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Aquatic risk assessment of alcohol ethoxylates in North America and Europe ☆

S.E. Belanger[a,*], P.B. Dorn[b], R. Toy[c], G. Boeije[d], S.J. Marshall[e], T. Wind[f], R. Van Compernolle[b], D. Zeller[g]

[a]*Procter & Gamble Company, Cincinnati, OH, USA*
[b]*Shell Global Solutions US, Houston, TX, USA*
[c]*Shell Chemicals, Ltd., UK*
[d]*Procter & Gamble, Brussels, Belgium*
[e]*Unilever Research, Merseyside, UK*
[f]*Henkel KgaA, Dusseldorf, Germany*
[g]*BASF, Ludwigshafen, Germany*

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Abstract

An environmental risk assessment for alcohol ethoxylates (AE) is presented that integrates wastewater treatment plant monitoring, fate, and ecotoxicity research with a new application of mixture toxicity theory based on simple similar concentration addition of AE homologs in a species-sensitivity distribution (SSD) context. AEs are nonionic surfactants composed of a homologous series of molecules that range in alkyl chain length from 12 to 18 carbons and ethoxylates from 0 to 18 units. Chronic ecotoxicity of AE is summarized for 17 species in 60 tests and then normalized to monitoring data for AE mixtures. To do so, chronic aquatic toxicity was first expressed as EC_{10} per species (the concentration predicted to cause a 10% reduction in an important ecological endpoint). Normalization integrated several new quantitative structure–activity relationships for algae, daphnids, fish, and mesocosms and provided an interpretation of toxicity test data as a function of individual homologs in an AE mixture. SSDs were constructed for each homolog and the HC_5 (hazardous concentration protective of 95% of species based on a small biological effect [the chronic EC_{10}]) was predicted. Total mass of AE in monitored effluents from 29 sites in Europe, Canada, and the United States averaged 6.8, 2.8, and 3.55 μg/L, respectively. For risk assessment purposes, correction of exposure to account for fatty alcohol derived from sources other than AE and for sorbed components based on experimental evidence was used to determine AE concentrations in undiluted (100%) effluents from North America and Europe. Exposure and effect findings were integrated in a toxic unit (TU)-based model that considers the measured distribution of individual AE homologs in effluent with their corresponding SSDs. Use of environmentally relevant exposure corrections (bioavailability and accounting for AE-derived alcohol) resulted in TUs ranging from 0.015 to 0.212. Low levels of risk are concluded for AE in the aquatic environments of Europe and North America.

Keywords: Alcohol ethoxylate; Nonionic surfactant; Species-sensitivity distribution; Environmental fate; Environmental mixtures; Toxic units

1. Introduction

Alcohol ethoxylates (AE) are nonionic surfactants and are common components of detergent formulations, with approximately 1.1 million metric tons produced annually worldwide (Hauthal, 2004). AE molecules have the general formula $CH_3(CH_2)_n(OCH_2\ CH_2)_yOH$, where n is generally 11–15, 17 and y is 0–18 and are formed by the reaction of fatty alcohol and ethylene oxide. Interest in the environmental risk of AE exposure is great due to their high production volume and global usage. AE have been the subject of several environmental risk assessments including those of A.D. Little (1977), Goyer et al. (1981), Talmadge (1994) and van de

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*Corresponding author. Fax: +1 513 627 1208.
E-mail address: belanger.se@pg.com (S.E. Belanger).

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Plassche et al. (1999). These assessments have become increasingly sophisticated with numerous advances in understanding analytical methods, exposure, fate, and effects. In the most recent assessment, conducted in the Netherlands, van de Plassche et al. (1999) concluded that there was no need for concern for AE, alcohol ethoxy sulfates (AES), and linear alkylbenzene sulfonate (LAS) under current use patterns. The present AE environmental risk assessment also occurs in the presence of intense regulatory interest surrounding other nonionic surfactants such as the alkyl phenol ethoxylates (APE) (Environment Canada, Health Canada, 2000; Servos, 1999; US Environmental Protection Agency, 2003). A difference between the surfactants evaluated in the Dutch risk assessment (AES, AE, LAS) and APE is the absence of endocrine modulating activity for AES, AE and LAS (Knepper et al., 2003; Routledge and Sumpter, 1996).

Monitoring of municipal wastewater treatment plant (WWTP) effluents containing AE by the newly advanced pyridinium derivatization method has been conducted in Europe and North America (Dunphy et al., 2001; Eadsforth et al., 2006; Morrall et al., 2006). Concentrations of total AE (including all sources of fatty alcohol) ranged from 1.6 to 16.8, 1.0 to 22.7, and 1.3 to 15.6 μg/L in Europe, Canada, and the United States, respectively. Total mass (μg/L), however, is an incomplete view of the relevant exposure as the environmental fingerprint of the homolog distribution is of fundamental importance in risk assessment. That is, rates of biodegradation, sorption, and formation of alcohols are different across the range of alkyl and ethoxylate chains. Further perspective on exposure in effluents was gained by determination of removal and metabolite formation by Wind et al. (2006) in a benchtop Continuous Activated Sludge (CAS) test and by Federle and Itrich (2006) in batch radiotracer studies. These data support an interpretation of extremely rapid biodegradation. Production of metabolites of toxicological interest includes the presence of alcohol. Fatty alcohol is known to occur in wastewater from natural microbial and metabolic processes (Leeming et al., 1994) including degradation of alcohol-based surfactants (Wind et al., 2006) and used directly in consumer products (Modler et al., 2002). Wind et al. (2006) demonstrated that biodegradation of AE is likely to be only a partial contributor to effluent fatty alcohol. Sorption of AE ethoxymers to organic carbon and wastewater solids is an additional mechanism of removal and follows a predictable pattern as a function of hydrophobicity (Van Compernolle et al., 2006).

The ecotoxicological literature has markedly increased since van de Plassche et al. (1999), who summarized effect conclusions from the Dutch surfactant risk assessment of the 1990s (AISE-CESIO, 1996). A series of mesocosm studies (Belanger et al., 2000; Dorn et al., 1996, 1997a, b; Wong et al., 2004) with numerous associated aquatic toxicity investigations were performed. Chronic QSARs for daphnids have been generated (Morrall et al., 2003) and refined further (Boeije et al., 2006). Algal and fish chronic QSARs have also been generated (Boeije et al., 2006; Wind and Belanger, 2006). Improvements in the use and interpretation of species-sensitivity distributions (SSDs) and extrapolations to ecosystems have also taken place (Posthuma et al., 2002; Versteeg et al., 1999). The wealth of new data clearly supports a major understanding of the ecological effects of AE and allows greater in-depth assessment and drawing of conclusions from this greatly expanded database.

The environmental behavior of AE is believed to be controlled by the nonionic surfactant's hydrophobic nature. A common feature of new chronic quantitative structure–activity relationships (QSARs, Boeije et al., 2006) and more recently developed fate insight into the behavior of AE in the environment is the relationship between structural estimators of hydrophobicity and environmental properties. Hydrophobicity is clearly related to the ecotoxicity of AE (Boeije et al., 2006). Octanol:water partition coefficients (K_{ow}) are a common means of estimating hydrophobicity but are extremely difficult to determine accurately for surfactants (Morrall et al., 1999). However, they can be successfully estimated by mathematical means demonstrated by Roberts (1991) and Roberts and Marshall (1995) by reapplication of the methods of Leo and Hansch (1979). Boeije et al. (2006) applied homolog-specific estimations of K_{ow} to correlate with the toxicity of AE mixtures. In addition, van Compernolle et al. (2006) developed a model built from radiolabel sorption studies of pure AE homologs (69 determinations for 34 different AEs), where chain length and ethoxylation were known, that can be reapplied to estimate changes in bioavailability for environmental fingerprints of AE mixtures. Sorption can play a potentially significant role in resulting environmental effects of AE as with other common hydrophobic contaminants (DiToro et al., 2000).

Detection of AE in the aquatic environment utilizes a highly specific and sensitive method whereby homologs are derivatized with 2-fluoro-*N*-methyl pyridinium *p*-toluene sufonate, which imparts a permanent positive charge to the molecule (Dunphy et al. 2001). In the process, all aliphatic alcohols are also derivatized and detected. This phenomenon creates a situation where all aliphatic alcohols, including those that naturally occur in the environment, are considered "AE." To understand how to evaluate this meshing of natural and synthetic aliphatic alcohol, a study was conducted to quantitatively determine the amount of alcohol that is produced either by biodegradation of AE through central ether cleavage or as part of the unethoxylated components present in AE technical mixtures (Wind et al., 2006). Benchtop CAS units were used as models of sewage treatment and fed influent that was either AE-free or amended at a level of AE found in typical sewage influent. Analysis of effluents then provided an estimate of fatty alcohol attributable to AE biodegradation. Wind et al. (2006) coined the termed "alcohol cap" as the amount of alcohol to be included in the AE risk assessment.

The alcohol cap is mathematically defined as the molar ratio (for each chain length) between the effluent alcohol that could have been derived from AE and the total

ethoxylated alcohols in the effluent (EO 1–18). The values derived from Wind et al. (2006) were 1.1361, 2.5943, 0.38199, 0.3969, 0.6273, and 0.11474 for C12, C13, C14, C15, C16, and C18, respectively.

The generation of new fate, exposure, and effects data have resulted in an opportunity to reassess the environmental risk assessment of AE. This paper presents a synthesis of the new findings and formulates a global (universally applicable) effects assessment integrated with site-specific, regional, and global exposure assessments for this important nonionic surfactant. The effects assessment will combine the use of new trophic-level-specific QSARs (Boeije et al., 2006) with knowledge of the homolog-specific distributions measured in European and North American WWTP effluents (Eadsforth et al., 2006; Morrall et al., 2006). These evaluations include an investigation of the two primary modifiers of exposure relevant to the environmental fingerprint: (a) the role and source of alcohol measured by the AE specific analytical method as reflected by the Wind et al. (2006) alcohol cap, and, (b) the potential role of sorption (affecting bioavailability) on predicted exposure and effects.

2. Materials and methods

2.1. AE nomenclature

An AE molecule consists of a fatty alcohol, which is ester-linked to a polyethylene glycol (or ethoxylate) chain. The general formula for AE is CH_3–$(CH_2)_x$–O–$(CH_2CH_2O)_y$–H. For typical commercial AE materials, x can range from 8 to 17 and y can range from 0 to >20. In this paper, AE are named in two ways: (a) for pure compounds the alkyl chain length is indicted by a single value as is the ethoxylate chain length (for example, C13E4 indicates an alkyl chain length of 13 with 4 ethoxylates), and (b) for technical mixtures the range of alkyl chain lengths is indicated with an average ethoxylation per mole of hydrophobe (e.g., C12–15E9 would indicate that the range of alkyl chains is 12–15 with an average number of ethoxylates of 9 per alkyl chain). This latter form of naming compounds is a means to describe the structure in shorthand, but incompletely describes the distribution involved, which can only be fully described by presenting a full matrix of individual homologs.

2.2. AE exposure

Treated wastewater effluents were collected from 29 sites and quantified as to the abundance and mass of AE homologs (the environmental fingerprints) that were present (Eadsforth et al., 2006; Morrall et al., 2006). For the purpose of environmental risk assessment these data can be used to evaluate AE on both a site-specific and a regional or national basis. In addition, several modifiers to exposure have been identified and can be incorporated, such as appropriate partitioning of the source of alcohol (the so-called alcohol cap; Wind et al., 2006) and sorption to organic carbon and suspended solids (van Compernolle et al., 2006) on an ethoxymer-specific basis. For this assessment, sorption based on predicted K_d (sorption coefficient between AE and suspended solids) per ethoxymer was utilized as described in van Compernolle et al. (2006), Table 7:

$$\log K_d = 0.331 \times (\text{alkyl chain length}) - 0.009 \times (\text{ethoxylate chain length}) - 1.126. \quad (1)$$

K_d was chosen (versus K_{oc}, or the sorption coefficient corrected for organic carbon content) because the regression was more reliable and the underlying data was dominated by measured K_d values. Because the studied effluents enter rivers of varying size, hydrology, flow, and ultimately dilution, the site-specific dilution of effluents will be ignored in these analyses in order to draw broad-based comparisons. Expressions of exposure will be as measured effluent concentrations (MEC) in this manuscript for each monitored site.

In addition to site-specific assessments, regional conditions were evaluated. Averages per ethoxymer were calculated for each region using the site-specific information and one-half the analytical method limit of quantitation (LOQ) when applicable (Eadsforth et al., 2006; Morrall et al., 2006). Regional differences are related to types of treatment processes monitored as well as the actual commercial materials used in each region. In Europe, activated sludge treatment only was monitored. In Canada, primarily activated sludge was assessed and two other treatment processes were included: trickling filter and rotating biological contactor. However, the relative effluent flow for each treatment type in Canada is not known; therefore simple averages were calculated, ignoring treatment. In the United Sates, the distribution of treatment is known (Rapaport, 1988; US Environmental Protection Agency, 1996) and the average ethoxymer distribution can be calculated weighted by the amount of wastewater by treatment type (Morrall et al., 2006).

2.3. Ecotoxicological data

Chronic toxicity data for AE were compiled from published literature and numerous industry reports. Industry data focused on standard test species (e.g., the green algae *Pseudokirchneriella subcapitata and Desmodesmus subspicatus*; the invertebrates *Daphnia magna* and *Ceriodaphnia dubia*; and fish, primarily fathead minnow and bluegill sunfish) and were universally conducted according to accepted international guidelines.

All raw data from ecotoxicity tests were compiled. Over time, a variety of statistical techniques were used by experimenters. Chronic EC_{10}s (the effective concentration causing a 10% reduction in a biological response) were determined for all studies by the nonlinear estimation method of Bruce and Versteeg (1992). Belanger and Dorn (2004) showed that AE EC_{10}s from this data set were in excellent agreement with NOEC trends. A total of 60 chronic toxicity studies of 25 unique distributions or pure materials were identified (Table 1). Distributions and tests were included if specific knowledge of the mixture composition was provided. Only 7 of the 60 studies were not accompanied by analytically confirmed exposure and all of these were algal studies. In 14 of 60 cases the EC_{10} could not be determined, primarily due to lack of convergence of the iterative regression, and the original NOECs were retained in the analysis.

2.4. Normalization based on the toxic unit concept

Because the commercial materials and pure AEs used in the ecotoxicological laboratory and mesocosm studies had homolog distributions that differed from environmental fingerprints measured in the effluent samples, a mechanism was needed to apply information on the diverse tested structures to the environmental fingerprints. Such a normalization process was previously utilized by van de Plassche et al. (1999) and has been reapplied here with several important modifications.

Normalization was used to reinterpret EC_{10}s for the tested AE mixtures to represent EC_{10}s of environmental fingerprints using a toxic unit (TU) concept (Fig. 1). Boeije et al. (2006) developed chronic QSARs for *Daphnia magna* (representing invertebrates) and fathead minnows (representing fish) and Wind and Belanger (2006) for *Selenastrum capricornutum* (= *Pseudokirchneriella subcapitata*) (representing algae). These were used to normalize toxicity test results within an appropriate taxonomic group (Fig. 1, Steps 1–3). A common feature of these QSARs is the use of $\log K_{ow}$ as a functional parameter that estimates hydrophobicity of an AE mixture. Contribution of each homolog to the overall hydrophobicity was estimated from the molar contribution of each ethoxymer in the mixture being considered (note that this could be a tested mixture or an environmental fingerprint). In van de Plassche et al. (1999) the $\log K_{ow}$ was estimated using a computed average structure for the tested and environmental distribution based on the mass (not mol)

Table 1
Chronic toxicity of alcohol ethoxylate (AE) to aquatic species

Species name	Common name	Taxonomic group for QSAR	Life stage	Compound	Most sensitive endpoint	Effect statistic	Effect conc (mg/L)	Citation
Chlorella vulgaris	Green algae	Algae	Vegetative	C12-15EO3	Growth rate	EC10	2.179	Turner (1988)*
Lemna minor	Duckweed	Algae	2-frond stage	C14-15EO7	Frond count	EC10	0.101	Bishop et al. (1980)*, Bishop and Perry (1981)
Microcystis aeruginosa	Blue-green algae	Algae	Vegetative	C14-15EO7	Cell density	EC10	0.154	Maziarz (1983a)*, Lewis and Hamm (1986)
Navicula pelliculosa	Diatom	Algae	Vegetative	C14-15EO7	Cell density	EC10	0.140	Maziarz (1983b)*, Lewis and Hamm (1986)
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C10EO8	Growth rate	EC10	8.087	Rieche (1997)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C12E2	Growth rate	EC10	0.030	Kirch (1997a)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C12E4	Growth rate	EC10	0.453	Werner (1997)a*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C12E8	Growth rate	EC10	0.325	Werner (1997b)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C16E2	Growth rate	EC10	0.042	Geisel (1998)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C16E8	Growth rate	EC10	0.096	Kirch (1997b)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C12-13E3	Growth rate	EC10	0.998	Neven (1993a)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C16-18E7	Growth rate	EC10	5.831	Neven (1993b)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C12-13E3	Growth rate	EC10	0.204	Hantsveit and Oldersma (1993)*
Scenedesmus subspicatus	Green algae	Algae	Vegetative	C12-14E7	Growth rate	EC10	0.137	Neven (1993c)*
Selenastrum capricornutum	Green algae	Algae	Vegetative	C8-10E5	Growth rate	EC10	9.791	Neven (1993d)*
Selenastrum capricornutum	Green algae	Algae	Vegetative	C14-15EO7	Cell density	EC10	0.092	Maziarz (1983c)*, Lewis and Hamm (1986)
Selenastrum capricornutum	Green algae	Algae	Vegetative	C12-14E9	Cell density	EC10	0.151	Yamane et al. (1984)
Brachionus calyciflorus	Rotifer	Invertebrate	Neonate	C10EO6	Population size	EC10	2.015	Versteeg et al. (1997), Morrall et al. (1999)
Brachionus calyciflorus	Rotifer	Invertebrate	Neonate	C12EO6	Population size	EC10	0.562	Versteeg et al. (1997), Morrall et al. (1999)
Brachionus calyciflorus	Rotifer	Invertebrate	Neonate	C14EO4	Population size	EC10	0.207	Versteeg et al. (1997), Morrall et al. (1999)
Brachionus calyciflorus	Rotifer	Invertebrate	Neonate	C14EO6	Population size	EC10	0.112	Versteeg et al. (1997), Morrall et al. (1999)
Brachionus calyciflorus	Rotifer	Invertebrate	Neonate	C14EO8	Population size	EC10	0.169	Versteeg et al. (1997), Morrall et al. (1999)
Brachionus calyciflorus	Rotifer	Invertebrate	Neonate	C12E3	Population size	EC10	0.733	Versteeg and Shorter (2000)*
Ceriodaphnia dubia	Water flea	Invertebrate	Neonate	C14-15EO7	Reproduction	EC10	0.328	Taylor (1984a, b)*
Ceriodaphnia dubia	Water flea	Invertebrate	Neonate	C14-15EO7	Reproduction	EC10	0.127	Taylor (1984a, b)*
Ceriodaphnia dubia	Water flea	Invertebrate	Neonate	C14-15EO7	Reproduction	EC10	0.464	Lewis (1989)*, Masters et al. (1991)
Ceriodaphnia dubia	Water flea	Invertebrate	Neonate	C14-15EO7	Reproduction	EC10	0.236	Lewis (1989)*, Masters et al. (1991)
Chironomus tentans	Insect	Invertebrate	Larvae	C9-11EO6	Survival	EC10	3.635	Dorn et al. (1997a)
Corbicula fluminea	Bivalve	Invertebrate	Juvenile	C12-15EO6	Length gain	EC10	0.062	Belanger et al. (1998)*, Belanger et al. (2000)
Daphnia magna	Water flea	Invertebrate	Neonate	C14-15EO7	Reproduction	EC10	0.140	Mank and Krueger (1998a)*, Morrall et al. (2003)
Daphnia magna	Water flea	Invertebrate	Neonate	C13-15EO5	Reproduction	EC10	0.082	Mank et al. (1999)*, Morrall et al. (2003)

Daphnia magna	Water flea	Invertebrate	Neonate	C12-15EO6	Reproduction	EC10	0.368	Mank and Krueger (1998b)*, Morrall et al. (2003)
Daphnia magna	Water flea	Invertebrate	Neonate	C12-13EO6.5	Reproduction	EC10	0.803	Gillespie et al. (1999)
Daphnia magna	Water flea	Invertebrate	Neonate	C14-15EO7	Reproduction	NOEC	0.790	Gillespie et al. (1999)
Daphnia magna	Water flea	Invertebrate	Neonate	C9-11EO6	Reproduction	EC10	2.579	Gillespie et al. (1999)
Daphnia magna	Water flea	Invertebrate	Neonate	C12-15E9	Reproduction	EC10	0.167	Kroese (1994)*
Daphnia magna	Water flea	Invertebrate	Neonate	C12-13EO6.5	Reproduction	EC10	0.355	Maki (1977)*, 1979
Daphnia magna	Water flea	Invertebrate	Neonate	C14-15EO7	Survival	EC10	0.255	Maki (1977)*, 1979
Elimia	Gastropod	Invertebrate	Junvenile	C12-15EO6	Weight gain	NOEC	0.259	Belanger et al. (1998)*, Belanger et al. (2000)
Hyallela azteca	Amphipod	Invertebrate	Larval	C9-11EO6	Survival	EC10	3.882	Dorn et al. (1997b)
Dugesia gonocephala	Flatworm	Invertebrate	Immature	C14E10	Survival	EC10	0.840	Patzner and Adams (1979)
Lepomis macrochirus	Bluegill	Fish	Juvenile	C9-11EO6	Survival	EC10	8.983	Dorn et al. (1997b)
Lepomis macrochirus	Bluegill	Fish	Juvenile	C12-13EO6.5	Reproduction	NOEC	0.880	Harrelson et al. (1997)
Lepomis macrochirus	Bluegill	Fish	Juvenile	C14-15EO7	Survival	NOEC	0.160	Kline et al. (1996)
Oncorhynchus mykiss	Rainbow trout	Fish	Egg-alevin	C12-15EO9	Dry weight	EC10	0.079	Wong et al. (2004)
Pimephales promelas	Fathead minnow	Fish	Egg-juvenile	C9-11EO6	Survival	NOEC	4.350	Harrelson et al. (1997)
Pimephales promelas	Fathead minnow	Fish	Egg-juvenile	C9-11EO6	Survival	NOEC	1.000	Harrelson et al. (1997)
Pimephales promelas	Fathead minnow	Fish	Egg-juvenile	C9-11EO6	Reproduction	NOEC	0.730	Harrelson et al. (1997)
Pimephales promelas	Fathead minnow	Fish	Egg-juvenile	C9-11EO6	Length	NOEC	1.010	Lizotte et al. (1999)
Pimephales promelas	Fathead minnow	Fish	Egg-juvenile	C12-13EO6.5	Survival	EC10	0.213	Holman (1975)*, Maki (1979)
Pimephales promelas	Fathead minnow	Fish	Egg-juvenile	C14-15EO7	Survival	EC10	0.121	Holman (1975)*, Maki (1979)
Pimephales promelas	Fathead minnow	Fish	Larva-juvenile	C12-13EO6.5	Survival	EC10	1.748	Lizotte et al. (1999)
Pimephales promelas	Fathead minnow	Fish	Larva-juvenile	C12-13EO6.5	Survival	NOEC	0.880	Dorn et al. (1997b)
Pimephales promelas	Fathead minnow	Fish	Larva-juvenile	C12-13EO6.5	Survival	NOEC	0.880	Dorn et al. (1997b)
Pimephales promelas	Fathead minnow	Fish	Larva-juvenile	C14-15EO7	Survival	EC10	1.441	Lizotte et al. (1999)
Pimephales promelas	Fathead minnow	Fish	Larva-juvenile	C14-15EO7	Survival	NOEC	0.160	Kline et al. (1996), Dorn et al. (1996)
Pimephales promelas	Fathead minnow	Fish	Larval	C14-15EO7	Survival	NOEC	0.160	Kline et al. (1996), Dorn et al. (1996)
Pimephales promelas	Fathead minnow	Fish	Adult	C14-15EO7	Survival	NOEC	0.160	Kline et al. (1996), Dorn et al. (1996)
Pimephales promelas	Fathead minnow	Fish	Larval	C14-15EO7	Survival	NOEC	0.280	Kline et al. (1996), Dorn et al. (1996)

Note: Unpublished internal industry reports are indicated with an asterisk.

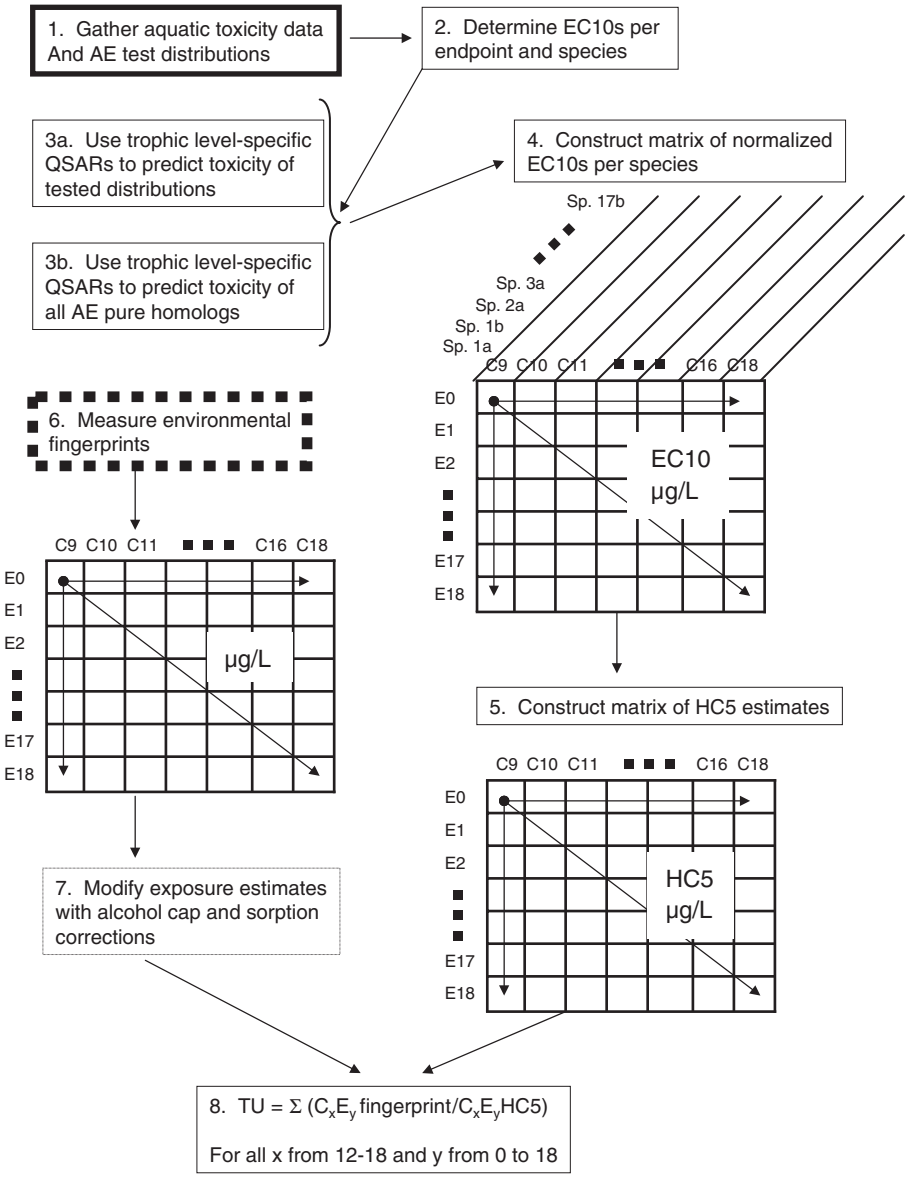

Fig. 1. Information flow for the determination of toxic-unit-based assessment of alcohol ethoxylates. Bold boxes indicate the initial stages for fate or effects. Solid boxes are effects-oriented portions of the assessment and dotted-line boxes are environmental-exposure-oriented portions of the assessment.

contribution of each ethoxymer in the distribution. As such, the shape of the AE distribution was not fully considered and a less accurate estimation of the toxicity of a mixture resulted (Boeije et al., 2006). The chronic QSARs (all in mol/L) used were as follows:

$$\log(21\,\text{d EC}_{20}\ \textit{Daphnia magna},\ \text{reproduction}) = -0.532 \times \log K_{ow} - 3.025, \quad (2)$$

$$\log(30\,\text{d EC}_{20}\ \textit{Pimephales promelas}) = -0.307 \times \log K_{ow} - 3.92, \quad (3)$$

$$\log(72\,\text{h E}_b\text{C}_{20}\ \text{algae}) = -0.378 \times \log K_{ow} - 4.073. \quad (4)$$

EC_{20} QSARs were developed for this purpose because these will be more robust than EC_{10}s. Confidence limits surrounding the EC_{20} are by definition narrower, indicating that the estimation is more precise when the same data structure is considered. Of course, the EC_{50} would have the smallest confidence intervals, but toxicologists are more concerned with more subtle effects (Stephan and Rogers, 1985) and this is also the case here. Normalization to an environmental fingerprint or distribution utilizes the equation

$$\text{normalized EC}_{env} = \text{reported EC}_{test} \times \left(\frac{\text{predicted EC}_{env}}{\text{predicted EC}_{test}}\right), \quad (5)$$

where normalized EC_{env} is normalized effect concentration for environmental distribution; reported EC_{test} is effect concentration from a toxicity test on a commercial distribution; predicted EC_{env} is effect concentration from a QSAR based on environmental distribution; predicted EC_{test} is effect concentration from a QSAR based on a commercial distribution.

For the purpose of normalization, the choice of EC_{10}, EC_{20} or EC_{50} is not extremely critical when the ratio of predictions above is being considered. For example, van de Plassche et al. (1999) normalized chronic toxicity data using the ratio of EC_{50}s from acute toxicity data sets. This assumes that the relative relationship of the endpoints being normalized using a chosen EC_x is robust across the value that was reported for a test (the reported EC_{test} above) and the x used in the predicted EC ratio. The QSARs were developed with this idea in mind and it is a reasonable assumption when comparing chronic EC_{10}s versus EC_{20}s. The normalization reported in the present research represents an improvement on van

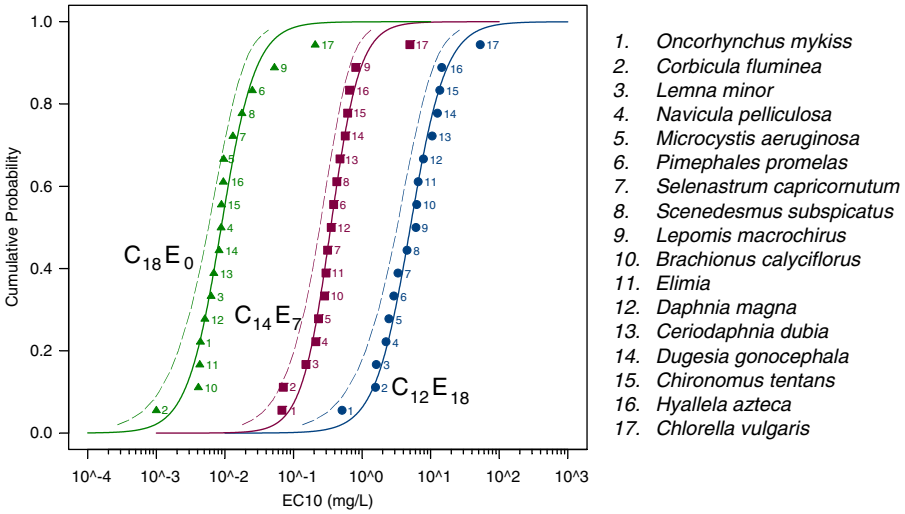

Fig. 2. Comparison of species-sensitivity distributions (SSDs) for several alcohol ethoxylates. The relative spread of ethoxymer-specific SSDs and order of normalized chronic EC10 values for represented species are indicated.

de Plassche et al. (1999) in several aspects: use of chronic time frames and QSARs, expression of effects on a homolog-specific molar basis, and use of a consistent statistical framework. Reevaluation of the AE chronic toxicity data included determinations of EC_{10}, EC_{20}, and EC_{50}. The ratio EC_{10}/EC_{20} for all tests and endpoints combined was 0.8 ± 0.3 indicating that the ratio is fairly consistent.

AE is a mixture of homologous chemicals and a means to clearly integrate environmental exposure determinations with ecotoxic effects was needed. The most meaningful way to accomplish this was to consider each homolog individually. AE acts as a nonpolar narcotic and is expected to behave according to a concentration addition (mixture toxicity) model (see Boeije et al., 2006, for a discussion). This approach allows the simple summation of predicted effects per homolog with the homolog's exposure concentration (Fig. 2). Because a large array of single-species data is available, an effects model based on the SSD approach (van de Plassche et al., 1999; Posthuma et al., 2002) was used to appropriately capture the array and variation of species sensitivity across all homologs. To do so, normalizations of each chronic toxicity result based on empirically determined EC_{10}s from tested distributions were made to each potential homolog in the environmental fingerprint (from $C_{12}EO_0$ to $C_{18}EO_{18}$) by Eq. (4) (Fig. 1, Steps 3–4). The geometric mean response per taxon was then calculated and a SSD analysis for each ethoxymer was conducted (Fig. 1, Step 5; Fig. 2). The hazardous concentration (HC) of the SSD predicted to be protective of 95% of species (the HC_5) was estimated by the method of Aldenberg and Jaworska (2000) and Van Vlaardingen et al. (2003). Programs to integrate QSARs and the HC_5 predictions per homolog were constructed in SAS (2000).

The matrix of HC_5s (all possible chain lengths and ethoxylates) was constructed from SSDs for direct comparison to the matrix of environmentally determined concentrations of AE homologs (Fig. 1, Steps 5 and 6; Fig. 2). The HC_5 matrix was applied using a TU approach (ECETOC (European Centre for Ecotoxicology and Toxicology of Chemicals), 2001) where the fractional contribution of each HC_5 is based on the measured environmental fingerprint (Fig. 1, Steps 7 and 8). In this application of TUs the ratio of the MEC (defined earlier as the measured effluent concentration) to the HC_5 based on EC_{10}s (equated to the PNEC or predicted no-effect concentration used in risk assessment) per ethoxymer was determined and summed:

$$\sum \mathrm{TU} = \left(\frac{\mathrm{MEC\ C_{12}E_0}}{\mathrm{HC_5C_{12}E_0}}\right) + \left(\frac{\mathrm{MEC\ C_{12}E_1}}{\mathrm{HC_5C_{12}E_1}}\right) + \left(\frac{\mathrm{MEC\ C_{12}E_2}}{\mathrm{HC_5C_{12}E_2}}\right) + \cdots + \left(\frac{\mathrm{MEC\ C_{18}E_{18}}}{\mathrm{HC_5C_{18}E_{18}}}\right). \quad (6)$$

Normalizations were conducted using different environmental exposure scenarios of environmental fingerprints to account for alcohol source and sorption (Fig. 1, Steps 7 and 8). Four scenarios were considered: (1) assessing all monitored alcohol and no bioavailability correction (sorption), (2) imposing an alcohol cap and no bioavailability correction, (3) including all alcohol and a bioavailability correction, and (4) imposing the alcohol cap and a bioavailability correction. Normalizations were developed on a site-specific and regional average basis.

Site-specific and regional average AE distributions were compared based on TU predictions as a characterization of risk from undiluted wastewater effluent. For sites with TUs greater than 1, some risk would be predicted and if less than 1, risk would not be expected.

2.5. Mesocosm-based predictions

Five mesocosm studies of similar experimental design and scope have been conducted with AE. A refined QSAR to predict the mesocosm NOEC (in mol/L) was developed by Boeije et al. (2006) as based on the underlying data reported by Belanger et al. (2000):

$$\log(\text{mesocosm NOEC}) = -0.74 \times K_{ow} - 2.78. \quad (7)$$

In this QSAR, the NOEC is used as opposed to an EC_{10} because the mesocosm NOEC results summarize responses of dozens to hundreds of taxa from each study, in addition to community function and structural measures (Belanger et al., 2000; Dorn et al., 1996, 1997a, b; Wong et al., 2004). Because no singular endpoint drives all the studies, best professional judgment is used to derive conclusions (Giddings et al., 2002). Each study contained sensitive taxa representative of low to unimpacted stream ecosystems. The mesocosm QSAR was used to predict effects of AE environmental fingerprints using $\log K_{ow}$ for the tested mixture and as a comparative check against the SSD-based analyses. As with the SSD-based TU approach, the mesocosm-based assessment used a TU approach only with the predicted mesocosm NOEC as the estimated PNEC. Exposure to the effluent environmental fingerprint was sequentially modified using the alcohol cap and corrections for bioavailability due to sorption as in the SSD approach.

2.6. Additional statistical analyses

SSDs with resulting HC_5s statistics were computed using the underlying assumption of a log-normal distribution (Aldenberg and Jaworska, 2000; Posthuma et al., 2002) and executed in SAS (2000). To evaluate the

influence of treatment type on HC_5-based TU predictions, paired *t*-tests using sites as the common feature with Bonferroni adjustments were used for the various exposure scenarios. To evaluate the influence of exposure scenarios on the overall HC_5-based TU predictions (all regions combined) two-way analysis of variance with interaction was performed. Comparisons of HC_5- and mesocosm-based TU predictions were conducted using linear regression with the various iterations of factors used to correct for exposure (alcohol cap and bioavailability due to sorption). In all statistical analyses significance was inferred with $\alpha = 0.05$ and performed using SAS (1999).

3. Results

Total AE concentrations averaged 3.554–6.799 μg/L depending on the region, with approximately 41–45% of the mass being fatty alcohol (Table 2). Imposing the alcohol cap reduced total AE to 2.367–4.782 μg/L or by 29% (Canada) to 33% (US). Bioavailability correction alone was somewhat less influential in reducing exposure concentrations in effluent with reductions of 18%, 24%, and 35% for Canada, Europe, and the United States on average, respectively. When the bioavailability correction was imposed in addition to the cap, total AE exposure was reduced on average 38% (Canada), 44% (Europe), and 53% (United States) compared to the fully monitored distribution. Note that these compiled totals do not reflect the distributions of homologs and as such are incomplete descriptors of exposure. Average AE distributions, which are often computed for commercial distributions (always on a wt% basis, not a mol% basis) is a convenient shorthand for comparison, but the average distribution notation is an inappropriate means to describe ecotoxic potential and exposure because the toxicity profile is nonlinear (note the use of $\log K_{ow}$ in all QSARs). Canada had the shortest average alkyl and longest ethoxylate chain lengths of the three regions. WWTP effluent concentrations (MEC) were determined for each site and with the various exposure modifications, in addition to determining regional averages (Table 3). Total MEC ranged from 0.0010 to 0.0227 mg/L when the entire environmental fingerprint was considered. Imposing the alcohol cap and correcting for sorption reduced the total MEC to 0.0007–0.0150 mg/L.

Predictions of SSDs of pure homologs were generated by the normalization process (Fig. 1, Table 4). HC_5 predictions ranged from 2.8656 mg/L (C_9EO_{18}, least toxic homologs) to 0.0017 mg/L ($C_{18}EO_0$, most toxic homologs) (Fig. 2). Depending on which pure AE homolog is being considered, the rank order of taxa sensitivity is altered. The reason for this observation is that the trophic-level-specific QSARs used in the normalization have slightly different slopes and intercepts. Fish, invertebrates, and algae are well interspersed within the sensitivity distribution. An analysis of variance with taxonomic group as the dependent and normalized EC_{10} values as the independent variable for the representative distributions in Fig. 2 ($C_{12}EO_{18}$, $C_{14}EO_7$, and $C_{18}EO_0$) indicate that no trophic group is uniquely sensitive or tolerant (*F*-statistics range from 1.58 to 1.97: *P*-values range from 0.14 to 0.22). The matrix of predicted HC_5 values using chronic ecotoxicity EC_{10} data as input provides the basis for the effects characterization in the subsequent TU-based risk characterization.

The risk characterization of environmental fingerprints on a site-specific and regional basis using the HC_5-based TU approach indicated that 6 of 29 sites had $TU > 1$ and regional averages were approximately 0.4–0.6 when the fully monitored environmental fingerprint (all alcohol and no sorption) was considered (Table 5). Recall that the MEC represents undiluted effluent in these calculations. Correction of exposures: (1) using the alcohol cap without bioavailability correction, (2) including all alcohol and including bioavailability correction, and (3) imposing both the alcohol cap and bioavailability correction reduce the number of sites ($n = 29$) exceeding 1 TU to 0, 3, and 0, respectively. In general the alcohol cap was more influential than sorption as an exposure correction scenario, although not universally so. This phenomenon can be traced to the individuality of AE environmental fingerprints and to which homologs are most influenced by each exposure

Table 2
Regional comparison of AE in effluents

Region	Total AE (μg/L) All alcohol included	Total AE (μg/L) Alcohol cap, no sorption	Total AE (μg/L) All alcohol with sorption	Total AE (μg/L) Alcohol cap and sorption	Average distribution (weight basis)
Canada					
All AE	6.799	4.782	5.571	4.199	CL: 13.6
Alcohol	2.811	0.794	2.091	0.719	EO: 8.4
United States					
All AE	3.554	2.367	2.269	1.608	CL: 14.7
Alcohol	1.459	0.273	0.884	0.222	EO: 9.1
Europe					
All AE	4.976	3.365	3.757	2.767	CL: 14.0
Alcohol	2.170	0.559	1.474	0.485	EO: 6.2

Note: These summaries do not fully incorporate detailed analyses of ethoxymer distributions but are intended to allow a broad comparison of effluents containing AE.

Table 3
Calculated WWTP effluent concentrations to estimate exposure with and without the use of the alcohol cap and bioavailability (sorption) corrections

Alcohol option Sorption option			Include all None	Capped None	Include all Include	Capped Include
Geography	Site	Treatment type	Effluent (mg/L)	Effluent (mg/L)	Effluent (mg/L)	Effluent (mg/L)
Canada	Vernon	TF	0.0131	0.0114	0.0113	0.0102
	Kelowna	AS	0.0027	0.0022	0.0022	0.0018
	Toronto	AS	0.0100	0.0084	0.0083	0.0073
	La Prairie	AS	0.0010	0.0008	0.0008	0.0007
	Victoriaville	AS	0.0012	0.0011	0.0010	0.0009
	Paris	AS	0.0014	0.0013	0.0011	0.0010
	Cardston	RBC	0.0227	0.0108	0.0182	0.0096
	Waterloo	AS	0.0022	0.0015	0.0018	0.0013
US	San Benito	L	0.0103	0.0064	0.0061	0.0036
	Rockaway	OD	0.0054	0.0043	0.0045	0.0038
	St. Clairsville	RBC	0.0041	0.0041	0.0034	0.0033
	Oskaloosa	TF	0.0122	0.0095	0.0095	0.0080
	Sedalia	TF	0.0208	0.0090	0.0148	0.0074
	Rosehill	L	0.0095	0.0068	0.0053	0.0040
	Lodi	AS	0.0017	0.0013	0.0007	0.0008
	Durham	AS	0.0023	0.0016	0.0013	0.0009
	Opelika	OD	0.0015	0.0009	0.0011	0.0008
Europe	Northwich (UK)	AS	0.0057	0.0041	0.0044	0.0034
	Cannock (UK)	AS	0.0016	0.0010	0.0012	0.0009
	Rushmoor (UK)	AS	0.0029	0.0020	0.0022	0.0017
	Kralingse Veer (NL)	AS	0.0054	0.0047	0.0045	0.0041
	De Meern (NL)	AS	0.0082	0.0077	0.0069	0.0066
	Horstermeer (NL)	AS	0.0058	0.0037	0.0041	0.0031
	Estepona (ES)	AS	0.0044	0.0019	0.0028	0.0150
	La Vibora (ES)	AS	0.0168	0.0067	0.0119	0.0051
	Munich (G)	AS	0.0036	0.0036	0.0030	0.0030
	Torino (IT)	AS	0.0018	0.0013	0.0015	0.0012
	Robecco (IT)	AS	0.0024	0.0018	0.0017	0.0014
	Ratingen (G)	AS	0.0011	0.0009	0.0008	0.0007
Canada	Average		0.0068	0.0048	0.0056	0.0042
US	Average		0.0036	0.0024	0.0023	0.0016
Europe	Average		0.0050	0.0034	0.0038	0.0023

scenario. When the alcohol cap and bioavailability correction are imposed the calculated TU are frequently 10–20% of those predicted from the fully monitored and uncorrected fingerprints. This observation is tied to the fact that low-ethoxylate (alcohol and EO1) and long-alkyl-chain homologs are both the most toxic and sorptive chemical species.

Two-way analysis of variance was used to ascertain the relative influence of exposure correction scenarios on resulting TUs as well as their potential interaction (data from Table 5). Imposing the alcohol cap, correcting for bioavailability through sorption, and their combined interaction were all significant (overall $F = 10.21$, $P < 0.0001$). Alcohol cap (F-statistic $= 21.22$, $P < 0.0001$), sorption (F-statistic $= 6.25$, $P = 0.0139$), and their interaction (F-statistic $= 4.49$, $P = 0.0470$) terms were all statistically significant.

Effluents of several types of wastewater treatment were investigated in this study and as Morrall et al. (2006) demonstrated, an overall pattern of treatment type on the AE fingerprint was discerned in the US monitoring study. To evaluate this further an analysis of variance using categorization of treatment type as the dependent variable and TU as the independent variable was conducted. The dominant treatment type monitored from all regions was activated sludge (AS, $n = 20$) (Table 3). AS treatment was compared to fixed biofilm treatment as represented by trickling filter and rotating biological contactor processes ($n = 5$). Comparisons of the two treatment types consistently showed that fixed biofilm treatment had higher TUs than activated sludge (Table 6). Differences were significant in all four exposure scenarios (alcohol cap imposed with and without bioavailability correction, Table 6). TU associated with activated sludge plants, all geographies combined, and with both the alcohol cap and correction for bioavailability had 0.071 ± 0.052 TUs compared to those of trickling filter and rotating biological contactor treatments employing fixed biofilm-based technologies with

Table 4
Predicted HC_5 values (mg/L) for pure homologs based on an EC_{10} determinations for 17 species

Ethoxylate chain length	Alkyl chain length								
	C9	C10	C11	C12	C13	C14	C15	C16	C18
0	0.1244	0.0881	0.0607	0.0405	0.0260	0.0161	0.0096	0.0056	0.0017
1	0.1574	0.1090	0.0736	0.0482	0.0305	0.0186	0.0110	0.0063	0.0019
2	0.2102	0.1442	0.0967	0.0630	0.0397	0.0242	0.0143	0.0081	0.0024
3	0.2706	0.1845	0.1232	0.0801	0.0505	0.0307	0.0181	0.0103	0.0031
4	0.3394	0.2305	0.1536	0.0998	0.0629	0.0384	0.0226	0.0129	0.0039
5	0.4174	0.2828	0.1882	0.1223	0.0773	0.0472	0.0279	0.0160	0.0048
6	0.5056	0.3421	0.2276	0.1481	0.0937	0.0575	0.0341	0.0196	0.0059
7	0.6050	0.4091	0.2723	0.1774	0.1126	0.0693	0.0412	0.0237	0.0072
8	0.7169	0.4846	0.3227	0.2106	0.1341	0.0828	0.0495	0.0286	0.0088
9	0.8425	0.5695	0.3795	0.2482	0.1585	0.0983	0.0590	0.0342	0.0106
10	0.9831	0.6647	0.4434	0.2906	0.1861	0.1159	0.0698	0.0407	0.0127
11	1.1404	0.7712	0.5151	0.3383	0.2173	0.1359	0.0823	0.0482	0.0151
12	1.3159	0.8903	0.5953	0.3918	0.2525	0.1585	0.0965	0.0568	0.0179
13	1.5115	1.0232	0.6850	0.4517	0.2920	0.1841	0.1126	0.0666	0.0212
14	1.7291	1.1712	0.7850	0.5187	0.3364	0.2129	0.1308	0.0778	0.0250
15	1.9709	1.3358	0.8963	0.5935	0.3860	0.2453	0.1515	0.0906	0.0294
16	2.2392	1.5186	1.0202	0.6768	0.4414	0.2816	0.1747	0.1050	0.0344
17	2.5365	1.7214	1.1577	0.7694	0.5032	0.3222	0.2009	0.1214	0.0402
18	2.8656	1.9461	1.3103	0.8723	0.5720	0.3676	0.2303	0.1399	0.0469

0.179 ± 0.068 TUs. Overall, TUs were higher for fixed biofilm treatment types versus activated sludge.

3.1. Mesocosm predictions

The TU predicted based on mesocosm NOECs were similarly influenced by the alcohol cap and bioavailability corrections as in TU assessments (Table 7). Predictions involving mesocosms were more conservative than those based on HC_5 SSDs and TUs. Fig. 3 shows the relationship for site-specific predictions when the entire fingerprint was used and when the fingerprint was modified using the alcohol cap and bioavailability correction. Both relationships are highly significant ($P<0.0001$), with r^2 values approximately 0.9 or greater. Overall, the mesocosm-based TU predictions were 5.1, 3.5, 4.3, and 2.9 times higher than the respective HC_5-based TU predictions for the series of exposure scenarios (the entire environmental fingerprint, imposing the alcohol cap without sorption, using sorption and no alcohol cap, and using the alcohol cap and correcting for bioavailability due to sorption, respectively). Sites employing fixed biofilm treatment processes were associated with the largest differences in TUs predicted from SSDs and mesocosms. Predictions involving mesocosms were universally more conservative than SSDs.

4. Discussion

A comprehensive environmental risk assessment of surfactants was conducted by the Dutch government (represented by RIVM and VROM) and the surfactant industry (represented by the Dutch Soap and Detergent Association, NVZ) in the 1990s (Feijtel et al., 1999; Matthijs et al., 1999; van de Plassche et al., 1999). AE were considered in this review, but was limited to homologs from C12 to C15, and ethoxylation of 3–20. Monitoring by best available analytical techniques (at the time, electrospray LC/MS, McAvoy et al., 2006) indicated that exposure in Dutch effluents averaged 6.2 μg/L with an average composition of $C_{13.3}E_{8.2}$. A predicted PNEC of 110 μg/L was determined using SSD analysis based on an average structure and a single QSAR (Könemann, 1981). The final risk assessment for AE was seen to be less certain at the time than that of the anionic surfactant LAS (AISE-CESIO, 1996), which had a larger fate and effect database. In the intervening years the data set for AE has been greatly expanded, giving greater certainty to the risk assessment. In addition, analytical techniques have been improved such that a greater range of alkyl chains ($C_{12–18}$) and detection of the lowest ethoxylated components ($E0_{0–3}$) was valid and possible (Dunphy et al., 2001). McAvoy et al. (2006) have since shown that historically published monitoring studies in the United States using outdated techniques could be realigned with current capabilities.

New monitoring data for the United States, Canada, and Europe have shown that the environmental fingerprint distributions for wastewater effluents have longer alkyl chains and shorter ethoxylation than was previously believed (Eadsforth et al., 2006; Morrall et al., 2006). A reinspection of toxicity predictions to be better aligned with the environmental fingerprint was therefore needed. Development of new AE-specific chronic toxicity QSARs for algae (Wind and Belanger, 2006), daphnids, and fish

Table 5
Risk characterization by the toxic unit approach for AE based on single-species sensitivity distributions (TU above 1 are in bold)

Alcohol option Sorption option			Include all None	Capped None	Include all Include	Capped Include
Geography	Site	Treatment type	TU	TU	TU	TU
Canada	Vernon	TF	0.822	0.291	0.428	0.205
	Kelowna	AS	0.152	0.055	0.082	0.036
	Toronto	AS	0.618	0.182	0.331	0.132
	La Prairie	AS	0.073	0.023	0.036	0.015
	Victoriaville	AS	0.093	0.027	0.040	0.019
	Paris	AS	0.096	0.055	0.048	0.030
	Cardston	RBC	**2.268**	0.292	**1.430**	0.220
	Waterloo	AS	0.181	0.064	0.101	0.042
US	San Benito	L	**1.425**	0.384	0.587	0.165
	Rockaway	OD	0.488	0.100	0.211	0.082
	St. Clairsville	RBC	0.119	0.106	0.061	0.057
	Oskaloosa	TF	**1.321**	0.327	0.577	0.201
	Sedalia	TF	**3.547**	0.347	**1.607**	0.212
	Rosehill	L	**1.470**	0.346	0.523	0.151
	Lodi	AS	0.249	0.078	0.095	0.037
	Durham	AS	0.399	0.092	0.140	0.039
	Opelika	OD	0.282	0.021	0.101	0.016
Europe	Northwich (UK)	AS	0.573	0.143	0.276	0.104
	Cannock (UK)	AS	0.258	0.036	0.110	0.026
	Rushmoor (UK)	AS	0.366	0.068	0.167	0.052
	Kralingse Veer (NL)	AS	0.292	0.160	0.175	0.110
	De Meern (NL)	AS	0.438	0.253	0.262	0.184
	Horstermeer (NL)	AS	0.961	0.159	0.397	0.106
	Estepona (ES)	AS	0.938	0.083	0.387	0.048
	La Vibora (ES)	AS	**2.625**	0.291	**1.290**	0.174
	Munich (G)	AS	0.246	0.246	0.123	0.123
	Torino (IT)	AS	0.142	0.045	0.091	0.031
	Robecco (IT)	AS	0.370	0.186	0.161	0.083
	Ratingen (G)	AS	0.098	0.054	0.049	0.031
Canada	Average		0.583	0.126	0.312	0.090
US	Average		0.536	0.120	0.212	0.058
Europe	Average		0.609	0.151	0.291	0.094

Table 6
Influence of treatment type (activated sludge versus fixed film processes) on AE toxic units

Treatment	TU All alcohol included	TU Alcohol cap, no sorption	TU All alcohol with sorption	TU Alcohol cap and sorption
Activated sludge ($n = 20$)	0.457 ± 0.571	0.115 ± 0.082	0.218 ± 0.276	0.071 ± 0.052
Fixed film ($n = 5$)	1.615 ± 1.333	0.272 ± 0.096	0.821 ± 0.667	0.179 ± 0.052
t-Statistic	−3.04	−3.73	−3.22	−3.9
P-value	0.0058	0.0011	0.0038	0.0007

Note: Mean ± 1 SD is indicated with significance inferred at $\alpha = 0.05$.

(Boeije et al., 2006) makes possible more comprehensive, trophic-level specific normalization that increases the validity of the AE risk assessment. The development of a mesocosm QSAR also enables the new approach to be compared to the highest tier risk assessment. The deployment of multiple QSARs and the use of SSDs (HC_5s) goes beyond their typical use in screening level risk assessments where large uncertainty factors are applied (European Union, 1995; Zeeman, 1995) and justifies not using additional uncertainty factors in this assessment. Given the breadth, quantity, and quality of monitoring, chronic toxicity and mesocosm effect data in the assessment combined with other properties of AE (anaerobically biodegradable, nonendocrine-disrupting) strongly support the use of no additional application factors applied to the present assessment. Further, the assessment does not yet

Table 7
Risk characterization by the toxic unit approach for AE based on mesocosm NOEC predictions (TU above 1 are in bold)

Alcohol option Sorption option			Include all None	Capped None	Include all Include	Capped Include
Geography	Site	Treatment type	TU	TU	TU	TU
Canada	Vernon	TF	**3.976**	0.912	1.643	0.522
	Kelowna	AS	0.696	0.179	0.293	0.092
	Toronto	AS	**2.904**	0.519	**1.267**	0.317
	La Prairie	AS	0.365	0.076	0.144	0.039
	Victoriaville	AS	0.505	0.086	0.181	0.048
	Paris	AS	0.445	0.215	0.186	0.097
	Cardston	RBC	**12.478**	0.787	**5.536**	0.465
	Waterloo	AS	0.809	0.207	0.358	0.108
US	San Benito	L	**7.786**	**1.682**	**2.811**	0.649
	Rockaway	OD	**2.714**	0.222	0.948	0.171
	St. Clairsville	RBC	0.490	0.402	0.198	0.171
	Oskaloosa	TF	**7.203**	**1.171**	**2.647**	0.555
	Sedalia	TF	**19.273**	**1.271**	**7.511**	0.610
	Rosehill	L	**8.724**	**1.497**	**2.862**	0.580
	Lodi	AS	**1.445**	0.357	0.491	0.141
	Durham	AS	**2.386**	0.410	0.780	0.155
	Opelika	OD	**1.747**	0.052	0.561	0.035
Europe	Northwich (UK)	AS	**2.937**	0.410	**1.151**	0.251
	Cannock (UK)	AS	**1.455**	0.109	0.531	0.065
	Rushmoor (UK)	AS	**1.973**	0.190	0.756	0.123
	Kralingse Veer (NL)	AS	**1.188**	0.510	0.565	0.273
	De Meern (NL)	AS	**1.795**	0.736	0.854	0.449
	Horstermeer (NL)	AS	**5.489**	0.522	**1.960**	0.283
	Estepona (ES)	AS	**5.398**	0.334	1.978	0.152
	La Vibora (ES)	AS	**13.418**	**1.043**	**5.605**	0.503
	Munich (G)	AS	**1.176**	**1.176**	0.479	0.479
	Torino (IT)	AS	0.560	0.140	0.314	0.077
	Robecco (IT)	AS	**2.201**	0.958	0.752	0.358
	Ratingen (G)	AS	0.473	0.216	0.191	0.098
Canada	Average		**2.772**	0.375	1.201	0.213
US	Average		**3.063**	0.512	1.072	0.207
Europe	Average		**3.157**	0.555	1.261	0.275

invoke site-specific dilution as an additional exposure modifier, which will only lower exposure overall.

The use of SSDs has been combined with the TU approach to formulate a risk assessment for AE. The reanalysis of chronic data used in this research was based on improved and consistent estimations of toxic effects relative to that of van de Plassche et al. (1999). This was accomplished using a suite of chronic QSARs applied appropriately to each trophic level based on a homolog-specific measure of hydrophobicity for an AE mixture. The matrix of pure homolog HC_5s can provide the basis to interpret any new or future AE environmental fingerprint for the purpose of risk assessment. The HC_5 matrix, based on a relatively large chronic single-species data set, will be robust to future additional data additions. The incremental change in adding new species to the data set was investigated using a Monte Carlo simulation and bootstrapping technique (G. Carr, Procter & Gamble, unpublished). The HC_5 is stable with a change of $<5\%$ from the present values expected if additional sensitive or tolerant species are added across the range of AE homologs. The robustness of the existing data set essentially anchors the distribution to become insensitive to additional data; that is to say, a broad range in sensitivity is incorporated that makes a solid data set not easily influenced by outliers or new data. Therefore, the present pure homolog HC_5 matrix will be useful in its present form for a considerable period of time.

Roberts (1991) demonstrated and later refined (Roberts and Marshall, 1995) that ecotoxicity of commercial nonionic surfactants, such as AE, can be modeled as multicomponent mixtures. A general nonpolar narcotic mode of action for all components makes the use of a simple similar concentration addition model possible (Nirmalakhandan et al., 1994). Similar findings of baseline toxicity have been given for pharmaceuticals, chlorinated benzenes, alcohols, and phenols (Broderius et al., 1995; Escher et al., 2002). Vighi et al. (2003) showed that the concentration addition model for similarly acting compounds fit exceptionally well for single species and

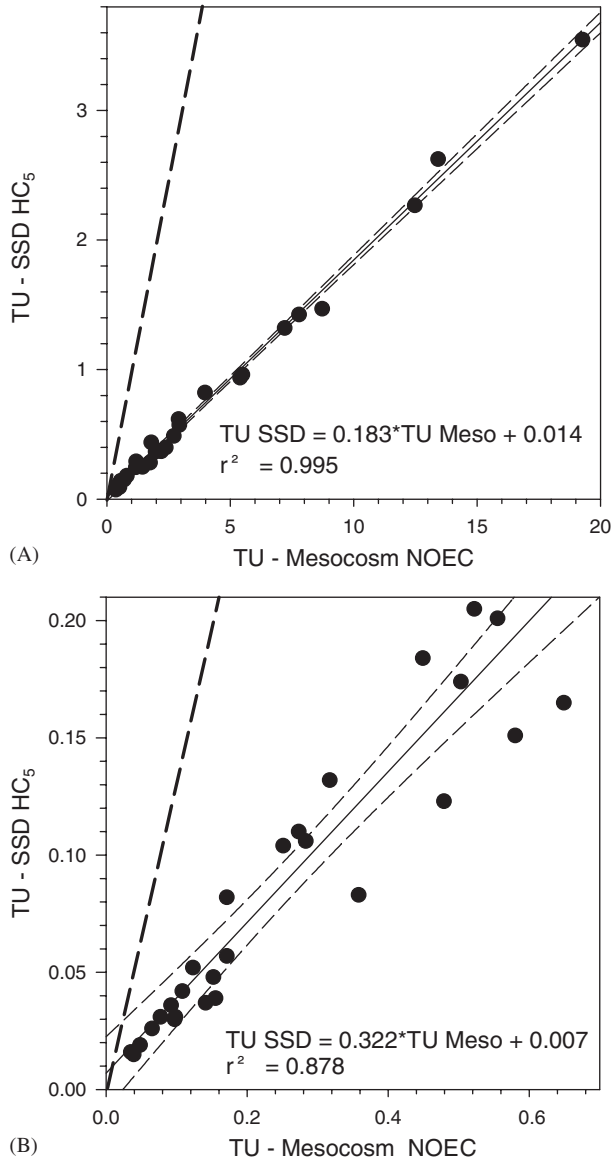

Fig. 3. Relative predictions of TU-based predictions versus MEC/Mesocosm NOEC. (A) Compares predictions for the entire AE environmental fingerprint and (B) compares predictions when exposure is modified by the alcohol cap and the bioavailability correction. The regression confidence intervals are indicated by a short dash line. The heavy dashed line is the line of 1:1 correspondence if predictions were exactly the same.

reasonably well for community level toxicity tests. AE are complex mixtures but fit well into the expectations for these models.

A unique problem in the AE risk assessment is how best to account for the alcohol measured in wastewater effluents. Fatty alcohol is a natural component of wastewater influent and effluent from normal biological processes. Fatty alcohol can be produced by the degradation of vegetable or animal matter and is an expected component of human waste (blackwater) (Leeming et al., 1994). Further, fatty alcohol is highly sorptive (van Compernolle et al., 2006) and the fraction that is "free" in solution is rapidly attacked and biodegraded (Federle and Itrich, 2006). In addition, fatty alcohol is used in a wide variety of consumer products and pharmaceutical applications and is a component of alcohol-based anionic surfactants such as alcohol sulfates and alcohol ether sulfates (or alkyl ethoxysulfates). The determination of an alcohol cap to quantitatively associate alcohol derived from the initial distribution of AE going down the drain or from AE biodegradation is a reasonable means to account for the fatty alcohol concentration measured as part of the AE environmental distribution (Wind et al., 2006). Sorption is also used in the definitive assessment because AE components measured in the sewage treatment plant effluent samples included the fraction initially associated with solids (Dunphy et al., 2001). The concentration of ecotoxicological concern is that which is free in solution, but because concentrations of the 114 ethoxymers are so low individually (often 10 ng/L or less), large sample volumes (4 L) must be concentrated during extraction, thereby disturbing the equilibrium between free and sorbed components. Correction for sorption is then used to account for the amount likely to be bioavailable in the effluent before sample preparation based on an AE-specific QSAR. The "free" or biologically available AE is then used in the risk characterization. Bioavailability of surfactants in wastewater is typically a function of alkyl chain length and the amount of suspended solids and sorption of hydrophobic surfactants to organic carbon and wastewater solids, either or both of which have been shown to directly influence the apparent toxicity of a sample (Traina et al., 1996; Versteeg and Shorter, 1992; Versteeg and Woltering, 1990).

The conclusions for the environmental risk assessment of AE utilized the full power of new analytical methods, corrected exposure for alcohol and sorption, and employed a revised effects database consisting of EC_{10} calculations from 60 studies on 17 different aquatic species. The TU approach based on homolog-specific interpretations of SSDs provides a powerful and convenient means to perform site-specific and regional risk assessments. Predictions based on these SSDs were consistent with five mesocosm studies summarized by an independently derived structure–activity relationship. For 29 monitored sites, the risk from exposure to AE is small, especially considering that exposure is based on undiluted effluent. Once dilution is considered the minimal risk will proportionately decline further. Regional evaluations for the United States, Canada, and Europe reach similar conclusions, with TU-based risk assessments ranging from 0.049 to 0.094, with 1.000 indicating acceptable risk of the use of AE.

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