**Exposure of aquatic receptors to Bisphenol A: Evidence that current risk models may not be sufficiently protective.**

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Bisphenol A, [BPA; 2,2-bis(4-hydroxyphenyl)propane], a xenoestrogen identified as an agonist of the estrogen receptor, is an industrially important chemical that is used as a primary raw material for the production of engineering plastics (e.g., polycarbonate/epoxy resins), food cans (i.e., lacquer coatings), and dental composites/sealants. BPA discharge into the aquatic environment occurs not only from the migration of BPA-based products (processing and production) into rivers and marine waters but, the primary route of BPA contamination in the aquatic environment is effluent from wastewater treatment plants and landfill sites. Despite its biodegradability and short half life, BPA has been implicated in various wildlife and human health endpoints; survival, growth and development and reproductive success and infertility, impaired reproduction, precocious puberty, endometriosis and production of breast, vaginal, prostate, and uterine cancer, respectively. Since BPA has been identified in surface waters it has been the subject of considerable research into its potential effects on aquatic organisms. Several studies have been done to establish environmental concentrations of BPA in various aqueous media. Further, wildlife studies have been done to establish acute and chronic toxicity endpoints. These aforementioned endpoints have been utilized to conduct and establish an aquatic hazard assessment. An aquatic hazard assessment establishes a “derived predicted no effect concentration” (PNEC) below which it is assumed that any aquatic receptors and the aquatic ecosystem will not suffer adverse effects from a particular chemical.

With this background, an aquatic hazard assessment of BPA was conducted using the weight of evidence approach. These studies yielded very broad “no observed effect concentrations” (NOEC’s) ranging from 0.002 µg/L to 8,400 µg/L and “lowest observed effect concentrations” (LOEC’s) ranging from 0.048 µg/L to 12,500 µg/L for various species of aquatic receptors including fishes, invertebrates and algae. This toxicity dataset for BPA thus suggests that effects on species specific aquatic receptor mortality, growth and development and reproduction may start to begin at extremely low concentrations that are environmentally relevant and have been reported in surface waters in China, Germany, Japan, the Netherlands, and the USA. Calculation of a refined PNEC of 0.0519 µg/L based on inclusion of previously disregarded and new NOEC’s and LOEC’s for an expanded aquatic receptor matrix strongly suggests that the currently accepted BPA PNEC of 8 µg/L or less is not sufficiently protective for some aquatic species.