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Derivation of aquatic predicted no-effect concentration (*PNEC*) for ibuprofen and sulfamethoxazole based on various toxicity endpoints and the associated risks

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HIGHLIGHTS

- PNECs of two pharmaceuticals were derived from various toxicity endpoints.
- The traditional endpoints could not be the most sensitive ones for pharmaceuticals.
- The most sensitive endpoint varies among pharmaceuticals we test.
- The probability of risk quotient of ibuprofen exceeding 0.1 was 11.2%.

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ABSTRACT

For pharmaceuticals, the ecological risk assessment based on traditional endpoints of toxicity could not be properly protective in the long run since the mode of action could vary because they are intended for different therapeutic uses. In this study, the predicted no-effect concentrations (*PNEC*s) of two selected pharmaceuticals, ibuprofen (IBU) and sulfamethoxazole (SMX), were derived based on either traditional endpoints of survival and growth data or some nonlethal endpoints such as reproduction, biochemical and molecular data. The *PNEC*s of IBU based on biochemical-cellular and reproduction data were 0.018 and 0.026 μg L^{-1} that were significantly lower than those derived from other endpoints, while the lowest *PNEC* for SMX derived from growth data with the concentration of 0.89 μg L^{-1}. Ecological risk assessment was performed for IBU and SMX to the aquatic environment by applying hazard quotient and probabilistic distribution based quotient (*DBQ*s) methods. The results showed that the probability of *DBQ*s of IBU exceeding 0.1 was 11.2%, while for SMX the probability was 0.9% that could be neglected.

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1. Introduction

During past decades, more and more concerns have been attracted by emerging contaminants due to their potential environmental occurrence and hazard, among which pharmaceuticals are one of the most important classes. Most pharmaceuticals are human designed chemicals that have potent biological activities (Snyder et al., 2003), and thus they may react with non-target receptors and result in unpredictable hazardous effects (Heberer, 2002; Kim et al., 2007; Ricart et al., 2010). Consequently, pharmaceuticals are often considered as a class of contaminants with 'subtle, potential, and cumulative effects' (Daughton and Ternes, 1999).

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Due to their large consumption as part of human welfare, an increasing detection of pharmaceuticals in the environment has been reported. For example, environmental sampling programs in China, UK, Europe, North America and elsewhere have shown the extensive occurrence of a large set of pharmaceuticals in various matrices (Bu et al., 2013; Crane et al., 2006; Sui et al., 2015, 2017; Wang and Wang, 2016; Yang et al., 2017). Considering both the potential hazard and extensive detection, it is vital to assess the ecological risk of pharmaceuticals.

Ecological risk assessment (ERA) has been extensively used to help understand the adverse effects posed by contaminants to the ecosystem. An important step in ERA is effect assessment, i.e. the determination of the maximum concentration at which the ecosystem is protected, well known as the predicted no-effect concentration (*PNEC*). To determine *PNEC*s, various measurement endpoints of toxicity could be employed. Although no identical criterion is available at present, the derivation of *PNEC*s in most study was usually based on all available measurement endpoints or traditional endpoints related to survival, development and growth of test organisms obtained from the measurement of a standard ecotoxicology test. These traditional endpoints were extensively used because they can be readily linked to population-concentration effects (Jin et al., 2014; Lei et al., 2012).

However, it has been argued that *PNEC*s derived from certain traditional endpoints are not guaranteed to protect aquatic organisms from being affected. Adverse effects may be observed at concentrations lower than *PNEC*s derived from traditional endpoints (Caldwell et al., 2008; Jin et al., 2014, 2015). For example, acute and chronic *PNEC*s of bisphenol A derived from the estrogenic effect data were much lower than that derived from all available endpoints (Feng et al., 2015). The similar result came from the study of Wang et al. (2013), which showed that bisphenol A posed a higher risk to the Chinese aquatic environment if the estrogenic effect was considered as the effect endpoint. Another study showed that reproduction was a more sensitive endpoint for nonylphenol, with *PNEC*s ranging from 0.12 to 0.60 μg L^{-1}, which was significantly lower than those derived from the traditional endpoints (Jin et al., 2014). The study of Caldwell et al. (2008) showed that the estrogen can cause the death of aquatic organisms at a concentration of mg/L, however, it could lead to an irreversible toxic effect on the reproduction of vertebrates at a concentration of the ng/L.

These above-mentioned facts indicated that some chemicals could act in a specific mode, and for these chemicals, nonlethal toxicity endpoints could be properly protective in the long run (Feng et al., 2015; Jin et al., 2014, 2015; Lei et al., 2012). This could especially be the truth for pharmaceuticals because they were designed to have a specific mode of action with target receptors in human beings or animals depending on therapeutic uses. In particular, few pharmaceuticals were identified as 'toxic' chemicals from the acute toxicity data acquired in the standard ecotoxicological test (Brausch et al., 2012; Corcoran et al., 2010; Fent et al., 2006). Nevertheless, the toxicity effects should not be ignored due to their reactive properties with non-target receptors. Literature studies have demonstrated that the same or similar target organs, tissues, cells and biomolecules in non-target organisms may be affected after exposure to pharmaceuticals (Daughton and Ternes, 1999; Fent et al., 2006; Jones et al., 2002; Seiler, 2002). Therefore, these raised one question that are *PNEC*s derived from traditional endpoints of pharmaceuticals properly protective to the environment? To answer this question, we hypothesize that for pharmaceuticals (I) the traditional toxicity endpoints were not always the sensitive ones, and (II) the most sensitive endpoint varies among different pharmaceuticals due to different therapeutic uses.

To test these hypotheses, we derived *PNEC*s for two selected pharmaceuticals, i.e. ibuprofen (IBU) and sulfamethoxazole (SMX), based on various toxicity endpoints. These two pharmaceuticals were identified by Bu et al. (2013) as potentially risky contaminants in the aquatic environment in China. In addition, IBU and SMX were selected because they belong to different therapeutic groups (anti-inflammatories and antibiotics, respectively) and experimental toxicity data of diverse endpoints are available for them at present. In addition, an assessment of the ecological risk posed by IBU and SMX to aquatic organisms throughout China was conducted by applying the hazard quotient (*HQ*) method considering the most sensitive toxicity endpoint and probabilistic ERA. In the probabilistic ERA, the risk probability was obtained by comparing distribution of both exposure and toxicity data.

2. Materials and methods

2.1. Toxicity data

Toxicity data for aquatic organisms exposed to IBU and SMX were retrieved were retrieved from multiple sources, including the United States Environmental Protection Agency ECOTOXicology Database (http://www.epa.gov/ecotox/), published literature and government documents. Data selection followed principles of accuracy, relevance, and reliability (Klimisch et al., 1997). When the no observed effect concentration (*NOEC*) was not available, the lowest observed effect concentration (*LOEC*) and/or data of lethal concentration (LC_x) or effect concentration (EC_x) was collected. For the same species with multiple toxicity data on the same endpoint, the geometric mean was used. All toxicity data were obtained from tests conducted in a freshwater system.

Toxicity data for IBU and SMX on aquatic organisms were divided into four categories in accordance with different endpoints as follows (Jin et al., 2014). Mortality data considered the effect of lethality to aquatic organisms. Growth data considered the effect of test chemicals on development, growth and morphology to aquatic organisms. In addition to the traditional endpoints, two categories of nonlethal endpoints, namely reproduction and molecular biology data, were considered. Reproduction data considered the effect of test chemicals on reproductive behavior, physiology, care of progeny and avian/reptile eggs to aquatic organisms. Biochemical and cellular data considered the effect of biochemical, enzyme, hormone, cellular, genetic and histology to aquatic organisms.

2.2. Derivation of PNECs

Species sensitivity distributions (SSDs) were constructed by fitting cumulative probability distribution under the assumption that the toxicity data were randomly sampled for all species and subject to a certain distribution (Wheeler et al., 2002; Zhao et al., 2017). A harmful concentration to x% species (HC_x) from the SSD was estimated, usually expressed as HC_5, at which less than 5% of species were affected (De Laender et al., 2008). Before the fitting procedure, the statistical distribution types of toxicity data for different species were tested by using the Kolmogorov-Smirnov test. In this study, log-normal distribution model was fitted to the toxicity data using the ETX 2.0, RIVM software, and then the value of HC_5 was derived. The final *PNEC*s were calculated as the derived HC_5 divided by a factor 5 (ECB, 2003).

2.3. ERA of IBU and SMX

Concentration data of IBU and SMX in the surface water of diverse water bodies were compiled by our previously literature review, including rivers, bays, harbors, lakes and coastal regions located in seven regions in China (Bu et al., 2013). A total of 36 exposure data of IBU were obtained with concentrations range

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