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Office of Pesticide Programs (OPP)
Regulatory Public Docket (7502P)
Environmental Protection Agency
1200 Pennsylvania Ave., NW.
Washington, DC 20460-0001

Regarding: Triclosan Risk Assessment; Docket EPA–HQ–OPP–2007–0513

To Whom It May Concern:

The Environmental Working Group (EWG) is a non-profit public health and environmental research and advocacy organization based in Washington, DC, and Oakland, CA. We focus much of our research on potential health risks from exposures to hazardous chemicals that contaminate food, water, and the environment, or that are used as ingredients in consumer products. With this letter, we provide detailed suggestions to EPA regarding its draft risk assessment of one such chemical, the antimicrobial agent triclosan, a potential developmental toxicant used in a wide range of consumer products.

Overall, we have very serious concerns with the Agency's assessment, particularly in its overt omission of numerous critical exposure routes and safety margins that in the end make triclosan exposures appear much safer than a reasonable and thorough risk assessment would conclude. The Agency omits critical routes of exposure for infants and/or young children, including breast milk, children's personal care products, toys and clothing and yet concludes that current exposures are safe. The Agency fails to consider potentially significant exposures routes, particularly inhalation exposures, which could present very serious risks to consumers. And in addition to these clear omissions, the Agency does not adopt an additional ten-fold safety margin, as required by the nation's pesticide law when exposure and toxicity data for infants and children are incomplete, as is clearly the case here based on data gaps identified by the agency in the draft assessment. Until these major failings are addressed, this risk assessment cannot be considered an adequate or accurate estimate of triclosan exposure and risk.

We do, however, applaud EPA for efforts to compile data on toxicity and the range of consumer products containing triclosan from all 3 agencies that regulate the safety of this pesticide – the Consumer Product Safety Commission for triclosan-containing consumer products not making a claim for antimicrobial action, the Food and Drug Administration for triclosan as a food packaging chemical, hospital disinfectant, and ingredient in personal care products, and your own agency for consumer products making antimicrobial claims.

We also applaud the use of biomonitoring, or body burden, data to represent the range of exposure to triclosan across the population. These direct measurements of triclosan concentrations in the human body capture many types of exposures for which it would be difficult to collect specific information from manufacturers and other agencies, given the broad range of consumer products for which manufacturers are not required to provide records of their use of this pesticide or proof of its safety. These body burden data provide the best available record of cumulative exposures to triclosan across the many possible sources people face daily, including consumer products and contamination in homes, food, and water. They show that at least 75% of the U.S. population has triclosan in their bodies at any given time, and highlight the necessity of a risk assessment that fully considers potential health risks from these cumulative exposures.

Unfortunately, as mentioned above, we have identified major gaps in the assessment that may leave the public at risk from triclosan exposures, particularly children who may be more vulnerable to its adverse effects. We recommend that EPA:

- **Assess cumulative risks for infants drinking triclosan-contaminated breast milk, and for all young children facing multiple exposures to this pesticide.** In the draft assessment, EPA has not evaluated the safety of cumulative exposures to infants and young children from contaminated breast milk and all other sources of the pesticide, including house dust, contaminated food, plastic toys, bibs and children's clothing, and children's body care products and toothpaste. The biomonitoring studies on which the Agency relies for cumulative exposure estimates do not include children under 6 years of age. Triclosan poses particular risks to infants and young children, as one study linked in utero exposures to the chemical to reduced fetal weight and irregular skull formation during development. Other studies link the pesticide to disruption of both the thyroid system and levels of calcium ion in the body, which could impact growth and development especially with respect to the brain and nervous system.

- **Provide an adequate margin of safety for children.** In the draft assessment, EPA has not provided the additional margin of safety of a factor of 10 that is required under the Food Quality Protection Act (FQPA) for children exposed to pesticides. Although triclosan is not approved as a food pesticide, it contaminates food and water consumed by children and is widely used in children's products. The Agency's failure to provide an additional safety margin of 10 to account for potential neurodevelopmental effects and other impacts to which children may be especially vulnerable violates the intent of the nation's pesticide law and leaves children at potential risk.

- **Assess inhalation risks and other cumulative exposures to triclosan.** In the draft assessment, EPA has not evaluated risks for children and adults who face the highest exposures and risks from triclosan, particularly consumers who use triclosan-containing products that can be inhaled, like powdered personal care products. EPA's assessment clearly shows that workers who apply triclosan-

treated paint using sprayers fall well outside standard safety margins, with a margin of exposure of only 54. It is important that EPA assess risk from the full range of Americans' inhalation exposures, both residential and occupational, since studies show that triclosan is most toxic when it is inhaled, with harmful effects at every dose tested. Although the biomonitoring data on which EPA relies capture a range of exposures across the population, they do not allow for an assessment of cumulative, high-risk inhalation exposures, and they do not encompass the most highly exposed in the population who would face the greatest risks. Cumulative exposure assessments that include occupational and household exposures to triclosan are particularly critical for commercial painters using airless spraying equipment, and pulp and paper workers using metering pump equipment.

- **Fully account for triclosan risks to human health.** In the draft assessment, EPA has not fully accounted for potential health risks posed by triclosan. The Agency based its health risk calculations on a study showing a NOAEL, or no observed adverse effect level, that is 4.6 times higher than the NOAEL reported for a study documenting triclosan's ability to cause liver toxicity in mice. The Agency's cancer risk assessment relies on second-hand information, as EPA has stated that Colgate has refused to provide them with its cancer study data upon request. EPA further takes the controversial position that the liver tumors linked to triclosan in the Colgate study are not relevant to human health, though there is no scientific consensus on this subject. EPA further neglects any assessment of triclosan's potential to disrupt the endocrine system, despite a growing body of research indicating that the pesticide may affect thyroid and reproductive hormone systems.

- **Fully consider triclosan risks to the environment**. In the draft assessment, EPA has not fully assessed the potential environmental risks posed by triclosan. Though triclosan is known to have significant acute effects on algae and other aquatic species, and is widely detected in streams across the U.S., EPA found no studies that assessed chronic ecotoxicological effects of the pesticide. Further, EPA's evaluation identifies data gaps including the lack of acceptable studies of triclosan's acute effects on freshwater invertebrates, marine organisms, and selected plants. The Agency further ignores the risks posed by triclosan that contaminates biosolids applied to agricultural lands. EPA also neglects to assess the impact of exposure of low levels of antimicrobial agents like triclosan on the biological wastewater treatment systems essential to preserving the water quality of rivers and lakes throughout the country, as well as clean water supplies for downstream communities.

- **Assess risks associated with triclosan's transformation products.** In the draft assessment, EPA has not assessed potential health and environmental risks posed by triclosan's many transformation products. In chlorinated or natural waters, triclosan can be transformed into a number of other chemicals, including methyl triclosan, chloroform, and even a form of dioxin. EPA's draft risk assessment

mentions some of these chemicals, but does not evaluate associated exposures or risks for any of them.

- **Assess risks relating to antimicrobial resistance.** In the draft assessment, EPA has not assessed potential health risks that could result through development of antimicrobial resistance to triclosan. Laboratory evidence and common sense indicate that microbes can develop resistance to the antibiotic effects of triclosan, rendering the pesticide ineffective.

EWG's previous research on triclosan includes studies of the pesticide in consumer products, the environment, and people, and confirms its common use in consumer products, its ubiquity as an environmental pollutant, and its widespread presence in the human body. As part of our online safety assessment of personal care products, Skin Deep, we have collected extensive information on body care products containing this ingredient, with 932 products altogether that include many marketed specifically for children's use (EWG 2008a). Our study of contaminants in urban wastewater documented detectable levels of triclosan in wastewater flowing from laundry facilities, medical and veterinary clinics, and manufacturing facilities, as well as in treated wastewater discharged to San Francisco Bay (EWG/EBMUD 2007). In addition, multiple EWG biomonitoring studies have documented the presence of triclosan in the blood and urine of everyday Americans (EWG 2008b), detecting triclosan in 42 of 49 people tested altogether.

EPA originally approved use of triclosan as a pesticide prior to November 1984 under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Now, over a quarter century later, triclosan is a ubiquitous pollutant in people, and has found its way into hundreds of consumer products. Expanded use and exposure has occurred despite the fact that FDA review of the existing research found no evidence that antibacterial products provide any health benefits over soap and water (FDA 2005). An updated risk assessment is now required to ensure that triclosan meets current scientific and regulatory standards for safety.

We urge EPA to amend its draft risk assessment to fully assess all exposures and risks to people, especially infants and young children, and to use this information to take steps to reduce exposures of everyday Americans to this pesticide. In fact, we believe that FDA should not allow triclosan in household hand soaps, as their own research indicates that it provides no health benefits to families. EPA must also evaluate the effects of triclosan in the larger environment, and take action to reduce its release into U.S. land and waterways.

Details and our rationale for these recommendations are provided below.

Assess cumulative risks for infants drinking triclosan-contaminated breast milk, and for all young children facing multiple exposures to this pesticide. EPA has failed to assess young children's exposures and risks. By relying on NHANES biomonitoring data that does not include any exposure information for children ages 5 and younger, EPA has ignored the very group that may be most vulnerable to

triclosan toxicity. Triclosan is found in breast milk, house dust, children's personal care products, children's toys and other items that raise particular concerns for children, and yet EPA assesses exposures and risks for almost none of these sources.

We provide results from a basic calculation on infant exposures to triclosan through breast milk. Using the highest measured level of triclosan in breast milk samples collected from 62 women in the U.S., 2100 µg/kg lipid (Dayan 2007), and the body weight-adjusted lipid intake calculated for the 95th percentile of 0-6 month infants, 6.35 g lipid/kg body weight/day (Arcus-Arth 2005), a significant number of American infants may consume more than 13.3 µg triclosan/kg body weight/day. This exposure alone is substantially greater than a "safe" daily dose calculated according to standard EPA requirements for children's protection under pesticide law (details on this calculation are in the subsequent section of these comments).

To ensure that infants are not exposed to potentially unsafe levels of triclosan, EPA should evaluate the full range of exposures to triclosan for exclusively breast-fed infants using a Monte Carlo statistical analysis that includes a full accounting of the range of infants' weight and breast milk consumption, and ensuring that 99.9% of this sensitive population is exposed below levels established as safe, as is the Agency's practice for protecting children from pesticides in food. This analysis should consider all other potential routes of exposure for infants in addition to contaminated breast milk, including triclosan-impregnated mattresses, blankets, mattress pads, mattress filling and sheets (Microban Products Company 1997; Sanitized Inc. 2003; Huntsman International 2007); plastic toys (Microban Products Company 1997; Ciba 2006; Huntsman International 2007); triclosan in body wash and diaper rash ointment (EWG 2008a); triclosan in sleepwear, caps, and other "general wear" clothing (Microban Products Company 1997, Sanitized Inc. 2003); and triclosan contamination in house dust (Canosa 2007). Inhalation exposures may be a particular concern for infants sleeping on triclosan mattresses, sheets and blankets, in addition to skin absorption and ingestion from mouthing. When children begin eating solid food, exposures through mouthing of triclosan-impregnated bibs should also be considered, as well as exposures through contaminated food.

As young children grow and develop, exposure to triclosan through ingestion of toothpaste may become an important exposure pathway. We can use calculated exposures to fluoride in toothpaste as a means of estimating exposure to triclosan in the same product. Typical two-year-olds can ingest an average of 0.055 mg/kg/day of fluoride through two daily brushings using a toothpaste with 1000 ppm fluoride (Levy 1999). Toothpastes with triclosan typically contain 3000 ppm of the pesticide, suggesting young children of average weight could ingest triclosan at levels as high as 0.17 mg/kg/day through normal use of toothpaste alone.

Infants' and children's combined exposures to triclosan via breast milk, toothpaste and other personal care products, clothing, toys, bedding, house dust and other sources must be considered for EPA's assessment to fully reflect potential health risks children under 6 face as the result of the widespread use of this pesticide in consumer products.

Provide an adequate margin of safety for children. EPA has not provided children with the additional margin of safety of 10 that is required under the Food Quality Protection Act (FQPA) for children exposed to pesticides. Triclosan contaminates food and water consumed by children and is widely used in children's products. As described below, studies provide evidence that triclosan poses risks during development, particularly for the brain and nervous system, yet a complete developmental neurotoxicity study has yet to be conducted. Thus, there is both a potential serious health risk for children and a substantial data gap that together support an additional margin of safety to protect children's health. The Agency's failure to provide an additional safety margin of 10 to account for potential neurodevelopmental effects and other impacts to which children may be especially vulnerable leaves them at potential risk.

Based on the most sensitive developmental study, which gives a NOAEL (no observed adverse effect level) of 25 mg/kg/d (MRID 43817501: citation missing from EPA 2008b), an appropriate "safe" or reference dose for oral exposures can be estimated by adjusting this NOAEL by EPA's proposed 100-fold margin of safety (Margin of Exposure, or MoE), with an additional 10-fold margin of safety, resulting in a reference dose of 0.0025 mg/kg/day, or 2.5 ug/kg/day. Data indicate that many infants are exposed to triclosan above this level from breast milk contamination alone.

Assess inhalation risks and other cumulative exposures to triclosan. While EPA notes that triclosan is acutely toxic via inhalation at low levels, it provides no assessment of inhalation risks in the home, and draws unwarranted conclusions regarding the occupational risk assessments it provides. EPA also neglects to estimate extreme exposures that could result from use of multiple, readily-available triclosan-treated consumer products.

A 21-day rat study on the effects of triclosan inhalation revealed toxicity at all dose levels examined, including dyspnea, nasal discharge, muscle spasms, pallor, and diarrhea, decreased body weight, decreased body weight gain, decreased food consumption, increased total leukocyte count, increased percentage of neutrophils and decreased lymphocytes, increased serum glutamic-pyruvic transaminase activity, increased alkaline phosphatase, decreased serum proteins (in males), and increased incidence of respiratory inflammation (MRID 0087996: citation missing from EPA 2008b). The LOAEL (lowest observed adverse effects level) calculated based on this study is 50 mg/m^3, or 3.21 mg/kg/day, for males, and 115 mg/m^3, or 9.91 mg/kg/day, for females. A NOAEL could not be established for males; the NOAEL for females is 50 mg/m^3 or 4.51 mg/kg/day. EPA incorrectly identifies the male rat LOAEL as a NOAEL in its summary table on triclosan toxicology (EPA 2008a).

EPA's own occupational exposure scenarios indicate that commercial painters using airless spraying equipment and pulp and paper workers using metering pump equipment could be exposed to levels of triclosan in the air not found safe in EPA's estimation. In the draft assessment, EPA gives the "safe" or reference dose for

inhalation exposures to triclosan as 1000 times lower than the LOAEL determined in animal studies (EPA 2008c). EPA calculates that painters may be exposed to a dose just 54 times less than the relevant lab animal dose, while pulp and paper workers may be exposed to a dose just 28 times less (EPA 2008c), many times higher than the agency's proposed safe dose.

EPA's calculation of risk related to non-occupational exposures to triclosan neglects risk associated with inhalation, instead focusing solely on risk associated with an oral exposure study that provides a NOAEL nearly ten times higher than the inhalation LOAEL. In fact, average Americans may inhale triclosan in a number of ways. Triclosan has been detected at trace levels in household dust (Canosa 2007), which can constitute a source of inhaled toxicants especially during vacuuming and cleaning, and for infants and toddlers who frequently mouth their hands, toys and other objects. Though a solid, triclosan has a measurable volatility, and may volatilize from home heating, ventilation, and air-conditioning (HVAC) systems or air filters or humidifiers/dehumidifiers treated with the pesticide. It could volatilize from mattresses, blankets, pillow and other bedding for potentially significant exposures for people whose faces are lying against or near these surfaces for 8 hours daily. Further exposure through inhalation could result following aerosolization of powder or pump action personal care products containing triclosan. Because triclosan is far more acutely toxic via inhalation than via other routes of exposure, risk assessment based an oral exposure study alone is inadequate.

In addition, EPA has failed to assess many other potentially significant sources of triclosan in the home, including:

- Mattresses – potential for dermal and inhalation exposure;
- Contaminated foods – contamination can occur through use of triclosan-treated kitchen implements (Canosa 2008), and may also arise through use of triclosan-treated mulch in home gardens, application of triclosan-contaminated biosolids in agricultural fields, and consumption of fish harvested from waterways that receive discharges of treated wastewater;
- Personal care products – significant oral exposure may occur through use of oral care products with triclosan, including many Colgate toothpastes; significant dermal exposure may occur through use of products applied over the entire body, such as Dial's Clean & Refresh Body Wash; and as mentioned above, significant inhalation exposures may occur through use of products in powder or aerosol form, such as Pinkie Swear Diva Dust, or Right Guard Sport Deodorant Aerosol.

Each of these must be assessed if the Agency is to understand how to mitigate exposures to triclosan so that human health is protected. By overlooking these and other sources of triclosan, EPA has failed to use best Agency practices in conducting its exposure assessment. The Agency must consider the full range of exposures and behaviors in its calculations of risk from particular products and sources (for instance, using a Monte Carlo simulation that is now typical Agency practice), and protect 99.9% of the most vulnerable population from non-cancer effects. EPA

performs such calculations regularly for food pesticides, and should apply the same level of scrutiny and protection for triclosan.

Fully account for triclosan risks to human health. The Agency's draft assessment of human health risks was not based on the most sensitive measure of toxicity reported, involved a cursory inspection of cancer risk, and completely neglected concerns about hormone disruption.

The most sensitive endpoint must be used for human health risk assessment. EPA calculates the reference dose it considers safe for humans based on a NOAEL 4.6 times higher than that identified in a 28-day study of liver toxicity in mice (Trutter 1993). The NOAEL EPA has selected for its calculation is also 3 times higher than studies that report cancer and both maternal and developmental effects in mice, discussed in further detail below (See 1996; MRID 43817501: citation missing from EPA 2008b). By ignoring studies performed using robust methodologies according to EPA's own guidelines, and setting "safe" doses higher than can be supported by the science, EPA's triclosan risk assessment could erode public health protections.

EPA must assess cancer risks. The Agency's cancer risk assessment was performed with a similar disregard for available evidence. In 2007, EPA reviewed cancer data for triclosan and determined it to be not a concern for humans (EPA 2008d). Rat and hamster studies revealed no increase in tumors. Liver adenomas and carcinomas were reported in a Colgate-Palmolive mouse study (See 1996), which the company refused to release to EPA. Lacking legal authority to demand the results, EPA was limited to reviewing data provided by FDA.

EPA took the position that the mode of action for carcinogenicity of triclosan is irrelevant to human health because it likely involves proliferation of peroxisomes and cell proliferation, making it "plausible" but "quantitatively implausible" in humans. However, the human relevance of liver tumors in animal, and particularly the issue of peroxisome proliferation, continues to be a highly controversial subject (Klaunig 2003; Peters 2005; Keshava 2006; NAS 2008).

There is no scientific consensus about the type of evidence required to support EPA's contention that PPAR activation alone is responsible for the observed tumors, and that these tumors do not signal any health concerns in humans. For example, transgenic mice that express PPARα in hepatocytes display many similar effects as wild-type mice with respect to PPAR activation, but do not get hepatocarcinoma. These findings indicate "that these effects alone are not sufficient to induce liver cancer" (Yang 2007). Given the controversy surrounding links between PPAR activation and cancer, EPA cannot dismiss the carcinogenic properties of triclosan observed in the laboratory as irrelevant to human health.

EPA must evaluate developmental risks. EPA's evaluation concludes that triclosan does not cause developmental toxicity, based on rat and rabbit studies that do not detect developmental effects under the dose ranges examined. EPA also

suggests that a developmental neurotoxicity study is not needed, as it found no evidence of neurological toxicity to adults.

In drawing these conclusions, EPA ignores the findings of a mouse study it has classified as "acceptable/guideline," which supports a developmental NOAEL associated with a maternal oral dose of 25 mg/kg/day, or an achieved dose of 11.2 mg/kg/day (MRID 43817501: citation missing from EPA 2008b). This study found irregular ossification of the skull and decreased fetal weight of 14 and 18% at the two highest administered doses. This developmental NOAEL is in fact lower than that used to calculate the reference dose for this assessment (25 vs. 30 mg/kg/day) (EPA 2008a).

A further deficit in EPA's reasoning, neurodevelopmental impacts include behavior, learning, memory and other types of disruption that are not probed in typical tests of adult animals. An EPA review of developmental neurotoxicity studies for pesticides found that such studies often identify toxicity at lower levels than other endpoints. As of 2006, 58 studies had been completed and in 8 cases were used for risk assessment (Raffaele 2006). Lead, mercury, and PCBs are well-known examples of developmental neurotoxicants that elicit effects specific to development, and not seen in studies of healthy adult animals (Grandjean 2006).

In the case of triclosan, evidence of thyroid disruption indicates a risk of disruption to the brain and nervous system during development. A study of frogs shows that this pesticide perturbs a fundamental thyroid hormone signaling mechanism that is nearly identical to that of humans (Veldhoen 2006). Triclosan, in concentrations of less than 1 part per billion commonly measured in U.S. streams, interferes with the timing of the expression of several genes that are crucial in early development. The developing brain of a child is particularly vulnerable to damage from thyroid hormone changes. An additional cause for concern, recent research using the ryanodine receptor type 1 (RyR1)-based bioassay reveals triclosan may have the ability to affect Ca^{2+} homeostasis (Ahn 2008), suggesting the pesticide has the potential to contribute to alteration of neurodevelopment and neuroplasticity function (Wong 2001).

EPA has not evaluated the potential for neurological impairment due to developmental triclosan exposure. EPA presents findings from 3 developmental toxicity studies, but the summary information provided indicates no assessment of neurodevelopmental outcomes. As outlined in OPPTS 870.6300, neurodevelopmental toxicity studies should assess dams for autonomic function, and offspring's motor activity, startle reflect, learning and memory, and neuropathology (EPA 1998).

By ignoring potential developmental effects that occur at low levels of exposure, this assessment is intrinsically biased in favor of reduced concern over triclosan. We strongly urge EPA to require a study of triclosan's effects on the developing brain and nervous system. In EPA's assessment of triclosan and in the absence of a full developmental neurotoxicity study, the Agency must include a 10-fold safety factor to account for unique vulnerabilities of exposures during pregnancy, infancy and

childhood, given that neurodevelopmental impacts cannot be ruled out based on the data available so far.

By refusing to consider triclosan as a developmental toxicant and incorrectly removing this additional safety factor, EPA justifies using the far less protective margin of error (MOE) of 100, rather than 1000, in calculating its "safe" dose for human exposure, as mentioned earlier. Using the lowest NOAEL from a study meeting EPA guidelines (6.48 mg/kg/day), and a MOE of 1000, results in calculation of a "safe" dose of 0.00648 mg/kg/day, 46 times lower than the dose suggested by EPA. According to NHANES data, more than 1 in 100 Americans is exposed to higher levels of triclosan than would be considered safe if EPA were to use more appropriate, health-protective decisions in its assessment of triclosan toxicity.

Neglecting these findings and data gaps thus results not only in rejection of a key toxic property of triclosan, but also in use of an inappropriate reference dose to establish the appropriate level of concern associated with exposures to this pesticide. It is essential that EPA use findings from all suitable studies in its assessment of triclosan's developmental toxicity, and demand additional study where necessary.

EPA must consider reproductive hormone disruption effects. A further concern for human health, emerging science indicates triclosan may affect reproductive hormone systems, as well as the thyroid system (Foran 2000; Ishibashi 2004; Matsumura 2005; Veldhoen 2006; Ahn 2008; Gee 2008). Hormone-disrupting chemicals can produce very different health effects at levels much lower than those associated with other measures of toxicity.

Many useful case studies are available that could help the Agency with its assessment of hormone disrupting effects of triclosan. For example, the well-known hormone disruptor bisphenol A has set a useful paradigm for evaluating dose response relationships for endocrine-disrupting chemicals. At high doses, those exceeding 5 mg/kg/day, bisphenol A can trigger gross toxic effects in laboratory animals including decreases in number of pups per litter, increases in fetal death, post-implantation loss, and reductions in fetal weight or growth during postnatal life (NTP 2008).

In contrast, at low doses, those less than 5 mg/kg/day, many effects emerged that were not observed in high-dose studies, including neural and behavioral alterations, especially related to the development of normal sex-based differences between males and females, tissue changes in mammary gland that may signal an increased susceptibility to develop mammary gland tumors later in life, and some evidence that perinatal exposure to BPA in rodents may alter prostate and urinary tract development and predispose the prostate to develop hormonally-induced pre-neoplastic lesions later in life (NTP 2008).

Like BPA, triclosan can produce significant hormonal alterations at levels of exposure far below those identified in the toxicological studies reviewed by EPA. By ignoring

hormonal effects that occur at lower levels of exposure, this assessment is intrinsically biased in favor of reduced concern over triclosan. EPA foot-dragging has delayed development of its formal Endocrine Disruptor Screening Program (EDSP) for over a decade. A list of 73 pesticides that EDSP suggests testing for hormone activity, released last year, does not include triclosan (EPA 2007). EDSP delays should not serve as an excuse to ignore consideration of serious hormone-related heath risks associated with exposures to triclosan.

Fully consider triclosan risks to the environment. EPA's evaluation identifies data gaps including the lack of acceptable studies of triclosan's acute effects on freshwater invertebrates, marine organisms, and selected plants. In addition, triclosan has not been assessed for chronic ecotoxicological effects for any aquatic species. Considering triclosan's toxicity to aquatic life, as well as the large volumes of the pesticide produced annually, this lack of chronic ecotoxicity assessment is especially egregious. According to EPA's Office of Pollution Prevention and Toxics Inventory Update, in 1998, the last reporting year for triclosan, between 1 million and 10 million pounds of this chemical were produced or imported into the U.S. (EPA 2002). In light of past industry trends and the ubiquity of triclosan-containing products on the market, it is reasonable to think that same or greater quantities are produced and released today.

Currently, 20 different concentrated triclosan products for various final manufacturing applications are actively registered with the EPA (NPIRS 2008). On the material safety data sheets accompanying these products, EPA requires a strict warning label about environmental hazards associated with triclosan: "This pesticide is toxic to fish and aquatic vertebrates. Do not discharge effluent containing this product into lakes, streams, ponds, estuaries, oceans, or public waters unless in accordance with requirements of a National Pollutant Discharge Elimination System (NPDES) permit and the permitting authority has been notified in writing prior to discharge. Do not discharge effluent containing this product to sewer systems without previously notifying the local sewage treatment plant authority." This stern toxicity warning to manufacturers stands in stark contrast to the embarrassingly incomplete ecotoxicity database in the current risk assessment for triclosan.

Moreover, although EPA has noted the potential for triclosan to impact endangered species, it has not yet completed an assessment of these impacts. EPA assumptions regarding the fate of triclosan as a component of biosolids derived from wastewater treatment are inconsistent with current research, described below, and must be amended given potential impacts to terrestrial wildlife.

The widespread detection of triclosan in streams across the U.S. makes a full environmental risk assessment for aquatic life essential. EPA's statements regarding detections described by Kolpin et al. (2002) are in error, and minimize the extent of contamination. Triclosan was detected in 49 of 85 streams tested, not 36 of an implied 139 streams tested, as described by EPA. The median level of triclosan detected in this study was 0.14 µg/L, and the maximum was 2.3 µg/L, not 0.040 µg/L and 0.280 µg/L as stated in the risk assessment.

As mentioned above, triclosan has been shown to have hormone-disrupting effects at low levels in aquatic species (Foran 2000; Ishibashi 2004; Matsumura 2005; Veldhoen 2006). These studies suggest that the levels of triclosan now found in many bodies of water across the U.S. may be harming aquatic populations at present. A complete, quantitative analysis of the effects of triclosan on the environment is thus a critical, and currently lacking, component of EPA's risk assessment. As well, EPA is required to assess the effects of triclosan use on endangered species, including "direct, indirect and habitat effects" (EPA 2008a).

An additional flaw in the draft ecological hazard and environmental risk assessment lies with EPA's inadequate treatment of terrestrial contamination through application of biosolids containing triclosan. EPA presents limited data on the concentrations of triclosan measured in biosolids taken from wastewater treatment facilities (EPA 2008e). This is followed by the allegation that less than 5% of the triclosan in influent is retained in biosolids, while the rest is biodegraded, based on laboratory experiments performed by Procter & Gamble scientists (Federle 2002).

EPA neglects to review an investigation of a real-world treatment plant that reports concentrations of triclosan in biosolids of 20 – 55 µg/g dry weight, higher than those reported by any study described in the risk assessment (Heidler 2007). This study further indicates that fully 50% of the triclosan present in wastewater influent does not degrade, but instead accumulates in the biosolids removed from the activated sludge treatment system.

EPA supports application of biosolids to agricultural land, though it lacks a comprehensive program to monitor the material for contaminants and analyze the risks it poses to human and ecological health, a situation that has led to lawsuits in recent years (Tollefson 2008). When spread over fields, biosolids become a source of terrestrial contamination with triclosan that EPA ignores in its risk assessment. Bioaccumulation of this pesticide in earthworms inhabiting soil amended with biosolids (Kinney 2008) provides further evidence to indicate a more detailed inspection of risks to terrestrial wildlife associated with triclosan is warranted.

A concern for both environmental and human health, the widespread use of triclosan in residential, commercial, and industrial settings results in trace levels of triclosan in the wastewater stream, as revealed by EWG's testing of 16 wastewater samples drawn from homes, businesses, and manufacturing facilities upstream of an urban water treatment facility in Oakland, California (EWG/EBMUD 2007). The effect of this antimicrobial agent on the industrial-scale biological systems used to treat wastewater prior to release into receiving bodies is ignored in this risk assessment.

While an initial study produced by Procter & Gamble claimed that the concentrations of triclosan found in untreated wastewater would have no effect on the microbial processes occurring in activated sludge wastewater treatment systems (Federle 2002), recent studies from independent scientists at universities around the world indicate that trace levels of triclosan could harm useful microbes inhabiting these

systems, and impair key biodegradation processes (Dokianakis 2004; Neumegen 2005; Stasinakis 2007, 2008). In addition, triclosan is not the only antimicrobial agent commonly found in consumer products and the wastewater stream; its effects should be evaluated in combination with other wastewater contaminants that could damage water treatment capacity.

Many communities in the U.S. depend on the drinking water drawn from water sources impacted by discharges from distant wastewater treatment plants. A pesticide that could reduce the effectiveness of common methods of wastewater treatment must be evaluated as a potential threat to public health.

Assess risks associated with triclosan's transformation products. EPA's risk assessment notes that triclosan is susceptible to biotransformation, resulting in detection of methyl triclosan in surface waters (EPA 2008e). However, EPA makes no attempt to assess the ecotoxicological risk associated with methyl triclosan. This biotransformation product is bioaccumulative (Adolfsson-Erici 2002; Lindstrom 2002); a recent European study found methyl triclosan in fish, especially concentrated in fatty tissue (Balmer 2004).

Few studies have probed the toxicological effects of methyl triclosan, but a recent publication reveals that the transformation product triggers acute toxic effects in the marine bacterium *Vibrio fischeri* at lower levels than triclosan (Farré 2008). Measurements of the inhibition of bioluminescence of this model organism result in an EC_{50} (50% effective concentration) of 0.28 µg/mL for triclosan and 0.21 µg/mL for methyl triclosan. The study documented a LOEC (lowest observed effect concentration) of 0.10 µg/mL for triclosan, and 0.075 µg/mL for methyl triclosan. EPA is obliged to require studies of the aquatic toxicity of this key triclosan transformation product as part of a thorough risk assessment of the pesticide.

Triclosan is known to degrade into a form of dioxin, a class of chemicals linked to a broad range of toxicities including cancer (Lores 2005). The Canadian government limits the levels of dioxins and furans allowed as impurities in personal care products that contain triclosan (Health Canada 2007). EPA makes no mention of potential health or environmental risks associated with impurities in its evaluation of triclosan, an evident shortcoming of its evaluation.

Finally, recent research indicates triclosan can react with residual chlorine from standard water disinfecting procedures to form a variety of chlorinated byproducts at low levels, including chloroform, a suspected human carcinogen (Fiss 2007). Again, EPA neglects to mention this critical finding, and provides no assessment of the risks associated with these transformation products. Given the widespread use of triclosan in consumer products, especially those that come into contact with tap water, an evaluation of the levels of exposure and health risks associated with these chlorinated byproducts is an essential component of a thorough investigation of triclosan's effects on human health.

Assess risks relating to antimicrobial resistance. The evolving interaction between microbes and antiseptic agents has led to concern that use of some antimicrobial ingredients may provoke development of strains of bacteria resistant to disinfection. Some strains of bacteria have acquired reduced susceptibility to triclosan (McMurry 1998; Chuanchuen 2001). The identification of a triclosan-resistant bacterial enzyme suggests that resistance to this antibacterial agent may develop more readily than to other agents (Heath 2000). In addition, exposing specific bacterial strains to triclosan appears to result in selection favoring bacteria that are resistant to multiple antibiotics (Chuanchuen 2001).

Concerns about antimicrobial resistance led to the official American Medical Association recommendation against using antibacterial products in the home: "The use of common antimicrobials for which acquired bacterial resistance has been demonstrated should be discontinued in consumer products unless data emerge to conclusively show that such resistance has no effect on public health and that such products are effective at preventing infection. Ultimately, antibiotic resistance must be controlled through judicious use of antibiotics by health care professionals and the public" (Tan 2002). Both FDA and the Centers for Disease Control and Prevention have reviewed the research on antimicrobial consumer products and concluded that these products provides no health benefits over soap and water (FDA 2005): "Currently, no evidence suggests that use of antibacterial soap containing 0.2% triclosan provides a benefit over plain soap in reducing bacterial counts and rate of infectious symptoms in generally healthy persons in the household setting" (Aiello 2005). Despite this fact, FDA continues to allow extensive application of the pesticide in a wide range of products.

The issues outlined above constitute serious defects in EPA's draft risk assessment for triclosan. However, we hope that the Agency will use the re-registration process as a chance to address all of the public health and environmental safety gaps associated with widespread use of this powerful antimicrobial chemical. We thank EPA for the opportunity to provide these comments, and urge the risk assessment team to address these issues thoroughly and promptly. We are confident that EPA will see the need to improve its evaluation of triclosan as we have outlined, as part of its mission to protect human health and the environment.

Sincerely,

[signed]

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