, N., J.M. Bayona and J. Albaiges. 1994. Determination of nonylphenols as pentafluorobenzyl derivatives by capillary gas chromatography with electron-capture and mass spectrometric detection in environmental matrices. J. Chromatogr. A 686:275-281.

Chee, K.K., M.K. Wong and H.K. Lee. 1996. Determination of 4-nonylphenol - Part 1: optimization of the gas chromatographic determination of 4-nonylphenol by orthogonal array design and electronic pressure programming. J. Microcolumn Separ. 8:29-36.

Chen, E.C.. 1989. Environmental assessment of the Canadian textile industry. Chemical Industries Division, Industrial Programs Branch, Environmental Protection Service Report EPS 5/TX/1, Department of the Environment, Ottawa, Ontario K1A 0H3, ISBN 0-662-16883-6, 104+ pp.

Chen, T.T. 1983. Identification and characterization of estrogen responsive gene products in the liver of the rainbow trout. Can. J. Biochem. Cell Biol. 61:802-810.

Christiansen, T., B. Korsgaard and A. Jespersen. 1998. Effects of nonylphenol and 17β-estradiol on vitellogenin synthesis, testicular structure and cytology in male eelpout (*Zoarces viviparus*). J. Exper. Biol. 201:179-192.

CITI. 1992. Biodegradation and bioaccumulation data of existing chemicals based on the CSCL Japan, Chemical Inspection and Testing Institute, Japan Chemical Industry Ecology-Toxicology and Information Center, Tokyo, Japan.

Clark, L.B., R.T. Rosen, T.G. Hartman, L.H. Alaimo, J.B. Louis, C. Hertz, C.-T. Ho and J.D. Rosen. 1991. Determination of non-regulated pollutants in three New Jersey publicly owned treatment works (POTWs). Res. J. Water Pollut. Contr. Fed. 63:104-113.

Clark, L.B., R.T. Rosen, T.G. Hartman, J.B. Louis, I.H. Suffet, R.L. Lippincott and J.D. Rosen. 1992. Determination of alkylphenol ethoxylates and their acetic acid derivatives in drinking water by particle beam liquid chromatography/mass spectrometry. Intern. J. Environ. Anal. Chem. 47:167-180.

Coldham, N.G., S. Sivapathasundaram, M. Dave, L.A. Ashfield, T.G. Pottinger, C. Goodall and M.J. Sauer. 1998. Biotransformation, tissue distribution, and persistence of 4-nonylphenol residues in juvenile rainbow trout (*Oncorhynchus mykiss*). Drug Metabol. Dispos. 26:347-354.

Colerangle, J.B. and D. Roy. 1996. Exposure of environmental estrogenic compound nonylphenol to Noble rats alters cell-cycle kinetics in the mammary gland. Endocrine 4:115-122.

Collyard, S.A., G.T. Ankley, R.A. Hoke and T. Golenstein. 1994. Influence of age on the relative sensitivity of *Hyalella azteca* to diazinon, alkylphenol ethoxylates, copper, cadmium and zinc. Arch. Environ. Contam. Toxicol. 26:110-113.

Comber, M.H.I., T.D. Williams and K.M. Stewart. 1993. The effects of nonylphenol on *Daphnia magna*. Water Res. 27:273-276.

Corti, A.., S. Frassinetti, G. Vallini, S. D'Antone, C. Fichi and R. Solaro. 1995. Biodegradation of nonionic surfactants. I. Biotransformation of 4-(1-nonyl)phenol by a *Candida maltosa*

isolate. Environ. Pollut. 90:83-87.

Crathorne, B., M. Fielding, C.P. Steel and C.D. Watts. 1984. Organic compounds in water: analysis using coupled-column high performance liquid chromatography and soft-ionization mass spectrometry. Environ. Sci. Technol. 18:792-802.

Crescenzi, C., A. Di Corcia, A. Marcomini and R. Samperi. 1997. Detection of poly(ethylene glycols) and related acidic forms in environmental waters by liquid chromatography/electrospray/mass spectrometry. Environ. Sci. Technol. 31:2679-2685.

Crescenzi, C., A. Di Corcia, R. Samperi and A. Marcomini. 1995. Determination of nonionic polyethoxylate surfactants in environmental waters by liquid chromatography/electrospray mass spectrometry. Anal. Chem. 67:1797-1804.

Cripe, G.M., A. Ingley-Guezou, L.R. Goodman and J. Forester. 1989. Effect of food availability on the acute toxicity of four chemicals to *Mysidopsis bahia* (Mysidacea) in static exposures. Environ. Toxicol. Chem. 8:333-338.

Cserhati, T., Z. Illes and I. Nemes. 1991. Effect of non-ionic tensides on the growth of some soil bacteria. Appl. Micro. Biotech. 35:115-118.

Dachs, J., D.A. Van Ry and S. J. Eisenreich. 1999. Occurrence of estrogenic nonylphenols in the urban and coastal atmosphere of the lower Hudson river estuary. Environmental Science & Technology, 39 (15): 2676-2679.

Danzo, B.J. 1997. Environmental xenobiotics may disrupt normal endocrine function by interfering with the binding of physiological ligands to steroid receptors and binding proteins. Environ. Health Perspect. 105:294-301.

Davis, E.M. and E.F. Gloyna. 1967. Biodegradability of nonionic and anionic surfactant by blue-green algae. Report of the Environmental Health Engineering Research Laboratory, University of Texas, Centre for Research in Water Resources (cited in Talmage, 1994).

Davis, E.M. and E.F. Gloyna. 1969. The role of algae in degrading detergent surface active agents. J. Water Pollut. Contr. Fed. 41:1494-1504.

Desbène, P.L. and B. Desmazieres. 1994. Analytical study of polyoxyethylene surfactants of high degree of condensation by normal-phase liquid chromatography on *p*-nitrophenyl-bonded silica. J. Chromatogr. A 661:207-213.

Desbrow, C., E. Routledge, G.C. Brighty, J.P. Sumpter, and M. Waldock. 1998. Identification of estrogenic chemicals in STP effluent. 1. Chemical fractionation and *in vitro* biological screening. Environ. Sci. Technol. 32:1549-1558.

Desbrow, C., E. Routledge, D. Sheehan, M. Waldock and J. Sumpter. 1996. The identification and assessment of oestrogenic substances in sewage treatment works effluents. MAFF Fisheries Laboratory and Brunel University report P2 - i490/7.

Dickey, P. 1997. Troubling bubbles - the case for replacing alkylphenol ethoxylate surfactants. A report of the Washington Toxics Coalition, 4516 University Way N.E., Seattle, WA, U.S.A. 98105, 88 pp.

Di Corcia, A., A. Costantino, C. Crescenzi, E. Marinoni and R. Samperi. 1998. Characterization of recalcitrant intermediates from biotransformation of the branched alkyl side chain of nonylphenol ethoxylate surfactants. Environ. Sci. Technol. 32:2401-2409.

Di Corcia, A., R. Samperi and A. Marcomini. 1994. Monitoring aromatic surfactants and their biodegradation intermediates in raw and treated sewages by solid-phase extraction and liquid chromatography. Environ. Sci. Technol. 28:850-858.

Ding, W.-H., Y. Fujita, R. Aeschimann and M. Reinhard M. 1996. Identification of organic residues in tertiary effluents by GC/EI-MS, GC/CI-MS and GC-TSQ-MS. Fresenius J. Anal. Chem. 354:48-55.

Ding, W.-H. and S.-H. Tzing. 1998. Analysis of nonylphenol polyethoxylates and their degradation products in river water and sewage effluent by gas chromatography - ion trap (tandem) mass spectrometry with electron impact and chemical ionization. J. Chromatogr. A 824:79-90.

Dorn, P.B., J.P. Salanitro, S.H. Evans and L. Kravetz. 1993. Assessing the aquatic hazard of some branched and linear nonionic surfactants by biodegradation and toxicity. Environ. Toxicol. Chem. 12:1751-1762.

Dwyer, F.J., L.C. Sappington, D.R. Buckler and S.B. Jones. 1995. Use of surrogate species in assessing contaminant risk to endangered and threatened fishes. EPA/600/R96/029. Final report, U.S. Environmental Protection Agency, Gulf Breeze, FL, U.S.A.

Ekelund, R., Å. Bergman, Å. Granmo and M. Berggren. 1990. Bioaccumulation of 4-nonylphenol in marine animals - a re-evaluation. Environ. Pollut. 64:107-120

Ekelund, R., Å. Granmo, K. Magnusson, M. Berggren and Å. Bergman. 1993. Biodegradation of 4-nonylphenol in seawater and sediment. Environ. Pollut. 79:59-61.

England, D.C. and J, Downing. 1995. Chronic toxicity of NPE 1.5 to *Ceriodaphnia dubia*. Analytical Bio Chemistry Laboratories. Final report (No. 41766) to the Chemical Manufacturers Association, Washington, DC, USA.

England, D.C. 1995. Chronic toxicity of nonylphenol to *Ceriodaphnia dubia*. Prepared for the Chemical Manufacturers Association by Analytical Bio Chemistry Laboratories Inc., Report No. 41756.

England, D.C. and J.B. Bussard. 1993. Toxicity of nonylphenol to the midge *Chironomus tentans*. Prepared for the Chemical Manufacturers Association by Analytical Bio Chemistry Laboratories Inc., Report No. 40597.

England, D.C., and J.B. Bussard. 1994. Toxicity of nonylphenol to the amphipod *Hyalella azteca* (Saussure). Prepared for the Chemical Manufacturers Association by Analytical Bio Chemistry Laboratories Inc., Report No. 41569.

Environment Canada 1997a. Environmental assessments of priority substances under the Canadian Environmental Protection Act. Guidance manual version 1.0. Environmental Protection Series Report EPS/2/CC/3E, Chemicals Evaluation Division, Commercial Chemicals Evaluation Branch, Environment Canada, Ottawa, Ontario K1A 0H3, ISBN 0-662-25403-1.

Environment Canada 1997b. Notice Respecting the second Priority Substances List and di(2-ethylhexyl) phthalate. Issued under authority of Section 16 of the Canadian Environmental Protection Act. Canada Gazette, Part 1. February 15, 1997, Vol. 131, No. 7, pp. 366-368, Ottawa, Ontario, Canada.

Environment Canada. 1998. Priority Substances List 2 Technical Report for Nonylphenol and its Ethoxylates (1995-1996 data). Protected Report. Use Patterns Section, Chemicals Control Division, Commercial Chemicals Evaluation Branch. July 1998.

Ernst, B., G. Julien, K. Doe and R. Parker. 1980. Environmental investigations of the 1980 spruce budworm spray program in New Brunswick. Environment Canada, Environmental Protection Service, Atlantic Region report EPS-5-AR-81-3, Halifax, Nova Scotia, 46+ pp.

Fairchild, W.L., E.O. Swansburg, J.T. Arenaut and S.B. Brown. 1999. Does an association between pesticide use and subsequent declines in catch of Atlantic Salmon *(Salmo solar)* represent a case of endocrine disruption? Environ. Health Perspect. 107:349-357.

Fent, K., G.E. Ackermann and J. Schwaiger. 1999. Analysis of vitellogenin mRNA by quantitative reverse transcription polymerase chain reaction (RT-PCR) in juvenile fish exposed for twelve months to nonylphenol. Tenth International Symposium on Pollutant Responses in Marine Organisms (PRIMO 10), Williamsburg, Virginia, April 25-29, 1999.

Field, J.A., D.J. Miller, T.M. Field, S.B. Hawthorne and W. Giger. 1992. Quantitative determination of sulfonated aliphatic and aromatic surfactants in sewage sludge by ion-pair/supercritical fluid extraction and derivatization gas chromatography/mass spectrometry. Anal. Chem. 64:3161-3167.

Field, J.A. and R.L. Reed. 1996. Nonylphenol polyethoxy carboxylate metabolites of nonionic surfactants in U.S. paper mill effluents, sewage treatment plant effluents, and river waters. Environ. Sci. Technol. 30:3544-3550.

Fischer, 1993. Personal communication. Cited in Talmage as per Gloxhuber, 1974.

Folmar, L.C., N.D. Denslow, V. Rao, M. Chow, D.A. Crain, J. Enblom, J. Marcino and L.J. Guillette, Jr. 1996. Vitellogenin induction and reduced serum testosterone concentrations in feral male carp (*Cyprinus carpio*) captured near a major metropolitan sewage treatment

plant. Environ. Health Perspect. 104:1096-1101.

Forbis, A.D. 1988. Chronic toxicity of octylphenol [4-(1,1,3,3-tetramethylbutyl) phenol] to *Daphnia magna* under flow-through test conditions. Report No. 36195, Analytical Bio Chemistry Laboratories, Columbia, MO, USA.

Forbis, A.D., G. Lori and D. Burgess. 1984. Acute toxicity of octylphenol to *Selenastrum capricornutum* Printz. Analytical Bio Chemistry Laboratories. Final report to the Chemical Manufacturers Association, Washington, DC, USA.

Frassinetti, S., A. Isoppo, A. Corti and G. Vallini. 1996. Bacterial attack of non-ionic aromatic surfactants: comparison of degradative capabilities of new isolates from nonylphenol polyethoxylate polluted wastewaters. Environ. Technol. 17:199-205.

Fujita, Y. and M. Reinhard. 1997. Identification of metabolites from the biological transformation of the nonionic surfactant residue octylphenoxyacetic acid and its brominated analog. Environ. Sci. Technol. 31:1518-1524.

Fytianos, K., S. Pegiadou, N. Raikos, I. Eleftheriadis and H. Tsoukali. 1997. Determination of non-ionic surfactants (polyethoxylated nonylphenols) by HPLC in waste waters. Chemosphere 35:1423-1429.

Giger, W. and M. Ahel. 1991. Behaviour of nonylphenol polyethoxylates and their metabolites in mechanical-biological sewage treatment. *In* Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP) (1991). Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, pp. 87-103.

Giger, W., M. Ahel and M. Koch. 1986. Behaviour of alkylphenol polyethoxylate surfactants in mechanical-biological sewage treatment. Vom Wasser 67:69-81.

Giger, W., M. Ahel, M. Koch, H.U. Laubscher, C. Schaffner and J. Schneider. 1987. Behaviour of alkylphenol polyethoxylate surfactants and of nitrilotriacetate in sewage treatment. Water Sci. Technol. 19:449-460.

Giger, W., P.H. Brunner and C. Schaffner. 1984. 4-Nonylphenol in sewage sludge: accumulation of toxic metabolites from nonionic surfactants. Science 225:623-625.

Giger, W., E. Stephanou and C. Schaffner. 1981. Persistent organic chemicals in sewage effluents: I. Identifications of nonylphenols and nonylphenolethoxylates by glass capillary column gas chromatography/mass spectrometry. Chemosphere 10:1253-1263.

Gilbert, J., M.J. Shepherd, J.R. Startin and M.A. Wallwork. 1982. Identification by gas chromatography-mass spectrometry of vinyl chloride oligomers and other low-molecular-weight components in poly(vinyl chloride) resins for food packaging applications. J. Chromatogr. 237:249-261.

Gimeno, S., H. Komen, W. Paul, M. Venderbosh and T. Bowmer. 1997. Distribution of sexual

differentiation in genetic male common carp (*Cyprinus carpio*) exposed to an alkylphenol during different life stages. Environ. Sci. Technol. 31:2884-2890.

Granmo, Å., R. Ekelund, M. Berggren and K. Magnusson. 1991. Toxicity of 4-nonylphenol to aquatic organisms and potential for bioaccumulation. *In* Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP) (1991). Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, pp. 53-75.

Granmo, Å., R. Ekelund, K. Magnusson and M. Berggren. 1989. Lethal and sublethal toxicity of 4-nonylphenol to the common mussel (*Mytilus edulis* L.). Environ. Poll. 59:115-127.

Granmo, Å. and S. Kollberg. 1976. Uptake pathways and elimination of a nonionic surfactant in cod (*Gadus morrhua* L.). Water Res. 10:189-194.

Gray, M.A. and C.D. Metcalfe. 1997. Induction of testes-ova in Japanese medaka (*Oryzias latipes*) exposed to *p*-nonylphenol. Environ. Toxicol. Chem. 16:1082-1086.

Guardiola, A., Ventura, F., Matia, L., Caixach, J. and Rivera, J. 1991. Gas chromatographic-mass spectrometric characterization of volatile organic compounds in Barcelona tap water. J. Chromatogr. 562:481-492.

Hall, W.S., J.B. Patoczka, R.J. Mirenda, B.A. Porter and E. Miller. 1989. Acute toxicity of industrial surfactants to *Mysidopsis bahia*. Arch. Environ. Contam. Toxicol. 18:765-772.

Häni, H. 1990. The analysis of inorganic and organic pollutants in soil with special regard to their bioavailability. Intern. J. Environ. Anal. Chem. 39:197-208.

Harries, J.E., D.A. Sheahan, S. Jobling, P. Matthiessen, P. Neall, E.J. Routledge, R. Rycroft, J.P. Sumpter and T. Tylor. 1997. A survey of estrogenic activity in U.K. inland waters. Environ. Toxicol. Chem. 16:534-542.

Haux, C. and B. Norberg. 1985. Effect of sexual maturation and estradiol-17β on liver content of protein, lipids, glycogen and nucleic acids in juvenile rainbow trout, *Salmo gairdneri*. Comp. Biochem. Physiol. B 81:275-279.

He, Y. and H.K. Lee. 1996. Separation of structural homologues of alkylphenols and isomers of 4-nonylphenol by cyclodextrin-modified micellar electrokinetic chromatography. J. Chromatogr. A 749:227-236.

Heinig, K., C. Vogt and G. Werner. 1996. Separation of ionic and neutral surfactants by capillary electrophoresis and high performance liquid chromatography. J. Chromatogr. A 745:281-292.

Heinis, L.J., M.L. Knuth, K. Liber, B.R. Sheedy, R. Tunell and G.T. Ankley. 1999. Persistence and distribution of 4-nonylphenol following repeated application to littoral enclosures. Environ. Toxicol. Chem. 18:363-375.

Hellyer, Y.P. 1991. EPA's chemical testing program. Nonylphenols in general, 4-nonylphenol

in particular. *In* Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP) (1991). Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, pp. 13-21.

Hewitt, M.L., L. Tremblay, G.J. Van Der Kraak, K.R. Solomon and M.R. Servos. 1998. Identification of the lampricide 3-trifluoromethyl-4-nitrophenol (TFM) as an agonist for the rainbow trout estrogen receptor. Environ. Toxicol. Chem. 17:425-432.

Hoglund, L.B. 1976. Avoidance reactions of the cod (*Gadus morrhua*) to certain surfactants. Food and Agriculture Organization (Rome) TF-TNT 173 Supplemental 1:132-154.

Holcombe, G.W., G.L. Phipps, M.L. Knuth and T. Felhaber. 1984. The acute toxicity of selected substituted phenols, benzenes and benzoic acid esters to fathead minnows *Pimephales promelas*. Environ. Poll. Ser. A Ecol. Biol. 35:367-381.

Holmes, S.B. and P.D. Kingsbury. 1980. The environmental impact of nonyl phenol and the Matacil formulation. Part I: aquatic ecosystems. Environment Canada, Canadian Forestry Service Report FPM-X-35, 52+ pp.

Holt, M.S., E.H. McKerrell, J. Perry and R.J. Watkinson. 1986. Determination of alkylphenol ethoxylates in environmental samples by high-performance liquid chromatography coupled to fluorescence detection. J. Chromatogr. 362:419-424.

Holt, M.S., G.C. Mitchell and R.J. Watkinson. 1992. The environmental chemistry, fate and effects of nonionic surfactants. *In* Hutzinger, O. (ed.), Handbook of environmental chemistry, Vol. 3, Part F, Anthropogenic compounds. Detergents. Springer-Verlag, Berlin, ISBN 3-540-53797-X, pp. 89-144.

Howard, P.H. 1989. Handbook of environmental fate and exposure data for organic chemicals. Vol. I. Large production and priority pollutants. Lewis Publ., Chelsea, MI 48118, U.S.A.

Hughes, A.I., J. Fisher and E. Brumbaugh. 1996. Biodegradation of NPE in soil. *In* Proc. 4th Surfactants Conf., Barcelona, Spain, June 3-7, 1996. Publ. Royal Society of Chemistry, Cambridge, UK, Vol. 4, pp. 365-372 (ISBN 0-85404-766-2), pp. 365-372.

Hüls, AG. 1987. Acute toxicity of nonylphenol to *Leuciscus idus*. Final study report. Marl, Germany.

Hüls, AG. 1992. Acute and chronic toxicity of nonylphenol to *Daphnia magna*. Final study report numbers DK-522, DL-143, Marl, Germany.

Hüls, AG. 1994. Product information sheet revision - nonylphenol (1/8/94). Marl, Germany.

Hüls, AG. 1996. Determination of the effects of nonylphenol on the growth of *Scenedesmus subspicatus*. 86.91.SAG (algal growth inhibition test according to UBA Feb. 1984). Report AW-185 (as cited in OECD, 1997).

Hunter, G.A., and E.M. Donaldson. 1983. Hormonal sex control and its application to fish

culture. *In* W.S. Hoar, D.J. Randall and E.M. Donaldson (eds.), Fish Physiology, Academic Press, New York, NY, USA. pp. 223-303.

Hyllner, S.J., D.O. Oppen-Bernsten, J.V. Helvik, B.T. Walther and C. Haux. 1991. Oestradiol-17β induces the major vitelline envelope proteins in both sexes in teleosts. J. Endocrinol. 131:229-236.

Ibrahim, N.M.A. and B.B. Wheals. 1996a. Determination of alkylphenol ethoxylate non-ionic surfactants in trade effluents by sublation and high performance liquid chromatography. Analyst 121:239-242.

Ibrahim, N.M.A. and B.B. Wheals. 1996b. Oligomeric separation of alkylphenol ethoxylate surfactants on silica using aqueous acetonitrile eluents. J. Chromatogr. A 731:171-177.

IEC International Environmental Consultants Ltd. 1982. Survey of textile wet processing and pollution abatement technology. Report No. EPS 3-WP-82-5, November 1982, Water Pollution Control Directorate, Environmental Protection Service, Environment Canada, ISBN 0-662-12270-4, Ottawa, Ontario K1A 0H3, 116+ pp.

Jandera, P., J. Urbánek, B. Prokeš and J. Churácek. 1990. Comparison of various stationary phases for normal-phase high performance liquid chromatography of ethoxylated alkylphenols. J. Chromatogr. 504:297-318.

Janicke, W., G. Bringmann and R. Kühn. 1969. Aquatic toxicology studies of the harmful action of ethoxylate nonionic surfactants. Gesundheitsingenieure 90:133-138.

Jobling, S. D. Sheahan, J.A. Osbourne, P. Matthiessen and J.P. Sumpter. 1996. Inhibition of testicular growth in rainbow trout (*Oncorhynchus mykiss*) exposed to estrogenic alkylphenolic chemicals. Environ. Toxicol. Chem. 15:194-202.

Jobling, S. and J.P. Sumpter. 1993. Detergent components in sewage effluent are weakly oestrogenic to fish: an *in vitro* study using rainbow trout (*Oncorhynchus mykiss*) hepatocytes. Aquat. Toxicol. 27:361-372.

Jobst, H. 1995. Chlorophenols and nonylphenols in sewage sludges. Part I: Occurrence in sewage sludges of Western Germany treatment plants from 1987 to 1989. Acta Hydrochim. Hydrobiol. 23:20-25.

Johnstone, R., T.H. Simpson and A.F. Youngson. 1978. Sex reversal in salmonid culture. Aquaculture 13:115-134.

Jones, F.W. and D.J. Westmoreland. 1998. Degradation of nonylphenol ethoxylates during the composting of sludges from wool scour effluents. Environ. Sci. Technol. 32:2623-2627.

Jones, P. and G. Nickless. 1978a. Characterization of non-ionic detergents of the polyethoxylated type from water systems. I. Evaluation of Amberlite XAD-4 resin as an extractant for polyethoxylated material. J. Chromatogr. 156:87-97.

Jones, P. and G. Nickless. 1978b. Characterization of non-ionic detergents of the polyethoxylated type from water systems. II. Isolation and examination of polyethoxylated material before and after passage through a sewage plant. J. Chromatogr. 156:99-110.

Jungclaus, G.A., V. Lopez-Avila and R.A. Hites. 1978. Organic compounds in an industrial wastewater: a case study of their environmental impact. Environ. Sci. Technol. 12:88-96.

Junk, G.A., H.J. Svec, R.D. Vick and M.J. Avery. 1974. Contamination of water by synthetic polymer tubes. Environ. Sci. Technol. 8:1100-1106.

Kahl, M.D., G.T. Ankley, P.A. Kosian and E.A. Makynen. 1997. Toxicity of *p*-nonylphenol in a life-cycle test with the freshwater macroinvertebrate, *Chironomus tentans*. Presented at the 17th Annual Meeting of the Society of Environmental Toxicology and Chemistry, Washington, DC, USA.

Kibbey, T.C.G., T.P. Yavaraski and K.F. Hayes. 1996. High performance liquid chromatographic analysis of polydisperse ethoxylated non-ionic surfactants in aqueous samples. J. Chromatogr. A 752:155-165.

Klugland, H., O. Anderson, G. Kisen, P. Alestrom and L. Tora. 1993. Estrogen-receptor binds to the salmon GnRH gene in a region with long palindromic sequences. Molec. Cell. Endocrinol. 95:147-154.

Knie, V.J., A. Halke, J. Juhnke and W. Schiller. 1983. Results of the studies of chemicals in four bioassays. Deutsche Gewaesserkundliche Mitteilungen 27:77-79.

Kopf, W. 1997. Wirkung endokriner stoffe in biotests mit wasserorganismen. In: Stoffe mit endokriner wirkung in wasser. Bayerisches landesamt für wasserwirt schaft, Institut für Wasserforschung, München, as cited in U.K. Environment Agency, 1998.

Kravetz, L., H. Chung, K.F. Guin, W.T. Shebs and L.S. Smith. 1984. Primary and ultimate biodegradation of an alcohol ethoxylate and a nonylphenol ethoxylate under average winter conditions in the United States. Tenside Detergents 21:1-6.

Kravetz, L., J.P. Salanitro, P.B. Dorn and K.F. Guin. 1991. Influence of hydrophobe type and extent of branching on environmental response factors of nonionic surfactants. J. Amer. Oil Chem. Soc. 68:610-618.

Kreisselmeier, A. and H.W. Dürbeck. 1997. Determination of alkylphenols, alkylphenol ethoxylates and linear alkylbenzene sulfonates in sediments by accelerated solvent extraction and supercritical fluid extraction. J. Chromatogr. A 775:187-196.

Krogh, P. H., Holstrump, M., Jensen, J. and Petersen, S. O. (1996). Økotoksikologisk vurdering af spildevandsslam i landbrugsjord. [Ecological assessment of sewage sludge on farm land- Report from the Danish EPA. In Danish with English summary]. Arbejdsrapport Nr. 43, 53 pp. Miljø-og Energiminisisteriet Miljøstyrelsen

Kubeck, E. and C.G. Naylor. 1990. Trace analysis of alkylphenol ethoxylates. J. Amer. Oil Chem. Soc. 67:400-405.

Kudoh, M., H. Ozawa, S. Fudano and K. Tsuiji. 1984. Determination of trace amounts of alcohol and alkylphenol ethoxylates by high performance liquid chromatography with fluorimetric detection. J. Chromatogr. 287:337-344.

Kujawa, M., Schnaak, W. and Küchler. T. 1997. Occurrence of organic pollutants in sewage sludge and influence of surfactants on their mobility in amended soils. *In* Abstracts of the Specialty Conference on Management and Fate of Toxic Organics in Sludge Applied to Land. Copenhagen, Denmark, 30 April - 2 May, 1997. Department of Environmental Science and Engineering, Technical University of Denmark.

Kurata, N., K. Koshida and T. Fujii. 1977. Biodegradation of surfactants in river water and their toxicity to fish. Yukagaku 26:115-118.

Kveštak, R. and M. Ahel. 1995. Biotransformation of nonylphenol polyethoxylate surfactants by estuarine mixed bacterial cultures. Arch. Environ. Contam. Toxicol. 29:551-556.

Kveštak, R., S. Terzic and M. Ahel. 1994. Input and distribution of alkylphenol polyethoxylates in a stratified estuary. Mar. Chem. 46:89-100.

Lech, J.J., S.K. Lewis and L. Ren. 1996. *In vivo* estrogenic activity of nonylphenol in rainbow trout. Fundamental Appl. Toxicol. 30:229-232.

Lee, H-B. 1999. Review of analytical methods for the determination of nonylphenol and related compounds in environmental samples. Water Qual. Res. J. Canada. 34:3-35.

Lee, H.-B. 1998. Unpublished observations. National Water Research Institute, Environment Canada, Burlington, Ontario L7R 4A6 Canada.

Lee, H-B. and T.E. Peart. 1999. Occurrence of nonylphenol ethoxylates and their metabolites in Canadian pulp and paper mill effluents and sludge. Water Qual. Res. J. Canada. 34:633-652.

Lee, H.-B. and T.E. Peart. 1995. Determination of 4-nonylphenol in effluent and sludge from sewage treatment plants. Anal. Chem. 67:1976-1980.

Lee, H.-B. and T.E. Peart. 1998. Occurrence and elimination of nonylphenol ethoxylates and metabolites in municipal wastewater and effluents. Water Qual. Res. J. Can. 33:389-402.

Lee, H.-B., T.E. Peart, D.T. Bennie and R.J. Maguire. 1997. Determination of nonylphenol polyethoxylates and their carboxylic acid metabolites in sewage treatment plant sludge by supercritical carbon dioxide extraction. J. Chromatogr. A 785:385-394.

Lee, H.-B., J. Weng, T.E. Peart and R.J. Maguire. 1998. Occurrence of alkylphenoxyacetic acids

in Canadian sewage treatment plant effluents. Water Qual. Res. J. Can. 33:19-29.

Lee, P.-C. and W. Lee. 1996. *In vivo* estrogenic action of nonylphenol in immature female rats. Bull. Environ. Contam. Toxicol. 57:341-348.

Leenheer, J.A., R.L. Wershaw, P.A. Brown and T.I. Noyes. 1991. Detection of poly(ethylene glycol) residues from nonionic surfactants in surface water by ^{1}H and ^{13}C nuclear magnetic resonance spectrometry. Environ. Sci. Technol. 25:161-168.

Lewis, J.C. and L. Jurd. 1972. Sporostatic action of cinnamylphenols and related compounds on *Bacillus megaterium*. Spores 5:384-389.

Lewis, M.A. 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, M.A. 1990. Chronic toxicities of surfactants and detergent builders to algae: a review and risk assessment. Ecotoxicol. Environ. Safety 20:123-140.

Lewis, S.K. and J.J. Lech. 1996. Uptake, disposition and persistence of nonylphenol from water in rainbow trout (*Oncorhynchus mykiss*). Xenobiotica 26:813-819.

Liber, K., L.M. Knuth and F.S. Stay. 1999a. An integrated evaluation of persistence and effects of 4-nonylphenol in an experimental littoral ecosystems. Environ. Toxicol. Chem. 18:357-363.

Liber, K., J.A. Gangl, L.J. Heinis, T.D. Corry and F.S. Stay. 1999b. Lethality and bioaccumulation of 4-nonylphenol in bluegill sunfish in littoral enclosures. Environ. Toxicol. Chem. 18:394-400.

Lussier, S., D. Champlin, J. LiVolsi, S. Poucher, R. Pruell and G. Thursby. 1996. Acute toxicity of 4-nonylphenol to saltwater animals. Soc. Environ. Toxicol. Chem, Annual Meeting, Washington, DC, U.S.A. (poster 408).

Macek, K.J. and S.F. Krzeminski. 1975. Susceptibility of bluegill sunfish (*Lepomis macrochirus*) to non-ionic surfactants. Bull. Environ. Contam. Toxicol. 13:377-384.

Mackay, L.G., M.Y. Croft, D.S. Selby and R.J. Wells. 1997. Determination of nonylphenol and octylphenol ethoxylates in effluent by liquid chromatography with fluorescence detection. J. Assoc. Offic. Anal. Chem. Inter. 80:401-407.

Madsen, S.S., A.B. Mathiesen and B. Korsgaard. 1997a. Effects of 17β-estradiol and 4-nonylphenol on smoltification and vitellogenesis in Atlantic salmon (*Salmo solar*). Fish Physiol. Biochem. 17:303-312.

Madsen, T., Kristensen, P., Samsøe-Petersen, L., Tørsløv, J. and Rasmussen, J.O. 1997b. Application of sludge on farmland - quality objectives, level of contamination and environmental risk assessment. *In* Abstracts of the Specialty Conference on Management

and Fate of Toxic Organics in Sludge Applied to Land, Copenhagen, Denmark, April 30 - May 2, 1997, Department of Environmental Science and Engineering, Technical University of Denmark.

Maguire, R.J., 1999. Review of the persistence of nonylphenol and nonylphenol ethoxylates in aquatic environments. Water Qual. Res. J. Canada. 34:37-78.

Maguire, R.J. 1991. Kinetics of pesticide volatilization from the surface of water. J. Agric. Food Chem. 39:1674-1678.

Maki, H., M. Fujita and Y. Fujiwara. 1996. Identification of final degradation product of nonylphenol ethoxylate (NPE) by river microbial consortia. Bull. Environ. Contam. Toxicol. 57:881-887.

Maki, H., N. Masuda, Y. Fujiwara, M. Ike and M. Fujita. 1994. Degradation of alkylphenol ethoxylates by *Pseudomonas sp.* strain TR01. Appl. Environ. Microbiol. 60:2265-2271.

Maki, H., H. Okamura, I. Aoyama and M. Fujita. 1998. Halogenation and toxicity of the biodegradation products of a nonionic surfactant, nonylphenol ethoxylate. Environ. Toxicol. Chem. 17:650-654.

Mann, A.H. and V.W. Reid. 1971. Biodegradation of synthetic detergents. Evaluation by community trials. Part 2: alcohol and alkylphenol ethoxylates. J. Amer. Oil Chem. Soc. 48:794-797.

Manzano, M.A., J.A. Perales, D. Sales and J.M. Quiroga. 1998. Effect of concentration on the biodegradation of a nonylphenol polyethoxylate in river water. Bull. Environ. Contam. Toxicol. 61:489-496.

Marchetti, R. 1965. The toxicity of nonylphenol ethoxylate to the developmental stages of the rainbow trout, *Salmo gairdneri* Richardson. Ann. Appl. Biol. 55:425-430.

Marcomini, A. 1991. Behaviour in sludge treated soils and landfills. *In* Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP) (1991). Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, pp. 153-167.

Marcomini, A., P.D. Capel, T. Lichtensteiger, P.H. Brunner and W. Giger. 1988b. Fate of organic pollutants in sludge amended soil and sludge-only landfills: linear alkylbenzenesulphonates, nonylphenols and polychlorinated biphenyls. *In* D. Quaghebeur, I. Temmerman and G. Angeletti (eds.), Organic contaminants in waste water, sludge and sediment: occurrence, fate and disposal. Elsevier Applied Science, New York, U.S.A., ISBN 1-85166-445-9, pp. 105-123.

Marcomini, A., P.D. Capel, T. Lichtensteiger, P.H. Brunner and W. Giger. 1989a. Behavior of aromatic surfactants and PCBs in sludge-treated soil and landfills. J. Environ. Qual.

18:523-528.

Marcomini, A., S. Capri, P.H. Brunner and W. Giger. 1988a. Mass fluxes of linear alkylbenzenesulphonates, nonylphenol, nonylphenol mono- and diethoxylate through a sewage treatment plant. *In* G. Angeletti and A. Bjørseth (eds.), Organic micropollutants in the aquatic environment, Proc. Fifth Eur. Symp., Rome, Italy, Oct. 20-22, 1987. Kluwer Academic Publishers, Dordrecht, The Netherlands, ISBN 90-277-2738-4, pp. 266-277.

Marcomini, A., S. Capri and W. Giger. 1987. Determination of linear alkylbenzene-sulphonates, alkylphenol polyethoxylates and nonylphenol in waste water by high-performance liquid chromatography after enrichment on octadecylsilica. J. Chromatogr. 403:243-252.

Marcomini, A., F. Cecchi and A. Sfriso. 1991. Analytical extraction and environmental removal of alkylbenzene sulphonates, nonyl phenol and nonylphenol monoethoxylate from dated sludge-only landfills. Environ. Technol. 12:1047-1054.

Marcomini, A., A. Di Corcia, R. Samperi and S. Capri. 1993. Reversed-phase high performance liquid chromatographic determination of linear alkylbenzene sulphonates, nonylphenol polyethoxylates and their carboxylic biotransformation products. J. Chromatogr. 644:59-71.

Marcomini, A., F. Filipuzzi and W. Giger. 1988c. Aromatic surfactants in laundry detergents and hard-surface cleaners: linear alkylbenzenesulphonates and alkylphenol polyethoxylates. Chemosphere 17:853-863.

Marcomini, A. and W. Giger. 1987. Simultaneous determination of linear alkylbenzenesulfonates, alkylphenol polyethoxylates and nonylphenol by high-performance liquid chromatography. Anal. Chem. 59:1709-1715.

Marcomini, A., B. Pavoni, A. Sfriso and A.A. Orio. 1990. Persistent metabolites of alkylphenol polyethoxylates in the marine environment. Mar. Chem. 29:307-323.

Marcomini, A., S. Stelluto and B. Pavoni. 1989b. Determination of linear alkylbenzenesulphonates and alkylphenol polyethoxylates in commercial products and marine waters by reversed- and normal-phase HPLC. Intern. J. Environ. Anal. Chem. 35:207-218.

Marcomini, A. and M. Zanette. 1996. Chromatographic determination of non-ionic aliphatic surfactants of the alcohol polyethoxylate type in the environment. J. Chromatogr. A 733:193-206.

Markarian, R.K., K.W. Pontasch, D.R. Peterson and A.I. Hughes. 1989. Review and analysis of environmental data on Exxon surfactants and related compounds. Technical Report, Exxon Biomedical Sciences, East Millstone, NJ, U.S.A.

Markert, J.R., D.A. Higgs, H.M. Due and D.W. MacQuarrie. 1977. Influence of bovine growth

hormone on growth rate, appetite, and food conversion of yearling coho salmon (*Oncorhyncus kisutch*) fed two diets of different composition. Can. J. Zool. 55:74-83

Maxwell, K.E. and W.D. Piper. 1968. Molecular structure of nonionic surfactants in relation to laboratory insecticidal activity. J. Econ. Entomol. 61:1633-1636.

McAllister, W.A., T. McLain and L. Schuster. 1986. Early life stage toxicity of *para*-tert-octylphenol to rainbow trout (*Salmo gairdneri*) in a flow-through system. Report No. 34452, Analytical Bio-Chemistry Laboratories, Columbia, MO, U.S.A.

McLeese, D.W., D.B. Sergeant, C.D. Metcalfe, V. Zitko and L.E. Burridge. 1980a. Uptake and excretion of aminocarb, nonylphenol, and pesticide diluent 585 by mussels (*Mytilus edulis*). Bull. Environ. Contam. Toxicol. 24:575-581.

McLeese, D.W., V. Zitko, C.D. Metcalfe and D.B. Sergeant. 1980b. Lethality of aminocarb and the components of aminocarb formulation to juvenile Atlantic salmon, marine invertebrates, and a freshwater clam. Chemosphere 9:79-82.

McLeese, D.W., V. Zitko, D.B. Sergeant, L. Burridge and C.D. Metcalfe. 1981. Lethality and accumulation of alkylphenols in aquatic fauna. Chemosphere 10:723-730.

Meldahl, A.C., K. Nithipatikom and J.J. Lech. 1996. Metabolism of several ^{14}C-nonylphenol isomers by rainbow trout (*Oncorhynchus mykiss*): *in vivo* and *in vitro* microsomal metabolites. Xenobiotica 26:1167-1180.

Menges, R.A., T.S. Menges, G.L. Bertrand, D.W. Armstrong and L.A. Spino. 1992. Extraction of nonionic surfactants from waste water using centrifugal partition chromatography. J. Liq. Chromatogr. 15:2909-2925.

Metcalfe, C.D. 1999. Personal communication. Trent University. Peterborough. ON.

Metcalfe, C., L. Hoover and S. Sang. 1996. Nonylphenol ethoxylates and their use in Canada. World Wildlife Fund Canada report, December 1996, World Wildlife Fund Canada, 90 Eglinton Avenue East, Suite 504, Toronto, Ontario M4P 2Z7, 33+ pp.

Miles-Richardson, S.R., S.L. Pierens, K.M. Nichols, V.J. Kramer, E.M. Snyder, S.A. Snyder, J.A. Render, S.D. Fitzgerald and J.P. Giesy. 1999. Effects of waterborne exposure to 4-nonylphenol and nonylphenol ethoxylate on secondary sex characteristics and gonads of fathead minnows (*Pimephales promelas*). Environ. Res. Sect. A, 80:S122-137.

Ministers' Expert Advisory Panel. 1995. Report of the Ministers' Expert Advisory Panel on the Second Priority Substances List under the Canadian Environmental Protection Act (CEPA). Environment Canada, Ottawa, Ontario, 26 pp.

Miszkiewicz, W. and J. Szymanowski. 1996. Analysis of nonionic surfactants with polyoxyethylene chains by high performance liquid chromatography. Crit. Rev. Anal. Chem. 25:203-246.

Monsanto Industrial Chemicals Co. 1985. Chemical hazard information profile: nonylphenol. Report to the Office of Toxic Substances, U.S. Environmental Protection Agency, Washington, DC, U.S.A.

Moody, R.P., P. Weinberger and R. Greenhalgh. 1983. Algal fluorometric determination of the potential phytotoxicity of environmental pollutants. *In* J.O. Nriagu (ed.), Aquatic Toxicology, John Wiley and Sons, New York, NY, U.S.A., pp. 503-512.

Moore, G. 1999. Personal Communication. Pest Management Regulatory Agency. Health Canada. Ottawa, Ontario, Canada.

Moore, S.B., R.A. Diehl, J.M. Barnhardt and G.B. Avery.1987. Aquatic toxicity of textile surfacctants. Text. Chem. Col. 19:29-32. As cited in Talmage, 1994.

Mueller, G.C. and U.H. Kim. 1978. Displacement of estradiol from estrogen receptors by simple alkyl phenols. Endocrinol. 102:1429-1435.

Müller, S., P. Schmid and C. Schlatter. 1998. Pharmacokinetic behavior of 4-nonylphenol in humans. Environ. Toxicol. Pharmacol. 5:257-265.

Nagel, S.C., F.S. vom Saal, K.A, Thayer, M.G. Dhar, M. Boechler and W.V. Welshons. 1997. Relative binding affinity-serum modified access (RBA-SMA) assay predicts the relative *in vivo* bioactivity of the xenoestrogens bisphenol A and octylphenol. Environ. Health Perspect. 105:70-76.

Narayanan, K.S., M. Kaminsky, D. Jon and R.M. Ianniello. 1997. Water in oil microemulsion aerosol systems for insecticidal compositions. *In* G.R. Goss, M.J. Hopkinson and H.M. Collins (eds.), Pesticide formulations and applications systems, ASTM STP 1328. American Society for Testing and Materials, Conshohocken, PA, U.S.A., ISBN 0-8301-2469-4, pp. 39-48.

National Research Council of Canada. 1982. Aminocarb: the effects of its use on the forest and the human environment. Associate Committee on Scientific Criteria for Environmental Quality. NRCC Report No. 18979, Ottawa, Ontario, K1A 0R6, ISSN-0316-0114, 253+ pp.

Naylor, C.G. 1992. Environmental fate of alkylphenol ethoxylates. Soap/Cosmetics/Chemical Specialties 68:27-32.

Naylor, C.G. 1995. Environmental fate and safety of nonylphenol ethoxylates. Textile Chemist and Colorist 27:29-33.

Naylor, C.G., J.P. Mieure, W.J. Adams, J.A. Weeks, F.J. Castaldi, L.D. Ogle and R.R. Romano. 1992. Alkylphenol ethoxylates in the environment. J. Amer. Oil Chem. Soc. 69:695-703.

Naylor, C.G., J.P. Mieure, I. Morici and R.R. Romano. 1992. Alkylphenol ethoxylates in the environment. *In* Proc. 3rd World Surfactants Congress, pp. 111-124.

Naylor, C.G., P.T. Varineau, D. England and J. Bucksath. 1997. Toxicity of alkylphenoxyacetic acid salts to aquatic invertebrates. Soc. Environ. Toxicol. Chem. 18th Ann. Meet., San Francisco, CA, U.S.A., Nov. 1997 (abstract 114).

Neufahrt, A., K. Hofmann and G. Tauber. 1987. Biodegradation of nonylphenolethoxylate (APEO). Commun. J. Com. Esp. Deterg. 18:183-198.

Nimrod, A.C. and W.H. Benson. 1996a. Environmental estrogenic effects of alkylphenol ethoxylates. Crit. Rev. Toxicol. 26:335-364.

Nimrod, A.C. and W.H. Benson. 1996b. Estrogenic responses to xenobiotics in channel catfish (*Ictalurus punctatus*). Mar. Environ. Res. 42:155-160.

Nyberg, H. 1976. The effects of some detergents on the growth of *Nitzschia holsatica* Hust. (Diatomaceae). Ann Bot. Fenn. 13:65-68.

Nyberg, H. 1985. Physiological effects of four detergents on the algae *Nitzschia actinastroides* and *Porphyridium purpureum*. Publication 12, Department of Botany, University of Helsinki, Helsinki, Finland.

Nyberg, H. 1988. Growth of *Selenastrum capricornutum* in the presence of synthetic surfactants. Water Res. 22:217-223.

OECD (Organization for Economic Cooperation and Development). 1997. SIDS initial Assessment report for 6th SIAM. Prepared by M. Thomas, Environment Agency, National Centre for Ecotoxicology and Hazardous Substances, London, U.K.

O'Halloran, S.L., K. Liber, J.A. Gangl and M.L. Knuth. 1998. Effects of repeated exposure to 4-nonylphenol on zooplankton community in littoral enclosures. Environ. Toxicol. Chem. 18:376-385.

Öman, C. and Hynning, P.-Å. 1993. Identification of organic compounds in municipal landfill leachates. Environ. Pollution 80:265-271.

Ontario Ministry of Environment and Energy and Agriculture, Food and Rural Affairs 1996. Guidelines for the utilization of biosolids and other wastes on agricultural land. March 1996.

Oppen-Berntsen, D.O., E. Gram-Jensen and B.T. Walther. 1992. Zona radiata proteins are synthesized by rainbow trout (*Oncorhynchus mykiss*) hepatocytes in response to oestradiol 17β. J. Endocrinol. 135:293-302.

Oslo and Paris Conventions for the Prevention of Marine Pollution Working Group on Diffuse Sources (DIFF) 1996. Draft background document concerning the elaboration of programmes and measures on nonylphenol/nonylphenolethoxylates (NP/NPEOs) and octylphenol/octylphenolethoxylates (OP/OPEOs). Presented by Sweden at Cadiz, Sept. 30 - Oct. 3, 1996. Oslo and Paris Commissions for the Prevention of Marine Pollution,

London WC2A 2JE, United Kingdom.

Otsuki, A. and H. Shiraishi. 1979. Determination of poly(oxyethylene)alkylphenyl ether nonionic surfactants in water at trace levels by reversed phase adsorption liquid chromatography and field desorption mass spectrometry. Anal. Chem. 51:2329-2332.

PARCOM (Paris Commission) 1992. PARCOM recommendation 92/8 on nonylphenol ethoxylates. Oslo and Paris Commissions for the Prevention of Marine Pollution, London WC2A 2JE, United Kingdom.

Patoczka, J. and G.W. Pulliam. 1990. Biodegradation and secondary effluent toxicity of ethoxylated surfactants. Water Res. 24:965-972.

Pattanaargsorn, S., P. Sangvanich, A. Petsom and S. Roengsumran. 1995. Determination of oligomer distribution of alkylphenol polyethoxylates and fatty alcohol polyethoxylates by positive ion atmospheric pressure chemical ionization mass spectrometry. Analyst 120:1573-1576.

Patterson, S.J., C.C. Scott and K.B.E. Tucker. 1967. Nonionic detergent degradation. I. Thin-layer chromatography and foaming properties of alcohol polyethoxylates. J. Amer. Oil Chem. Soc. 44:407-412.

Patterson, S.J., C.C. Scott and K.B.E. Tucker. 1968. Nonionic detergent degradation. II. Thin-layer chromatography and foaming properties of alkyl phenol polyethoxylates. J. Amer. Oil Chem. Soc. 45:529-532.

Patterson, S.J., C.C. Scott and K.B.E. Tucker. 1970. Nonionic detergent degradation. III. Initial mechanism of the degradation. J. Amer. Oil Chem. Soc. 47:37-41.

Paxéus, N. 1996. Organic pollutants in the effluents of large wastewater treatment plants in Sweden. Water Res. 30:1115-1122.

Paxéus, N. and H.F. Schröder. 1996. Surfactants in domestic wastewaters in Sweden and their behaviour in the wastewater treatment process. *In* Proc. 4th Surfactants Conf., Barcelona, Spain, June 3-7, 1996. Publ. Royal Society of Chemistry, Cambridge, UK, Vol. 4, pp. 463-474 (ISBN 0-85404-766-2), pp. 463-474.

Piferrer, F. and E.M. Donaldson. 1989. Gonadal ontogenesis in coho salmon, *Oncorhynchus kisutch*, after a single treatment with androgen or estrogen during ontogenesis. Aquaculture 77:251-262.

Pilc, J.A. and P.A. Sermon. 1987. Chromatographic separation of a wide range of ethoxylated non-ionic surfactants and their sulphonates using high-performance liquid chromatography. J. Chromatogr. 398:375-380.

Poremski, H.-J. 1991. Use pattern of nonylphenol ethoxylates and their application properties. *In* Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP)

(1991). Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, pp. 23-40.

Prasad R. 1989. Effect of nonylphenol adjuvant on macrophytes. Adjuvants Agrochem. 1:51-61.

Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP). 1991. Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, 265 pp.

Portmann, J.E. and K.W. Wilson. 1971. Shellfish information leaflet. Ministry of Agriculture, Fisheries and Food, U.K. (as cited in Talmage 1994).

Purdom, C.E., P.A. Hardiman, V.J. Bye, N.C. Eno, C.R. Tyler and J.P. Sumpter. 1994. Estrogenic effects of effluents from sewage treatment works. Chem. Ecol. 8:275-285.

Quiroga, J.M., M.A. Manzano, D. Sales and J.A. Perales. 1996. Biodegradation of a nonylphenol polyethoxylate (NPEO) in river water. *In* Proc. 4th Surfactants Conf., Barcelona, Spain, June 3-7, 1996. Publ. Royal Society of Chemistry, Cambridge, UK, Vol. 4, pp. 417-425 (ISBN 0-85404-766-2).

Reed, H.W.B. 1978. Alkylphenols. *In* Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed., Vol. 2, ISBN 0-471-02038-0, John Wiley and Sons, Inc., New York, NY, U.S.A., pp. 72-96.

Reiff, B., R. Lloyd, M.J. How, D. Brown and A.S. Alabaster. 1979. The acute toxicity of eleven detergents to fish: results of an interlaboratory exercise. Water Res. 13:207-210.

Reinhard, M., N. Goodman and K.E. Mortelmans. 1982. Occurrence of brominated alkylphenol polyethoxy carboxylates in mutagenic wastewater concentrates. Environ. Sci. Technol. 16:351-362.

Ren, L., S.K. Lewis and J.J. Lech. 1996. Effects of estrogen and nonylphenol on the post-transcriptional regulation of vitellogenin gene expression. Chemico-Biol. Inter. 100:67-76.

Ren, L., M.A. Marquardt and J.J. Lech. 1997. Estrogenic effects of nonylphenol on pS2, ER and MUCI gene expression in human breast cancer cells-MCF-7. Chemico-Biol. Inter. 104:55-64.

Ribosa, I. M.T. Garcia, J. Sanchez Leal and J.J. Gonzalez. 1993. *Photobacterium phosphoreum* test data of non-ionic surfactants. Toxicological and Environmental Chem. 39:237-241.

Roberts, D.W. 1991. QSAR issues in aquatic toxicity of surfactants. Sci. Total Environ. 109/110:557-568.

Roderer, 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.

Rodier, D. 1996. RM-1 document for *para*-nonylphenol. U.S. Environmental Protection Agency, Washington, DC, U.S.A., 43 pp.

Rivera, J., F. Ventura, J. Caixach, M. de Torres, A. Figueras and J. Guardiola. 1987. GC/MS, HPLC and FAB mass spectrometric analysis of organic micropollutants in Barcelona's water supply. Intern. J. Environ. Anal. Chem. 29:15-35.

Romano, R.R. 1991. Current studies on nonylphenol - physical/chemical, biodegradation and aquatic effects. *In* Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP) (1991). Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, pp. 233-239.

Routledge E., D. Sheahan, C. Desbrow, G.C. Brighty, M. Waldock and J.P. Sumpter. 1998. Identification of estrogenic chemicals in STP effluent. 2. *In vivo* responses in trout and roach. Environ. Sci. Technol. 32:1559-1565.

Routledge, E.J. and J.P. Sumpter. 1997. Structural features of alkylphenolic chemicals associated with estrogenic activity. J. Biol. Chem. 272:3280-3288.

Rudling, L. and P. Solyom. 1974. The investigation of biodegradability of branched nonyl phenol ethoxylates. Water Res. 8:115-119.

Rutherford, L.A., Hennigar, P.A., Doe, K.G., Nicol, M.L., Holmes, M.M.E., MacDonald, B.C. and Horne, W.H. 1992. Chemical characterization, aquatic toxicity and environmental impact of untreated effluent discharges from three textile mills in the Atlantic Region. Environment Canada Report EPS-5-AR-93-1, Environmental Protection Branch, Atlantic Region, Dartmouth, Nova Scotia B2Y 2N6.

Salanitro, J.P. and L.A. Diaz. 1995. Anaerobic biodegradability testing of surfactants. Chemosphere 30:813-830.

Salanitro, J.P., L.A. Diaz and L. Kravetz. 1995. Aerobic biodegradability of surfactants at low concentrations using an automated pressure transducer system. Chemosphere 31:2827-2837.

Salanitro, J.P., G.C. Langston, P.B. Dorn and L. Kravetz. 1988. Activated sludge treatment of ethoxylate surfactants at high industrial use concentrations. Water Sci. Technol. 20(11/12):125-130.

Scarlett, M.J., J.A. Fisher, H. Zhang and M. Ronan. 1994. Determination of dissolved nonylphenol ethoxylate surfactants in waste waters by gas stripping and isocratic high performance liquid chromatography. Water Res. 28:2109-2116.

Schmude, K.L., K. Liber, T.D. Corry and F.S. Stay. 1998. Effects of 4-nonylphenol on benthic macroinvertebrates and insect emergence in littoral enclosures. Environ. Toxicol. Chem.,

in press.

Schöberl, P. and H. Mann. 1976. The influence of temperature on the degradation of non-ionic tensides in sea- and freshwater. Arch. Fisch. Wiss. 27:149-158.

Schübauer-Berigan, M.K. and Ankley, G.T. 1991. The contribution of ammonia, metals and nonpolar organic compounds to the toxicity of sediment interstitial water from an Illinois River tributary. Environ. Toxicol. Chem. 10:925-939.

Schuurmann, G. 1990. QSAR analysis of the acute toxicity of oxyethylated surfactants. Chemosphere 21:467-478.

Schuurmann, G. 1991. Acute aquatic toxicity of alkylphenol ethoxylates. Ecotoxicol. Environ. Safety 21:227-233.

Scullion, S.D., M.R. Clench, M. Cooke and A.E. Ashcroft. 1996. Determination of surfactants in surface water by solid-phase extraction, liquid chromatography and liquid chromatography - mass spectrometry. J. Chromatogr. A 733:207-216.

Serak, K.R. and Y. Zhixing. 1997. Aerobic river die-away of ^{14}C-nonylphenol ethoxylate standard method for biodegradation by a shake-flask die-away method. Analytical Bio Chemistry Laboratories, Inc. report No. 42889, to the Chemical Manufacturers Association, Analytical Bio Chemistry Laboratories, Inc., 7200 E. ABC Lane, Columbia, MO 65202-8015, U.S.A., 150 pp.

Servos, M.R., 1999a. Review of the aquatic toxicity and bioaccumulation of alkylphenols and alkylphenol polyethoxylates. Water Qual. Res. J. Canada. 34:123-177.

Servos, M.R. 1999b. Personal communication. National Water Research Institute, Burlington, ON.

Shang, D.Y., R.W. MacDonald and M.G. Ikonomou. 1999. Persistence of nonylphenol ethoxylate surfactants and their primary degradation products in sediments from near a municipal outfall in the Strait of Georgia, British Columbia, Canada. Environ. Sci. Technol. 33:1366-1372.

Sheldon, L.S. and Hites, R.A. 1978. Organic compounds in the Delaware River. Environ. Sci. Technol. 12:1188-1194.

Sheldon, L.S. and Hites, R.A. 1979. Sources and movement of organic chemicals in the Delaware River. Environ. Sci. Technol. 13:574-579.

Shell Chemical Company. 1987. Neodol product guide for alcohols, ethoxylates and ethoxysulfates. SC-7-87 (as cited in Staples 1998).

Sherrard, K.B., P.J. Marriott, R.G. Amiet and M.J. McCormick. 1996. Spectroscopic analysis of heterogeneous photocatalysis products of nonylphenol- and primary alcohol ethoxylate

nonionic surfactants. Chemosphere 33:1921-1940.

Sherrard, K.B., P.J. Marriott, M.J. McCormick, R. Colton and G. Smith. 1994. Electrospray mass spectrometric analysis and photocatalytic degradation of polyethoxylate surfactants used in wool scouring. Anal. Chem. 66:3394-3399.

Sithole, B.B. and Allen, L.H. 1989. Determination of nonionic nonylphenol ethoxylate surfactants in pulp and paper mill process samples by spectrophotometry and liquid chromatography. J. Assoc. Off. Anal. Chem. 72:273-276.

Soto, A.M., K.L. Chung and C. Sonnenschein. 1994. The pesticides endosulfan, toxaphene and dieldrin have estrogenic effects on human estrogen-sensitive cells. Environ. Health Perspect. 102:380-383.

Soto, A.M., H. Justicia, J.W. Wray and C. Sonnenschein. 1991. *p*-Nonyl-phenol: an estrogenic xenobiotic released from "modified" polystyrene. Environ. Health Perspect. 92:167-173.

Soto, A.M., C. Sonnenschein, K.L. Chung, M.F. Fernandez, N. Olea and F.O. Serrano. 1995. The E-screen assay as a tool to identify estrogens: an update on estrogenic environmental pollutants. Environ. Health Perspect. 103:113-122.

Specker, J.L. and C.V. Sullivan. 1993. Vitellogenesis in fishes: status and perspectives. *In* K.G. Davey, R.E. Peter and S.S. Tobe (eds.), Perspectives in Comparative Endocrinology, National Research Council of Canada publication NRCC-0038000, Ottawa, Ontario, Canada, pp. 304-315.

Staples, C.A., J. Weeks, J.F. Hall and C.G. Naylor. 1998. Evaluation of the aquatic toxicity and bioaccumulation of C8 and C9-alkylphenol ethoxylates. Environ. Toxicol. Chem. 17:2470-2480.

Stegeman, J.J. and M.E. Hahn. 1994. Biochemistry and molecular biology of monooxygenases: current perspectives on forms, function, and regulation of cytochrome P450 in aquatic species. *In* D.C. Malins and G.K. Ostrander (eds.), Aquatic Toxicity: Molecular, Biochemical, and Cellular Perspectives. CRC Press, Boca Raton, FL, U.S.A., pp. 87-174.

Stephanou, E. 1984a. Identification of nonionic detergents by GC/CI-MS: 1. A complementary method or an attractive alternative to GC/EI-MS and other methods? Chemosphere 13:43-51.

Stephanou, E. 1984b. Chemical ionization mass spectra of alkylphenol and linear alcohol polyethoxylates. Org. Mass Spectrom. 19:510-513.

Stephanou, E. 1985. Environmental determination of alkylphenol polyethoxylate refractory residues using mass spectrometric techniques. Intern. J. Environ. Anal. Chem. 20:41-54.

Stephanou, E. and W. Giger. 1982. Persistent organic chemicals in sewage effluents. 2. Quantitative determinations of nonylphenols and nonylphenol ethoxylates by glass

capillary gas chromatography. Environ. Sci. Technol. 16:800-805.

Stephanou, E., M. Reinhard and H.A. Ball. 1988. Identification and quantification of halogenated and non-halogenated octylphenol polyethoxylate residues by gas chromatography/mass spectrometry using electron ionization and chemical ionization. Biomed. Environ. Mass Spectrom. 15:275-282.

Stiff, M.J., R.C. Rootham and G.E. Culley. 1973. The effect of temperature on the removal of non-ionic surfactants during small scale activated-sludge sewage treatment - I. Comparison of alcohol ethoxylates with a branched-chain alkyl phenol ethoxylate. Water Res. 7:1003-1010.

Struijs, J. and J. Stoltenkamp. 1994. Testing surfactants for ultimate biodegradability. Chemosphere 28:1503-1523.

Sturm, R.N. 1973. Biodegradability of nonionic surfactants: screening test for predicting rate and ultimate biodegradation. J. Amer. Oil Chem. Soc. 50:159-167.

Sumpter, J.P. 1992. Control of growth in rainbow trout (*Oncorhynchus mykiss*). Aquaculture 100:299-321.

Sumpter, J.P. and S. Jobling. 1995. Vitellogenesis as a biomarker of estrogenic contamination in the aquatic environment. Environ. Health Perspect. 103:173-178.

Sundaram, K.M.S. and S. Szeto. 1981. The dissipation of nonylphenol in stream and pond water under simulated field conditions. J. Environ. Sci. Health B16:767-776.

Sundaram, K.M.S., S. Szeto, R. Hindle and D. MacTavish D. 1980. Residues of nonylphenol in spruce foliage, forest soil, stream water and sediment after its aerial application. J. Environ. Sci. Health. B15:403-419.

Swedmark, M. 1968. Resistens hos fisk mot glyko, tesider och en vanlig tensidravara. Vatten 5:430-443.

Swedmark, M., B. Braaten, E. Emanuelsson and Å. Granmo. 1971. Biological effects of surface active agents on marine animals. Mar. Biol. 9:183-201.

Swedmark, M., Å. Granmo and S. Kollberg. 1976. Toxicity testing at Kristinberg Marine Biology Station. *In* Pollutants in the Aquatic Environment. Food and Agriculture Organization FAO/SIDA/TF 108, Supplement 1, Rome, Italy.

Sweetman, A.J. 1994. Development and application of a multi-residue analytical method for the determination of *n*-alkanes, linear alkylbenzenes, polynuclear aromatic hydrocarbons and 4-nonylphenol in digested sewage sludges. Water Res. 28:343-353.

Syracuse Research Corp. 1998. Information from the World Wide Web, Dec. 8, 1998 (http://esc.syrres.com/~ESC/kowexpdb.htm).

Szymanski, A., Z. Swit and Z. Lukaszewski. 1995. Studies of preservation of water samples for the determination of non-ionic surfactants. Anal. Chim. Acta 311:31-36.

Talmage, S.S. 1994. Environmental and human safety of major surfactants - alcohol ethoxylates and alkylphenol ethoxylates. Lewis Publ., Boca Raton, FL, U.S.A., 374+ pp., ISBN 1-56670-017-5.

Tanghe, T., G. Devriese and W. Verstraete. 1998. Nonylphenol degradation in lab scale activated sludge units is temperature dependent. Water Res. 32:2889-2896.

Thiele, B., K. Günther and M.J. Schwuger. 1997. Alkylphenol ethoxylates: trace analysis and environmental behavior. Chem. Rev. 97:3247-3272.

Tideström, H. 1997. Swedish regulation on the use of sewage sludge in agriculture. *In* Abstracts of the Specialty Conference on Management and Fate of Toxic Organics in Sludge Applied to Land, Copenhagen, Denmark, April 30 - May 2, 1997, Department of Environmental Science and Engineering, Technical University of Denmark.

Tobin, R.S., F.I. Onuska, D.H.J. Anthony and M.E. Comba. 1976. Nonionic surfactants: conventional biodegradation test methods do not detect persistent polyglycol products. Ambio 5:30-31.

Tomiyama, S. 1974. Effects of surfactants on fish. Bull Jap. Soc. Sci. Fish. 40:1291-1296.

Topp, Ed. 1999. Personal Communication. Agriculture Canada.

Tremblay, L., X. Yoa and G. Van Der Kraak. 1995. Interactions of the environmental estrogens nonylphenol and β-sitosterol with liver estrogen receptors in fish. Proc. 5th Inter. Symp. Reproductive Physiol. Fish, Austin, TX, U.S.A., July 2-8, 1995, eds. F.W. Goetz and P. Thomas, p. 202.

Tremblay, L. and G. Van Der Kraak. 1998. Use of a series of homologous *in vitro* and *in vivo* tests to evaluate the endocrine modulating actions of β-sitosterol in rainbow trout. Aquat. Toxicol., in press.

Trocmé, M., J. Tarradellas and J.-C. Védy. 1988. Biotoxicity and persistence of nonylphenol during incubation in a compost-sandstone mixture. Biol. Fertil. Soils 5:299-303.

Trudeau, V.L., R.E. Peter and B.D. Soley. 1991. Testosterone and estradiol potentiate the serum gonadotropin release to gonadotropin releasing hormone in goldfish. Biol. Reprod. 48:951-960.

Tyler, C.R. 1991. Vitellogenesis in salmonids. *In* Reproductive Physiology of Fish, eds. A.P. Scott, J.P. Sumpter, D.A. Kime and M.S. Rolfe, Fish Symposium 91, Sheffield, England, pp. 297-301.

Tyler, C.R., J.P. Sumpter and N.R. Bromage. 1988. The selectivity of protein sequestration by vitellogenic oocytes of the rainbow trout *Salmo gairdneri in vivo*. J. Exper. Zool.

248:199-206.

Tyler, C.R., J.P. Sumpter and N.R. Bromage. 1988. *In vivo* ovarian uptake and processing of vitellogenin in the rainbow trout *Salmo gairdneri*. J. Exper. Zool. 246:171-179.

U.K. Department of the Environment. 1993. Uses, fate and entry to the environment of nonylphenol ethoxylates. Final report, Consultants in Environmental Sciences, Ltd., 78+ pp.

U.K. Environment Agency. 1997. Draft comprehensive risk assessment report for nonylphenol. National Centre for Ecotoxicity and Hazardous Substances, A3.37 Romney House, 43 Marsham Street, London SW1P 3PY, United Kingdom.

U.K. Environment Agency. 1998. Risk assessment of 4-nonylphenol (branched) and nonylphenol. Draft of September 1998. Romney House, 43 Marsham Street, London SW1P 3PY, United Kingdom.

Ukeles, R. 1965. Inhibition of unicellular algae by synthetic surface-active agents. J. Phycol. 1:102-110.

Unilever Research Laboratories. 1977. Unpublished data cited in Arthur D. Little (1977) (as cited in Talmage, 1994).

Union Carbide Corp. 1994. Environmental Fate/Effects Database. As cited in Williams *et al.*, 1996.

U.S. Environmental Protection Agency. 1985. Chemical hazard information profile - nonylphenol. Draft report, September 17, 1985, Washington, DC, 20460, U.S.A., 41+ pp.

U.S. National Toxicology Program. 1998. Information from the World Wide Web, Dec. 8, 1998 (http://ntp-server.niehs.nih.gov/cgi/iH_Indexes/Chem_H&S/iH_Chem_H&S_Frames.html).

Valls, M., P. Fernandez, J.M. Bayona and J. Albaiges. 1988. Broad spectrum analysis of organic contaminants in urban wastewaters and coastal receiving systems. *In* Organic Contaminants in Waste Water, Sludge and Sediment: Occurrence, Fate and Disposal, D. Quaghebeur, I. Temmerman and G. Angeletti (eds.), Elsevier Applied Science, New York, NY, U.S.A., pp. 19-34.

van der Hurk, R. and G.A. Slof. 1981. A morphological and experimental study of gonadal sex differentiation in rainbow trout, *Salmo gairdneri*. Cell Tissue Res. 218:487-497.

Van Der Kraak, G. 1999. Personal Communication. University of Guelph, Guelph, Ontario.

Van Emden, H.M., C.C.M. Kroon, E.N. Schoeman and H.A. Van Secenter. 1974. The toxicity of some detergents tested on *Aedes aegypti* L., *Lebistes reticulatus* Peters, and *Biomphalaria glabrata* Say. Environ. Pollut. 6:297-308.

Veith, G.P. and L.M. Kiwus. 1977. An exhaustive steam-distillation and solvent extraction unit for pesticides and industrial chemicals. Bull. Environ. Contam. Toxicol. 17:631-636.

Ventura, F., J. Caixach, A. Figueras, I. Espadaler, D. Fraisse and J. Rivera. 1989. Identification of surfactants in water by FAB mass spectrometry. Water Res. 23:1191-1203.

Ventura, F., J. Caixach, J. Romero, I. Espadaler and J. Rivera. 1992. New methods for the identification of surfactants and their acidic metabolites in raw and drinking water: FAB-MS and MS/MS. Water Sci. Technol. 25:257-264.

Ventura, F., A. Figueras, J. Caixach, I. Espadaler, J. Romero, J. Guardiola and J. Rivera. 1988. Characterization of polyethoxylated surfactants and their brominated derivatives formed at the water treatment plant of Barcelona by GC/MS and FAB mass spectrometry. Water Res. 22:1211-1217.

Ventura, F., D. Fraisse, J. Caixach and J. Rivera. 1991. Identification of [(alkyloxy)poly-ethoxy]carboxylates in raw and drinking water by mass spectrometry/mass spectrometry and mass determination using fast atom bombardment and nonionic surfactants as internal standards. Anal. Chem. 63, 2095-2099.

Wahlberg, C., L. Renberg and U. Wideqvist. 1990. Determination of nonylphenol and nonylphenol ethoxylates as their pentafluorobenzoates in water, sewage sludge and biota. Chemosphere 20:179-195.

Waldock, M.J. and Thain, J.E. 1986. Environmental considerations of 4-nonylphenol following dumping of anaerobically digested sewage sludges: a preliminary study of occurrence and acute toxicity. International Council for the Exploration of the Sea, Marine Environmental Quality Committee Report CM 1986/E:16.

Waldock, M.J. and J.E. Thain. 1991. Environmental concentrations of 4-nonylphenol following dumping of anaerobically digested sewage sludges: a preliminary study of occurrence and acute toxicity. Report to the Ministry of Agriculture, Fisheries and Food, Fisheries Laboratory, Burnham-on-Crouch, Essex, U.K.

Wallace, R.A. 1985. Vitellogenesis and oocyte growth in non-mammalian vertebrates. *In* L.W. Browder (ed.), Developmental Biology, Vol. 1, Plenum Publishing Corporation, New York, NY, U.S.A., pp. 127-177.

Wang, Z. and M. Fingas. 1993a. Rapid separation of non-ionic surfactants of polyethoxylated octylphenol and determination of ethylene oxide oligomer distribution by C^1 column reversed-phase liquid chromatography. J. Chromatogr. 673:145-156.

Wang, Z. and M. Fingas. 1993b. Quantitative analysis of polyethoxylated octylphenol by capillary supercritical fluid chromatography. J. Chromatogr. 641:125-136.

Wang, Z. and M. Fingas. 1993c. Analysis of polyethoxylated nonylphenols by supercritical fluid chromatography and high performance liquid chromatography. J. Chromatogr. Sci.

31:509-518.

Ward T.J. and R.L. Boeri. 1990a. Acute static toxicity of nonylphenol to the marine alga (*Skeletonema costatum*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8970-CMA, Hampton, NH, U.S.A.

Ward T.J. and R.L. Boeri. 1990b. Acute static toxicity of nonylphenol to the freshwater alga (*Selenastrum capricornutum*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8969-CMA, Hampton, NH, U.S.A.

Ward, T.J. and R.L. Boeri. 1990c. Acute flow through toxicity of nonylphenol to the mysid (*Mysidopsis bahia*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8974-CMA, Hampton, NH, U.S.A.

Ward, T.J. and R.L. Boeri. 1990d. Acute flow through toxicity of nonylphenol to the sheepshead minnow (*Cyprinodon variegatus*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8972-CMA, Hampton, NH, U.S.A.

Ward, T.J. and R.L. Boeri. 1991a. Bioconcentration test with nonylphenol and the fathead minnow (*Pimephales promelas*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8975-CMA, Hampton, NH, U.S.A.

Ward, T.J. and R.L. Boeri. 1991b. Early life stage toxicity of nonylphenol to the fathead minnow (*Pimephales promelas*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8979-CMA, Hampton, NH, U.S.A.

Ward T.J. and R.L. Boeri. 1991c. Chronic toxicity of nonylphenol to the mysid (*Mysidopsis bahia*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8977-CMA, Hampton, NH, U.S.A.

Ward, T.J. and R.L. Boeri. 1992. Toxicity of nonylphenol to the tadpole (*Rana catesbiana*). Prepared for the Chemical Manufacturers Association by Resource Analysts, Study No. 8981-CMA, Hampton, NH, U.S.A.

Washburn, B.S., J.S. Krantz, E.H. Avery and R.A. Freedland. 1993. Effects of estrogen on gluconeogenesis and related parameters in male rainbow trout. Amer. J. Physiol. 264:R720-R725.

Water Technology International Corporation. 1998a. Report on alkylphenol ethoxylate persistence in biosolids-treated field soil. Report prepared for the National Water Research Institute, Project 71773, March, 1998, Burlington, Ontario L7R 4A6, Canada.

Water Technology International Corporation. 1998b. Interim report - characterization of selected Canadian sewage treatment plants for the occurrence, fate and release of endocrine-disrupting compounds. Report prepared for the National Water Research Institute, April 1998, Burlington, Ontario L7R 4A6, Canada.

Watkinson, R.J. and M.S. Holt. 1991. Environmental aspects of ethoxylates based on alkylphenol and primary alcohols. *In* Proceedings of the Seminar on Nonylphenol Ethoxylates (NPE) and Nonylphenol (NP) (1991). Saltsjöbaden, Sweden, Feb. 6-8, 1991, Ingvar Bingman, publ., Stockholm, ISBN 91-620-3907-5, pp. 169-177.

Webster, L. (1998). Personal Communication. Plant Health and Protection Division, Canadian Food Inspection Agency, Ottawa, Ontario, Canada.

Weeks, J.A., W.J. Adams, P.D. Guiney, J.F. Hall and C.G. Naylor. 1996. Risk assessment of nonylphenol and its ethoxylates in U.S. river water and sediment. *In* Proc. 4th Surfactants Conf., Barcelona, Spain, June 3-7, 1996. Publ. Royal Society of Chemistry, Cambridge, UK, Vol. 3, pp. 276-291 (ISBN 0-85404-761-1).

Weinberger, P. 1984. Screening for potential ecotoxicology of solvents, surfactants and detergents: final contract report. Environment Canada DSS file 24SU, KN107-2-4304.

Weinberger, P., C. DeChacin and M. Czuba. 1987. Effects of nonyl phenol, a pesticide surfactant, on some metabolic processes of Chlamdomona segnis. Can.J. Bot. 65:696-702.

Weinberger, R. and M. Rea. 1981. Nonylphenol: a perturbant additive to an aquatic ecosystem. *In* N. Bermingham, C. Blaise, P. Couture, B. Hummel, G. Joubert and M. Speyer (eds.), Proc. 7th Ann. Aquat. Tox. Workshop, Can. Tech. Rep. Fish Aquat. Sci. No. 990, pp. 369-381.

Wheeler, T.F., J.R. Heim, M.R. LaTorre and A.B. Janes. 1997. Mass spectral characterization of *p*-nonylphenol isomers using high resolution capillary GC-MS. J. Chromatogr. Sci. 35:19-30.

White, R., S. Jobling, S.A. Hoare, J.P. Sumpter and M.G. Parker. 1994. Environmentally persistent alkylphenolic compounds are estrogenic. Endocrinol. 135:175-182.

Williams, J.B. 1997. Aerobic degradation and aquatic toxicity of alkylphenol ether carboxylates. Presentation to the Canadian seminar on nonylphenol ethoxylates, Toronto, Ontario, Canada, Dec. 12, 1997.

Williams, J.B., R.L. Blessing and P.T. Varineau. 1996. Aquatic fate and effects testing: data on alkylphenol ether carboxylates. Union Carbide Corporation, Infrastructure and Central Technology project report, file No. 42064, October 28, 1996, South Charleston, VA, U..S.A., 14 pp.

Winther-Nielsen, M., Pedersen, U.S., Rasmussen, H.B. and Madsen, T. 1997. Effects of organic chemicals in sludge applied to soil. Degradation and toxicity to organisms living in soil. *In* Abstracts of the Specialty Conference on Management and Fate of Toxic Organics in Sludge Applied to Land. Copenhagen, Denmark, April 30 - May 2, 1997. Department of Environmental Science and Engineering, Technical University of Denmark.

Wong, S.L. 1985. Algal assay evaluation of trace contaminants in surface water using the

nonionic surfactant, Triton X-100. Aquat. Toxicol. 6:115-131.

World Wildlife Fund Canada. 1997. A review of initiatives to phase out nonyl phenol ethoxylates in Europe. Report, September 1997, 27+ pp.

Xiong, F., D. Liu, H. Elsholtz and C.L. Hew. 1994. The chinook salmon gonadotropin IIb subunit gene contains a strong minimal promoter with a proximal negative element. Molec. Endocrinol. 8:771-781.

Xiong, F., D. Liu, Y.L. Drean, H.P. Elsholtz and C.L. Hew. 1994. Differential recruitment of steroid hormone response elements may dictate the expression of the pituitary gonadotropin IIb subunit gene during salmon maturation. Molec. Endocrinol. 8:782-793.

Yasuhara, A., H. Shiraishi, M. Tsuji and T. Okuno. 1981. Analysis of organic substances in highly polluted river water by mass spectrometry. Environ. Sci. Technol. 15:570-573.

Yoshimura, K. 1986. Biodegradation and fish toxicity of nonionic surfactants. J. Amer. Oil Chem. Soc. 63:1590-1596.

Zhou, C., A. Bahr and G. Schwedt. 1990. Separation and determination of non-ionic surfactants of the nonylphenol polyglycol ether type by liquid chromatography. Anal. Chim. Acta 236:273-280.

Zimniak, P. and D.J. Waxman. 1993. Liver cytochrome P450 metabolism of endogenous steroid hormones, bile acids, and fatty acids. *In* J.B. Schenkman and H. Greim (eds.), Cytochrome P450: Handbook in Experimental Pharmacology, Vol. 105, Springer-Verlag, Berlin, pp. 125-144.

Zoller, U. 1992. Distribution and survival of nonionic surfactants in the surface, sea and groundwater of Israel. J. Environ. Sci. Health A27:1521-1533.

Zoller, U. 1994. Non-ionic surfactants in reused water: are activated sludge/soil aquifer treatments sufficient? Water Res. 28:1625-1629.

Zoller, U. and Romano, R. 1983. Determination of nonionic detergents in municipal wastewater. Environ. Intern. 9:55-61.

Table 1. Properties of nonylphenol and octylphenol.[1]

Property/ Specification	Nonylphenol	Octylphenol
CAS Registry Number	84852-15-3	140-66-9
molecular formula	$C_{15}H_{24}O$	$C_{14}H_{22}O$
molecular weight	220.3	206.3
melting point, °C	-8[2]	81-83
boiling point, °C (kPa)	295-320 (101.3)	280-302 (101.3)
colour	colourless to pale straw (liquid)	white (solid)
specific gravity	0.953	0.922
pK_a	10.7[3]	
vapour pressure	$(4.55\pm3.5)\times10^{-3}$ Pa[3]	
solubility, mg/L	5.4[4]	12.6[4]
log K_{ow}	4.48[5], 4.2[6]	4.12[5]
Henry's Law constant (Pa m^3/mol)	11.02[7]	

[1]from Reed (1978) except where noted. Note that "nonylphenol" refers to a mixture, while "octylphenol" refers to 4-(1,1,3,3-tetramethylbutyl)phenol. CAS means Chemical Abstracts Service. Other physical and chemical properties may be found in U.S. Environmental Protection Agency (1985).
[2]Hüls (1994)
[3]Romano (1991).
[4]Ahel and Giger (1993a).
[5]Ahel and Giger (1993b).
[6]McLeese *et al.* (1981).
[7]U.K. Environment Agency (1997).

Table 2. Methods for the determination of APEs in water/wastewater and sediment/sludge samples.

Analyte	Matrix	Extraction	Analysis	Detection limit	Reference
NP	sewage	steam distillation	GC/EI/MS, OV-1		Giger *et al.* (1981)
NP	sewage	continuous liquid-liquid extraction (DCM)	GC/FID, OV-73 GC/EI/MS	10 µg/L	Stephanou and Giger (1982)
NP	sludge	steam distillation (cyclohexane)	GC/FID, OV-73 LC/UV, Lichrosorb NH2	< 80 µg/g	Giger *et al.* (1984)
NP	wastewater, sludge	steam distillation (cyclohexane)	LC/UV, Lichrosorb NH2	0.5 µg/L, water low µg/g, sludge	Ahel and Giger (1985a)
NP	sewage, wastewater	SPE (ODS)	LC/FL, Lichrosorb RP-8		Marcomini *et al.* (1987); Brunner *et al.* (1988)
NP	sediment, sludge	Soxhlet (MeOH), sediment steam distillation, sludge	LC/UV, Lichrosorb RP-8 LC/FL, Hypersil APS	65 ng	Marcomini *et al.* (1987); Brunner *et al.* (1988)
NP	wastewater, sludge	DCM for water acetone/water/ether for sludge	GC/ECD and GC/EI/NCI/MS of PFB and HFB derivatives		Wahlberg *et al.* (1990)
NP	sludge	Soxhlet (hexane)	LC/FL, Hypersil APS		Marcomini (1991)
NP	river water, river sediment	steam distillation	LC/FL, Microsorb CN	0.107 µg/L, water 2.93 µg/g, sediment	Naylor *et al.* (1992)
NP	sludge	steam distillation	LC/UV, Hypersil APS		Sweetman (1994)
NP	sewage, river water	steam distillation	LC/UV, Hypersil APS	10 g/L	Ahel *et al.* (1994b,c, 1996)
NP	sewage	SPE (graphitized carbon black)	LC/FL, C-8		Di Corcia *et al.* (1994)
NP	sludge, river sediment	Soxhlet (DCM/MeOH, 2:1), pentafluorobenzyl ether derivatives	GC/ECD, DB-5 GC/EI/MS, DB-5MS GC/NCI/MS, DB-5MS	2.9 pg/g, ECD 1.4 pg/g, EI/MS 0.3 pg/g, NCI/MS	Chalaux *et al.* (1994)
NP	spiked sediment	SFE (CO_2 modified by MeOH)	LC/FL, RP-18	10 ng	Kreisselmeier and Dürbeck (1997)
NP, OP	wastewater, sediment	DCM, water Soxhlet (isopropanol), sediment	GC/FID, 3% SP-2100, SE-52		Jungclaus *et al.* (1978)

Analyte	Matrix	Extraction	Analysis	Detection limit	Reference
NP, OP	sludge	petroleum ether or SFE (CO_2) with in situ acetylation	GC/EI/MS of acetyl derivatives, HP-5MS	0.1 µg/L or µg/g (NP) 0.01 µg/L or µg/g (OP)	Lee and Peart (1995)
NP, OP	river water	SPE (ODS)	GC/EI/MS, DB-5	30-200 ng/L (NP) 50-250 ng/L (OP)	Blackburn and Waldock (1995)
NP, OP	wastewater	SPE (ODS)	GC/EI/MS	0.5 µg/L	Paxéus (1996)
OP and BrOP	sewage	diethyl ether, methylation	GC/EI/MS, DB-5	0.005 µM	Ball *et al.* (1989)
NP1EO	sludge	Soxhlet (hexane)	LC/FL, Hypersil APS		Marcomini *et al.* (1991)
NP1EO, NP2EO	wastewater, sludge	steam distillation	LC/UV, Lichrosorb-NH_2	0.5 µg/L, water low µg/g, sludge	Ahel and Giger (1985a)
OP1EO/OP2EO NP1EO/NP2EO	wastewater	SPE (ODS)	GC/EI/MS, DB-5	0.5 µg/L	Paxéus (1996)
NPnEO	wastewater	SPE (ODS)	LC/UV, Bondapak C-18 FD/MS	low µg/L	Otsuki and Shiraishi (1979)
NPnEO	sewage, wastewater	SPE (ODS)	LC/FL, Lichrosorb RP-8	4 µg/L	Marcomini *et al.* (1987); Brunner *et al.* (1988)
NPnEO	sediment, sludge	Soxhlet (MeOH), sediment steam distillation, sludge	LC/UV, Lichrosorb RP-8 LC/FL, Hypersil APS	95 ng	Marcomini and Giger (1987); Brunner *et al.* (1988)
NPnEO	wastewater	centrifugal partition chrom.	UV and FL detection	0.5-3 mg/L	Menges *et al.* (1992)
NPnEO	sewage	SPE (ODS)	LC/FL, Lichrosorb RP-18		Marcomini *et al.* (1993)
NPnEO	sewage	SPE (graphitized carbon black)	LC/FL, C-8		Di Corcia *et al.* (1994)
NPnEO	wastewater	SPE (graphitized carbon black)	LC/API/ES/MS		Crescenzi *et al.* (1995)
NPnEO and halogenated derivatives	raw and tap water	SPE (granular activated carbon), then Soxhlet (DCM)	GC/EI/MS, BP-5, FAB/MS LC, Lichrosorb RP-18, FAB/MS	qualitative	Ventura *et al.* (1988, 1989)
NPnEO (n = 1-3)	sewage	steam distillation	GC/EI/MS, OV-1		Giger *et al.* (1981)
NPnEO (n = 1-3)	sewage	continuous liquid-liquid extraction with dichloromethane (DCM)	GC/FID, OV-73 GC/EI/MS, OV-73	10 µg/L	Stephanou and Giger (1982)
NPnEO (n = 1-6)	wastewater,	DCM	GC/ECD, GC/EI/NCI/MS of		Wahlberg *et al.* (1990)

Analyte	Matrix	Extraction	Analysis	Detection limit	Reference
	sludge	acetone/water/ether	PFB and HFB derivatives		
NPnEO (n = 1-13)	wastewater	ethyl acetate sublation	LC/UV, Resolve CN		Scarlet *et al.* (1994)
NPnEO (n = 1-17)	river water, sediment	steam distillation (NP1EO) SPE (NP2EO - NP17EO)	LC/FL, Microsorb CN	0.067-1.58 µg/L (water), 2.26 µg/g (sediment)	Naylor *et al.* (1992)
NPnEO (n = 1-17)	sludge	SFE (CO_2 modified with water)	LC/FL, Hypersil APS	0.2 to 2 µg/g	Lee *et al.* (1997)
NPnEO (n = 1-18)	wastewater	SPE (ODS and ion exchange resins)	LC/FL, Microsorb CN		Kubeck and Naylor (1990)
NP1EO, NP2EO NPnEO (n = 3-20)	sewage, river water	steam distillation (lower NPnEO) solvent sublation (higher NPnEO)	LC/UV, Hypersil APS	10 ng/L	Ahel *et al.* (1994b;c; 1996)
NPnEO/OPnEO (n = 2-8)	drinking water	continuous liquid-liquid extraction (DCM)	LC/PB/MS, C-18	1 ng/L	Clark *et al.* (1992)
NPnEO (n=13) Triton X-100 (OP9.4EO)	spiked sediment	SFE (CO_2 modified with MeOH)	LC/FL, RP-18	10 ng	Kreisselmeier and Dürbeck (1997)
OPnEO/NPnEO (n = 1-16)	sewage	SPE (graphitized carbon black)	LC/FL and LC/API/ES/MS, C-18	5 µg/L	Mackay *et al.* (1997)
NPnEO/OPnEO (n = 1-18)	wastewater	solvent sublation	LC/UV, Lichrosorb-NH_2, Hypersil APS	1-3 µg/L	Ahel and Giger (1985b)
OPnEO (n = 1-5)	wastewater	DCM	GC/PCI/MS, DB-5	qualitative	Stephanou (1984a)
OPnEO (n = 1-5) and Br derivatives	sewage	diethyl ether	GC/EI/MS, DB-5	0.005 µM	Ball *et al.* (1989)
Triton X-100 (OP9.4EO)	water	SPME (Carbowax/template resin coating)	LC/UV, Supelcosil LC-NH_2	low µg/L	Boyd-Boland and Pawliszyn (1996)
APEO	river water	SPE (ODS and SAX)	LC/FL, TMS LC/APCI/MS, TMS	0.05 µg/L	Scullion *et al.* (1996)
APEO	wastewater	solvent sublation	LC/FL, Spherisorb silica	100 µg/L	Ibrahim and Wheals (1996a)
APnEO (n = 1-16)	sewage	solvent sublation	LC/FL, Zorbax-NH_2, Partisil 5 PAC	0.2 ng of each APEO oligomer	Holt *et al.* (1986)

Analyte	Matrix	Extraction	Analysis	Detection limit	Reference
NP1EC, NP2EC	sewage	$CHCl_3$ or gaseous stripping, methylation	GC/EI/MS, SE-54 LC/UV, Lichrosorb NH_2	1 µg/L	Ahel *et al.* (1987)
NP1EC, NP2EC	sewage	SPE (ODS)	LC/FL, Lichrosorb RP-18		Marcomini *et al.* (1993)
NP1EC, NP2EC	sewage, river water	$CHCl_3$	LC/UV, Lichrosorb NH_2	1 µg/L (1994) 100 ng/L (1996)	Ahel *et al.* (1994b;c; 1996)
NP1EC, NP2EC	sludge	SFE (CO_2 modified by water), methylation	GC/EI/MS, HP-5MS	0.5 µg/g	Lee *et al.* (1997)
NP1EC, OP1EC	sewage	SPE (ODS), methylation	GC/EI/MS, HP-5MS	0.5 µg/L (NP1EC) 0.05 µg/L (OP1EC)	Lee *et al.* (1998)
brominated OP1EC	ground water	DCM, methylation	GC/EI/MS, DB-5		Fujita and Reinhard (1997)
NPnEC	raw and drinking water	SPE (XAD-2) or granular activated carbon	FAB/MS and FAB/MS/MS	qualitative	Ventura *et al.* (1991; 1992)
NPnEC (n = 1-4)	sewage, paper mill effluent	SPE (SAX), methylation	GC/PCI/MS, SE-54	0.2-2 µg/L	Field and Reed (1996)
NPnEC (n = 1-4) and halogenated derivatives	raw and tap water	granular activated carbon then Soxhlet (DCM)	GC/EI/MS, BP-5 FAB/MS	qualitative	Ventura *et al.* (1988)
NPnEC (n = 1-10)	sewage	SPE (graphitized carbon black)	LC/FL, C-8		Di Corcia *et al.* (1994)
OPnEC (n = 1-6) and Br derivatives	sewage	diethyl ether, methylation	GC/EI/MS, DB-5	0.005 µM	Ball *et al.* (1989)
brominated APEC	wastewater	SPE (XAD-8), methylation	GC/EI/MS, SE-52		Reinhard *et al.* (1982)

Abbreviations: APCI = atmospheric pressure chemical ionization; API = atmospheric pressure ionization; $CHCl_3$ = chloroform; DCM = dichloromethane; ECD = electron capture detector; EI = electron impact; FAB = fast atom bombardment; FD = field desorption; FID = flame ionization detector; FL = fluorescence; GC = gas chromatography; LC = liquid chromatography; MeOH = methanol; MS = mass spectrometry; NCI = negative ion chemical ionization; ODS = octadecylsilane; PCI = positive ion chemical ionization; PFB = pentafluorobenzyl bromide; SAX = strong anion exchange; SFE = supercritical fluid extraction; SPE = solid phase extraction; UV = ultraviolet.

Table 3. Examples of commercial APEO preparations.

Product name	Type	Average no. of ethoxy units	Manufacturer
Igepal CA-210	OPEO*	2	GAF Corp.
Igepal CA-520	OPEO	5	
Igepal CA-720	OPEO	12	
Igepal CO-210	NPEO	2	
Igepal CO-520	NPEO	5	
Igepal CO-720	NPEO	12	
Igepal CO-890	NPEO	40	
Igepal CO-990	NPEO	100	
Imbentin-N/7A	NPEO	5	W. Kolb, AG
Imbentin-N/200	NPEO	20	
Imbentin-O/050	OPEO	5	
Imbentin-O/200	OPEO	20	
Lissapol TN 450	NPEO	8-9	ICI Chemicals
Lissapol TN XP	NPEO	8-9	
Marlophen 810	NPEO	11	Chemisch Werke Hüls
Marlophen 83	NPEO	3.15	
Surfonic N-40	NPEO	4	Texaco
Surfonic N-95	NPEO	9	
Synperonic OP10	OPEO	10	ICI Chemicals
Tergitol NP15	NPEO	15	Union Carbide
Tergitol NP40	NPEO	40	
Terric N2	NPEO	2	ICI Chemicals
Terric N4	NPEO	4	
Triton N-101	NPEO	9.5	Rohm and Haas
Triton X-15	OPEO	1.2	
Triton X-35	OPEO	3.0	
Triton X-45	OPEO	4.3	
Triton X-114	OPEO	7.4	
Triton X-100	OPEO	9.4	
Triton X-102	OPEO	11.6	
Triton X-165	OPEO	14.7	

* OPEO = octylphenol ethoxylates; NPEO = nonylphenol ethoxylates

Table 4. Amount of nonylphenol and its ethoxylates produced, imported, exported and available for use in Canada in 1995 and 1996.

Amount	Year	
	1995	1996
	tonnes NPE	
Produced in Canada	30,000	23,600
Imported to Canada	3,800	4,600
Exported from Canada	12,300	11,000
Available for use in Canada	**21,500**	**17,200**

Note:
Amount available for use = Amount produced + amount imported - amount exported. Results are based on survey responses received from 189 companies that reported involvement with NPEs of 1 tonne or more.

Source: Environment Canada (1998).

Table 5. Releases of nonylphenol and its ethoxylates to various environmental media, by industry sector in Canada in 1996.

Industry Sector	# of sites	Total Released to				Total, Range of kg released from all sites
		Air	Stream	Wastewater	Landfill/or Deep well*	
		kg				
Formulators and distributors of surfactants	4		-			25,000-60,000
Industrial users of cleaning products, degreasers and detergents	3	-			-	25,000-60,000
Producers of paints, protective coatings, resins and adhesives	19		-			5,000-9,999
Formulators of industrial, institutional and domestic cleaning products, degreasers and detergents	22					100-4,999
Pulp and paper mills	3	-		-	-	100-4,999
Oil and gas recovery	2		-	-		100-4,999
Production of wastewater treatment productsH	2		-			100-4,999
Formulators and distributors of products for the pulp and paper industry	6		-			100-4,999
Miscellaneous	4					100-4,999
TOTAL	65					

Notes:

* = indicates release is to deep well.

# of sites: in some cases several sites may be owned by one company

H = products used in pulp and paper, steel, oil and gas, hydropower and wastewater treatment facilities.

Textile mills are greatly under-represented. In 1996, approximately 227 textile mills operated in Canada, however only 97 received the Section 16 survey and of these only 22 responded that they were involved with NPEs above the 1 tonne trigger quantity. Releases of NPEs were reported to be zero by the 22 respondents, although detectable levels of NPEs have been found in untreated textile mill effluents. The lack of release information from the textile mills is a shortcoming in this assessment

Source: Environment Canada (1998). For reasons of confidentiality, only ranges are reported.

Table 6. Occurrence of alkylphenols, alkylphenol polyethoxylates and alkylphenol polycarboxylates in the Canadian environment.

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
Nonylphenolics				
4-NP	beluga whale (*Delphinapterus leucas*)	< 0.020 - 0.12	St. Lawrence River	Bennie (1998)
4-NP	carp (*Cyprinus carpio*)	< 0.020 - 0.020	Hamilton Harbour	Bennie (1998)
4-NP	rainbow trout (*Oncorhynchus mykiss*)	< 0.020 - 0.043	Lake Ontario	Bennie (1998)
4-NP	fresh water	< 0.010 - 0.92	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
4-NP	fresh water	< 0.010	St. Lawrence River	Bennie *et al.* (1997a)
NP	fresh water	11 - 2600	Canagagigue Creek, Ontario	Carey *et al.* (1981)
4-NP	oil refinery effluent	< 0.020 (n = 2)		Bennie (1998)
4-NP	pulp mill effluent	< 0.020 - 26.2	pulp mills	Bennie (1998)
4-NP	sediment	0.17 - 72	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
4-NP	sediment	0.36 - 0.72	St. Lawrence River	Bennie *et al.* (1997a)
4-NP	sediment	< 0.005 - 0.064	Lower Fraser River basin	Brewer *et al.* (1998)
4-NP	sediment	< 0.0015 - 0.021	Thompson River sub-basin	Brewer *et al.* (1998)
4-NP	sediment	< 0.002 - 0.570	Upper Fraser River basin	Brewer *et al.* (1998)
4-NP	sediment	41.1	Hamilton Harbour	Lee and Peart (1995)
4-NP	sediment	1.29 - 6.73	Hamilton Harbour	Lee and Peart (1995)
4-NP	sediment	0.78	Humber River, Ontario	Lee and Peart (1995)
4-NP	sediment	0.29 - 1.28	Kaministiquia River, Ontario	Lee and Peart (1995)
4-NP	sludge-amended soil	2.72	Ontario	Water Technology International Corporation (1998a)
4-NP	MWWTP final effluent	< 0.020 - 13	Canadian MWWTPs	Bennie *et al.* (1998)
4-NP	MWWTP final effluent	0.8 - 15.1	Toronto area MWWTPs	Lee and Peart (1995)
4-NP	MWWTP final effluent	0.99 - 1.85	Canadian MWWTPs	Water Technology International Corporation (1998b)
4-NP	MWWTP primary effluent	2.8 - 30	Toronto area MWWTPs	Lee and Peart (1995)
4-NP	MWWTP raw sewage	0.69 - 155	Canadian MWWTPs	Bennie *et al.* (1998)
4-NP	MWWTP raw sewage	6.0 - 21	Canadian MWWTPs	Water Technology International Corporation (1998b)
4-NP	MWWTP sludge	8.4 - 850	Canadian MWWTPs	Bennie *et al.* (1998)
4-NP	MWWTP sludge	137 - 470	Toronto area MWWTPs	Lee and Peart (1995)
4-NP	MWWTP sludge	290 - 818	Canadian MWWTPs	Water Technology International Corporation (1998b)
4-NP	textile mill effluent	1.1 - 13.3	Canadian textile mills	Bennie (1998)
NP1EO	fresh water	< 0.020 - 7.8	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
NP1EO	fresh water	< 0.020 - 0.15	St. Lawrence River	Bennie *et al.* (1997a)
NP1EO	pulp mill effluent	< 0.015 - 25.9	Canadian pulp mills	Bennie (1998)
NP1EO	sediment	< 0.015 - 38	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
NP1EO	sediment	< 0.015 - 2.6	St. Lawrence River	Bennie *et al.* (1997a)
NP1EO	sludge-amended soil	trace	Ontario	Water Technology International Corporation (1998a)

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
NP1EO	MWWTP final effluent	0.072 - 26	Canadian MWWTPs	Bennie *et al.* (1998)
NP1EO	MWWTP final effluent	1.9, 10	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP1EO	MWWTP raw sewage	2.9 - 43	Canadian MWWTPs	Bennie *et al.* (1998)
NP1EO	MWWTP raw sewage	12.3, 40	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP1EO	MWWTP sludge	3.9 - 437	Canadian MWWTPs	Bennie *et al.* (1998)
NP1EO	MWWTP sludge	28 - 304	Canadian MWWTPs	Lee *et al.* (1997)
NP1EO	MWWTP sludge	136, 140	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP1EO	textile mill effluent	0.74 - 260	Canadian textile mills	Bennie (1998)
NP2EO	fresh water	< 0.020 - 10	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
NP2EO	fresh water	< 0.020 - 0.023	St. Lawrence River	Bennie *et al.* (1997a)
NP2EO	pulp mill effluent	< 0.015 - 35.9	Canadian pulp mills	Bennie (1998)
NP2EO	sediment	< 0.015 - 6.0	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
NP2EO	sediment	< 0.015 - 0.48	St. Lawrence River	Bennie *et al.* (1997a)
NP2EO	sludge-amended soil	trace	Ontario	Water Technology International Corporation (1998a)
NP2EO	MWWTP final effluent	0.099 - 21	Canadian MWWTPs	Bennie *et al.* (1998)
NP2EO	MWWTP final effluent	1.5, 8.9	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP2EO	MWWTP raw sewage	0.26 - 24	Canadian MWWTPs	Bennie *et al.* (1998)
NP2EO	MWWTP raw sewage	38, 10.8	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP2EO	MWWTP sludge	1.5 - 297	Canadian MWWTPs	Bennie *et al.* (1998)
NP2EO	MWWTP sludge	4 - 118	Canadian MWWTPs	Lee *et al.* (1997)
NP2EO	MWWTP sludge	10.6, 22	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP2EO	textile mill effluent	0.64 - 590	Canadian textile mills	Bennie (1998)
NPnEO (Igepal CO 610)	unbleached pulp mill whitewater	310	Canadian pulp mill	Sithole and Allen (1989)
NPnEO (n = 3 - 17)	sludge-amended soil	trace	Ontario	Water Technology International Corporation (1998a)
NPnEO (n = 3 - 17)	MWWTP final effluent	4.4, 3.9	Canadian MWWTPs	Water Technology International Corporation (1998b)
NPnEO (n = 3 - 17)	MWWTP raw sewage	1300, 534	Canadian MWWTPs	Water Technology International Corporation (1998b)
NPnEO (n = 3 - 17)	MWWTP sludge	9 - 169	Canadian MWWTPs	Lee *et al.* (1997)
NPnEO (n = 3 - 17)	MWWTP sludge	60, 27	Canadian MWWTPs	Water Technology International Corporation (1998b)
NPnEO (n = 3 - 17)	textile mill effluent	50.2 - 8600	Canadian textile mills	Bennie (1998)
NP1EC	sludge-amended soil	< 0.538	Ontario	Water Technology International Corporation (1998a)
NP1EC	MWWTP final effluent	3.2 - 703	Canadian MWWTPs	Lee *et al.* (1998)
NP1EC	MWWTP final effluent	1.9 - 22	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP1EC	MWWTP primary effluent	2.4 - 17.7	Canadian MWWTPs	Lee *et al.* (1998)
NP1EC	MWWTP raw sewage	0.9 - 8.3	Canadian MWWTPs	Lee *et al.* (1998)
NP1EC	MWWTP raw sewage	2.0 - 15	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP1EC	MWWTP sludge	< 0.5 - 25	Canadian MWWTPs	Lee *et al.* (1997)
NP1EC	MWWTP sludge	2.8 - 6.6	Canadian MWWTPs	Water Technology International Corporation (1998b)

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
NP1EC	MWWTP sludge	11.8	Canadian MWWTP	Water Technology International Corporation (1998a)
NP2EC	sludge-amended soil	< 0.538	Ontario	Water Technology International Corporation (1998a)
NP2EC	MWWTP final effluent	11.1 - 565	Canadian MWWTPs	Lee *et al.* (1998)
NP2EC	MWWTP final effluent	11 - 32	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP2EC	MWWTP primary effluent	3.5 - 39.3	Canadian MWWTPs	Lee *et al.* (1998)
NP2EC	MWWTP raw sewage	1.7 - 20.1	Canadian MWWTPs	Lee *et al.* (1998)
NP2EC	MWWTP raw sewage	1.9 - 35	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP2EC	MWWTP sludge	< 0.5 - 38	Canadian MWWTPs	Lee *et al.* (1997)
NP2EC	MWWTP sludge	7.1 - 23	Canadian MWWTPs	Water Technology International Corporation (1998b)
NP2EC	MWWTP sludge	11.8	Canadian MWWTP	Water Technology International Corporation (1998a)
Octylphenolics				
OP	beluga whale (*Delphinapterus leucas*)	< 0.010	St. Lawrence River	Bennie (1998)
OP	carp (*Cyprinus carpio*)	< 0.010	Hamilton Harbour	Bennie (1998)
OP	rainbow trout (*Oncorhynchus mykiss*)	< 0.010	Lake Ontario	Bennie (1998)
OP	fresh water	< 0.005 - 0.47	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
OP	fresh water	< 0.005 - 0.013	St. Lawrence River	Bennie *et al.* (1997a)
OP	pulp mill effluent	< 0.005	Canadian pulp mills	Bennie (1998)
OP	sediment	0.010 - 1.8	Ontario lakes, rivers and harbours	Bennie *et al.* (1997a)
OP	sediment	< 0.010 - 0.28	St. Lawrence River	Bennie *et al.* (1997a)
OP	sediment	0.07 - 0.40	Hamilton Harbour	Lee and Peart (1995)
OP	sediment	< 0.005	Humber River, Ontario	Lee and Peart (1995)
OP	sediment	< 0.005 - 0.028	Kaministiquia River, Ontario	Lee and Peart (1995)
OP	sediment	0.91	Hamilton Harbour	Lee and Peart (1995)
OP	MWWTP final effluent	< 0.005 - 0.37	Canadian MWWTPs	Bennie *et al.* (1998)
OP	MWWTP final effluent	0.12 - 1.7	Toronto area MWWTPs	Lee and Peart (1995)
OP	MWWTP final effluent	0.016 - 0.13	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP	MWWTP primary effluent	0.41 - 2.5	Toronto area MWWTPs	Lee and Peart (1995)
OP	MWWTP raw sewage	< 0.005 - 21	Canadian MWWTPs	Bennie *et al.* (1998)
OP	MWWTP raw sewage	0.61 - 1.22	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP	MWWTP sludge	< 0.010 - 20	Canadian MWWTPs	Bennie *et al.* (1998)
OP	MWWTP sludge	9.2 - 12.1	Toronto area MWWTPs	Lee and Peart (1995)
OP	MWWTP sludge	11 - 15	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP	MWWTP sludge	14.9	Canadian MWWTP	Water Technology International Corporation (1998a)
OP	textile mill effluent	0.26 - 9.0	Canadian textile mills	Bennie (1998)
OPnEO (Triton X-100)	textile mill effluent	5400 - 50000	Canadian textile mills	Rutherford *et al.* (1992)
OP1EC	sludge-amended soil	trace	Ontario	Water Technology International Corporation (1998a)
OP1EC	MWWTP final effluent	0.29 - 4.61	Canadian MWWTPs	Lee *et al.* (1998)

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
OP1EC	MWWTP final effluent	0.050 - 7.37	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP1EC	MWWTP primary effluent	0.18 - 1.18	Canadian MWWTPs	Lee *et al.* (1998)
OP1EC	MWWTP raw sewage	0.70 - 1.14	Canadian MWWTPs	Lee *et al.* (1998)
OP1EC	MWWTP raw sewage	0.56 - 1.33	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP1EC	MWWTP sludge	0.05 - 1.2	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP1EC	MWWTP sludge	< 0.538	Canadian MWWTP	Water Technology International Corporation (1998a)
OP2EC	sludge-amended soil	trace	Ontario	Water Technology International Corporation (1998a)
OP2EC	MWWTP final effluent	0.54 - 7.74	Canadian MWWTPs	Lee *et al.* (1998)
OP2EC	MWWTP final effluent	0.12 - 4.0	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP2EC	MWWTP primary effluent	0.12 - 1.60	Canadian MWWTPs	Lee *et al.* (1998)
OP2EC	MWWTP raw sewage	0.14 - 1.62	Canadian MWWTPs	Lee *et al.* (1998)
OP2EC	MWWTP raw sewage	0.24 - 2.3	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP2EC	MWWTP sludge	0.04 - 0.30	Canadian MWWTPs	Water Technology International Corporation (1998b)
OP2EC	MWWTP sludge	0.31	Canadian MWWTP	Water Technology International Corporation (1998a)

Table 7. Range of Concentrations of NPEs and OPEs in the Canadian Environment (total number of sites, total number of samples) [2]

Environmental Compartment	Site Type		4-NP	NP1EO	NP2EO	NP3-17EO	NP1EC	NP2EC	4-t-OP	OP1EC	OP2EC
Effluents (µg/L)	Textiles	untreated	2.68-13.33 (2,5)	37.17-257.09 (2,5)	106.31-591.98 (2,5)	798.42-8811.24 (2,5)	<0.45 (1,2)	<0.45 (1,2)	1.85-9.01 (2,5)	0.68-0.83 (1,2)	1.26-2.53 (1,2)
		on-site 2° treatment	0.09-3.56 (2,4)	1.12-4.10 (1,2)	0.93-3.92 (1,2)	2.07-315.45 (2,3)	0.74-5.2 (2,4)	<0.45-55.13 (2,4)	<0.02-0.22 (2,4)	<0.05-0.43 (2,4)	<0.05-11.81 (2,4)
		going to MWWTP	0.23-25.62 (9,14)	0.74-69.15 (10,14)	0.64-284.51 (10,14)	50.18-5767.65 (10,14)	<0.45-1.90 (5,7)	<0.45-2.80 (5,7)	<0.02-0.65 (9,14)	<0.05-0.08 (5,7)	<0.05 (5,7)
	Pulp and Paper	prior to 1998	<0.02-26.20 (14,33)	<0.02-3780.00 (13,32)	<0.02-67.84 (14,33)	-	-	-	<0.005 (13,29)	-	-
		after 1998	<0.10-4.3 (19,19)	<0.10-6.90 (3,3)	<0.10-35.60 (3,3)	5.90-28.80 (3,3)	<1.00-10.13 (15,15)	<1.00-32.32 (15,15)	<0.005 -0.06 (19,19)	<0.10-3.95 (15,15)	<0.10-3.22 (15,15)
	MWWTP	primary	<0.02-62.08 (8,21)	0.07-56.13 (10,26)	0.34-36.33 (10,26)	4.81-735.20 (8,22)	1.17-11.00 (3,7)	1.01-5.20 (3,7)	<0.005 -1.23 (9,21)	0.19-0.46 (3,7)	0.19-0.37 (3,7)
		secondary	0.12-4.79 (21, 54)	<0.02-43.37 (20, 46)	<0.02-32.62 (20,46)	1.00-52.82 (16,36)	2.15-74.97 (14,34)	2.15-45.40 (14,34)	<0.005 -0.57 (21,54)	0.19-9.98 (14,34)	0.19-13.09 (14,34)
		tertiary	<0.02-3.20 (7,37)	0.30-26.4 (7,37)	0.25-12.45 (7,37)	0.40-18.00 (6,35)	2.15-48.58 (6,34)	2.15-59.46 (6,34)	<0.005 -0.28 (7,37)	<0.05-29.31 (6,34)	0.12-9.04 (6,34)
		lagoon	0.75-2.15 (5,5)	0.34-0.90 (5,5)	0.03-0.90 (5,5)	1.00-2.10 (4,4)	2.15-2.6 (4,4)	2.15-3.00 (4,4)	0.08-0.12 (5,5)	0.19 (4,4)	0.19 (4,4)
Aquatic (µg/L)	Rivers		<0.02-4.25 (25,90)	<0.02-2.30 (12, 51)	<0.02-2.45 (12,51)	0.11-17.56 (3, 27)	0.44-3.17 (1,37)	0.81-4.30 (1,37)	<0.003-0.61 (25,90)	0.09-0.51 (1,37)	0.07-0.30 (1,37)
	Lakes		<0.02-0.06 (5,5)	<0.02-5.07 (4,4)	<0.02 (4,4)	-	-	-	<0.003 (5,5)		
	Harbours		<0.02-0.98 (12,31)	<0.02-10.29 (12,26)	<0.02-10.43 (12,26)	-	-	-	<0.003-0.096 (12,31)	-	-
Benthic (µg/g)			<0.02-72.20 (23,58)	<0.02-38.12 (6,14)	<0.02-6.02 (6,14)	0.02-0.17 (1, 4)	-	-	<0.01-23.7 (20,52)	-	-
Soil/Sludge			0.74-1260	2.90-1825.29	1.52-297.21	0.43-215	<0.30-8.70	<0.30-26.0	<0.04-19.97	0.03-1.2	<0.03-2.33

2 Bennie 1998; Bennie *et al.* 1998; Water Technology International Corporation 1998a and 1998b; Lee and Peart 1995; Lee *et al.* 1998; Bennie *et al.* 1997a and 1997b; Brewer *et al.* 1998; Bennet and Metcalfe, 1998; Lee *et al.* 1997

Environmental Compartment	Site Type	4-NP	NP1EO	NP2EO	NP3-17EO	NP1EC	NP2EC	4-t-OP	OP1EC	OP2EC
(μg/g)		(30, 107)	(28,90)	(28,90)	(28,90)	(17,66)	(17,66)	(30,106)	(15,64)	(15,64)

Table 8. Occurrence of alkylphenols, alkylphenol polyethoxylates and alkylphenol polycarboxylates in the environment outside Canada.

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
Nonylphenolics				
NP	chemical plant effluent	600	Philadelphia, PA, U.S.A.	Sheldon and Hites (1979)
NP	drinking water	N.D. - 0.14	Barcelona, Spain	Guardiola *et al.* (1991)
NP	drinking water	N.D.	Philadelphia, PA, U.S.A.	Sheldon and Hites (1979)
NP	duck (*Anas boscas*)	< 0.03 - 1.20	Swiss rivers	Ahel *et al.* (1993)
4-NP	estuarine water	< 0.03 - 5.2	UK estuaries	Blackburn and Waldock (1995)
4-NP	estuarine water	N.D. - 15	Thames Estuary, UK	Waldock and Thain (1986)
NP	barbel (*Barbus barbus*)	< 0.03 - 0.98	Swiss rivers	Ahel *et al.* (1993)
NP	rainbow trout (*Oncorhynchus mykiss*)	0.15 - 1.6	Swiss rivers	Ahel *et al.* (1993)
NP	chub (*Squalius cephalus*)	0.98 - 1.4	Swiss rivers	Ahel *et al.* (1993)
NP	fresh water	< 0.5 - 1.5	Swiss rivers	Ahel and Giger (1985a)
4-NP	fresh water	< 0.3 - 45	Swiss rivers	Ahel *et al.* (1994a)
NP	fresh water	0.7 - 26	Swiss rivers	Ahel *et al.* (1996)
4-NP	fresh water	< 0.9 - 180	UK rivers	Blackburn and Waldock (1995)
NP	fresh water	N.D. - 11.7	Llobregat River, Spain	Guardiola *et al.* (1991)
NP	fresh water	< 0.11 - 0.64	U.S. rivers	Naylor *et al.* (1992)
NP	fresh water	0.04 - 2	Delaware River, U.S.A.	Sheldon and Hites (1978)
NP	fresh water	N.D. - 1	Delaware River, U.S.A.	Sheldon and Hites (1979)
NP	fresh water	1.2 - 3.4	Lake Geneva, Switzerland	Stephanou (1985)
NP	groundwater	< 0.1 - 33	Glattfelden and Engelburg, Switzerland	Ahel *et al.* (1996)
4-NP	groundwater	0.79	Falmouth, MA, U.S.A.	Barber *et al.* (1988)
NP	marine resuspended solid material	N.D. - 5.6	lagoons of Venice, Italy	Marcomini *et al.* (1990)
NP	marine sediment	0.005 - 0.042	lagoons of Venice, Italy	Marcomini *et al.* (1990)
4-NP	municipal landfill leachate	< 10 - 107	Gryta, Vasteras, Sweden	Oman and Hynning (1993)
4-NP	macrophyte (*Cladophera glomerata*)	3.5 - 38	Swiss rivers	Ahel *et al.* (1993)
4-NP	macrophyte (*Fontinalis antipyretica*)	4.2	Swiss rivers	Ahel *et al.* (1993)
4-NP	macrophyte (*Potamogeton crispus*)	2.5	Swiss rivers	Ahel *et al.* (1993)
4-NP	sediment	0.19 - 13.1	Swiss rivers	Ahel *et al.* (1994a)
NP	sediment	6 - 69	Mediterranean Sea near Barcelona	Chalaux *et al.* (1994)
NP	sediment	19 - 44	Nile estuary, Egypt	Chalaux *et al.* (1994)
NP	sediment	0.9	Rhine River, Switzerland	Marcomini and Giger (1987)
NP	sediment	< 0.003 - 2.96	U.S. rivers	Naylor *et al.* (1992)
NP	sediment pore water	55	Illinois River at Chicago, IL, U.S.A.	Schubauer-Berigan and Ankley (1991)
NP	sludge-amended soil	0.5	Liebefeld, Switzerland	Hani (1990)
NP	sludge-amended soil	1.6	Switzerland	Marcomini and Giger (1987)
NP	MWWTP final effluent	8 - 11	Swiss MWWTPs	Ahel and Giger (1985a)
NP	MWWTP final effluent	1 - 14	Swiss MWWTPs	Ahel *et al.* (1987)
4-NP	MWWTP final effluent	< 0.2 - 330	UK MWWTPs	Blackburn and Waldock (1995)
4-NP	MWWTP final effluent	2.7	Swiss MWWTPs	Brunner *et al.* (1988)
NP	MWWTP final effluent	0.7 - 2.6	Italian MWWTP	Di Corcia *et al.* (1994)
NP	MWWTP final effluent	0.8 - 2.5	High Point, NC, U.S.A. MWWTPs	Kubeck and Naylor (1990)
NP	MWWTP final effluent	30	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1987)
NP	MWWTP final effluent	2.7 - 5.6	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP	MWWTP final effluent	0.5 - 3	Swedish MWWTPs	Paxéus (1996)

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
NP	MWWTP final effluent	40	Philadelphia, PA, U.S.A.	Sheldon and Hites (1979)
NP	MWWTP final effluent	35.8 - 69.3	Swiss MWWTPs	Stephanou (1985)
NP	MWWTP final effluent	N.D. - 35	Swiss MWWTPs	Stephanou and Giger (1982)
4-NP	MWWTP final effluent	< 0.002 - 0.021	UK MWWTPs	Waldock and Thain (1986)
NP	MWWTP final effluent	1587	U.S.A.	Amato and Wayment (1998)
NP	MWWTP primary effluent	21 - 49	Swiss MWWTPs	Ahel *et al.* (1987)
4-NP	MWWTP primary effluent	14	Swiss MWWTPs	Brunner *et al.* (1988)
NP	MWWTP primary effluent	150	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1987)
NP	MWWTP primary effluent	15 - 15	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP	MWWTP raw sewage	14	Swiss MWWTPs	Ahel and Giger (1985a)
NP	MWWTP raw sewage	21 - 57	Swiss MWWTPs	Ahel *et al.* (1987)
4-NP	MWWTP raw sewage	20	Swiss MWWTPs	Brunner *et al.* (1988)
NP	MWWTP raw sewage	2.7 - 7.5	Italian MWWTP	Di Corcia *et al.* (1994)
NP	MWWTP raw sewage	280	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1987)
NP	MWWTP raw sewage	21 - 50	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP	MWWTP sludge	1000	Swiss MWWTPs	Ahel and Giger (1985a)
4-NP	MWWTP sludge	1500	Swiss MWWTPs	Brunner *et al.* (1988)
NP	MWWTP sludge	20 - 350	Barcelona, Spain	Chalaux *et al.* (1994)
NP	MWWTP sludge	370	Los Angeles, CA, U.S.A.	Chalaux *et al.* (1994)
4-NP	MWWTP sludge	890	Swiss MWWTP	Giger *et al.* (1984)
4-NP	MWWTP sludge	22.1 - 1193	German MWWTP	Jobst (1995)
NP	MWWTP sludge	67 - 214	Brandenburg, Germany	Kujawa *et al.* (1997)
NP	MWWTP sludge	1200	Swiss MWWTPs	Marcomini and Giger (1987)
NP	MWWTP sludge	1500 - 1570	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
4-NP	MWWTP sludge	256 - 823	UK MWWTPs	Sweetman (1994)
4-NP	MWWTP sludge	30 - 4000	UK MWWTPs	Waldock and Thain (1986)
NP1EO	drinking water	0.077	New Jersey, U.S.A.	Clark *et al.* (1992)
NP1EO	drinking water	N.D. - 1.10	Barcelona, Spain	Guardiola *et al.* (1991)
NP1EO	duck (*Anas boscas*)	< 0.03 - 2.10	Swiss rivers	Ahel *et al.* (1993)
NP1EO	barbel (*Barbus barbus*)	0.06 - 3.1	Swiss rivers	Ahel *et al.* (1993)
NP1EO	rainbow trout (*Oncorhynchus mykiss*)	0.42 - 7.0	Swiss rivers	Ahel *et al.* (1993)
NP1EO	chub (*Squalius cephalus*)	0.18 - 1.8	Swiss rivers	Ahel *et al.* (1993)
NP1EO	fresh water	< 0.5 - 16	Swiss rivers	Ahel and Giger (1985a)
NP1EO	fresh water	< 3 - 69	Swiss rivers	Ahel *et al.* (1994a)
NP1EO	fresh water	2.0 - 20	Swiss rivers	Ahel *et al.* (1996)
NP1EO	fresh water	N.D. - 4.80	Llobregat River, Spain	Guardiola *et al.* (1991)
NP1EO	fresh water	< 0.06 - 0.60	U.S. rivers	Naylor *et al.* (1992)
NP1EO	fresh water	1.1 - 4.1	Lake Geneva, Switzerland	Stephanou (1985)
NP1EO	groundwater	< 0.1 - 4.9	Glattfelden and Engelburg, Switzerland	Ahel *et al.* (1996)
NP1EO	marine resuspended solid material	0.2 - 6.6	lagoons of Venice, Italy	Marcomini *et al.* (1990)
NP1EO	marine sediment	0.009 - 0.082	lagoons of Venice, Italy	Marcomini *et al.* (1990)
NP1EO	macrophyte (*Cladophera glomerata*)	1.8 - 80	Swiss rivers	Ahel *et al.* (1993)
NP1EO	macrophyte (*Fontinalis antipyretica*)	0.9	Swiss rivers	Ahel *et al.* (1993)
NP1EO	macrophyte (*Potamogeton crispus*)	1.1	Swiss rivers	Ahel *et al.* (1993)
NP1EO	sediment	0.10 - 8.85	Swiss rivers	Ahel *et al.* (1994a)
NP1EO	sediment	0.80	Rhine River, Switzerland	Marcomini and Giger (1987)
NP1EO	sediment	<0.002 - 0.175	U.S. rivers	Naylor *et al.* (1992)
NP1EO	sludge-amended soil	0.40	Swiss MWWTPs	Marcomini and Giger (1987)
NP1EO	MWWTP final effluent	30 - 65	Swiss MWWTPs	Ahel and Giger (1985a)
NP1EO	MWWTP final effluent	4 - 78	Swiss MWWTPs	Ahel *et al.* (1987)

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
NP1EO	MWWTP final effluent	2.6 - 3.8	High Point, NC, U.S.A. MWWTPs	Kubeck and Naylor (1990)
NP1EO	MWWTP final effluent	19 - 41	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP1EO	MWWTP final effluent	1 - 11	Swedish MWWTPs	Paxéus (1996)
NP1EO	MWWTP final effluent	24.9 - 189.4	Swiss MWWTPs	Stephanou (1985)
NP1EO	MWWTP final effluent	N.D. - 133	Swiss MWWTPs	Stephanou and Giger (1982)
NP1EO	MWWTP final effluent	508	U.S.A.	Amato and Wayment (1998)
NP1EO	MWWTP primary effluent	13 - 35	Swiss MWWTPs	Ahel *et al.* (1987)
NP1EO	MWWTP primary effluent	30 - 71	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP1EO	MWWTP raw sewage	18	Swiss MWWTPs	Ahel and Giger (1985a)
NP1EO	MWWTP raw sewage	23 - 140	Swiss MWWTPs	Ahel *et al.* (1987)
NP1EO	MWWTP raw sewage	36 - 69	High Point, NC, U.S.A. MWWTPs	Kubeck and Naylor (1990)
NP1EO	MWWTP raw sewage	30 - 75	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP1EO	MWWTP sludge	79	Swiss MWWTPs	Ahel and Giger (1985a)
NP1EO	MWWTP sludge	60 - 680	Swiss MWWTPs	Brunner *et al.* (1988)
NP1EO	MWWTP sludge	220	Swiss MWWTPs	Marcomini and Giger (1987)
NP1EO	MWWTP sludge	130 - 140	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP2EO	drinking water	0.147	New Jersey, U.S.A.	Clark *et al.* (1992)
NP2EO	drinking water	N.D. - 0.25	Barcelona, Spain	Guardiola *et al.* (1991)
NP2EO	duck (*Anas boscas*)	< 0.03 - 0.35	Swiss rivers	Ahel *et al.* (1993)
NP2EO	barbel (*Barbus barbus*)	< 0.03 - 2.3	Swiss rivers	Ahel *et al.* (1993)
NP2EO	rainbow trout (*Oncorhynchus mykiss*)	0.05 - 3.0	Swiss rivers	Ahel *et al.* (1993)
NP2EO	chub (*Squalius cephalus*)	< 0.03 - 1.4	Swiss rivers	Ahel *et al.* (1993)
NP2EO	fresh water	< 0.5 - 11	Swiss rivers	Ahel and Giger (1985a)
NP2EO	fresh water	< 0.3 - 30	Swiss rivers	Ahel *et al.* (1994a)
NP2EO	fresh water	0.8 - 21	Swiss rivers	Ahel *et al.* (1996)
NP2EO	fresh water	N.D. - 1.90	Llobregat River, Spain	Guardiola *et al.* (1991)
NP2EO	fresh water	< 0.07 - 1.2	U.S. rivers	Naylor *et al.* (1992)
NP2EO	fresh water	1.3 - 5.8	Lake Geneva, Switzerland	Stephanou (1985)
NP2EO	groundwater	< 0.1 - 23	Glattfelden and Engelburg, Switzerland	Ahel *et al.* (1996)
NP2EO	marine resuspended solid material	N.D. - 1.5	lagoons of Venice, Italy	Marcomini *et al.* (1990)
NP2EO	marine sediment	0.003 - 0.020	lagoons of Venice, Italy	Marcomini *et al.* (1990)
NP2EO	macrophyte (*Cladophera glomerata*)	1.3 - 29	Swiss rivers	Ahel *et al.* (1993)
NP2EO	macrophyte (*Fontinalis antipyretica*)	0.6	Swiss rivers	Ahel *et al.* (1993)
NP2EO	macrophyte (*Potamogeton crispus*)	1.9	Swiss rivers	Ahel *et al.* (1993)
NP2EO	sediment	N.D. - 2.72	Swiss rivers	Ahel *et al.* (1994a)
NP2EO	sediment	0.70	Rhine River, Switzerland	Marcomini and Giger (1987)
NP2EO	sludge-amended soil	0.07	Swiss MWWTPs	Marcomini and Giger (1987)
NP2EO	MWWTP final effluent	47 - 77	Swiss MWWTPs	Ahel and Giger (1985a)
NP2EO	MWWTP final effluent	4 - 66	Swiss MWWTPs	Ahel *et al.* (1987)
NP2EO	MWWTP final effluent	15 - 31	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP2EO	MWWTP final effluent	0.5 - 6	Swedish MWWTPs	Paxéus (1996)
NP2EO	MWWTP final effluent	36.6 - 198.4	Swiss MWWTPs	Stephanou (1985)
NP2EO	MWWTP final effluent	N.D. - 70	Swiss MWWTPs	Stephanou and Giger (1982)
NP2E0	MWWTP final effluent	2602	U.S.A.	Amato and Wayment (1998)
NP2EO	MWWTP primary effluent	17 - 55	Swiss MWWTPs	Ahel *et al.* (1987)
NP2EO	MWWTP primary effluent	17 - 55	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP2EO	MWWTP raw sewage	18	Swiss MWWTPs	Ahel and Giger (1985a)
NP2EO	MWWTP raw sewage	30 - 67	Swiss MWWTPs	Ahel *et al.* (1987)
NP2EO	MWWTP raw sewage	16 - 65	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NP2EO	MWWTP sludge	N.D.	Swiss MWWTPs	Ahel and Giger (1985a)

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
NP2EO	MWWTP sludge	20 - 280	Swiss MWWTPs	Brunner *et al.* (1988)
NP2EO	MWWTP sludge	30	Swiss MWWTPs	Marcomini and Giger (1987)
NP2EO	MWWTP sludge	35 - 50	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1988a)
NpnEO	marine sediment	0.1 - 6.6	Mediterranean Sea near Barcelona	Valls *et al.* (1988)
NpnEO	seawater	0.845	Mediterranean Sea near Barcelona	Valls *et al.* (1988)
NpnEO	MWWTP final effluent	4.7 - 9.7	Italian MWWTP	Di Corcia *et al.* (1994)
NpnEO	MWWTP final effluent	< 5	Australian MWWTPs	Mackay *et al.* (1997)
NpnEO	MWWTP raw sewage	64 - 115	Italian MWWTP	Di Corcia *et al.* (1994)
NpnEO	MWWTP raw sewage	86 - 450	Australian MWWTPs	Mackay *et al.* (1997)
NpnEO	MWWTP raw sewage	37 - 123	Barcelona MWWTP, Spain	Valls *et al.* (1988)
NPnEO (n = 0 - 2)	sludge-amended soil	1.50 - 4.933	Denmark	Winther-Nielsen *et al.* (1997)
NPnEO (n = 0 - 2)	MWWTP sludge	150 - 370	Danish MWWTPs	Winther-Nielsen *et al.* (1997)
NPnEO (n = 1-13)	seawater	0.5 - 4.5	lagoons of Venice, Italy	Marcomini *et al.* (1990)
NPnEO (n = 1-15)	commercial liquid cleaning products	3.28 to 22.82 %	products available in Italy and Switzerland	Marcomini *et al.* (1988c)
NPnEO (n = 1-16)	seawater	19.55	lagoon of Venice, Italy	Marcomini *et al.* (1989b)
NPnEO (n = 1-17)	granular laundry detergent	N.D. - 6.0 %	products available in Switzerland	Marcomini *et al.* (1988b)
NPnEO (n = 1-17)	liquid hard-surface cleaner	7.0 - 25.1 %	products available in Switzerland	Marcomini *et al.* (1988b)
NPnEO (n = 1-18)	MWWTP final effluent	56 - 102	High Point, NC, U.S.A. MWWTPs	Kubeck and Naylor (1990)
NPnEO (n = 1-18)	MWWTP raw sewage	1600 - 2520	High Point, NC, U.S.A. MWWTPs	Kubeck and Naylor (1990)
NPnEO (n = 1 - 18)	MWWTP final effluent	230	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1987)
NPnEO (n = 1 - 18)	MWWTP primary effluent	1230	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1987)
NPnEO (n = 1 - 18)	MWWTP raw sewage	1920	Zurich-Glatt MWWTP, Switzerland	Marcomini *et al.* (1987)
NPnEO (n = 3 - 17)	fresh water	<1.6 - 14.9	U.S. rivers	Naylor *et al.* (1992)
NPnEO (n = 3 - 18)	MWWTP final effluent	980 - 1067	Swiss MWWTPs	Ahel and Giger (1985b)
NPnEO (n = 3 - 18)	MWWTP primary effluent	29 - 369	Swiss MWWTPs	Ahel and Giger (1985b)
NPnEO (n = 3 - 18)	MWWTP raw sewage	844 - 2250	Swiss MWWTPs	Ahel and Giger (1985b)
NPnEO (n = 3 - 20)	fresh water	< 1 - 7.1	Swiss rivers	Ahel *et al.* (1994a)
NPnEO (n = 3 - 7)	drinking water	0.501	New Jersey, U.S.A.	Clark *et al.* (1992)
NPnEO (n = avg. of 10)	MWWTP final effluent	3000 - 4000	Israeli MWWTPs	Zoller and Romano (1983)
NPnEO (n = 3-16)	MWWTP final effluent	N.D. - 813	U.S.A.	Amato and Wayment (1998)
NP1EC	fresh water	< 1 - 45	Swiss rivers	Ahel *et al.* (1994a)
NP1EC	fresh water	8.4 - 20	Swiss rivers	Ahel *et al.* (1996)
NP1EC	fresh water	< 0.2 - 2.0	U.S. rivers	Field and Reed (1996)
NP1EC	groundwater	< 0.1 - 13.1	Glattfelden and Engelburg, Switzerland	Ahel *et al.* (1996)
NP1EC	paper mill effluent	< 0.2 - 140	Fox River, WI, U.S.A.	Field and Reed (1996)
NP1EC	MWWTP final effluent	N.D. - 224	Swiss MWWTPs	Ahel *et al.* (1987)
NP1EC	MWWTP final effluent	1.5 - 3.9	Italian MWWTP	Di Corcia *et al.* (1994)
NP1EC	MWWTP final effluent	7.6 - 29.4	Fox River, WI, U.S.A.	Field and Reed (1996)
NP1EC	MWWTP primary effluent	N.D.	Swiss MWWTP	Ahel *et al.* (1987)
NP1EC	MWWTP raw sewage	N.D.	Swiss MWWTP	Ahel *et al.* (1987)
NP1EC	MWWTP raw sewage	N.D.	Italian MWWTP	Di Corcia *et al.* (1994)

Compound	Medium	Concentration Water/Effluent (µg/L) Sediment/Sludge (µg/g) Tissue (µg/g)	Location	Reference
NP2EC	drinking water	0.164	New Jersey, U.S.A.	Clark *et al.* (1992)
NP2EC	fresh water	2 - 71	Swiss rivers	Ahel *et al.* (1994a)
NP2EC	fresh water	20.6 - 28.7	Swiss rivers	Ahel *et al.* (1996)
NP2EC	fresh water	< 0.4 - 11.8	U.S. rivers	Field and Reed (1996)
NP2EC	groundwater	< 0.1 - 23.2	Glattfelden and Engelburg, Switzerland	Ahel *et al.* (1996)
NP2EC	paper mill effluent	< 0.4 - 931	Fox River, WI, U.S.A.	Field and Reed (1996)
NP2EC	MWWTP final effluent	71 - 233	Swiss MWWTPs	Ahel *et al.* (1987)
NP2EC	MWWTP final effluent	5.1 - 9.4	Italian MWWTP	Di Corcia *et al.* (1994)
NP2EC	MWWTP final effluent	64.1 - 144	Fox River, WI, U.S.A.	Field and Reed (1996)
NP2EC	MWWTP primary effluent	6 - 17	Swiss MWWTPs	Ahel *et al.* (1987)
NP2EC	MWWTP raw sewage	N.D. - 14	Swiss MWWTPs	Ahel *et al.* (1987)
NP2EC	MWWTP raw sewage	N.D.	Italian MWWTP	Di Corcia *et al.* (1994)
NP3EC	fresh water	< 0.2	U.S. rivers	Field and Reed (1996)
NP3EC	paper mill effluent	< 0.2 - 172	Fox River, WI, U.S.A.	Field and Reed (1996)
NP3EC	MWWTP final effluent	24.8 - 105	Fox River, WI, U.S.A.	Field and Reed (1996)
NP4EC	fresh water	< 0.2	U.S. rivers	Field and Reed (1996)
NP4EC	paper mill effluent	< 0.2 - 26.7	Fox River, WI, U.S.A.	Field and Reed (1996)
NP4EC	MWWTP final effluent	9.7 - 29.2	Fox River, WI, U.S.A.	Field and Reed (1996)
NPnEC (n = 3 - 7)	drinking water	0.062	New Jersey, U.S.A.	Clark *et al.* (1992)
Octylphenolics				
OP	chemical plant effluent	5000	Philadelphia, PA, U.S.A.	Sheldon and Hites (1979)
OP	drinking water	0.01	Philadelphia, PA, U.S.A.	Sheldon and Hites (1979)
OP	fresh water	0.2 - 2	Delaware River, U.S.A.	Sheldon and Hites (1978)
OP	fresh water	N.D. - 3	Delaware River, U.S.A.	Sheldon and Hites (1979)
OP	groundwater	0.36	Falmouth, MA, U.S.A.	Barber *et al.* (1988)
OP	MWWTP final effluent	N.D. - 1	Swedish MWWTPs	Paxéus (1996)
OP	MWWTP final effluent	200	Philadelphia, PA, U.S.A.	Sheldon and Hites (1979)
OP	MWWTP raw sewage	400	Philadelphia, PA, U.S.A.	Sheldon and Hites (1979)
OP1EO	MWWTP final effluent	< 0.5 - 0.9	Palo Alto, CA, U.S.A. MWWTP	Ball and Reinhard (1985)
OP1EO	MWWTP final effluent	N.D. - 1.5	Swedish MWWTPs	Paxéus (1996)
OP2EO	drinking water	0.002	New Jersey, U.S.A.	Clark *et al.* (1992)
OP2EO	MWWTP final effluent	1.5 - 6.0	Palo Alto, CA, U.S.A. MWWTP	Ball and Reinhard (1985)
OP2EO	MWWTP final effluent	N.D. - 0.5	Swedish MWWTPs	Paxéus (1996)
OpnEO	MWWTP final effluent	< 5	Australian MWWTPs	Mackay *et al.* (1997)
OpnEO	MWWTP raw sewage	< 5 - 23	Australian MWWTPs	Mackay *et al.* (1997)
OPnEO (n = 3 - 8)	drinking water	0.124	New Jersey, U.S.A.	Clark *et al.* (1992)
OPnEO (n=1-17)	granular laundry detergent	N.D. - 2.9 %	Products available in Switzerland	Marcomini *et al.* (1988a)
OPnEO (n=1-17)	liquid hard-surface cleaner	N.D.	Products available in Switzerland	Marcomini *et al.* (1988a)
OP1EC	MWWTP final effluent	4.9 - 11	Palo Alto, CA, U.S.A. MWWTP	Ball and Reinhard (1985)
OP2EC	drinking water	0.040	New Jersey, U.S.A.	Clark *et al.* (1992)
OP2EC	MWWTP final effluent	24 - 84	Palo Alto, CA, U.S.A. MWWTP	Ball and Reinhard (1985)
OPnEC (n = 3 - 4)	drinking water	0.012	New Jersey, U.S.A.	Clark *et al.* (1992)

Table 9. Toxicity of APEs, APs and other degradation products to freshwater organisms.

Chemical	Freshwater Organism		Endpoint		Value (µg/L)	Reference	Comments
	Fish - Acute Toxicity						
NP	Fathead minnow	*Pimephales promelas*	LC_{50}	96 h	135	Holcombe *et al.* (1984)	I
NP	Fathead minnow		LC_{50}	96 h	128	Brooke (1993a)	I
NP	Fathead minnow		LC_{50}	96 h	300	Ward and Boeri (1991b); Naylor (1995)	I
NP	Fathead minnow		LC_{50}	24/96 h	330/270	Dwyer *et al.* (1995)	II
NP	Fathead minnow		LC_{50}	24/48/96 h	700/520/300	Analytical Bio Chemistry (1981)	I
NP	Rainbow trout	*Oncorhynchus mykiss*	LC_{50}	96 h	221	Brooke (1993b)	I
NP	Rainbow trout		EC_{50}	96 h	109 (100-118)		
NP	Rainbow trout		LC_{50}	24/96 h	480/230	Naylor (1995)	II
NP	Rainbow trout		LC_{50}	96 h	230	Armstrong and Kingsbury (1979)	III
NP	Rainbow trout		LC_{50}	24/96 h	480/560-920	Ernst *et al.* (1980)	III
NP	Rainbow trout		LC_{50}	24/96 h	300/190	Dwyer *et al.* (1995)	II
NP	Brook trout	*Salvelinus fontinalis*	LC_{50}	96 h	145	Armstrong and Kingsbury (1979)	III
NP	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	209	Brooke (1993b)	I
NP	Golden orfe	*Leuciscus idus*	LC_{50}	48 h	950	Hüls (1987) (1)	III
NP	Stickleback	*Gasterosteus aculeatus*	LC_{50}	96 h	370	Granmo *et al.* (1991)	II
NP	Greenback cutthroat trout	*Oncorhynchus clarki stomias*	LC_{50}	24/96 h	300/150	Dwyer *et al.* (1995)	II
NP	Lahontan cutthroat trout	*Oncorhynchus clarki henshawi*	LC_{50}	24/96 h	250/180		
NP	Apache trout	*Oncorhynchus apache*	LC_{50}	24/96 h	240/170		
NP	Bonytail chub	*Gila elegans*	LC_{50}	24/96 h	490/290		
NP	Colorado squawfish	*Ptychocheilus lucius*	LC_{50}	24/96 h	280/260		
NP	Razorback sucker	*Xyrauchen texanus*	LC_{50}	24/96 h	220/170		
NP	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	1400	Yoshimura (1986)	III
	Fish - Chronic Toxicity						
NP	Fathead minnow (early life stage)	*Pimephales promelas*	NOEC (survival)	33 d	7.4	Ward and Boeri (1991b)	I
NP	Fathead minnow (early life stage)		NOEC (growth)	28 d	23		
NP	Fathead minnow (juvenile)		NOEC	28 d	77.5	Brooke (1993b)	I
NP	Rainbow trout	*Oncorhynchus mykiss*	NOEC (survival)	91 d	6.0	Brooke (1993b)	I
NP	Bluegill sunfish	*Lepomis macrochirus*	NOEC (growth)	28 d	59.5		
NP	Japanese medaka (embryos)	*Oryzias latipes*	LC_{50}	14 d	460	Gray and Metcalfe (1997)	III
	Invertebrates						
NP	Daphnia	*Daphnia magna*	LC_{50}	48 h	190	Naylor (1995)	III
NP	Daphnia		NOEC	48 h	77		
NP	Daphnia		LC_{50}	48 h	440		

Chemical	Freshwater Organism		Endpoint		Value (µg/L)	Reference	Comments
NP	Daphnia		EC_{50} (immobilization)	24 h	300	Comber *et al.* (1993)	I
NP	Daphnia		EC_{50} (immobilization)	48 h	190		
NP	Daphnia		LC_{50}	7 d	120		
NP	Daphnia		LC_{50}	14 d	120		
NP	Daphnia		LC_{50}	21 d	100		
NP	Daphnia		NOEC (reproduction)	21 d	24		
NP	Daphnia		NOEC (growth)	21d	39		I
NP	Daphnia		MATC (reproduction)	21 d	71	Baldwin *et al.* (1997)	I
NP	Daphnia		LC_{50}	48 h	93 (78-111)	Brooke (1993b)	III
NP	Daphnia		EC_{50} (reproduction)	48 h	85	Brooke (1993b)	I
NP	Daphnia		NOEC (reproduction)	21 d	116		
NP	Daphnia		LC_{50}	48 h	140	Hüls (1992) [1]	I
NP	Daphnia		NOEC (reproduction)	21 d	100		
NP	Daphnia		LC_{50}	48 h	180	Bringmann and Kühn (1982) [3]	III
NP	Daphnia		EC_{50} (immobilization)	48 h	440	Monsanto (1985) [3]	III
NP	Daphnia		LC_{50}	48 h	470	Ankley *et al.* (1990)	III
NP	Daphnia	*Daphnia pulex*	LC_{50}	48 h	140-190	Ernst *et al.* (1980)	III
NP	Ceriodaphnia	*Ceriodaphnia dubia*	EC_{50}	96 h	69	Weeks *et al.* (1996)	I
NP	Ceriodaphnia		LC_{50}	7 d	258		
NP	Ceriodaphnia		MATC (reproduction)	7 d	134		
NP	Ceriodaphnia		MATC (mortality)	7 d	276		
NP	Ceriodaphnia		LC_{50}	96 h	>300	England, 1995	I
NP	Ceriodaphnia		EC_{50} (total adverse effects)	7 d	27		
NP	Ceriodaphnia		NOEC (reproduction)	7 d	100		
NP	Amphipod	*Hyalella azteca*	LC_{50}	96 h	20.7(16.8-25.6)	Brooke (1993b)	I
NP	Amphipod		LC_{50}	96 h	170	England and Bussard (1994)	I
NP	Amphipod		EC_{50} (immobilization)	96 h	150		
NP	Freshwater clam	*Anodonta cataractae*	LC_{50}	6 d	1700	McLeese *et al.* (1980b)	III
NP	Snail	*Physella virgata*	LC_{50}	96 h	774	Brooke (1993b)	I
NP	Dragonfly	*Ophiogomphus sp.*	LC_{50}	96 h	>768		
NP	Annelid	*Lumbriculus variegatus*	LC_{50}	96 h	342 (296-396)		
NP	Copepods	*Calanoida* and *Paracyclops*	NOEC (abundance)	83 d	5	O'Halloran *et al.* (1998)	
	Aquatic plants and algae						
NP	Duckweed	*Lemna minor*	NOEC ("# fronds)	96 h	901	Brooke (1993b)	I
NP	Duck weed		LC_{50}	24 h	5500	Weinberger (1984)	III
			total ATP	24 h	2500 (µg/g dw)		
NP	Duck weed		LOEC (growth	24 h	125	Prasad, 1989	I

Chemical	Freshwater Organism		Endpoint		Value (µg/L)	Reference	Comments
			reduction)				
NP	Alga	*Chlamydomonas segnis*	LC_{50}	24 h	1500	Weinberger (1984)	III
			(inhibition of photosynthesis)		500-750		
NP	Alga		EC (cell division, zoospore release, dw)	16-24h	2500	Weinberger *et al.* (1987)	I
NP	Green alga	*Selenastrum capricornutum*	EC_{50}	96 h	410	Ward and Boeri (1990b); Naylor (1995); Weeks *et al.* (1996)	I
NP	Green alga		NOEC	96 h	92		
NP	Green alga		NOEC (biomass)	96 h	694	Brooke (1993b)	
NP	Green alga	*Scenedesmus subspicatus*	EC_{50} (biomass)	72 h	56.3	Kopf (1997) as cited in UK 1998	III
NP	Green alga		EC_{10} (biomass)	72 h	3.3		
NP	Green alga		EC_{50} (growth rate)	72 h	323		
NP	Green alga		EC_{10} (growth rate)	72 h	25.1		
NP	Green alga		EC_{50} (growth)	72 h	1300	Hüls (1996) [5]	III
NP	Green alga		EC_{10} (growth)	72 h	500		
NP	Green alga	*Chamydomonas reinhardii*	membrane disruption		500-700	Weinberger and Rea (1981)	I
NP	Green alga		growth		750	Moody *et al.* (1983)	III
NP	Green alga	*Chlorella pyrenoidosa*	LC_{50}	24 h	1500	Weinberger and Rea (1981)	III
NP	SMP	submitochondrial particles	EC_{50}		1800	Argese *et al.* (1994)	I
Other chemicals	**Toxicity to various organisms**						
NP1EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	3000	Yoshimura (1986)	III
NP1EO	Mosquito	*Culex pipiens*	EC_{50}	emergence	80000	Maxwell and Piper (1968)	III
NP1.5EO	Ceriodaphnia	*Ceriodaphnia dubia*	LC_{50}	48 h	1040	Ankley *et al.* (1990)	III
NP1.5EO	Ceriodaphnia	*Ceriodaphnia dubia*	EC_{50}	96 h	626	England and Downing (1995); Weeks *et al.* (1996)	I
NP1.5EO	Ceriodaphnia		LC_{50}	96 h	1016		
NP1.5EO	Ceriodaphnia		EC_{50}	7 d	319		
NP1.5EO	Ceriodaphnia		LC_{50}	7 d	886		
NP1.5EO	Ceriodaphnia		EC_{50} (total adverse effects)	7 d	319		
NP1.5EO	Ceriodaphnia		NOEC (reproduction)	7 d	285		
NP1.5EO	Ceriodaphnia		NOEC (mortality)		285		
NP1.5EO	Ceriodaphnia		LC_{50}	96 h	1016	England and Downing (1995)	I
NP1.5EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		67000	Maxwell and Piper (1968)	III
NP2EO	Daphnia	*Daphnia magna*	LC_{50}	48 h	148 (115-198)	Maki *et al.* (1998)	III
NP3.3EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	2500	Yoshimura (1986)	III
NP4EO	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	1300	Macek and Krzeminiski (1975)	III
NP4EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		6000-8000	Maxwell and Piper (1968)	III
NP4EO	Green alga	*Scenedesmus spp.*	Survival		6000	Janicke *et al.* (1969) [1]	III
NP5EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	3600	Yoshimura (1986)	III
NP5EO	Bluegill sunfish	*Lepomis*	LC_{50}	96 h	2400-2800	Macek and Krzeminiski (1975)	III

Chemical	Freshwater Organism		Endpoint		Value (µg/L)	Reference	Comments
		macrochirus					
NP6EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		6000	Maxwell and Piper (1968)	III
NP6EO	Green alga	*Scenedesmus spp.*	survival		10000	Janicke *et al.* (1969) [1]	III
NP6EO	Green alga	*Selenastrum capricornutum*	NOEC (growth)	21d	500000	Nyberg (1988)	III
NP6.4EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	5400	Yoshimura (1986)	III
NP7EO	Fathead minnow	*Pimephales promelas*	LC_{50}	96 h	3200	Markarian *et al.* (1989) [3]	III
NP7EO	Daphnia	*Daphnia magna*	LC_{50}	96 h	4100		
NP7EO	Green alga	*Scenedesmus spp.*	survival		16000	Janicke *et al.* (1969) [1]	III
NP7EO	Green alga	*Selenastrum capricornutum*	EC_{50} (growth)	96 h	>1000000	Markarian *et al.* (1989) [1,3]	III
NP8EO	Rainbow trout	*Oncorhynchus mykiss*	LC_{50}	96 h	4700	Calamari and Marchetti (1973) [3]	III
NP8.4EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	11600	Yoshimura (1986)	III
NP8.8EO	SMP	submitochondrial particles	EC_{50}		1300	Argese *et al.* (1994)	I
NP8.9EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	11200-14000	Yoshimura (1986)	III
NP9EO	Fathead minnow	*Pimephales promelas*	LC_{50}	96h	6600	Union Carbide Corp (1994) as cited in Williams *et al.* (1996)	III
NP9EO	Fathead minnow		LC_{50}	96 h	4600(4300-5000)	Dorn *et al.* (1993)	I
NP9EO	Fathead minnow		NOEC (survival)	7 d	1800		
NP9EO	Fathead minnow		NOEC (growth)	7 d	1000		
NP9EO	Fathead minnow		LC_{50}	96 h	1600	Shell Chemical Company (1987); Salanitro *et al.* (1988) [3]	
NP9EO	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	7900	Macek and Krzeminiski (1975)	III
NP9EO	Goldfish	*Carassius auratus*	LC_{50}	48 h	18000	Tomiyama (1974) [3]	III
NP9EO	Golden orfe	*Leuciscus idus*	LC_{50}	48h	7000	Fischer (1973) [3]	III
NP9EO	Daphnia	*Daphnia magna*	EC_{50}	48 h	14000(13000-16000)	Dorn *et al.* (1993)	I
NP9EO	Daphnia		NOEC (survival)	7 d	10000		
NP9EO	Daphnia		NOEC (growth)	7 d	10000		
NP9EO	Green alga	*Selenastrum capricornutum*	EC_{50} (growth)	96 h	12000		
NP9EO	Green alga		NOEC (growth)	96 h	8000		
NP9EO	Green alga		EC_{50} (growth)	96 h	50000	Lewis (1986)	III
NP9EO	Green alga		NOEC (growth)	21d	500000	Nyberg (1988)	III
NP9EO	Green alga		growth inhibition	5 d	<100000		III
NP9EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		10000	Maxwell and Piper (1968)	III
NP9EO	Microtox	*Photobacterium phosphoreum*	EC_{50}	5 min	60600 (35600-103100)	Dorn *et al.* (1993)	I
NP9(br)EO	Daphnia	*Daphnia pulex*	LC_{50}	48 h	2900	Shell Chemical Company(1987) [3]; Salanitro *et al.* (1988) [3]	III
NP9.5EO	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	7600	Macek and Krzeminiski (1975)	III
NP9.5EO	Harlequin fish	*Rasbora heteromorpha*	LC_{50}	48/96 h	11300/8600	Reiff *et al.* (1979)	III
NP9.5EO	Brown trout	*Salmo trutta*	LC_{50}	48/96 h	2700/1000		
NP9.5EO	Golden orfe	*Leuciscus idus*	LC_{50}	48 h	4900-11300		
NP9.5EO	Golden orfe		LC_{50}	96 h	7000-11200		
NP9.5EO	Goldfish	*Carassius auratus*	LC_{50}	6 h	6900	Reiff *et al.* (1979)	III
NP9.5EO	Rainbow trout	*Oncorhynchus mykiss*	LC_{50}	96 h	7500-12500 4500-5900	Unilever Research Laboratories (1977) [3]	III
NP9.5EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		19000	Maxwell and Piper (1968)	III

Chemical	Freshwater Organism		Endpoint		Value (µg/L)	Reference	Comments
NP9.5EO	Mosquito		EC_{50} (emergence)		7000		III
NP9.5EO	Green and blue-green algae		growth		20000	Davis and Gloyna (1967) [3]	III
NP10EO	Rainbow trout	*Oncorhynchus mykiss*	LC_{50}	3 h	2500-62000	Marchetti (1965)	III
NP10EO	Brown trout	*Salmo trutta*	LC_{50}	48 h	2700	Unilever Research laboratories (1977) [3]	III
NP10EO	Goldfish	*Carassius auratus*	LC_{50}	48 h	13800		
NP10EO	Goldfish		LC_{50}	48 h	5400	Kurata *et al.* (1977) [3]	III
NP10EO	Harlequin fish	*Rasbora heteromorpha*	LC_{50}	48 h	11300	Unilever Research Laboratories (1977) [3]	III
NP10EO	Golden orfe	*Leuciscus idus*	LC_{50}	48 h	7400. 9500		
NP10EO	Minnow	*Phoxinus phoxinus*	LC_{50}	96 h	8800		
NP10EO	Daphnia	*Daphnia magna*	LC_{50}	24 h	44200		
NP10EO	Daphnia	*Daphnia pulex*	LC_{50}	48 h	12500	Burlington Research (1985) [3]; Moore *et al.* (1987) [3]	III
NP10EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		19000	Maxwell and Piper (1968)	III
NP11(br)EO	Guppy	*Lebistes reticulatus*	LC_{100}	24 h	52000-64000	Van Emden *et al.* (1974)	III
NP11(br)EO	Mosquito	*Aides aegyptii*	LC_{50}	24 h	500000		
NP11(br)EO	Snail	*Biomphalaria glabrata*	LC_{100}	24h	23000		
NP13.1EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	48000	Yoshimura (1986)	III
NP15EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		9000	Maxwell and Piper (1968)	III
NP16.6EO	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	110000	Yoshimura (1986)	III
NP20EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		>200000	Maxwell and Piper (1968)	III
NP20EO	Mosquito		EC_{50} (emergence)		120000		
NP20EO	Mosquito		EC_{50} (emergence)		28000		
NP20EO	Green alga	*Scenedesmus* spp.	survival		125000	Janicke *et al.* (1969) [1]	III
NP30EO	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	>1000000	Macek and Krzeminiski (1975)	III
NP30EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		>400000	Maxwell and Piper (1968)	III
NP30EO	Green alga	*Scenedesmus* spp.	survival		5000000	Janicke *et al.* (1969) [1]	III
NP50EO	Mosquito	*Culex pipiens*	EC_{50} (emergence)		>400000	Maxwell and Piper (1968)	III
NP1EC	Fathead minnow	*Pimephales promelas*	LC_{50}	96h	2000	Williams *et al.* (1996); Williams (1997)	III
NP1EC	Fathead minnow		NOEC	96h	1000		
NP1EC	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	9600	Yoshimura (1986)	III
NP1EC	Daphnia	*Daphnia magna*	LC_{50}	48 h	14000	Naylor *et al.* (1997)	III
NP1EC	Daphnia		NOEC	48 h	11000		
NP1EC	Ceriodaphnia	*Ceriodaphnia dubia*	LC_{50}	96 h	17000		
NP1EC	Ceriodaphnia		NOEC (reproduction)	7 d	2200		
NP1EC	Ceriodaphnia		NOEC (mortality)	7 d	8400		
NP1EC	SMP	submitochondrial particles	EC_{50}		8200	Argese *et al.* (1994)	I
NP2EC	Japanese killifish	*Oryzias latipes*	LC_{50}	48 h	8900	Yoshimura (1986)	
NP2EC	Daphnia	*Daphnia magna*	LC_{50}	48 h	990 (770-1295)	Maki *et al.* (1998)	III
OP	Fathead minnow	*Pimephales promelas*	LC_{50}	96 h	250	Analytical Bio Chemistry (1984a)	I
OP	Fathead minnow		LC_{50}	24 h	290 (150-630)		
OP	Fathead minnow		NOEC	24 h	150		

Chemical	Freshwater Organism		Endpoint		Value (µg/L)	Reference	Comments
OP	Rainbow trout	*Oncorhynchus mykiss*	LC_{50}	24 h	450 (320-710)	Analytical Bio Chemistry (1984b)	I
OP	Rainbow trout		NOEC	24 h	170		
OP	Rainbow trout		LC_{50}	14 d	120		
OP	Rainbow trout		NOEC	14 d	84		
OP	Rainbow trout (early life stage)		NOEC	90 d	6.1	McAllister *et al.* (1986)	
OP	Rainbow trout (early life stage)		LOEC	90 d	11		
OP	Daphnia	*Daphnia magna*	LC_{50}	24/48 h	260/270	Analytical Bio Chemistry (1984c)	I
OP	Daphnia	*Daphnia magna*	EC_{50} (survival)	21 d	340	Forbis (1988)	I
OP	Daphnia		NOEC (reproduction)	21 d	37		
OP	Green alga	*Selenastrum capricornutum*	EC_{50}	96 h	1900 (1000-2700)	Forbis *et al.* (1984)	I
OP	Green alga		NOEL	96 h	<1000		
OP5EO	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	2800-3200	Macek and Krzeminiski (1975)	III
OP5EO	Mysid	*Mysidopsis bahia*	LC_{50}	48 h	1830	Hall *et al.* (1989)	II
OP9.5EO	Green alga	*Selenastrum capricornutum*	EC_{50} (growth)	21d	300000-500000	Nyberg (1988)	III
OP9.5EO	Alga	*Nitzschia actinastroides*	NOEC (growth)	5 d	10000-15000	Nyberg (1976) [1]	III
OP9.5EO	Alga	*Nitzschia holsatica*	NOEC (growth)	5 d	10000	Nyberg (1985) [1]	III
OP9.5EO	Alga	*Phorphyridium purpureum*	NOEC (growth)	5 d	5000-10000		III
OP10EO	Fathead minnow	*Pimephales promelas*	LC_{50}	96 h	8900	Williams *et al.* (1996); Williams (1997)	I
OP10EO	Fathead minnow		NOEC	96 h	3300		
OP10EO	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	12000	Macek and Krzeminiski (1975)	III
OP10EO	Chrysophyte	*Poterioochromonas malhamensis*			12400	Roderer (1987)	III
OP10EO	Green alga	*Chlorella fusca*	NOEC (growth)	14 d	131000-525000	Wong (1985)	III
OP10EO	Alga	*Microcysis aeruginos*	EC_{50} (growth)	96 h	7400	Lewis (1986)	III
OP30EO	Bluegill sunfish	*Lepomis macrochirus*	LC_{50}	96 h	531000	Macek and Krzeminiski (1975)	III
OP30EO	Green alga	*Selenastrum capricornutum*	EC_{50} (growth)	21d	500000	Nyberg (1988)	III
OP40EO	Chrysophyte	*Poterioochromonas malhamensis*			18000000	Roderer (1987)	III
OP1EC	Fathead minnow	*Pimephales promelas*	LC_{50}	96 h	5000	Williams *et al.* (1996); Williams (1997)	I
OP1EC	Fathead minnow		NOEC	96 h	1600		
OP1EC	Daphnia	*Daphnia magna*	LC_{50}	48 h	20500	Naylor *et al.* (1997)	III
OP1EC	Daphnia		NOEC	48 h	10600		
OP1EC	Ceriodaphnia	*Ceriodaphnia dubia*	LC_{50}	96 h	<48000		
OP1EC	Ceriodaphnia		NOEC	96 h	24000		
OP1EC	Ceriodaphnia		NOEC (reproduction)	7 d	3400		
OP1EC	Ceriodaphnia		NOEC (mortality)	7 d	3300		

Comments:
I. high confidence, reports study conditions, measured values, renewal or flow through tests
II does have all data reported
III data not sufficient to give confidence
References followed by parentheses were not available and were reported as cited in:

(1) Staples *et al.* (1998); (2) RM-1 document for para-nonylphenol (Rodier ,1996); (3) Talmage (1994); (4) Lewis (1990); (5) OECD (1997)

Table 10. Toxicity of APEs, APs and other degradation products to marine organisms.

Chemical	Marine Organism		Endpoint		Value (µg/L)	Reference	Comments
	Fish - Acute Toxicity						
NP	Winter flounder	*Pleuronectes americanus*	LC_{50}	96 h	17	Lussier *et al.* (1996)	III
NP	Cod	*Gadus morrhua*	LC_{50}	96 h	300	Swedmark (1968)	III
NP	Atlantic salmon	*Salmo salar*	LC_{50}	96 h	130-160/190	McLeese *et al.* (1981)	II
NP	Atlantic salmon		LC_{50}	96 h	900	McLeese *et al.* (1980b)	II
NP	Sheepshead minnow	*Cyprinodon variegatus*	LC_{50}	96 h	310	Ward and Boeri (1990d); Naylor (1995)	I
NP	Sheepshead minnow		LC_{50}	96 h	142	Lussier *et al.* (1996)	III
NP	Inland silverside	*Menidia beryllina*	LC_{50}	96 h	70		
NP	Hook-nose	*Agonus cataphractus*	LC_{50}	96 h	510	Waldock and Thain (1991) [3]	III
	Fish - Chronic Toxicity						
NP	Cod	*Gadus morrhua*	LC_{50}	21 d	<100	Swedmark (1968)	III
NP	Hook-nose	*Agonus cataphractus*	LC_{50}	7 d	360	Waldock and Thain (1991)	III
	Invertebrates						
NP	Amphipod	*Leptocheirus plumulosus*	LC_{50}	96 h	62	Lussier *et al.* (1996)	III
NP	Mysid	*Mysidopsis bahia*	LC_{50}	96 h	61		
NP	Mysid		LC_{50}	96 h	43	Ward and Boeri (1991c) [1]; Naylor (1995); Weeks *et al.* (1996)	I
NP	Mysid		NOEL (survival)	28 d	6.7	Ward and Boeri (1991); Naylor (1995); Weeks *et al.* (1996)	I
NP	Mysid		NOEL (reproduction)	28 d	6.7		
NP	Mysid		NOEL (length)	28 d	3.9		
NP	Coot clam	*Mulinia lateralis*	LC_{50}	96 h	38	Lussier *et al.* (1996)	III
NP	Sand shrimp	*Crangon septemspinosa*	LC_{50}	96 h	300	McLeese *et al.* (1981)	III
NP	Grass shrimp	*Paleomonetes pugio*	LC_{50}	96 h	59	Lussier *et al.* (1996)	III
NP	Brown shrimp	*Crangon crangon*	LC_{50}	96 h	600	Granmo *et al.* (1991)	III
NP	Brown shrimp		LC_{50}	96 h	420	Waldock and Thain (1991) [3]	III
NP	Brown shrimp		LC_{50}	7 d	340		
NP	Mud crab	*Dyspanopeus sayi*	LC_{50}	96 h	>195	Lussier *et al.* (1996)	III
NP	Crustacean	*Nitocra spinipes*	LC_{50}		118-139	Bergstrom (1984) [3]	III
NP	American lobster	*Homarus americanus*	LC_{50}	96 h	71	Lussier *et al.* (1996)	III
NP	American lobster		LC_{50}	96 h	170	McLeese *et al.* (1980b)	III
NP	Common mussel	*Mytilus edulis*	LC_{50}	96 h	3000	Granmo *et al.* (1989, 1991)	II
NP	Common mussel		LC_{50}	15 d	500		
NP	Common mussel		LC_{50}	32 d	140		
NP	Common mussel		NOEC (fertility, early development)	72 hr	200		
NP	Soft shell clam	*Mya arenaria*	LC_{50}	144 h	>700	McLeese *et al.* (1980b)	III
NP	Soft shell clam		LC_{50}	15 d	>700		
	Aquatic plants and algae						
NP	Marine alga	*Skeletonema costatum*	EC_{50} (growth)	96 h	27	Ward and Boeri (1990a); Naylor (1995); Weeks *et al.* (1996)	I
NP	Marine alga		NOEC	96 h	10		
Other chemicals	**Toxicity to various organisms**						
NP1.5EO	Mysid	*Mysidopsis bahia*	LC_{50}	48 h	110	Hall *et al.* (1989)	II
NP9EO	Mysid		LC_{50}	48 h	900-2000		

Chemical	Marine Organism		Endpoint		Value (µg/L)	Reference	Comments
NP9EO	Mysid		LC_{50}	48 h	1230	Patoczka and Pulliam (1990)	II
NP9EO	Marine alga		EC_{50} (growth)	14 d	10000	Ukeles (1965)	III
NP10EO	Cod	*Gadus morrhua*	LC_{50} (6-8/15-17°C)	96 h	6000/2500	Swedmark *et al.* (1971)	II
NP10EO	Cod		avoidance behaviour		>20	Hoglund (1976)	III
NP10EO	Flatfish	*Pleuronected flesus*	LC_{50}(15-17°C)	96 h	3000	Swedmark *et al.* (1971)	II
NP10EO	Barnacle	*Balanus balanoides*	LC_{50} (adult)	96 h	<25000		
NP10EO	Barnacle		LC_{50} (nauplius)	96 h	1500		
NP10EO	Cockle	*Cardium edule*	LC_{50}	96 h	<10000		
NP10EO	Clam	*Mya arenaria*	LC_{50}	96 h	<10000		
NP10EO	Clam		LC_{50}	96 h	<10000-18000	McLeese *et al.* (1980b)	III
NP10EO	Mussel	*Mytilus edulis*	LC_{50}	96 h	<10000	Swedmark *et al.* (1971)	II
NP10EO	Scallop	*Pecten maximus*	LC_{50}	96 h	<5000,<10000		
NP10EO	Shore crab	*Carcinus maenas*	LC_{50}	96 h	>100000	Swedmark *et al.* (1968); Swedmark *et al.* (1971)	II
NP10EO	Hermit crab	*Eupagarus bernhardus*	LC_{50}	96 h	>100000	Swedmark *et al.* (1971)	II
NP10EO	Spider crab	*Hyas araneus*	LC_{50}	96 h	>100000		
NP10EO	Cockle	*Cardium edule*	LC_{50}	96 h	5000		
NP10EO	Decapod	*Leander squilla*	LC_{50}	96 h	>100000		
NP10EO	Decapod	*Leander adspersus*	LC_{50} (6-8/5-17°C)	96 h	>100000/10000-15000		
NP10EO	Decapod		LC_{50} (intermoult)	96 h	50000		
NP10EO	Decapod		LC_{50} (postmoult)	96 h	10000		
NP12EO	Pink shrimp	*Pandalus montagui*	LC_{50}	48 h	19300	Portmann and Wilson (1971)	III
NP12EO	Brown shrimp	*Crangon crangon*	LC_{50}	48 h	89500		
NP12EO	Shore crab	*Carcinus maenas*	LC_{50}	48 h	>100000		
NP12EO	Shore crab		LC_{50}	48 h	>100000	Waldock and Thain (1991) [3]	III
NP12EO	Cockle	*Cardium edule*	LC_{50}	96 h	92500	Portmann and Wilson (1971)	III
NP15EO	Mysid	*Mysidopsis bahia*	LC_{50}	48 h	2570	Hall *et al.* (1989)	II
NP30EO	Marine alga		EC_{50} (growth)	14 d	1000000	Ukeles (1965)	III
NP40EO	Mysid	*Mysidopsis bahia*	LC_{50}	48 h	>100000	Hall *et al.* (1989)	II
NP50EO	Mysid		LC_{50}	48 h	>4110000		
NP1EC	Mysid		LC_{50}	96 h	9400	Naylor *et al.* (1997)	III
NP1EC	Mysid		NOEC	96 h	4100		
OP	Mysid		LC_{50}	96 h	55-113	Cripe *et al.* (1989)	I
OP1.5EO	Mysid		LC_{50}	48 h	7070	Hall *et al.* (1989)	II
OP5EO	Mysid		LC_{50}	48 h	1830		
OP1EC	Mysid		LC_{50}	96 h	10000	Naylor *et al.* (1997)	III
OP1EC	Mysid		NOEC	96 h	5000		

Comments:

I. high confidence, reports study conditions, measured values, renewal or flow through tests

II does have all data reported

III data not sufficient to give confidence

References followed by parentheses were not available and were reported as cited in:

(1) Staples *et al.* (1998); (2) RM-1 document for para-nonylphenol (Rodier ,1996); (3) Talmage (1994); (4) Lewis (1990); (5) OECD (1997)

Table 11. Toxicity of NP to freshwater organisms in sediment-water exposure systems.

Chemical	Species		Endpoint	Conditions	Duration	Value	Reference	Comments
NP	midge	*Chironomus tentans*	LC_{50}	dosed water	14 d	119 (µg/L)	England and Bussard 1993	I
NP	midge		LOEC (growth and survival)			150 (µg/L)		
NP	midge		NOEC (growth and survival)			76 (µg/L)		
NP	midge		MATC			107 (µg/L)		
NP	midge		EC_{50}			95 (µg/L)		
NP	midge		LC_{50}	interstitial water	14 d	75 (µg/L)		
NP	midge		LOEC (survival)			81 (µg/L)		
NP	midge		LOEC (growth)			39 (µg/L)		
NP	midge		NOEC (survival)			39 (µg/L)		
NP	midge		NOEC (growth)			21 (µg/L)		
NP	midge		MATC (survival)			56 (µg/L)		
NP	midge		MATC (growth)			30 (µg/L)		
NP	midge		MATC (growth and survival)			250 (µg/kg) concentration in sediment		
NP	midge		EC_{50} (total adverse effects)			41 (µg/L)		
NP	midge		LC_{50}	dosed sediment	14 d	> 34 (mg/kg)		
NP	midge		LOEC (survival and growth)			34 (mg/kg)		
NP	midge		NOEC (survival and growth)			20 (mg/kg)		
NP	midge		MATC (survival and growth)			26 (mg/kg)		
NP	midge		LOEC (survival -20 d larvae)	dosed water		91 (µg/L)	Kahl *et al.* 1997	I
NP	midge		NOEC (survival -20 d larvae)			42 (µg/L)		
NP	midge		NOEC (developmental and reproductive)			≥91 (µg/L)		
NP	tadpole	*Rana catesbiania*	LC_{50}	sediment	30 d	260 (mg/kg in sediment)	Ward and Boeri 1992	I
NP	tadpole		NOEL			155 (mg/kg)		
NP	tadpole		LOEL			390 (mg/kg)		
NP	tadpole		EC_{50}			220 (mg/kg)		

Table 12. Summary of relative toxicity and relative estrogenicity based on endocrine disrupting effects.

Chemical	Relative Potency to E2 Vg Induction, trout hepatocytes (1)	Vg induction in rainbow trout µg/L (2)	Relative Potency to E2 YES assay (3)	Relative Potency to E2 YES assay (4)	Relative Potency to E2 YES assay (5)	Relative Binding to E2 receptor; Kd (M) (6)	Binding to trout ER (7)	Relative Estrogenicity to NP (Selected for assessment)	Relative toxicity (Based on acute and chronic data)
NP	9.0 E-6	10	2 E-4	1.4 E-4	8.9 E-5	5 E-5	2.54 E-4	**1**	**1**
NP1EO					2 E-6		5.23 E-6	**0.67**	**0.5**
NP2EO	6.0E-6			6.6 E-6		0	3.93 E-5	**0.67**	**0.5**
NPnEO (≥9)	2 E-7			0				**0 (0.02)**	**0.005**
NP1EC	6.3E-6		8 E-6	4.0 E-5	0	2 E-4	1.68 E-4	**0.63**	**0.005**
NP2EC				4.0 E-5				**0.63**	**0.005**
OP	3.7 E-5	3	1.8 E-3	6.6 E-4		1.1 E-5	6.37 E-4	**4.1**	**1**
OP1EC								**0.63**	**0.005**
OP2EC								**0.63**	**0.005**

E2 = 17β-estradiol

(1) Jobling and Sumpter 1993
(2) Jobling *et al.* 1996
(3) Burnison, 1998
(4) Routledge *et al.* 1998
(5) Metcalfe, 1999
(6) White *et al.*, 1994
(7) Van Der Kraak, 1999

Table 13. Bioaccumulation of NP and NPEs in aquatic organisms.

Chemical	Species		BCF	BAF	$t_{1/2}$	Reference	Comments
NP	fathead minnow	*Pimephales promelas*	271		1.4 d	Ward and Boeri (1991a)	
NP	fathead minnow		344		1.2 d	Ward and Boeri (1991a)	
NP	fathead minnow		741			Brooke (1993b)	
NP	rainbow trout	*Oncorhynchus mykiss*	24-98		0.8 d	Lewis and Lech (1996)	
NP	rainbow trout			6		Ahel *et al.* (1993)	1
NP	rainbow trout				0.49-5.8 h (α) 40.2-99.0 h (β)	Coldham *et al.* (1998)	
NP	Atlantic salmon	*Salmo salar*	280 (k_1/k_2)		4 d	McLeese *et al.* (1981)	
NP	bluegill sunfish	*Lepomis macrochirus*	220			Brooke (1993a)	
NP	bluegill sunfish			87		Liber *et al.* (1999b)	
NP	carp	*Cyprinus carpio*	0.9-2.2			CITI (1992)	
NP	stickleback	*Gasterosteus aculeatus*	1250			Ekelund *et al.* (1990)	2
NP	squalus	*Squalus cephalus*		7		Ahel *et al.* (1993)	1
NP	barbus	*Barbus barbus*		15			
NP	alga	*Cladophora glomerata*		487		Ahel *et al.* (1993)	1
NP	aquatic plant	*Fontinalis antipyretica*		54			
NP	aquatic plant	*Potamogeton crispus*		32		Ahel *et al.* (1993)	
NP	mussel	*Mytilus edulis*		340		Granmo *et al.* (1991)	
NP	mussel		3430			Ekelund *et al.* (1990)	1
NP	shrimp	*Crangon crangon*	100			Ekelund *et al.* (1990)	1
NP	mussel	*Mytilus edulis*	10 (k_1/k_2)		0.3 d	McLeese *et al.* (1980)	
NP1EO	rainbow trout	*Oncorhynchus mykiss*		3		Ahel *et al.* (1993)	
NP1EO	barbus	*Barbus barbus*		19		Ahel *et al.* (1993)	
NP1EO	squalus	*Squalus cephalus*		1		Ahel *et al.* (1993)	
NP1EO	mussel	*Mytilus edulis*		170		Granmo *et al.* (1991)	
NP1EO	alga	*Cladophora glomerata*		10		Ahel *et al.* (1993)	1

Chemical	Species		BCF	BAF	$t_{1/2}$	Reference	Comments
NP1EO	aquatic plant	*Fontinalis antipyretica*		2		Ahel *et al.* (1993)	
NP1EO	aquatic plant	*Potamogeton crispus*		2		Ahel *et al.* (1993)	
NP2EO	rainbow trout	*Oncorhynchus mykiss*		0.8		Ahel *et al.* (1993)	
NP2EO	barbus	*Barbus barbus*		37		Ahel *et al.* (1993)	
NP2EO	squalus	*Squalus cephalus*		2		Ahel *et al.* (1993)	
NP2EO	mussel	*Mytilus edulis*		100		Granmo *et al.* (1991)	
NP2EO	aquatic plant	*Fontinalis antipyretica*		3		Ahel *et al.* (1993)	1
NP2EO	aquatic plant	*Potamogeton crispus*		10		Ahel *et al.* (1993)	1
NP2EO	algae	*Cladophora glomerata*		23		Ahel *et al.* (1993)	1
NP3EO	mussel	*Mytilus edulis*		60		Granmo *et al.* (1991)	

(1) Staples *et al.* (1998) calculated wet weight equivalent BAF based on 95% water.
(2) Caution because not corrected for >80% of radioactivity as metabolites.

Table 14: Most sensitive organisms in each trophic level for nonylphenol.

Trophic Level	Water Fresh/ Marine	Exposure Length	Species	Endpoint	CTV (µg/L)	Hyper-conservative AF	Hyper-conservative ENEV (µg/L)	Conser-vative AF	Conser-vative ENEV (µg/L)	Refs.
Fish	F	acute	Fathead minnow *(Pimephales promelas)*	96-h LC_{50}	128	100	1.28	50	2.6	Brooke 1993b
	F	chronic	Rainbow trout *(Oncorhynchus mykiss)*	91-d NOEC	6.0	10	0.60	10	0.60	Brooke 1993b
	M	acute	Winter Flounder *(Pleuronectes americanus)*	96-h LC_{50}	17	100	0.17	50	0.34	Lussier *et al.* 1996
	M	chronic	no primary study available							
Invertebrates	F	acute	*Hyalella azteca*	96-h LC_{50}	20.7	100	0.21	50	0.42	Brooke 1993b
	F	chronic	*Calanoida* and *Paracyclops*	NOEL (protect all species)	5	10	0.5	10	0.5	O'Halloran *et al.* 1998
	M	acute	Mysid shrimp *(Mysidopsis bahia)*	96-h LC_{50}	43	100	0.43	100	0.43	Ward and Boeri, 1990c
	M	chronic	Mysid shrimp *(Mysidopsis bahia)*	28-d NOEC (survival and reproduction)	6.7	10	0.67	10	0.67	Ward and Boeri, 1991b
				28-d NOEC (length)	3.9	10	0.39	10	0.39	
Algae	F		Algae *(Scenedesmus subspicatus)*	72-h EC_{50} (biomass)	56.3	100	0.56	100	0.56	Kopf 1997 as cited in UK 1998
				72-h EC_{10}	3.3	10	0.33	10	0.33	

Trophic Level	Water Fresh/ Marine	Exposure Length	Species	Endpoint	CTV (µg/L)	Hyper-conservative AF	Hyper-conservative ENEV (µg/L)	Conser-vative AF	Conser-vative ENEV (µg/L)	Refs.
				(biomass)						
				72-h EC_{50} (growth rate)	323	100	3.23	100	3.23	
				72-h EC_{10} (growth rate)	25.1	10	2.51	10	2.51	
	F		Algae *(Selenastrum capricornutum)*	96-h EC_{50} (growth)	410	100	4.1	100	4.1	Ward and Boeri, 1990a
				96-h NOEC	92	10	9.2	10	9.2	
	M		Algae *(Skeletonema costatum)*	96-h EC_{50} (cell growth)	27	100	0.27	100	0.27	Ward and Boeri, 1990d
				96-h NOEC	10	10	1.0	10	1.0	
Sediment Invertebrate	F	chronic	Midge *(Chironomus tentans)*	14-d NOEC (growth and survival)	20 mg/kg	10	2.0 mg/kg	10	2.0 mg/kg	England and Bussard 1993
Sediment Amphibian	F	chronic	Tadpole *(Rana catesbiana)*	30-d LC_{50}	260 mg/kg	100	2.6 mg/kg	100	2.6 mg/kg	Ward and Boeri 1992
				30-d NOEC (survival, sublethal effects; weight)	155 mg/kg	10	15.5 mg/kg	10	15.5 mg/kg	
Soil Invertebrate			Earthworm *(Apporectodea calignosa)*	21-d EC_{10} (reproduction)	3.44 mg/kg	10	0.34 mg/kg	10	0.34 mg/kg	Krogh *et al.* 1996
				21-d EC_{50} (reproduction)	13.7 mg/kg	10	1.37 mg/kg	10	1.37 mg/kg	

CTV = Critical toxicity value; AF = Application Factor; ENEV = Estimated No Effect Value

Key study- winter flounder inconsistently lower than other 19 species of fish tested.

Safety factor 50 chosen because more than 3 trophic levels reported in literature.

Table 15: Most Sensitive Aquatic Organisms for NPEs and NPECs

Substance	Trophic Level	Water (Fresh/ Marine)	Exposure Length	Species	Endpoint	CTV (ug/L)	AF	ENEV (ug/L)	Reference	Confidence
NP1EO	Fish	F	acute	Japanese killifish (*Oryzias latipes)*	48-h LC_{50}	3000	100	30	Yoshimura 1986	low
		F/M	chronic	NA						
	Invertebrates	F/M	acute	NA						
		F	chronic	Mosquito (*Culex pipiens)*	EC_{50} emergence	80000	100	800	Maxwell and Piper 1968	very low
	Algae	F/M	acute/chronic	NA						
NP1.5EO (mixture 1,2)	Fish	F/M	acute/chronic	NA						
	Invertebrates	M	acute	Mysid shrimp (*Mysidopis bahia)*	48-h LC_{50}	110	100	1.1	Hall *et al.* 1989	moderate
		M	chronic	NA						
		F	acute	*Ceriodaphnia dubia*	96-h EC_{50}	626	100	6.26	England and Downing 1995	high
		F	chronic	*Ceriodaphnia dubia*	7-d NOEC reproduction	285	10	28	England and Downing 1995	high
	Algae	F/M	acute/chronic	NA						
NP2EO	Fish	F/M	acute	NA						
		F/M	chronic	NA						
	Invertebrates	F	acute	*Daphnia magna*	48-h LC_{50}	148	100	1.48	Maki *et al.* 1998	low
		M	acute	NA						
		F/M	chronic	NA						
	Algae	F/M	acute/chronic	NA						
NP9EO	Fish	F	acute	Fathead minnow (*Pimephales promelas)*	96-h LC_{50}	4600	100	46	Dorn *et al.* 1993	high
		F	chronic	Fathead minnow (*Pimephales promelas)*	7-d NOEC growth	1000	10	100	Dorn *et al.* 1993	high
		M	acute (NP10EO)	*Gadus morhua*	96-h LC_{50}	2500	100	25	Swedmark *et al.* 1971	moderate
		M	chronic	NA						

Substance	Trophic Level	Water (Fresh/ Marine)	Exposure Length	Species	Endpoint	CTV (ug/L)	AF	ENEV (ug/L)	Reference	Confidence
	Invertebrates	F	acute	*Daphnia magna*	48-h EC_{50}	14000	100	140	Dorn *et al.* 1993	high
		F	chronic	*Daphnia magna*	7-d NOEC growth	10000	10	1000	Dorn *et al.* 1993	high
		M	acute	Mysid shrimp (*Mysidopis bahia)*	48-h LC_{50}	900	100	9.0	Hall *et al.* 1989	moderate
		M	chronic	NA						
	Algae	F	acute	*Selenastrum capricornutum*	EC_{50}	12000	100	120	Dorn *et al.* 1993	high
		F	chronic	NA						
		M	acute	NA						
		M	chronic	marine alga	14-d EC_{50} growth	10000	100	100	Ukeles 1965	low
NP1EC	Fish	F	acute	Fathead minnow (*Pimephales promelas)*	96-h NOEC growth	1000	10	100	Williams 1996	moderate/low
		F	chronic	NA						
		M	acute/chronic	NA						
	Invertebrates	F	acute	*Daphnia magna*	48-h LC_{50}	14000	100	140	Naylor *et al.* 1997	low
		F	chronic	*Ceriodaphnia dubia*	7-d NOEC reproduction	2200	10	220	Naylor *et al.* 1997	low
		M	acute	Mysid shrimp (*Mysidopis bahia)*	96-h LC_{50}	9400	100	94	Naylor *et al.* 1997	low
		M	chronic	NA						
	Algae	F/M	acute/chronic	NA						
NP2EC	Fish	F/M	acute/chronic	NA						
	Invertebrates	F	acute	*Daphnia magna*	48-h LC_{50}	990	100	9.9	Maki *et al.* 1998	low
		M	acute	NA						
		F/M	chronic	NA						
	Algae	F/M	acute/chronic	NA						

CTV = Critical toxicity value; AF = Application Factor; ENEV = Estimated No Effect Value; NA = No studies available

Table 16: Summary of the selected endpoints.

Compound	Assessment Level[1]	Endpoint	Species	CTV (µg/L)	AF	ENEV (µg/L)	Reference
NP	1	LC_{50} 96-h	Winter flounder *(Pleuronectes americanus)*	17	100	0.17	Lussier *et al.* 1996
	2	NOEC growth	Mysid *(Mysidopsis bahia)*	3.9	10	0.39	Ward and Boeri 1991c
	3	Chronic effects in less than 5% of species	Based on plotted acute data and applying an acute to chronic ratio of 4:1	10	10	1	
NP1EO	1	LC_{50} 48-h	Mysid *(Mysidopsis bahia)*	110	100	1.1	Hall *et al.* 1989
	2	LC_{50} 48-h	Mysid *(Mysidopsis bahia)*	110	100	1.1	Hall *et al.* 1989
	3	Chronic effects in less than 5% of species	Based on relative toxicity to NP's ENEV	1/0.5		2	
NP2EO	1	LC_{50} 48-h (from NP1EO)	Mysid *(Mysidopsis bahia)*	110	100	1.1	Hall *et al.* 1989
	2	LC_{50} 48-h (from NP1EO)	Mysid *(Mysidopsis bahia)*	110	100	1.1	Hall *et al.* 1989
	3	Chronic effects in less than 5% of species	Based on relative toxicity to NP's ENEV	1/0.5		2	
NP9EO	1	LC_{50} 48-h	Mysid *(Mysidopsis bahia)*	900	100	9.0	Hall *et al.* 1989
	2	LC_{50} 48-h	Mysid *(Mysidopsis bahia)*	900	100	9.0	Hall *et al.* 1989
	3	Chronic effects in less than 5% of species	Based on relative toxicity to NP	1/0.005		200	
NP1EC	1	NOEC 96-h	Fathead minnow *(Pimephales promelas)*	1000	10	100	Williams 1997
	2	NOEC 96-h	Fathead minnow *(Pimephales promelas)*	1000	10	100	Williams 1997
	3	Chronic effects in less than 5% of species	Based on relative toxicity to NP's ENEV	1/0.005		200	
NP2EC	1	LC_{50} 48-h	Daphnia *(Daphnia magna)*	990	100	9.9	Maki *et al.* 1998
	2	NOEC 96-h (from NP1EC)	Fathead minnow *(Pimephales promelas)*	1000	10	100	Williams 1997
	3	Chronic effects in less than 5% of species	Based on relative toxicity to NP's ENEV	1/0.005	10	200	

[1]Level 1= hyperconservative assessment; 2 = conservative assessment; 3 = distributional assessment

Table 17: Number of Canadian Effluent sites where risk quotients exceeded 1 for NPEs using a hyperconservative assessment. (total number of sites, total number of samples).

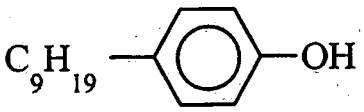
Environmental Compartment	Sites		NP	NP1EO	NP2EO	NP9EO	NP1EC	NP2EC	TEQ
Effluents	Textiles	untreated	2 (2,5)	2 (2,5)	2 (2,5)	2 (2,5)	0 (1,2)	0 (1,2)	2 (2,5)
		on-site treatment	2 (2,4)	1 (1,2)	1 (1,2)	2 (2,3)	0 (2,4)	1 (2,4)	2 (2,4)
		going to MWWTP	9 (9,14)	10 (10,14)	10 (10,14)	10 (10,14)	0 (5,7)	0 (5,7)	10 (10,15)
	Pulp and Paper	prior to 1998	4 (14,33)	13 (13,32)	4 (14,33)	-	-	-	14 (14,33)
		after 1998	5 (19,19)	2 (3,3)	2 (3,3)	2 (3,3)	0 (15,15)	2 (15,15)	6 (19,19)
	MWWTP	primary	8 (8,21)	10 (10,26)	10 (10,26)	7 (8,22)	0 (3,7)	0 (3,7)	10 (10,26)
		secondary	19 (21,54)	17 (20, 46)	17 (20,46)	8 (16,35)	0 (14,34)	12 (14,34)	21 (21,54)
		tertiary	6 (7,37)	7 (7,37)	7 (7,37)	3 (6,34)	0 (6,34)	5 (6,34)	7 (7,37)
		lagoon	5 (5,5)	5 (5,5)	4 (5,5)	0 (4,4)	0 (4,4)	0 (4,4)	5 (5,5)
	Refinery		0 (2,1)						

Table 18: Number of Canadian sites where risk quotients for NPEs exceeded 1 using a conservative assessment. (total number of sites, total number of samples).

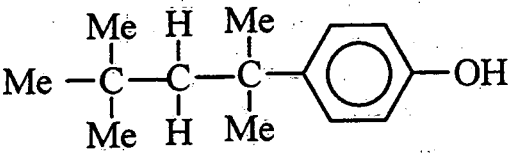
Environmental Compartment	Sites		NP	NP1EO	NP2EO	NP9EO	NP1EC	NP2EC	TEQ
Effluents	Textiles	untreated	1 (2,5)	2 (2,5)	2 (2,5)	2 (2,5)	0 (1,2)	0 (1,2)	2 (2,5)
		on-site treatment	0 (2,4)	1 (1,2)	1 (1,2)	2 (2,3)	0 (2,4)	0 (2,4)	1 (2,4)
		going to MWWTP	na	na	na	na	na	na	na
	Pulp and Paper	prior to 1998	3 (14,33)	12 (13,32)	4 (14,33)	-	-	-	7 (14, 33)
		after 1998	1 (19,19)	2 (3,3)	2 (3,3)	0 (3,3)	0 (15,15)	0 (15,15)	3 (19,19)
	MWWTP	primary	3 (8,21)	9 (10,26)	9 (10,26)	5 (8,22)	0 (3,7)	0 (3,7)	9 (10,26)
		secondary	1 (21, 54)	13 (20, 46)	12 (20,46)	0 (16,35)	0 (14,34)	0 (14,34)	7 (21,54)
		tertiary	0 (7,37)	6 (7,37)	5 (7,37)	0 (6,34)	0 (6,34)	0 (6,34)	3 (7,37)
		lagoon	0 (5,5)	0 (5,5)	0 (5,5)	0 (4,4)	0 (4,4)	0 (4,4)	0 (5,5)
Aquatic	Rivers		2 (25,90)	6 (12,51)	3 (12,51)	2 (3,27)	0 (1,37)	0 (1,37)	4 (25,90)
	Lakes		0 (5,5)	2 (4,4)	0 (4,4)	-	-	-	1 (5,5)
	Harbours		1 (12,31)	9 (12,26)	4 (12,26)	-	-	-	7 (12,31)
Benthic			5 (24,58)	1 (6,14)	1 (6,14)	0 (1,4)	-	-	5 (24,58)
Soil/Sludge			18 (30, 107)	8 (28,90)	3 (28,90)	0 (28,90)	0 (17,66)	0 (17,66)	18 (30,107)

Table 19: Number of Canadian effluent and freshwater sites where risk quotients for NPEs exceeded 1 using a distributional assessment. (# of exceedances/total number of sites)

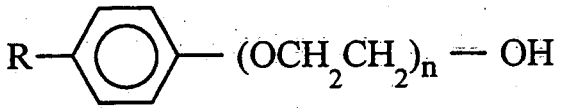
Environmental Compartment	Sites		NP	NP1EO	NP2EO	NP9EO	NP1EC	NP2EC	TEQ
Effluents	Textiles	untreated	1/2	2 /2	2/2	2/2	0/1	0/1	2/2
		on-site treatment	0/2	0/1	0/1	0/2	0/2	0/2	0/2
		going to MWWTP	na	na	na	na	na	na	na
	Pulp and Paper	prior to 1998	1/14	2/13	2/14	-	-	-	4/14
		after 1998	0/19	0/3	1/3	0/3	0/15	0/15	1/19
	MWWTP	primary	3/8	2/10	1/10	0/8	0/3	0/3	5/10
		secondary	0/21	1/20	0/20	0/16	0/14	0/14	1/21
		tertiary	0/7	0/7	0/7	0/6	0/6	0/6	0/7
		lagoon	0/5	0/5	0/5	0/4	0/4	0/4	0/5
Aquatic	Rivers		1/25	1/12	0/12	0/3	0/1	0/1	2/25
	Lakes		0/5	2/4	0/4	-	-	-	2/5
	Harbours		0/12	1/12	1/12	-	-	-	4/12



4-nonylphenol (NP)

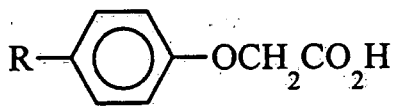


4-*tert*-octylphenol (OP)



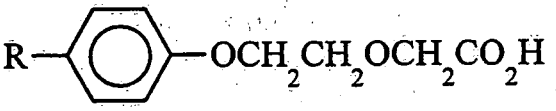
R=C_9H_{19}, nonylphenol ethoxylates (NPEO)

R=C_8H_{17}, octylphenol ethoxylates (OPEO)



R=C_9H_{19}, nonylphenoxyacetic acid (NP1EC)

R=C_8H_{17}, octylphenoxyacetic acid (OP1EC)



R=C_9H_{19}, nonylphenoxyethoxyacetic acid (NP2EC)

R=C_8H_{17}, octylphenoxyethoxyacetic acid (OP2EC)

Figure 1. Chemical structures for NP, OP, APEs, AP1EC, and AP2EC.

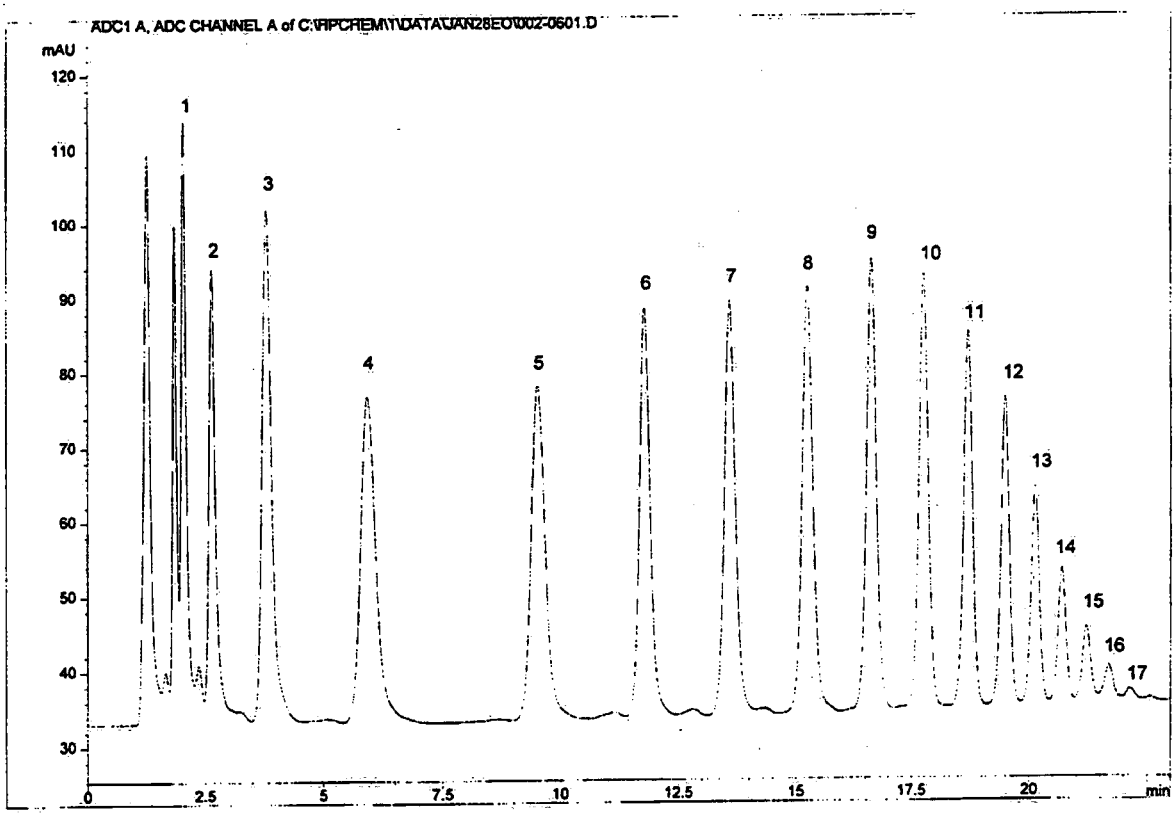


Figure 2. The separation of NPnEO (n=1-17) oligomers in a standard extract on a Hypersil APS column.

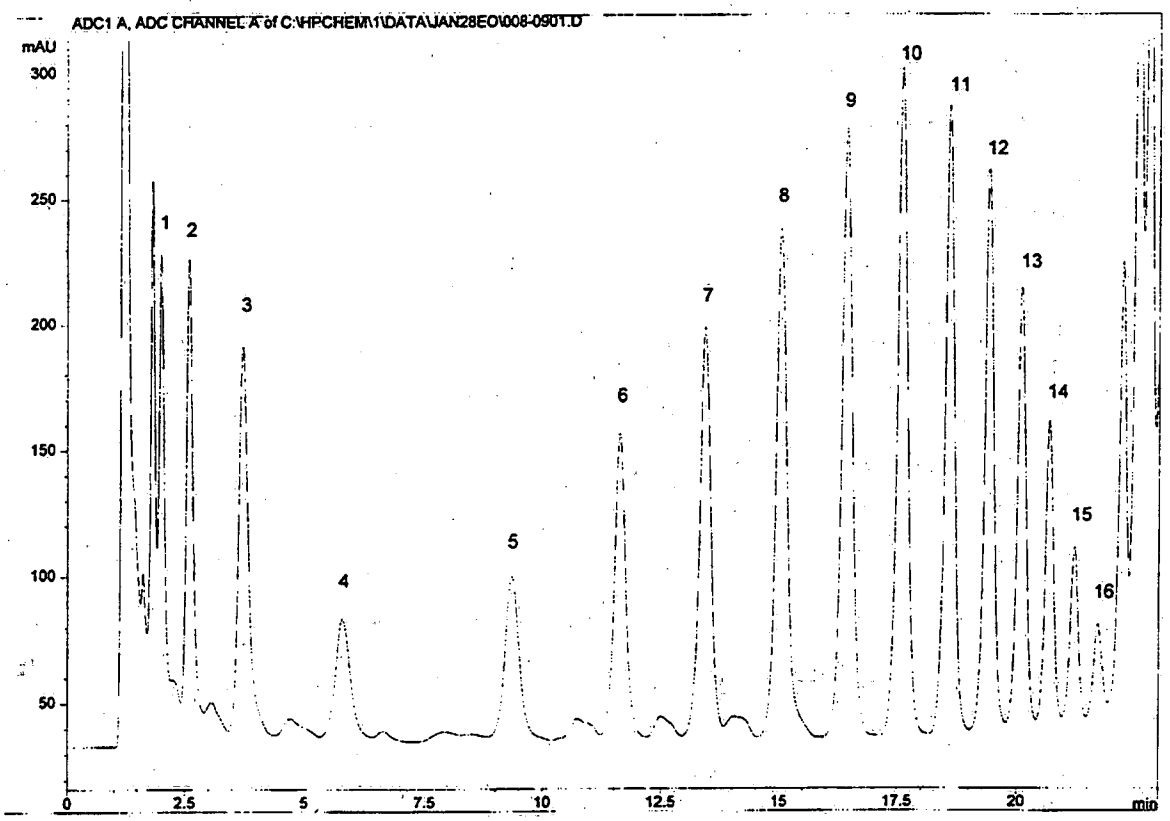


Figure 3. The separation of NPnEO (n=1-17) oligomers in a sludge extract on a Hypersil APS column.

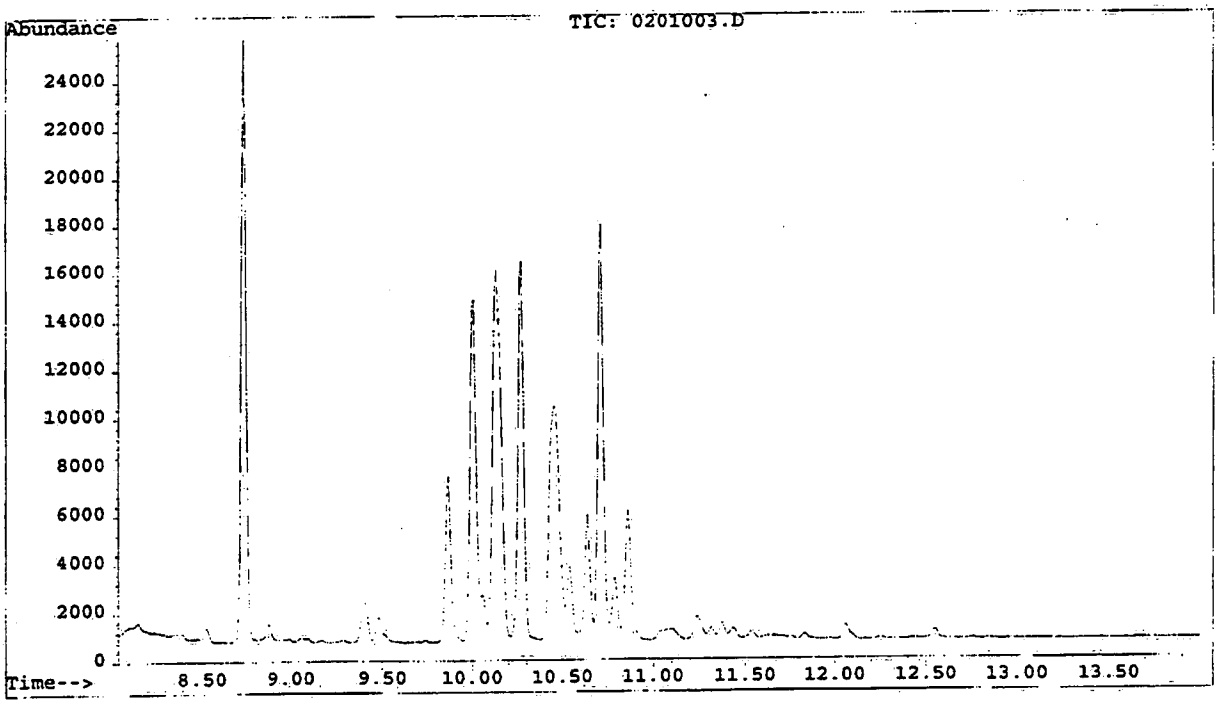


Figure 4. Total ion chromatogram of an acetylated NP/OP standard.

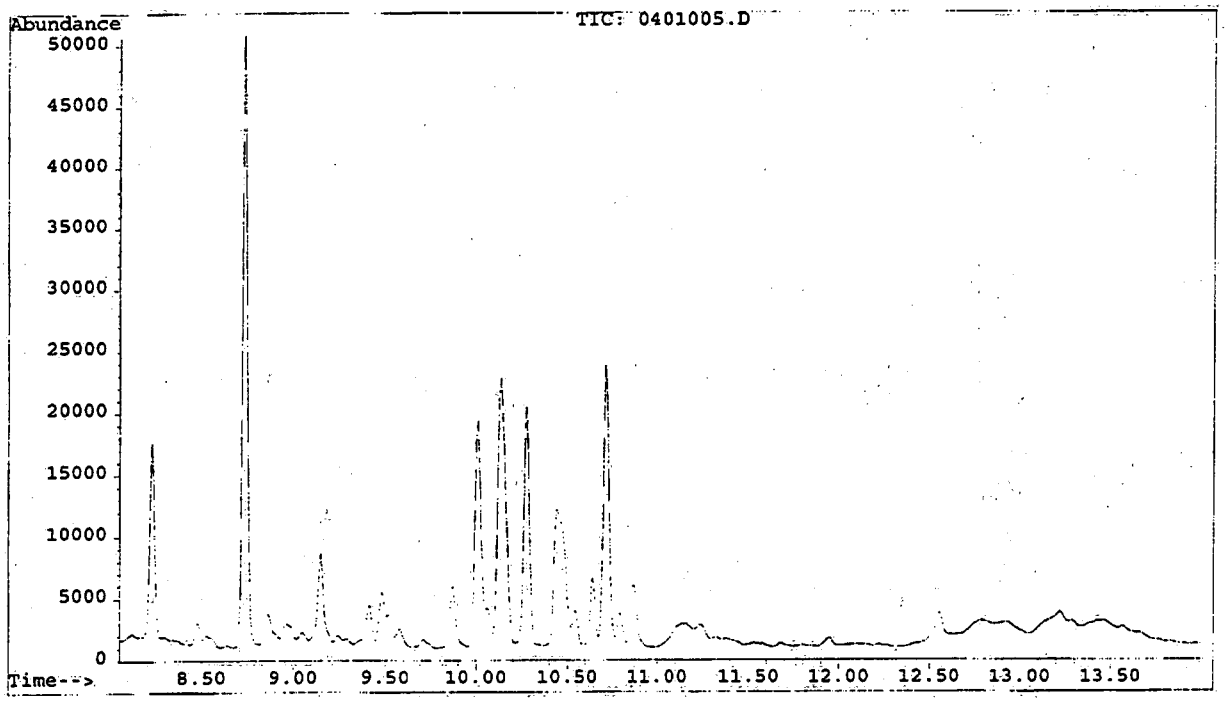


Figure 5. Primary sewage effluent extract depicting the occurrence of NP and OP.

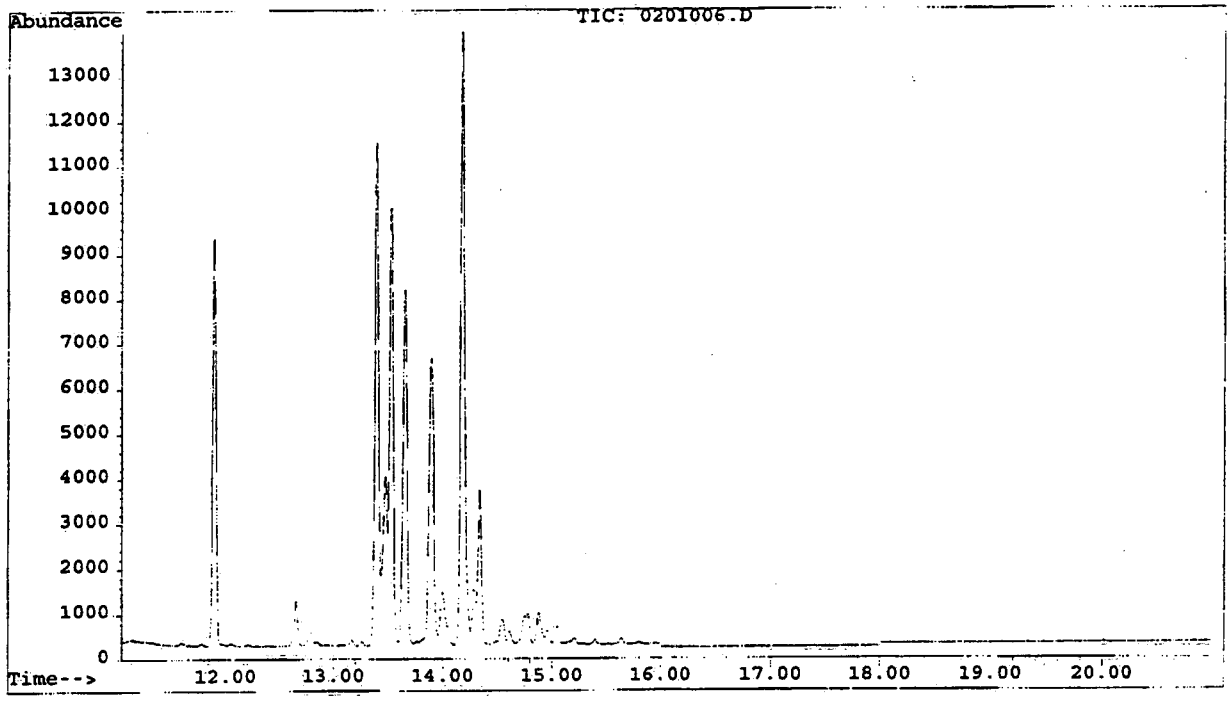


Figure 6. APEC methyl esters.

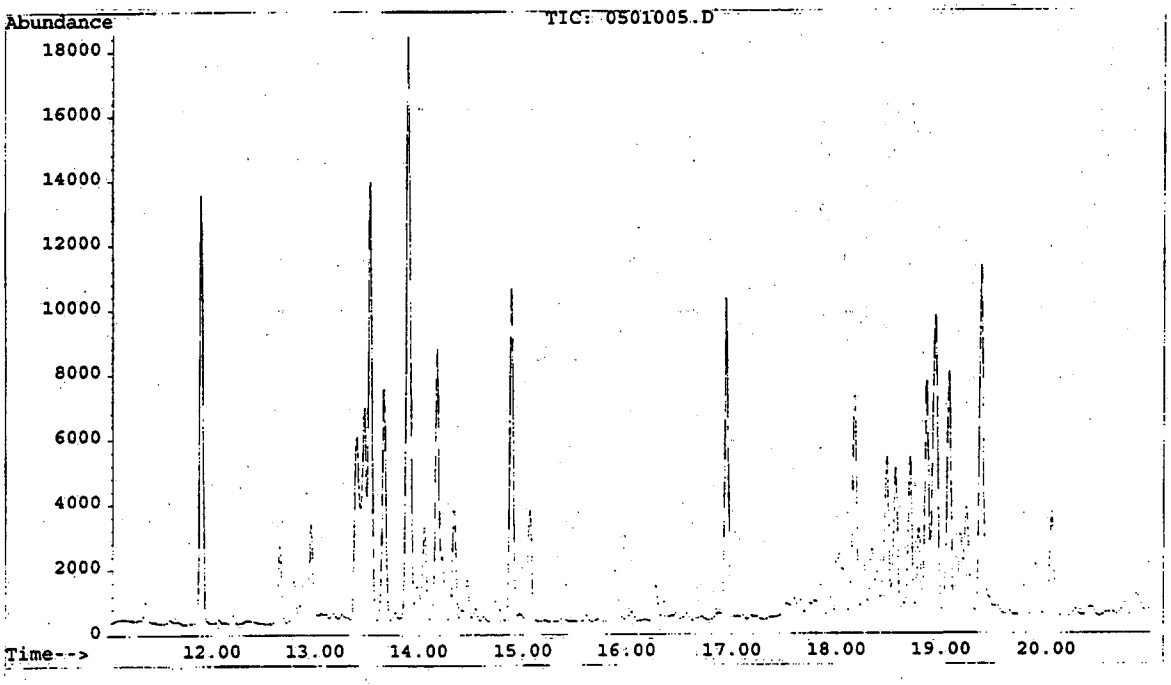
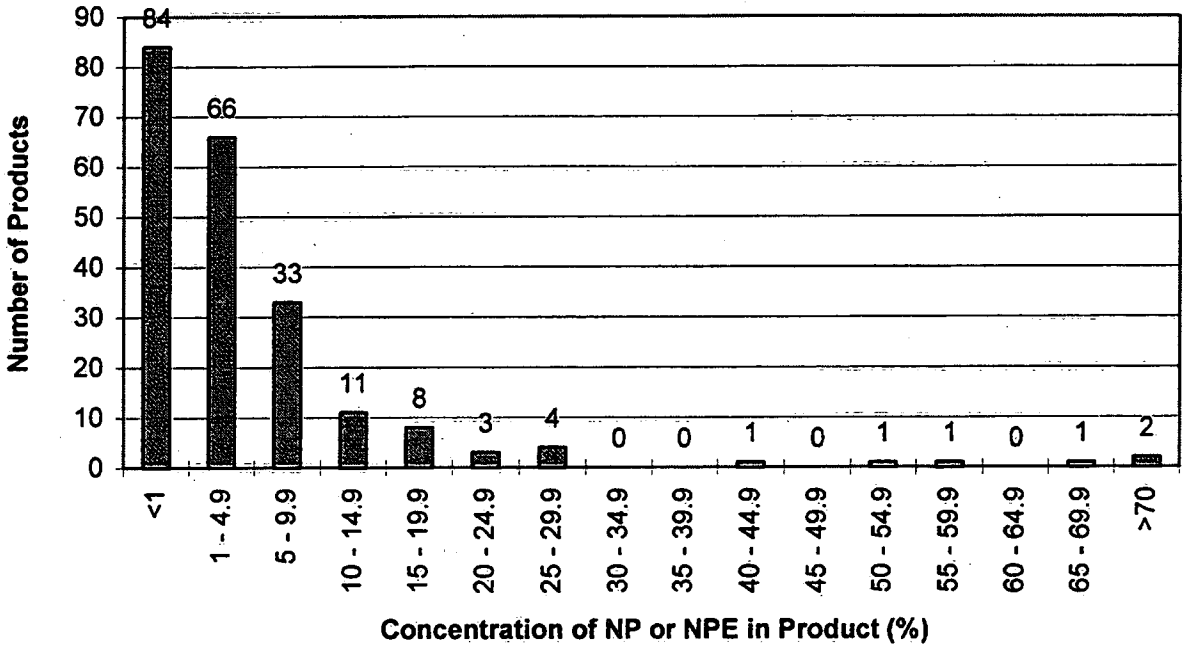


Figure 7. Final sewage effluent extract depicting the occurrence of OP1EC/NP1EC and OP2EC/NP2EC.



Number of products (total) = 211; NP appeared in 7 products and NPEs appeared in 207 products

Identified uses of products (number of products): acaracide (41); adjuvant (10); air sanitizer (1); disinfectant (2); antifouling paint (4); antisapstain (1); fungicide (22); herbicide (61); insect repellent (5); insecticide (84); joinery and remedial wood preservative (1); laundry additive (1); material preservative (9); plant growth regulator (2); sanitizer (12); slimicide (10); swimming pool bactericide (1)*

*Note: the total number of products does not equal the total number of products in their various uses since some products may have more than one use.

Figure 8. Nonylphenol and nonylphenol ethoxylate concentrations in pesticide products.

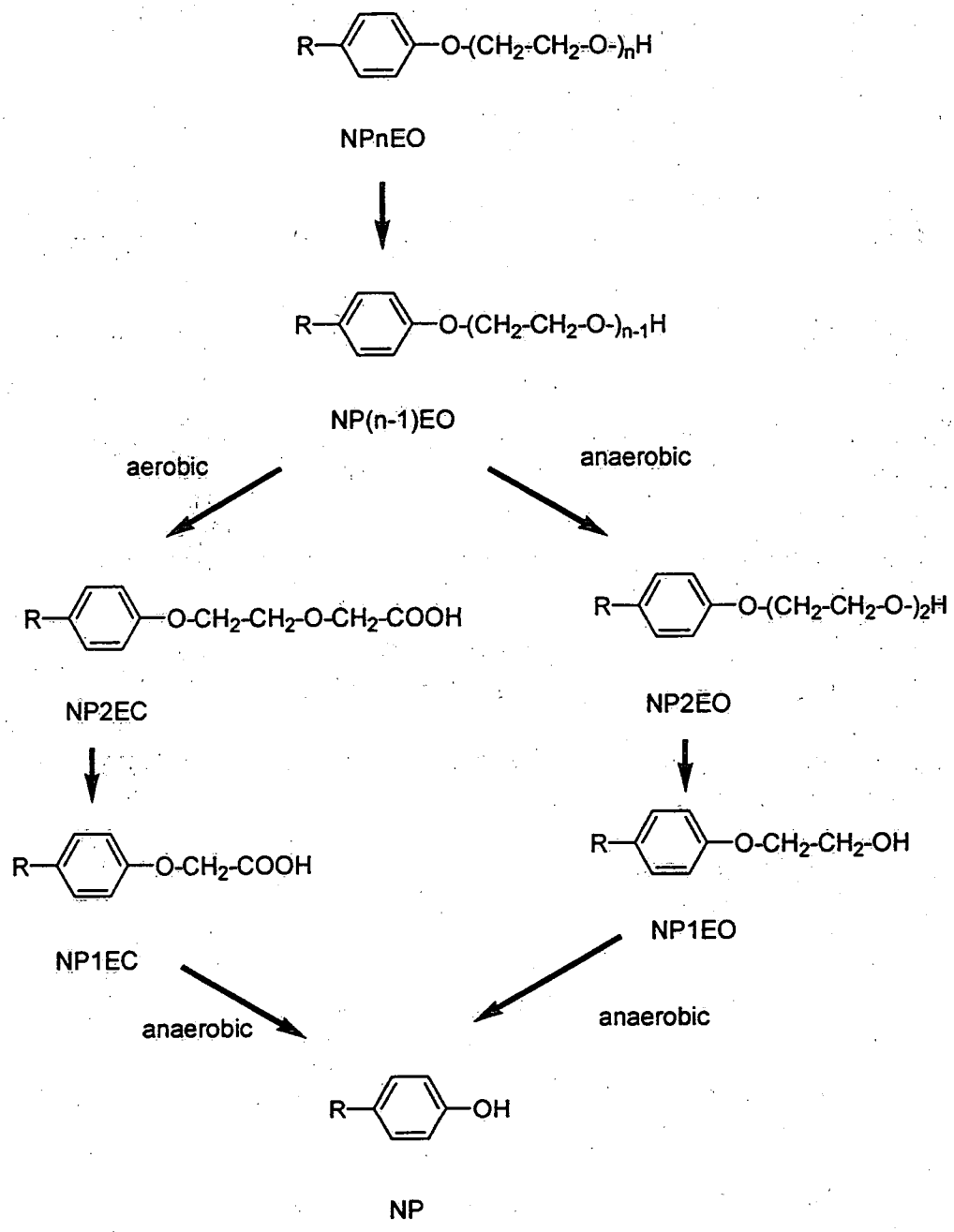


Figure 9. Biological degradation pathway for NPEs

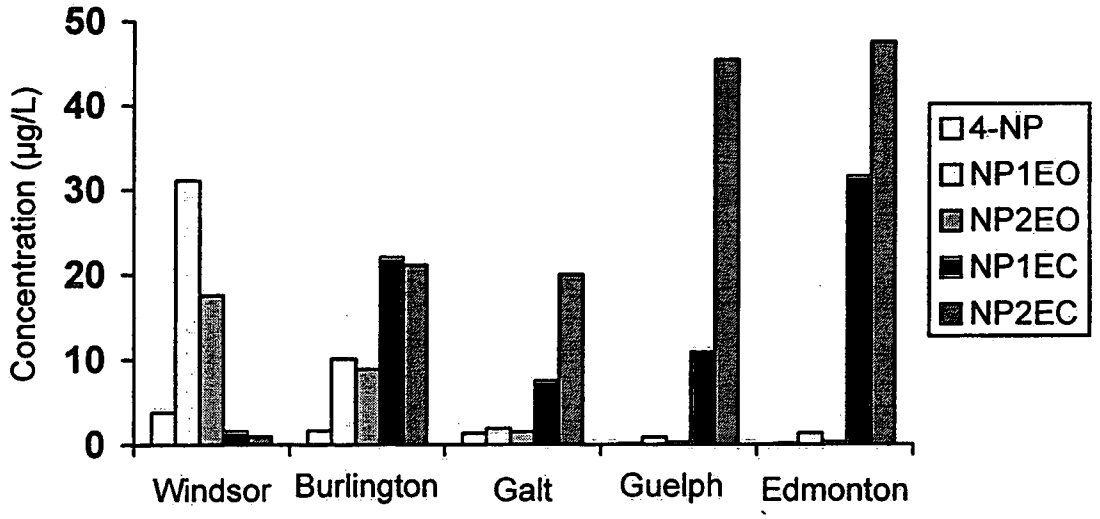


Figure 10. Concentration of NP, lower-chain NPEs and NPECs in various types of municipal wastewater treatment plant effluents (Windsor = primary treatment; Burlington = secondary treatment; Galt, Guelph and Edmonton = tertiary treatment) (data from Bennie, 1998a)

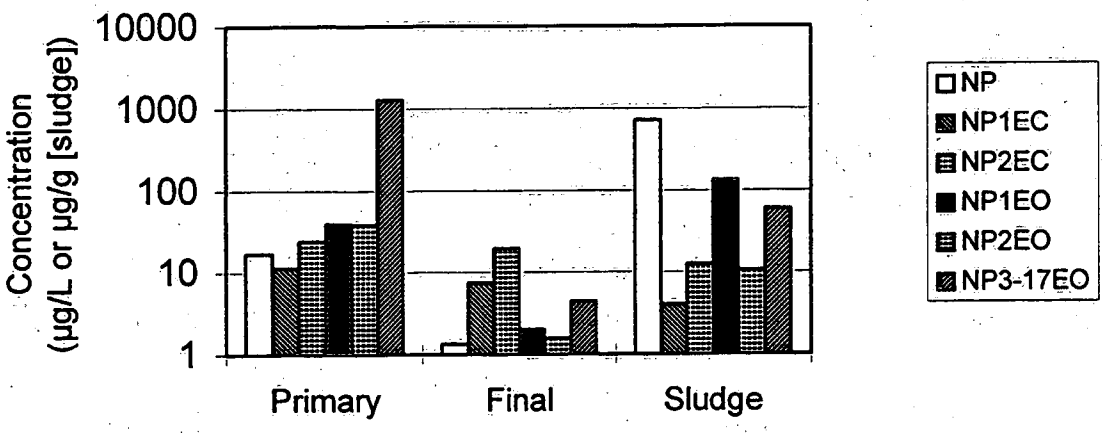


Figure 11. Distribution of NP, NPEs and lower-chain NPECs in effluent and sludge from a tertiary-treated municipal wastewater treatment plant. Sample taken September 1997, representing the mean of eight 24-hour composites (Water Technology International Corporation, 1998b)

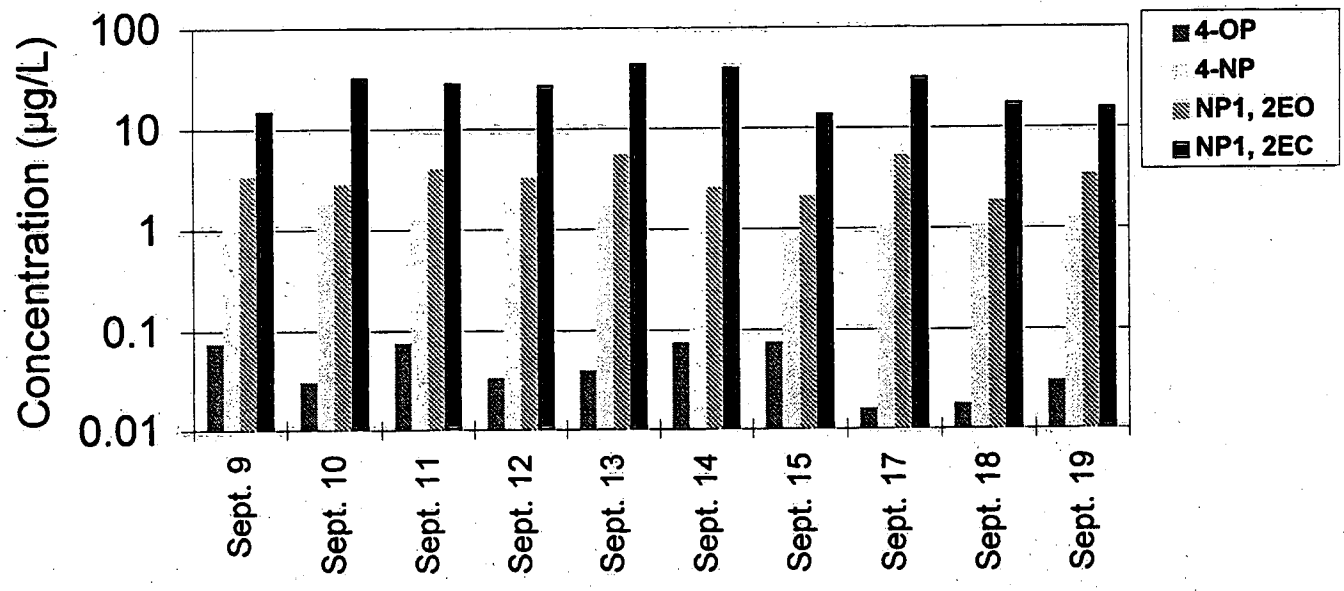


Figure 12. The daily variability of alkylphenol concentrations in final effluent from a tertiary treated MWWTP (Site A) 1997, n=10. (Water Technology Corporation International, 1998b)

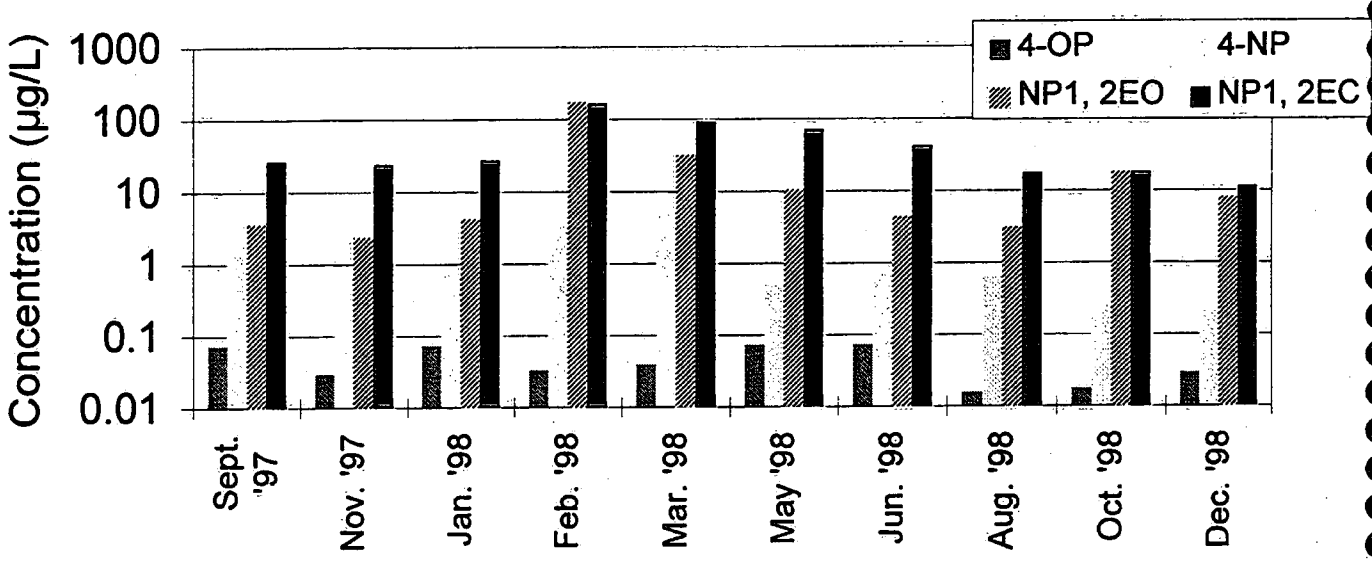


Figure 13. The seasonal variability of alkylphenol concentrations in final effluent from a tertiary treated MWWTP (Site A) 1997, n=10. (Water Technology Corporation International, 1998b)

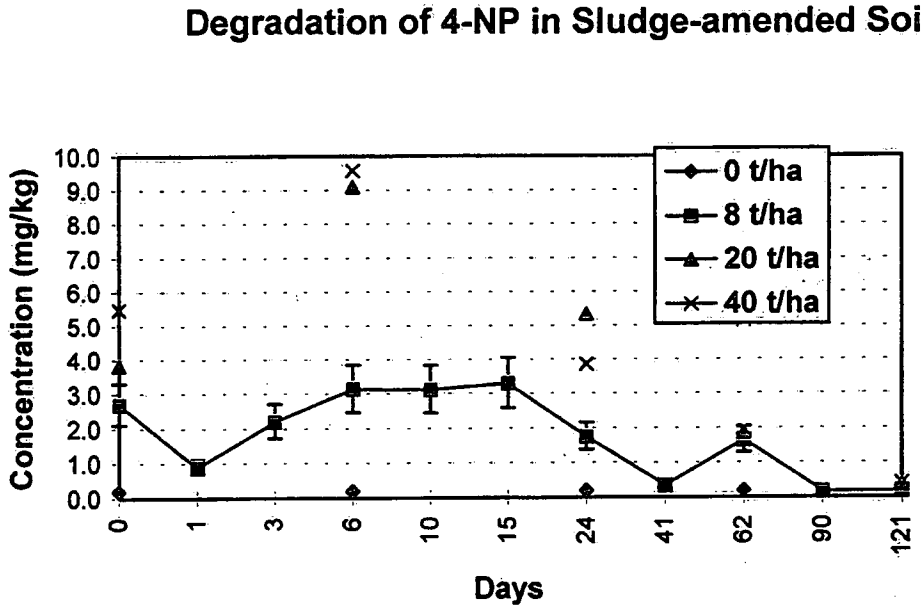


Figure 14. The disappearance of NP in soil plots after the application of sewage sludge.

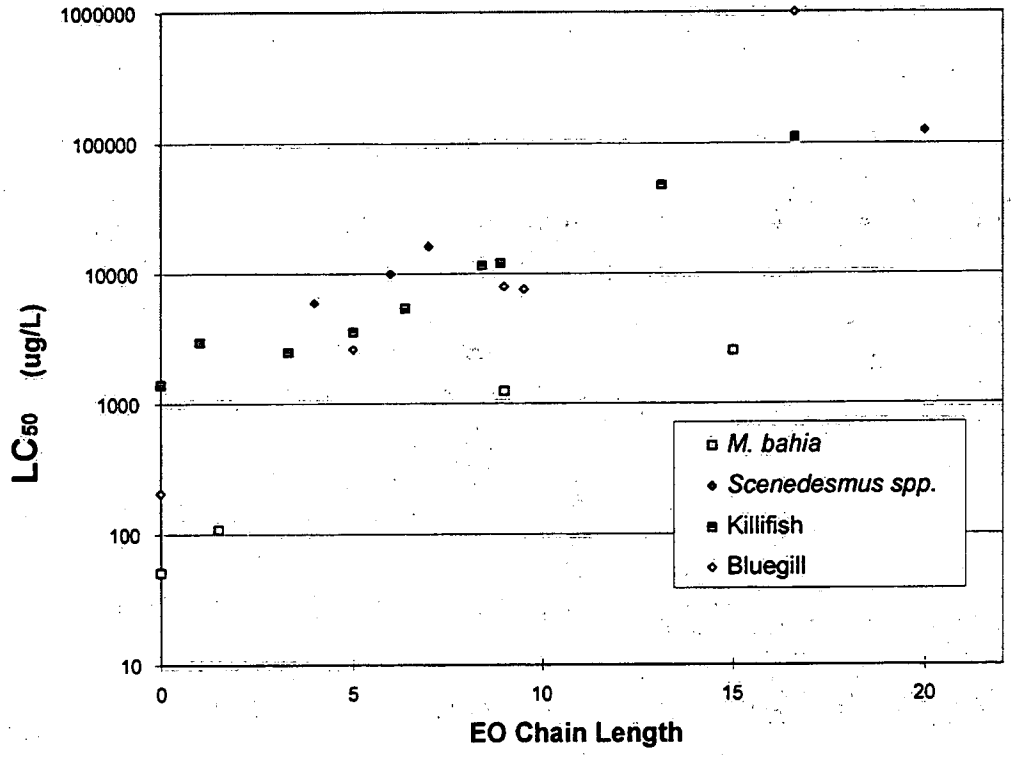


Figure 15. Relationship between EO chain length and toxicity in algae (*Scenedesmus* spp. - Janicke *et al.*, 1969), mysids (*Mysidopsis bahia*, 48 h - Hall *et al.*, 1989), Japanese killifish (*Oryzias latipes*, 48 h - Yoshimura, 1986) and bluegill sunfish (*Lepomis macrochirus*, 96 h - Brooke, 1993a,b; Macek and Krzeminiski, 1975).

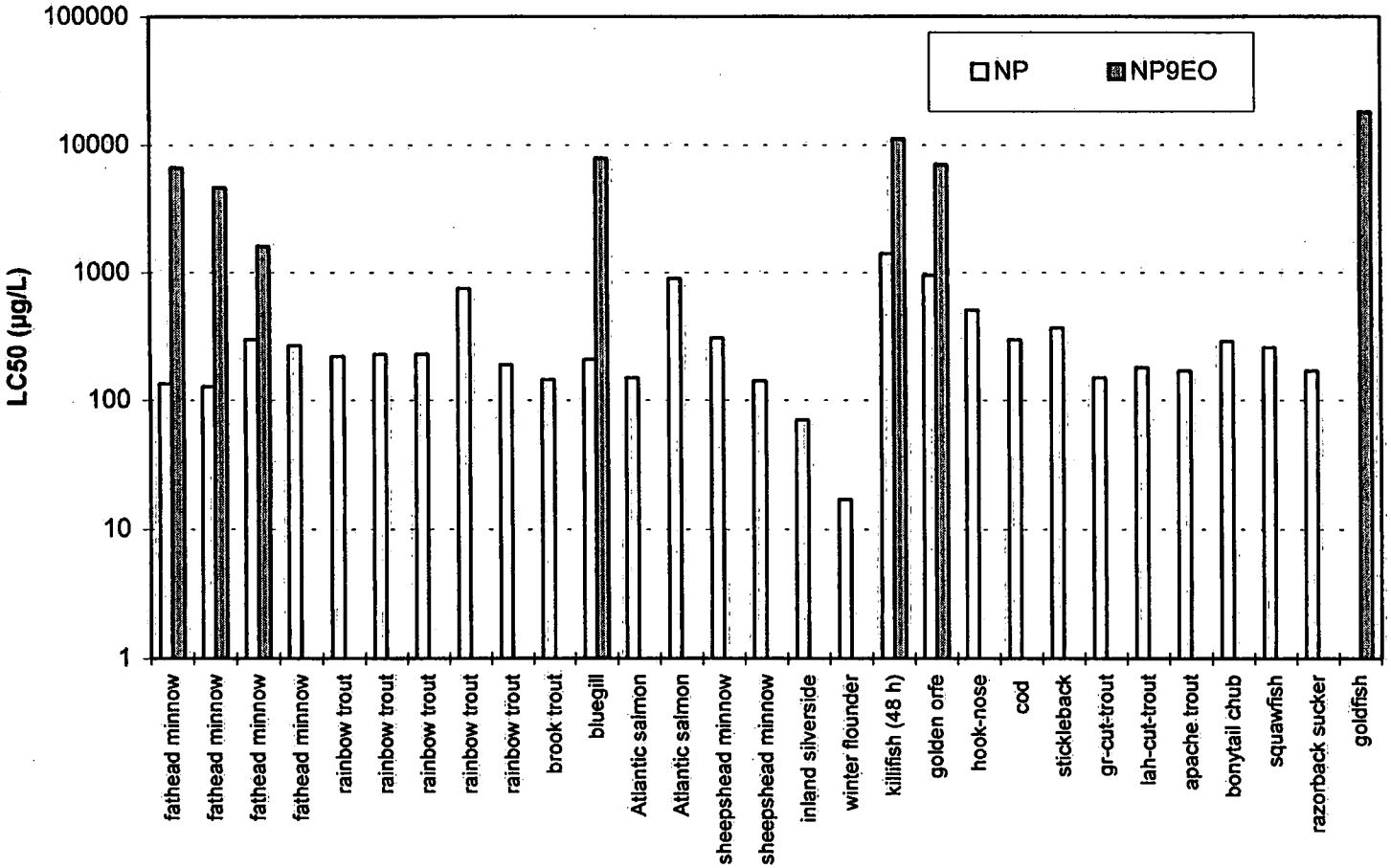


Figure 16. Acute toxicity of NP and NP9EO to various fish species.

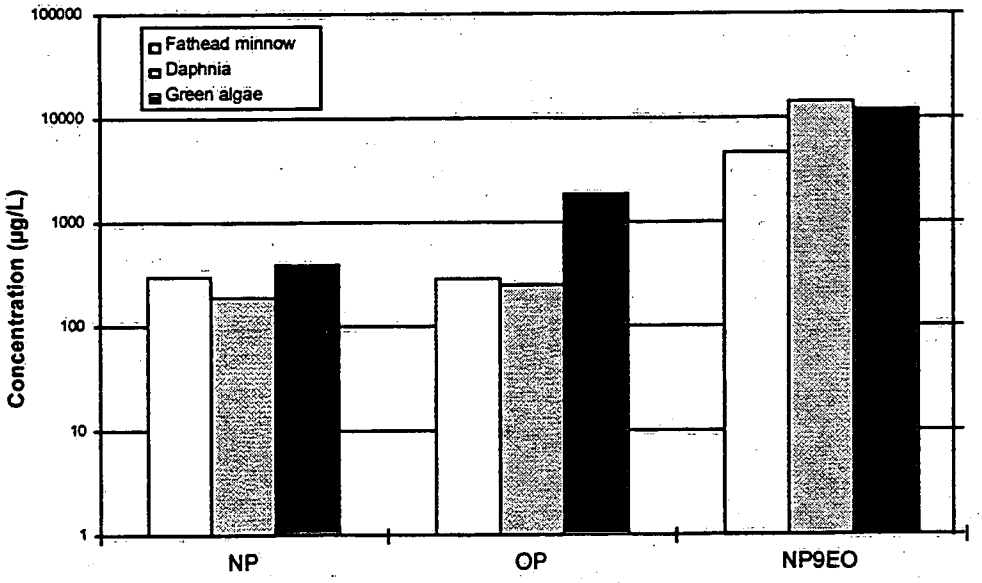


Figure 17. Relative toxicity of NP and NP9EO to the fathead minnow, *Pimephales promelas* (96-h LC_{50}), water flea, *Daphnia magna* (48-h LC_{50}), and the green alga, *Selenastrum capricornutum* (96-h EC_{50}) Based on the data generated by the Chemical Manufacturers Association (Weeks *et al.*, 1996; Naylor, 1995).

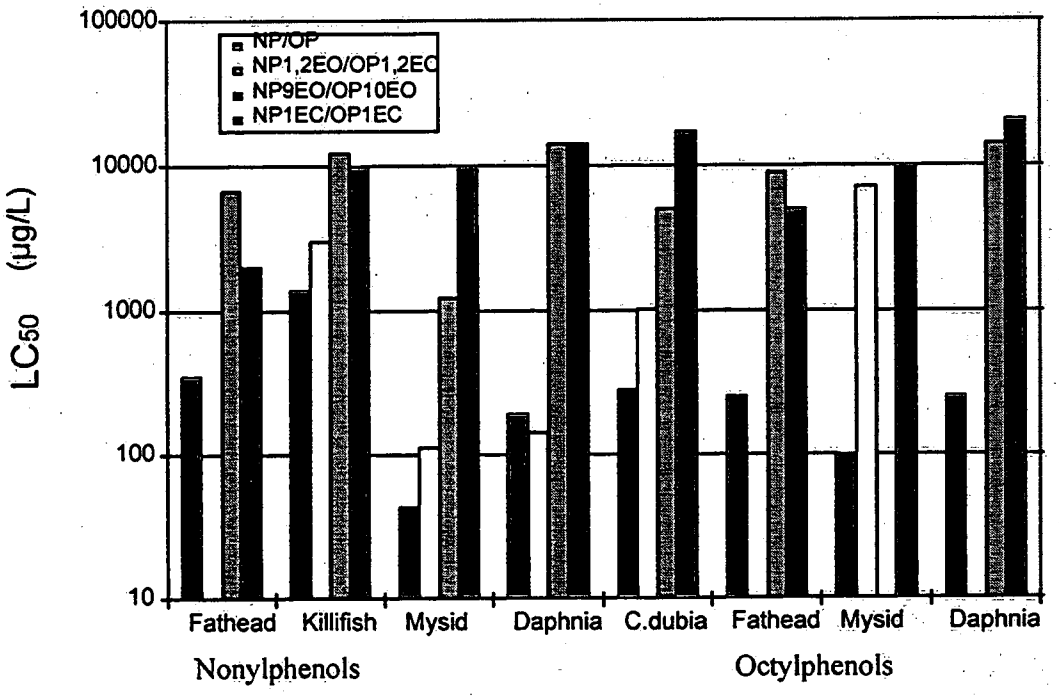


Figure 18. Relative toxicity of APs, APEs and APECs in fathead minnow, *Pimephales promelas* (96-h LC_{50}), killifish, *Oryzias latipes* (48-h LC_{50}), Daphnia, *Daphnia magna*, (48-h LC_{50}), *Ceriodaphnia dubia* (7-d LC_{50}), Mysids, *Mysidopsis bahia* (96-h LC_{50}).

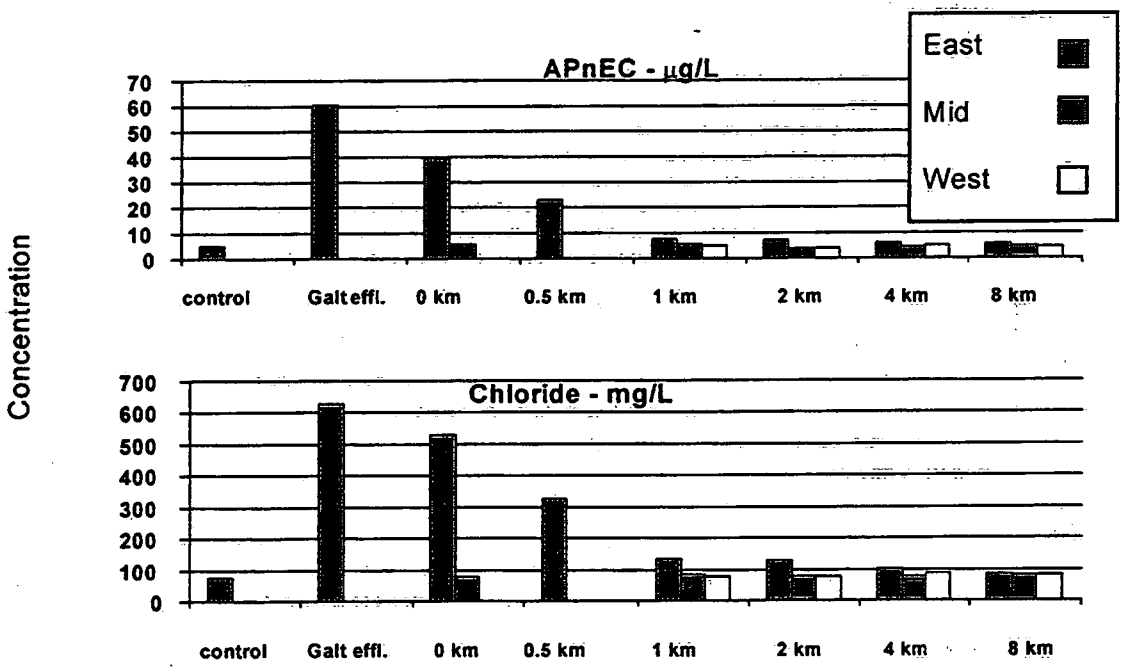


Figure 19. Concentrations of alkylphenols relative to chloride concentrations in the Grand River

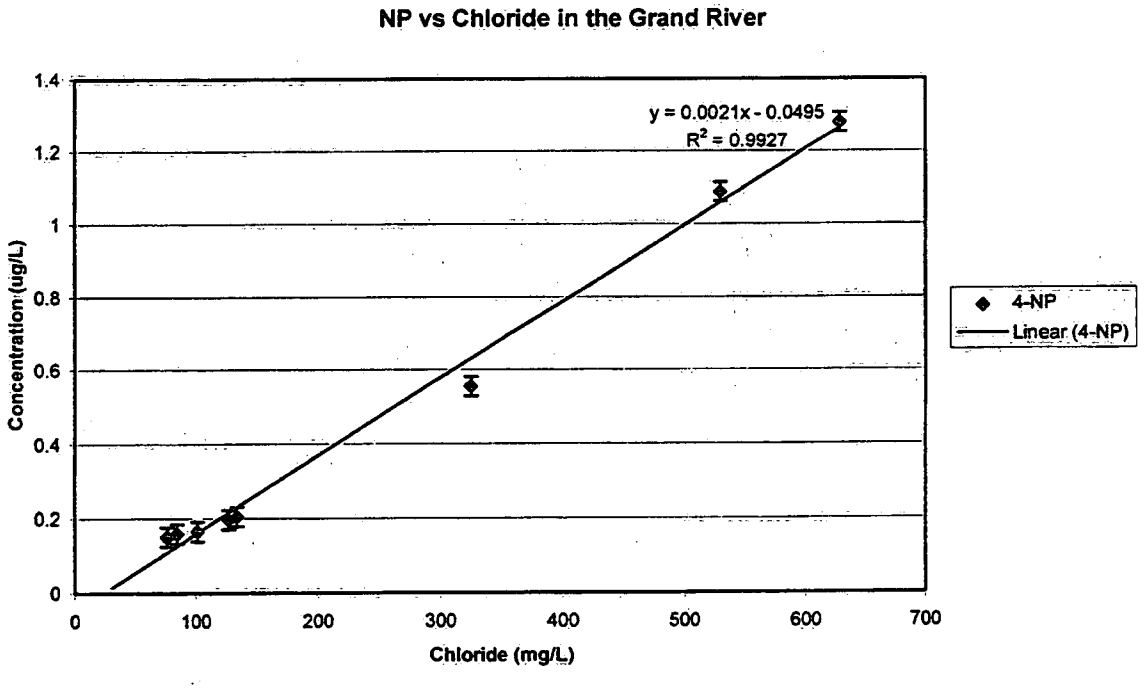


Figure 20. The concentration of NP downstream of the Galt municipal wastewater treatment plant in comparison to a conservative tracer, chloride.

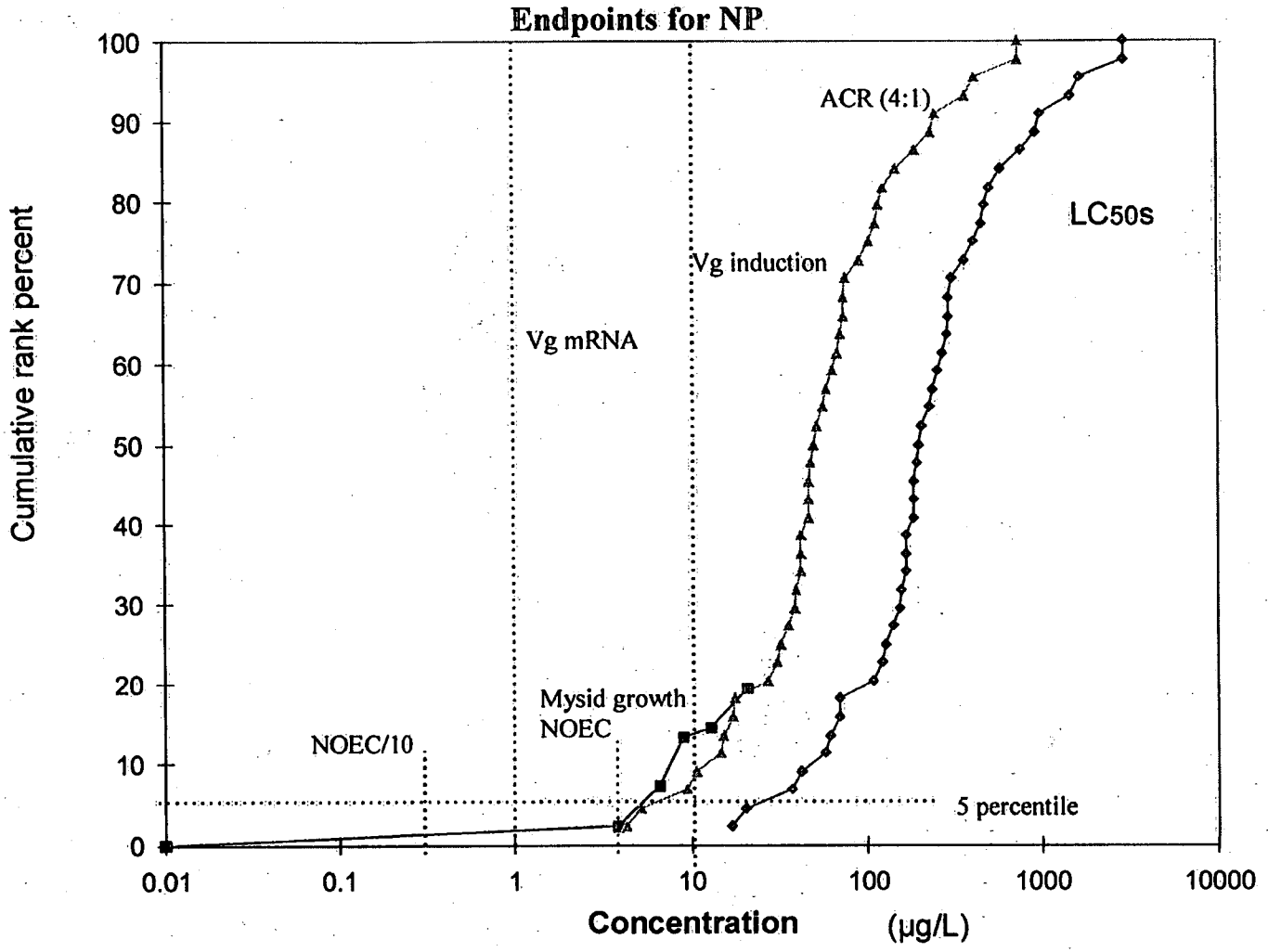


Figure 21. Assessment endpoints for nonylphenol. NOEC indicated represents growth.

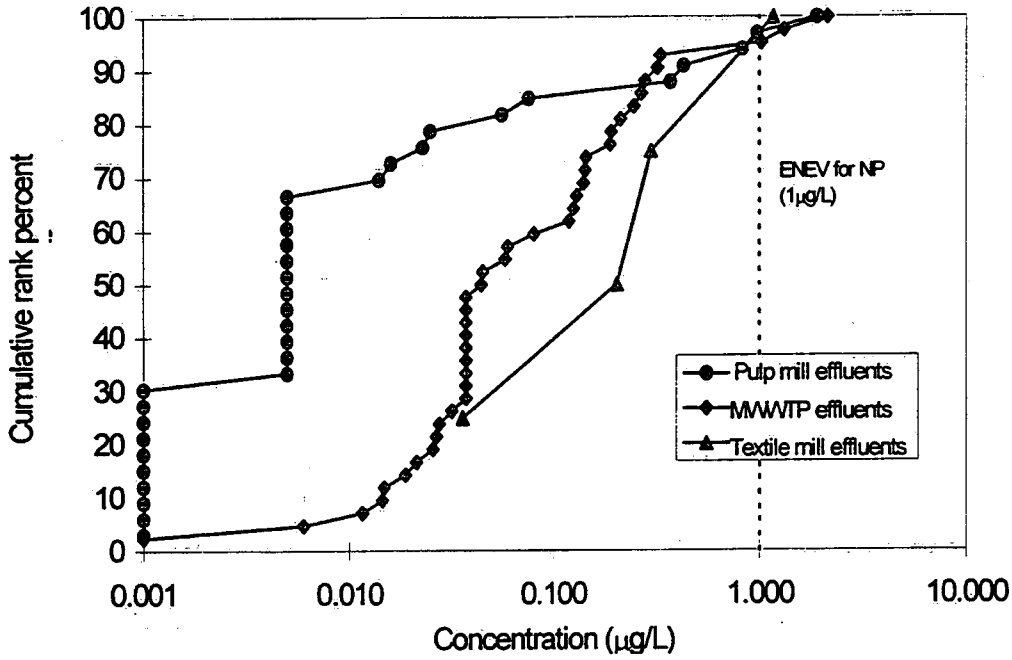


Figure 22. Environmental concentrations of NP in pulp mill, municipal wastewater treatment plant and textile mill effluents based on site averages and a dilution of 10:1.

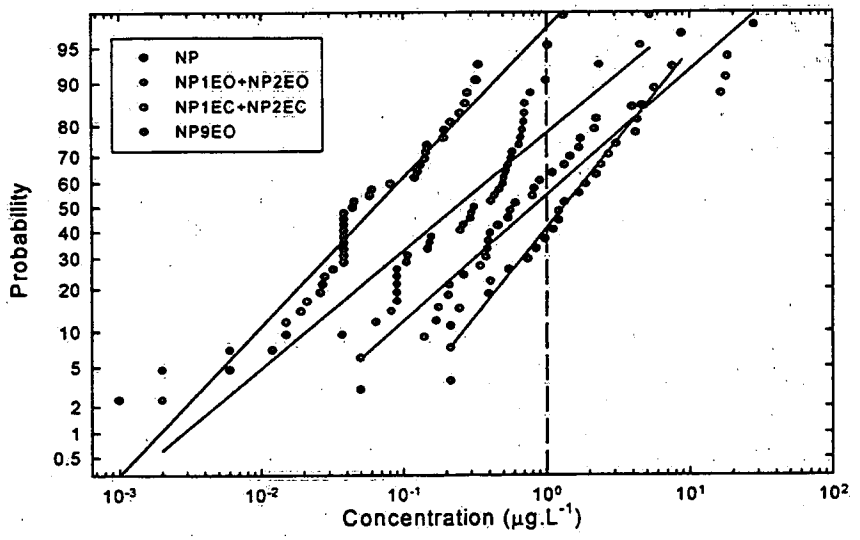
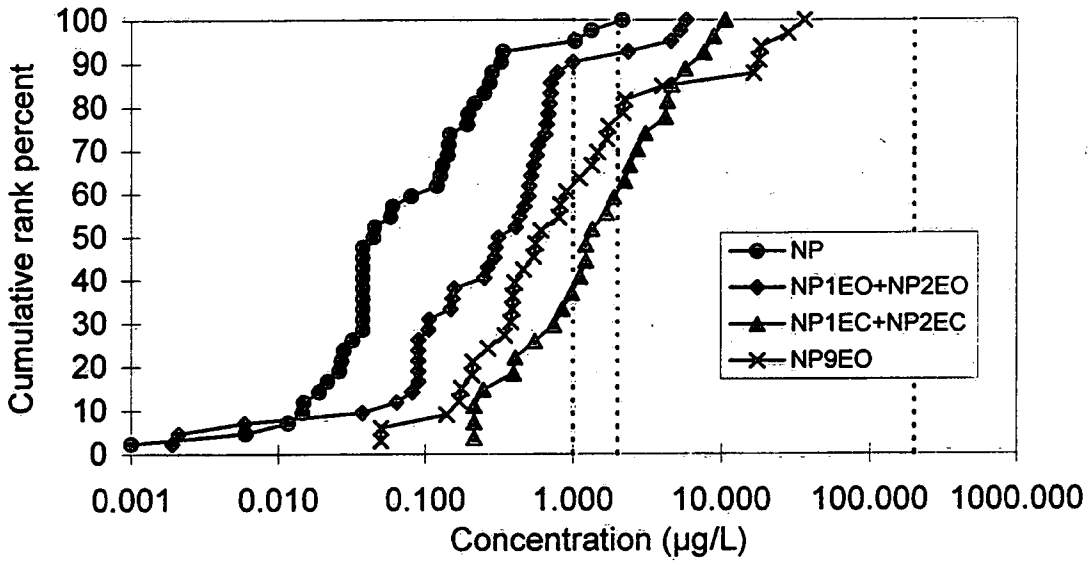
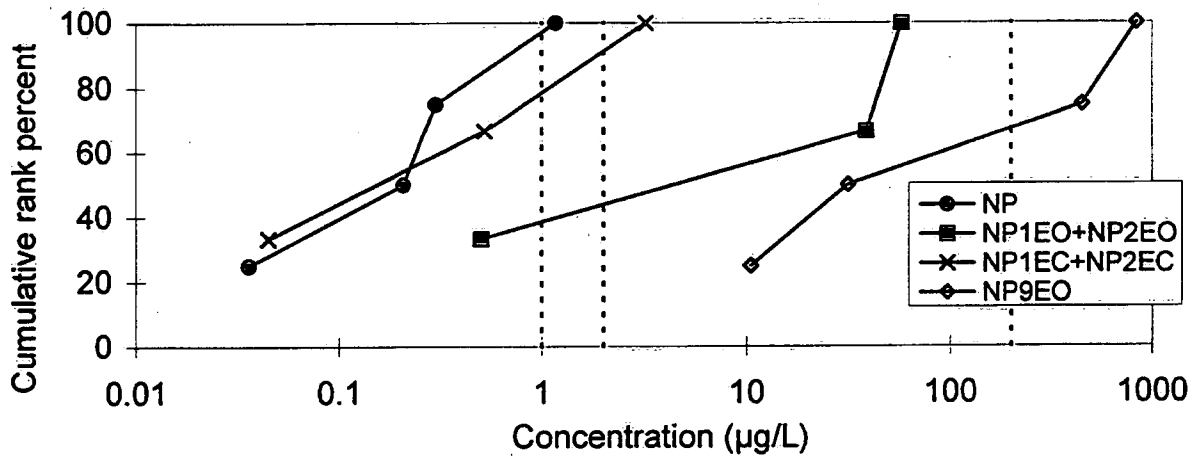


Figure 23. The distribution of predicted concentrations of NP and NPEs near municipal wastewater treatment plants (all treatment types combined).



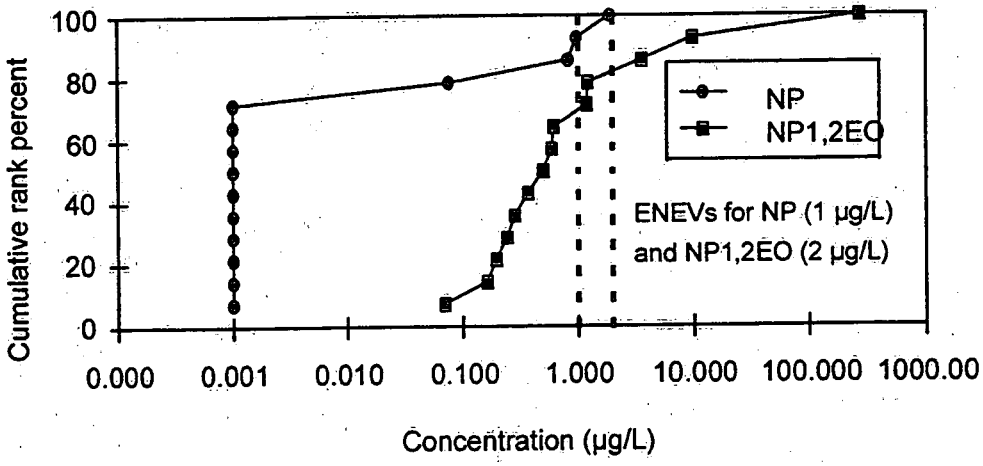
—— = ENEVs for NP(1 µg/L), NP1,2EO (2 µg/L) and NP1,2EC or NP9EO (200 µg/L)

Figure 24. Estimated environmental concentrations of NP, NP1EO+NP2EO, NP1EC+NP2EC and NP9EO near municipal wastewater treatment plants (primary, secondary and tertiary)



—— = ENEVs for NP (1 µg/L); NP1,2EO (2 µg/L); NP1,2EC or NP9EO (200 µg/L)

Figure 25. Estimated environmental concentrations of NP, NP1EO+NP2EO, NP1EC+NP2EC, NP9EO near textile mills discharging to the environment.



(----- = ENEVs for NP (1 µg/L), NP1,2EO (2 µg/L) and NP1,2EC or NP9EO (200 µg/L))

Figure 26. Estimated environmental concentrations of NP and NP1EO+NP2EO near pulp mills prior to 1998

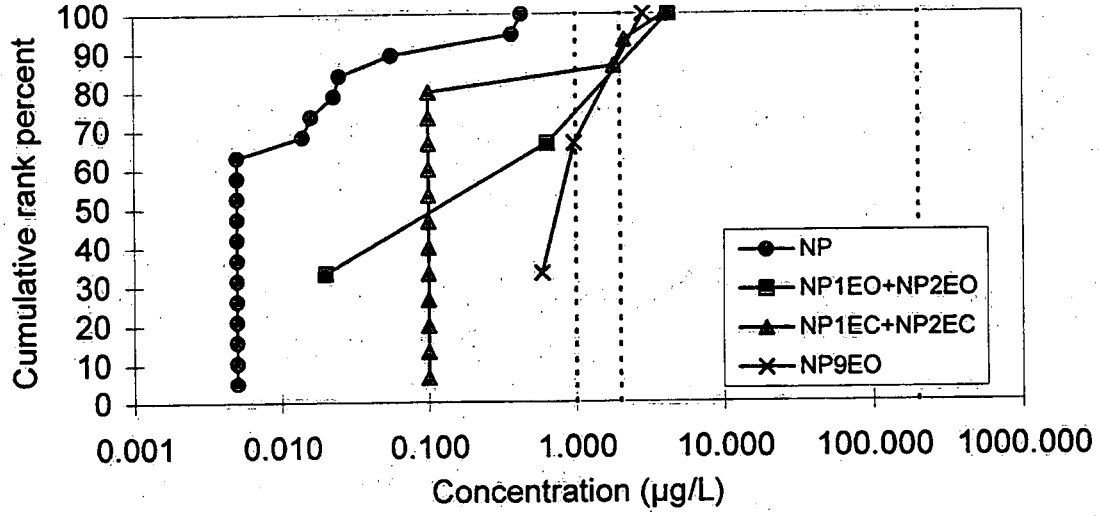


Figure 27. Estimated environmental concentrations of NP, NP1EO+NP2EO, NP1EC+NP2EC and NP9EO near pulp mills after 1998

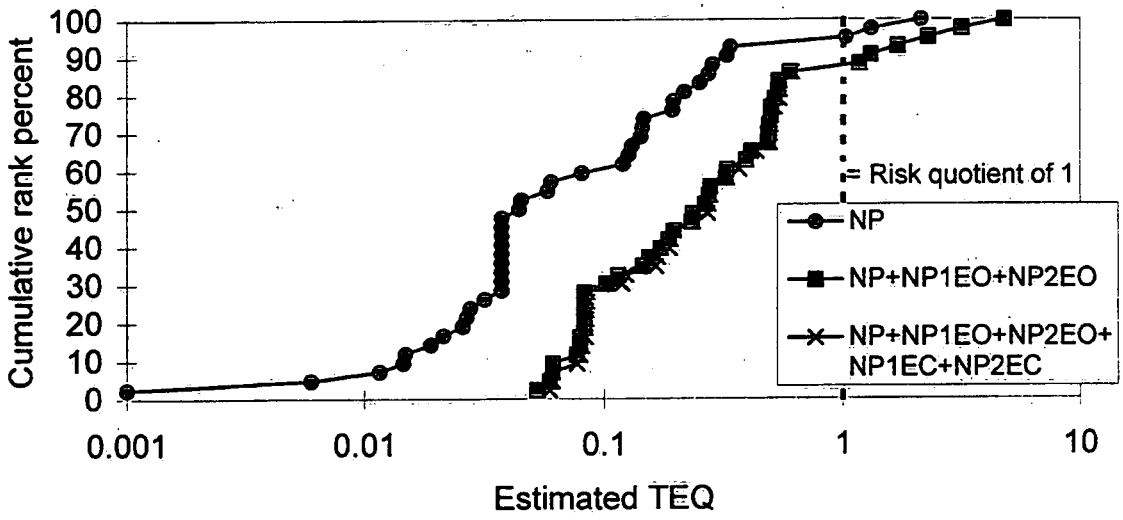


Figure 28. Estimated toxic equivalency quotients (TEQ) of NPEs near municipal wastewater treatment plants.

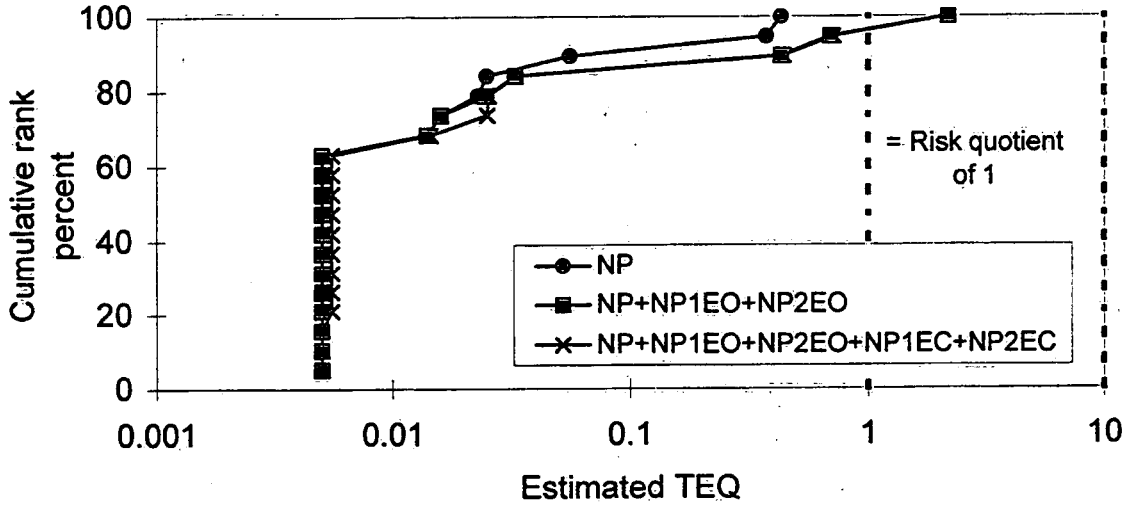


Figure 29. Estimated toxic equivalency quotients (TEQ) of NPEs near pulp mills after 1998.

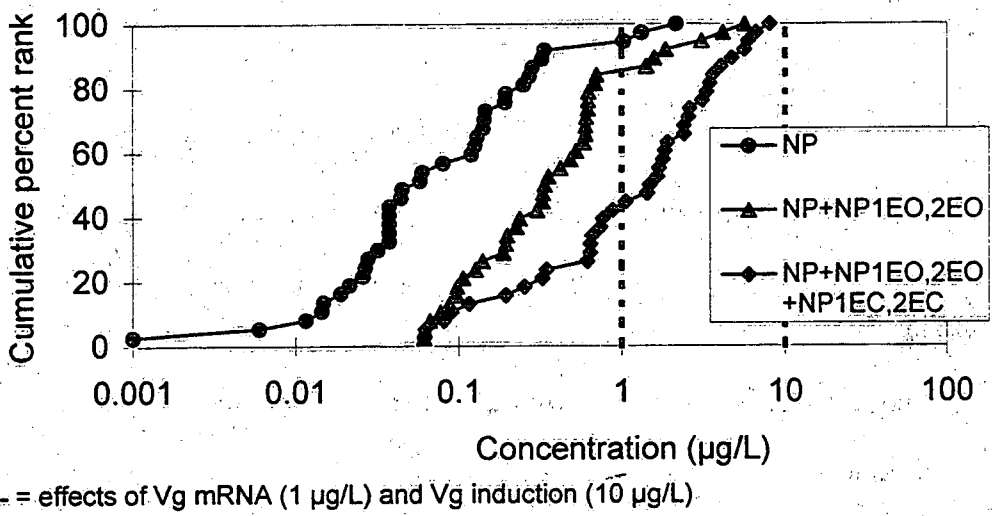


Figure 30. The estimated estrogenic equivalency (EEV_{EEQ}) of NPEs near municipal wastewater treatment plants

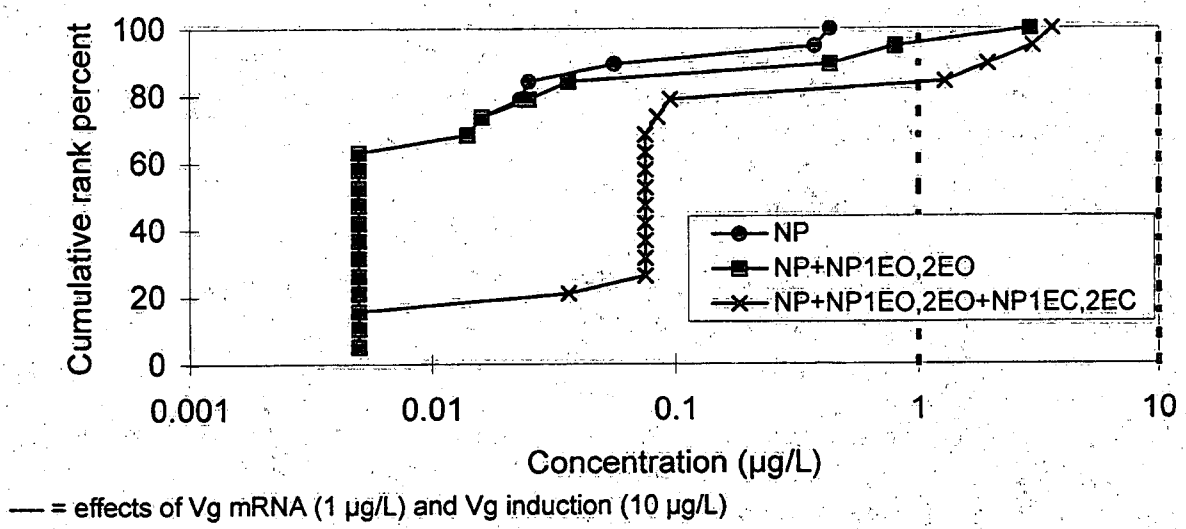


Figure 31. The estimated estrogenic equivalency (EEV_{EEQ}) of NPEs near pulp mills after 1998.

Appendix A: Tables of data and quotients used in the risk characterization. The following appendices are available as Excel spreadsheets in the electronic version of the supporting document.

Table A1. The concentration of AP/APEs in effluents from various sites by sector.

Table A2. The risk quotient values for the hyperconservative assessment; no dilution applied.

Table A3. The effluent concentration data for conservative assessment, using ½ detection limits, and a dilution factor of 10:1

Table A4. Quotient values for conservative assessment of effluents.

Table A5 Receiving water concentrations reported.

Table A6. Receiving water risk quotients for the conservative assessment.

Table A7. Sediment concentrations reported.

Table A8. Sediment risk quotients for the conservative assessment. Comparison to ENEV based on acute toxicity to chironomids. APEs converted to relative toxicity of NP and compared to NP to ENEV based on acute toxicity to chironomids.

Table A9. Sludge concentrations reported.

Table A10. Sludge quotients for sludge applied to fields. Based on a dilution factor of 0.004 in soil. Predicted concentrations are compared to the NP ENEV based on the earthworm EC_{10} for reproduction.

Table A11.Risk quotient values for environmental concentrations estimated from effluent concentration (dilution factor of 10:1) based on endocrine disruption endpoints using a level of concern of 1 µg/L for NP and relative NP estrogenic equivalents

Table A12. Risk quotient values for receiving waters based on endocrine disruption endpoints using a level of concern of 1 µg/L for NP and relative NP estrogenic equivalents.

Data sources for the Appendix A include:

1. Bennie (1998b) personal communication, unpublished data
2. Lee and Peart (1999)
3. Bennie (1998a)
4. Water Technology Incorporated (1998a)
5. Lee *et al.* (1997)
6. Bennie *et al.* (1997)
7. Brewer *et al.* (1999)
8. Bennett *et al.* (1999)

Appendix B: Scientific and Technical Literature Databases Searched for Nonylphenol and Its Ethoxylates

The following databases were searched for information on nonylphenol and its ethoxylates for the period 1960-1998.

National Pollutant Release Inventory
Domestic Substances List
CPI Profile
CCINFO (Chemical Evaluation Sourcing and Retrieval System), RTECS, MSDS, CHEMINFO)
Current Contents
ENVIRODAT
Databases: Government of Canada
Databases: Provinces and Territories
ENVIROLINE
POLLUTION ABSTRACTS
TOXLINE
Chemical Abstracts Service
Biological Abstracts
Primary journals

The assessment included data from research commissioned specifically for use in this assessment. The data were obtained following December, 1998. Additional literature was considered for the assessment as the authors became aware of its existence. A separate PSL assessment of textile mill effluents was initiated concurrently and is ongoing.

Appendix C: Contributors

Environmental Resource Group

D.T. Bennie, National Water Research Institute, Environment Canada
P. Cureton, Commercial Chemicals Evaluation Branch, Environment Canada
H.-B. Lee, National Water Research Institute, Environment Canada
K. Lloyd, Commercial Chemicals Evaluation Branch, Environment Canada
R.J. Maguire, National Water Research Institute, Environment Canada
M.R. Servos, National Water Research Institute, Environment Canada

PRINTED IN CANADA
IMPRIMÉ AU CANADA

ON RECYCLED PAPER
SUR DU PAPIER RECYCLÉ

National Water Research Institute
Environment Canada
Canada Centre for Inland Waters
P.O. Box 5050
867 Lakeshore Road
Burlington, Ontario
L7R 4A6 Canada

National Hydrology Research Centre
11 Innovation Boulevard
Saskatoon, Saskatchewan
S7N 3H5 Canada

NATIONAL WATER RESEARCH INSTITUTE
INSTITUT NATIONAL DE RECHERCHE SUR LES EAUX

Institut national de recherche sur les eaux
Environnement Canada
Centre canadien des eaux intérieures
Case postale 5050
867, chemin Lakeshore
Burlington, Ontario
L7R 4A6 Canada

Centre national de recherche en hydrologie
11, boul. Innovation
Saskatoon, Saskatchewan
S7N 3H5 Canada

Environment Canada Environnement Canada

Canada