ENVIRONMENTAL WORKING GROUP

1436 U Street NW, Suite
100 Washington, DC 20009
T: 202.667.6982
F: 202.232.2592

April 6, 2009

EPA needs to protect people and the environment from contamination with antimicrobial pesticides

Office of Pesticide Programs
Environmental Protection Agency
1200 Pennsylvania Ave, NW,
Washington DC 20460-0001

Regarding: Proposed Rule for Data Requirements for Antimicrobial Pesticides
Docket EPA-HQ-OPP-2008-0110

Environmental Working Group (EWG) is a non-profit public health and environmental research and advocacy organization based in Washington, DC. We focus much of our research on potential health risks from chemical contamination of food, water, consumer products, and the environment. With this letter, we urge the Environmental Protection Agency (EPA) to address the health and safety gaps in its proposed rule on antimicrobial pesticides in order to protect human and environmental health from excessive exposures to these potent chemicals.

Antimicrobial pesticides and preservatives serve a useful public health function, especially in medical, commercial, and industrial applications. But their increasing and unnecessary use in a wide range of consumer products has been associated with environmental pollution, potential human health risks, and toxicity to ecosystems (Aiello 2007; Focazio 2008; James 2009). With the proposed rule, EPA revises and updates the existing data requirements for information that manufacturers need to submit to the Agency during the pesticide registration process. The new data requirements should allow EPA to make regulatory decisions so as to protect human health and the environment from potentially harmful, unintended effects of antimicrobial pesticide products, many of which are powerful toxicants.

EWG supports the overall approach the Agency has taken with the proposed data rule that incorporates tiered toxicology, exposure, and environmental fate studies. However, several shortcomings of the draft rule threaten to undermine its future regulatory effectiveness, since it allows pesticide manufacturers to avoid responsibility for the full scope of environmental health risks due to antimicrobial pesticide pollution. Furthermore, the final EPA rule needs to account for the overall presence of antimicrobial products in commerce, whatever their original mode of registration. EWG urges EPA to strengthen the new rule in three key areas that should:

- Protect human health from cumulative exposure to antimicrobial pesticides;
- Require monitoring of pesticide contamination in the environment and in people, including in cord blood and statistically representative samples from other vulnerable populations;
- Ensure that aquatic and terrestrial ecosystems are protected from chronic pesticide toxicity.

Details and supporting information for these recommendations are described below.

1. Protect human health from cumulative exposure to antimicrobial pesticides

Research by government scientists at the Centers for Disease Control and Prevention (CDC) and the U.S. Geological Survey, independent academic researchers, and public health advocacy groups demonstrated that already, the bodies of people and environment at large have been contaminated with antimicrobial pesticides and related chemicals used as preservatives, such as antibacterial soap ingredients triclosan and triclocarcban, agricultural antimicrobial pesticide ortho-phenylphenol, and wood deck preservative arsenic (Caldwell 2008; CDC 2005; Chu 2007; Coogan 2008; EWG 2008a; Focazio 2008; Halden 2005; Kingsbury 2008; Kolpin 2002). For example, the bodies of nearly 75% of Americans are polluted with triclosan (Calafat 2008). The reason for this extensive contamination is clear: present system of pesticide registration creates an ambiguous and dangerous situation whereby antimicrobial pesticide manufacturers can produce and include these toxic chemicals in a wide variety of consumer products without any scrutiny from EPA. Simply by avoiding the use of the word "pesticide" on the product label, manufacturers can add antimicrobial chemicals to any consumer product they choose. As a result, people may be exposed to multiple sources of antimicrobial pesticides in their own homes, often without their knowledge and without a chance to make an informed decision (EWG 2008b). This loophole must be closed so as to protect the health of American families.

Hand soap ingredients triclosan and triclocarban illustrate the unacceptable weakness of the current EPA approach to antimicrobial pesticide regulation. Triclosan and triclocarban share a common pattern of household usage, heavy disposal into wastewater plants, accumulation in wastewater sludge, and contamination of effluent, which eventually leads to contamination of ambient water bodies and drinking water with these pesticides (Halden 2005; Young 2008). Once released into the environment, they pose grave risks to aquatic life (Coogan 2008; Lawrence 2009; Oliveira 2009; Orvos 2002; Tatarazako 2004; Yang 2008). Additionally, these antimicrobials have been associated with adverse effects on thyroid, estrogen, and testosterone hormone function (Ahn 2008; Chen 2008; James 2009; Kumar 2008; Zorrilla 2008);

Despite these well-documented risks, the majority of triclosan and triclocarban-containing products, such as household cleaners and hand soaps, completely evade EPA oversight since they officially fall under FDA's jurisdiction. In the case of triclosan, some of its' antimicrobial uses (excluding hand-soaps and cleaners) are reviewed by EPA. In contrast, triclocarban effectively slips under the radar of regulatory agencies. Although previously triclocarban has been registered with EPA for use as a pesticide, triclocarban registration has been later cancelled (NPIRS 2008), but its use in commerce has continued. Triclocarban is still found in numerous antibacterial bar soaps on the market. EWG's Cosmetic Safety database lists 55 different products containing triclocarban, including 51 different bar soaps (EWG 2009) and the actual numbers are likely to be higher (Perencevich 2001).

From both scientific and health perspectives, it is completely irrelevant whether exposure to an antimicrobial pesticide is due to the presence of this chemical in a product that is regulated by EPA, FDA, or any other government agency – what matters is whether or not the overall exposures are safe over life-time. The proposed rule fails to remedy the current regulatory impasse whereby EPA cannot enforce manufacturer responsibility for the full range of

antimicrobial pesticide exposures faced by people and the environment. The final EPA rule needs to account for the overall presence of antimicrobial products in commerce, whatever their original mode of registration. So long as potent pesticide compounds appear in consumer products with little or no review from EPA, public health remains at risk.

Antimicrobial pesticides and preservatives serve a useful public health function, especially in medical applications where they can prevent the spread of life-threatening diseases. In contrast, antimicrobials do not bring additional health benefits for residential indoor and consumer product applications; instead, they may contribute to potential development of antimicrobial resistance (Birosova 2009; Cottell 2009; Levy 2001). Increasing and unnecessary use of antimicrobials in a wide range of consumer products raises concerns, because of the prospect of reduced medical usefulness due to antimicrobial resistance. Spread of triclosan- and triclocarban-tolerant bacterial strains could result in these chemicals becoming ineffective in their important medical applications (Aiello 2007). As summarized in a recent review, "of major concern is the possibility that triclosan resistance may contribute to reduced susceptibility to clinically important antimicrobials, due to either cross-resistance or co-resistance mechanisms… Thus, widespread use of triclosan may represent a potential public health risk in regard to development of concomitant resistance to clinically important antimicrobials" (Yazdankhah 2006). Ensuring EPA oversight over the full use of an antimicrobial compound is the first step towards avoiding potential development of antimicrobial resistance in future.

Environmental and human health risks of antimicrobial pesticides should be addressed with a comprehensive health and safety assessment that would not hide behind the artificial separation of pesticide regulation between FDA and EPA. This divided responsibility for the same pesticide between separate government agencies does not serve the public interest since it allows a large percentage of products containing antimicrobial chemicals to be manufactured and used with no effective health and safety controls. The only solution that would protect the health of people and the environment is for EPA to enforce pesticide manufacturers' accountability for all uses of their products in commerce.

EPA would be able to protect public health and the environment from antimicrobials only if the Agency would consider aggregate risks and exposures. Furthermore, since there are so many uses of the same pesticide that EPA currently has no way to track, long-term downstream monitoring of people and the environment is critical.

2. Require monitoring of pesticide contamination in the environment and in people, including in cord blood and statistically representative samples from other vulnerable populations

Assessing human and environmental health risks due to antimicrobial pesticides requires both toxicity and exposure data. The proposed rule includes a solid and reliable panel of tests that would produce information about the pesticide's potential for mammalian toxicity. In contrast, the draft rule has only minimal and woefully insufficient data requirements for monitoring pesticide contamination of people and the environment. As a result, both EPA and the general public would lack information on cumulative pesticide exposures which leaves at risk

potentially vulnerable populations such as pregnant women, the developing fetus, and young children, as well as certain groups of workers who may be exposed to high levels of antimicrobial pesticides in their workplace.

While acknowledging the importance of real-life biomonitoring of human and environmental exposure to antimicrobial pesticides, the proposed rule does not actually require manufacturers to conduct these studies as a part of pesticide registration process. For some antimicrobials, such as triclosan, biomonitoring data may be availble thanks to the work of the CDC, government- and university-funded academic scientists, and non-profit organization. These studies clearly demonstrate the widespread triclosan contamination of water, soils, aquatic life and the bodies of people (Calafat 2008; Cha 2009; Coogan 2008; EWG 2008c; Halden 2005). For other pesticides, such as triclocarban, data on environmental contamination are available, but no human biomonitoring studies have been conducted as yet (Cha 2009; Coogan 2007; Coogan 2008; Sapkota 2007)}. Moreover, as new antimicrobial pesticides are developed and introduced into consumer products, the same story repeats itself, with both EPA and public health researchers constantly forced to play catch up and do detective work so as to discover which antimicrobials are becoming new, emergent contaminants and polluting bodies of people and the environment.

To address these glaring data gaps, manufacturers should be responsible for conducting biomonitoring studies on an ongoing basis. Understandably, such data would not be available for a recently discovered antimicrobial going through its first registration at EPA. However, collection of biomonitoring data should be a mandatory condition for a pesticide to remain in commerce. These data must be publicly available, allowing all concerned stakeholders – water utilities, health agencies, researchers, and parents – to make informed, data-driven decisions on risk mitigation.

In the proposed rule, monitoring of representative U.S. waters and monitoring of pesticide body burden in people in residential and occupational settings are both included as a "conditional requirement," meaning that there is less than 50% chance for biomonitoring to be required during a pesticide registration process. This weak condition creates a convenient loophole that allows manufacturers to continue using persistent polluting antimicrobial pesticides in consumer products so long as environmental contamination studies have not been conducted or made publicly available. To resolve this problem and to avoid present and future contamination, EPA should require manufactures to monitor and publicly release all data for pesticide contamination in soils, aquatic life, ambient waters, and wastewater sludge and effluent. EPA should also require mandatory monitoring of antimicrobial pesticide levels in umbilical cord blood, so as to detect any risks to the developing fetus, and in statistically representative samples from other vulnerable populations.

3. Ensure that aquatic and terrestrial ecosystems are protected from chronic pesticide toxicity

By their very design, antimicrobial pesticides are meant to kill. In most situations, these pesticides provide beneficial features such as decreasing potential spread of infections in medical, food storage and processing, and food-preparation settings; protecting equipment and building

materials from mold and rotting; controlling wood degradation; avoiding bacterial growth in public water systems, swimming pools, and aquatic areas as well as many other applications. Yet, whether used in flow-through water systems that flush high volumes of pesticides down the drain or included as preservatives in a material that slowly leaches pesticides over time, a significant portion of antimicrobial pesticides eventually ends up in wastewater plants. Following wastewater treatment, these pesticides pollute sludge, water, sediment, and soils, where they pose direct toxicity to aquatic life, and, with food and water, risks to human health as well.

EWG applauds EPA for working towards a system of tiered toxicology, exposure, and environmental fate studies that, ideally, would provide necessary information for human and ecosystem risk assessment. EWG supports the overall approach of the Agency that ranks the need for test data based on the toxicity and persistence of the antimicrobial pesticide. However, EWG is very concerned that in the proposed rule, ecological toxicity assessments ("nontarget organism data requirements") are primarily based on acute toxicity studies. Although the proposed rule considers a "conditional requirement" for studies that would provide chronic toxicity information, high-production volume pesticides that are used in consumer products would fall outside of this requirement. This would allow environmental contamination to continue and leave aquatic and terrestrial ecosystems unprotected from chronic pesticide toxicity.

In order to assure environmental safety of pesticides, EPA should require manufacturers to provide to the Agency data on both environmental exposure and chronic ecotoxicity of antimicrobial pesticides. As discussed in the previous section, EPA needs to have access to up-to-date environmental monitoring results as a key first step for protecting water, soils, and wildlife from toxic pollutants. Second, environmental risk assessment needs to consider chronic exposure of aquatic life to antimicrobial pesticides. For example, triclosan and triclocarban have been shown to accumulate in algae three orders of magnitude above the ambient levels (Coogan 2007). Triclocarban and triclosan also accumulate in snails (Coogan 2008). Bioaccumulation of triclosan metabolites in fish five orders of magnitude above the ambient levels has been reported (Balmer 2004). Triclocarban is even more persistent than triclosan and thus may pose greater chronic toxicity risks (Cha 2009; Heidler 2006; Miller 2008). Triclosan and triclocarban associate with sludge in wastewater treatment plants and with sediment in the water column. Sediment may serve as a constant-release delivery mechanism for high doses of antimicrobials, especially for bottom-dwelling organisms such as worms, mussels and clams, crabs, and some herbivorous fish species (Binelli 2009; Miller 2008). Furthermore, larvae of many aquatic organisms are filter-feeding. Thus, during the early, most sensitive stage of their life cycle, they may be exposed to levels of antimicrobial pesticides that have been associated with embryotoxicity, teratogenic developmental changes, and hatching delay (Oliveira 2009).

Due to their toxicity towards a wide spectrum of microbial and algal species, antimicrobials may disrupt critical ecological processes performed by beneficial microorganisms in nature (Dokianakis 2004; Johnson 2009; Lawrence 2009; Miller 2008; Neumegen 2005; Waller 2009). Especially worrisome are recent reports that indicate significant presence of antimicrobial pesticides in agricultural soils, due to soil application of triclosan- and triclocarban-containing sludge and biosolids from wastewater treatment plants (Cha 2009). EPA's latest Targeted

National Sewage Sludge Survey found triclosan in 92.4 % of public wastewater treatment plants tested nationwide; triclocarban was found in 100% of wastewater plants (U.S. EPA 2009). In some cases, triclocarban levels in dried wastewater sludge were as high as 441 microgram/kilogram (parts per million, ppm), while triclosan levels reached 133 ppm (U.S. EPA 2009). These findings are of great concern because triclosan and triclocarban can inhibits soil respiration and nitrification processes that are essential for preserving soil fertility (Waller 2009). They can also disrupt oxygen-producing algal communities in waterways and form toxic degradation by-products (Aranami 2007; Lawrence 2009). All of these harmful effects pose chronic toxicity risks. Acute toxicity studies alone are insufficient for addressing the damaging effects of long-term environmental contamination with antibacterial pesticides.

EPA should develop a data-based approach for comprehensive assessment and mitigation of chronic ecotoxicity risks. These studies should be driven by the real-life findings of pesticide levels in the environment and their effects on the ecosystems. Simply carrying chronic toxicity tests on laboratory organisms would not be sufficient to answer this question; instead, ecosystem health monitoring should be used as a key parameter for addressing ecological risks of antimicrobial pesticides. EPA should stand by its mandate as *Environmental* Protection Agency and ensure that chronic exposures to antimicrobial pesticides would not pose a risk to long-term survival and stability of aquatic and terrestrial ecosystems and productivity of agricultural soils.

In conclusion, EWG strongly urges EPA to strengthen the proposed data requirements for antimicrobial pesticides so as to address outstanding gaps in key areas such as cumulative human and environmental exposures, potential risks to human health, and chronic ecosystem toxicity. Future decisions on the use of antimicrobial pesticides should be based on extensive research and data collection that would provide necessary information for risk assessment and mitigation. We look forward to working with the Agency on the issues of pesticide safety for humans and the environment and we will be happy to provide our feedback and comments as EPA continues to assess toxicity data for antimicrobial pesticides. We are certain that with concerted effort and involvement of multiple stakeholders, many of the present risks of indoor pesticide use would be avoided and better health protection implemented, while preserving the use of antibacterials in the settings where they are truly needed.

With best regards,

Olga V. Naidenko, Ph.D.
Senior Scientist
Environmental Working Group

References

Ahn KC, Zhao B, Chen J, Cherednichenko G, Sanmarti E, Denison MS, et al. 2008. In vitro biologic activities of the antimicrobials triclocarban, its analogs, and triclosan in bioassay screens: receptor-based bioassay screens. Environ Health Perspect 116(9): 1203-10.

Aiello AE, Larson EL, Levy SB. 2007. Consumer antibacterial soaps: effective or just risky? Clin Infect Dis 45 Suppl 2: S137-47.
Aranami K, Readman JW. 2007. Photolytic degradation of triclosan in freshwater and seawater. Chemosphere 66(6): 1052-6.
Balmer ME, Poiger T, Droz C, Romanin K, Bergqvist PA, Muller MD, et al. 2004. Occurrence of methyl triclosan, a transformation product of the bactericide triclosan, in fish from various lakes in Switzerland. Environ Sci Technol 38(2): 390-5.
Binelli A, Cogni D, Parolini M, Riva C, Provini A. 2009. Cytotoxic and genotoxic effects of in vitro exposure to Triclosan and Trimethoprim on zebra mussel (Dreissena polymorpha) hemocytes. Comp Biochem Physiol C Toxicol Pharmacol.
Birosova L, Mikulasova M. 2009. Development of triclosan and antibiotic resistance in Salmonella enterica serovar Typhimurium. J Med Microbiol 58(Pt 4): 436-41.
Calafat AM, Ye X, Wong LY, Reidy JA, Needham LL. 2008. Urinary concentrations of triclosan in the U.S. population: 2003-2004. Environ Health Perspect 116(3): 303-7.
Caldwell KL, Jones RL, Verdon CP, Jarrett JM, Caudill SP, Osterloh JD. 2008. Levels of urinary total and speciated arsenic in the US population: National Health and Nutrition Examination Survey 2003-2004. J Expo Sci Environ Epidemiol.
CDC. 2005. Centers for Disease Control and Prevention. Third National Report on Human Exposure to Environmental Chemicals. NCEH Pub. No. 05-0570 Available: http://www.cdc.gov/exposurereport/ [accessed December 17 2008].
Cha J, Cupples AM. 2009. Detection of the antimicrobials triclocarban and triclosan in agricultural soils following land application of municipal biosolids. Water Res.
Chen J, Ahn KC, Gee NA, Ahmed MI, Duleba AJ, Zhao L, et al. 2008. Triclocarban enhances testosterone action: a new type of endocrine disruptor? Endocrinology 149(3): 1173-9.
Chu S, Metcalfe CD. 2007. Simultaneous determination of triclocarban and triclosan in municipal biosolids by liquid chromatography tandem mass spectrometry. J Chromatogr A 1164(1-2): 212-8.
Coogan MA, Edziyie RE, La Point TW, Venables BJ. 2007. Algal bioaccumulation of triclocarban, triclosan, and methyl-triclosan in a North Texas wastewater treatment plant receiving stream. Chemosphere 67(10): 1911-8.
Coogan MA, La Point TW. 2008. Snail bioaccumulation of triclocarban, triclosan, and methyltriclosan in a North Texas, USA, stream affected by wastewater treatment plant runoff. Environ Toxicol Chem 27(8): 1788-93.
Cottell A, Denyer SP, Hanlon GW, Ochs D, Maillard JY. 2009. Triclosan-tolerant bacteria: changes in susceptibility to antibiotics. J Hosp Infect.
Dokianakis SN, Kornaros ME, Lyberatos G. 2004. On the effect of pharmaceuticals on bacterial nitrite oxidation. Water Sci Technol 50(5): 341-6.
EWG. 2008a. Environmental Working Group Human Toxome Project: Triclosan. Available: http://www.ewg.org/sites/humantoxome/chemicals/chemical.php?chemid=100376 [accessed December 19 2008].

EWG. 2008b. Pesticide in Soap, Toothpaste and Breast Milk - Is It Kid-Safe? Available: http://www.ewg.org/reports/triclosan [accessed July 23 2008].
EWG. 2008c. Teen Girls' Body Burden of Hormone-Altering Cosmetics Chemicals. Available: http://www.ewg.org/reports/teens [accessed December 18 2008].
EWG. 2009. Environmental Working Group's Skin Deep cosmetic safety database. Ingredient report: triclocarban. Available: http://skindeep.ewg.org/ingredient.php?ingred06=706622 [accessed April 6 2009].
Focazio MJ, Kolpin DW, Barnes KK, Furlong ET, Meyer MT, Zaugg SD, et al. 2008. A national reconnaissance for pharmaceuticals and other organic wastewater contaminants in the United States - II) Untreated drinking water sources. Sci Total Environ 402(2-3): 201-16.
Halden RU, Paull DH. 2005. Co-occurrence of triclocarban and triclosan in U.S. water resources. Environ Sci Technol 39(6): 1420-6.
Heidler J, Sapkota A, Halden RU. 2006. Partitioning, persistence, and accumulation in digested sludge of the topical antiseptic triclocarban during wastewater treatment. Environ Sci Technol 40(11): 3634-9.
James MO, Li W, Summerlot DP, Rowland-Faux L, Wood CE. 2009. Triclosan is a potent inhibitor of estradiol and estrone sulfonation in sheep placenta. Environ Int: in press.
Johnson DR, Czechowska K, Chevre N, van der Meer JR. 2009. Toxicity of triclosan, penconazole and metalaxyl on Caulobacter crescentus and a freshwater microbial community as assessed by flow cytometry. Environ Microbiol.
Kingsbury JA, Delzer GC, Hopple JA. 2008. Anthropogenic Organic Compounds in Source Water of Nine Community Water Systems that Withdraw from Streams, 2002–05. U.S. Geological Survey Scientific Investigations Report 2008–5208. Available: http://pubs.usgs.gov/sir/2008/5208/ [accessed December 10 2008].
Kolpin DW, Furlong ET, Meyer MT, Thurman EM, Zaugg SD, Barber LB, et al. 2002. Pharmaceuticals, hormones, and other organic wastewater contaminants in U.S. streams, 1999-2000: a national reconnaissance. Environ Sci Technol 36(6): 1202-11.
Kumar V, Balomajumder C, Roy P. 2008. Disruption of LH-induced testosterone biosynthesis in testicular Leydig cells by triclosan: probable mechanism of action. Toxicology 250(2-3): 124-31.
Lawrence JR, Zhu B, Swerhone GD, Roy J, Wassenaar LI, Topp E, et al. 2009. Comparative microscale analysis of the effects of triclosan and triclocarban on the structure and function of river biofilm communities. Sci Total Environ: in press.
Levy SB. 2001. Antibacterial household products: cause for concern. Emerg Infect Dis 7(3 Suppl): 512-5.
Miller TR, Heidler J, Chillrud SN, DeLaquil A, Ritchie JC, Mihalic JN, et al. 2008. Fate of triclosan and evidence for reductive dechlorination of triclocarban in estuarine sediments. Environ Sci Technol 42(12): 4570-6.
Neumegen RA, Fernandez-Alba AR, Chisti Y. 2005. Toxicities of triclosan, phenol, and copper sulfate in activated sludge. Environ Toxicol 20(2): 160-4.
NPIRS. 2008. National Pesticide Information Retrieval System Available: http://ppis.ceris.purdue.edu/ [accessed June 13 2008].

Oliveira R, Domingues I, Koppe Grisolia C, Soares AM. 2009. Effects of triclosan on zebrafish early-life stages and adults. Environ Sci Pollut Res Int: in press.
Orvos DR, Versteeg DJ, Inauen J, Capdevielle M, Rothenstein A, Cunningham V. 2002. Aquatic toxicity of triclosan. Environ Toxicol Chem 21(7): 1338-49.
Perencevich EN, Wong MT, Harris AD. 2001. National and regional assessment of the antibacterial soap market: a step toward determining the impact of prevalent antibacterial soaps. Am J Infect Control 29(5): 281-3.
Sapkota A, Heidler J, Halden RU. 2007. Detection of triclocarban and two co-contaminating chlorocarbanilides in US aquatic environments using isotope dilution liquid chromatography tandem mass spectrometry. Environ Res 103(1): 21-9.
Tatarazako N, Ishibashi H, Teshima K, Kishi K, Arizono K. 2004. Effects of triclosan on various aquatic organisms. Environ Sci 11(2): 133-40.
U.S. EPA. 2009. Targeted National Sewage Sludge Survey: Sampling and Analysis Technical Report. Statistical Analysis Report. Available: http://www.epa.gov/waterscience/biosolids/tnsss-overview.html#results [accessed February 17, 2009].
Waller NJ, Kookana RS. 2009. Effect of triclosan on microbiological activity in Australian soils. Environ Toxicol Chem: in press.
Yang LH, Ying GG, Su HC, Stauber JL, Adams MS, Binet MT. 2008. Growth-inhibiting effects of 12 antibacterial agents and their mixtures on the freshwater microalga Pseudokirchneriella subcapitata. Environ Toxicol Chem 27(5): 1201-8.
Yazdankhah SP, Scheie AA, Hoiby EA, Lunestad BT, Heir E, Fotland TO, et al. 2006. Triclosan and antimicrobial resistance in bacteria: an overview. Microb Drug Resist 12(2): 83-90.
Young TA, Heidler J, Matos-Perez CR, Sapkota A, Toler T, Gibson KE, et al. 2008. Ab initio and in situ comparison of caffeine, triclosan, and triclocarban as indicators of sewage-derived microbes in surface waters. Environ Sci Technol 42(9): 3335-40.
Zorrilla LM, Gibson EK, Jeffay SC, Crofton KM, Setzer WR, Cooper RL, et al. 2008. The Effects of Triclosan on Puberty and Thyroid Hormones in Male Wistar Rats. Toxicol Sci: in press.