Minnesota Department of Health

Adopted as Rule: November 2015

Toxicological Summary for: Triclosan

CAS: **3380-34-5**

Synonyms: 5-Chloro-2-(2, 4-dichlorophenoxy)phenol; 2,4,4'-trichloro-2'-hydroxydiphenyl ether; 5-chloro-(2,4-dichlorophenoxy)phenol; trichloro-2'-hydroxydiphenyl ether; CH-3565; Lexol 300; Irgasan DP 300

Acute Non-Cancer Health Risk Limit ($nHRL_{Acute}$) = Not Derived (Insufficient Data)

Short-term Non-Cancer Health Risk Limit ($nHRL_{Short\text{-}term}$) = 50 µg/L

$$= \frac{\text{(Reference Dose, mg/kg/d) x (Relative Source Contribution) x (Conversion Factor)}}{\text{(Short-term intake rate, L/kg-d)}}$$

$$= \frac{\text{(0.067 mg/kg/d) x (0.2*) x (1000 µg/mg)}}{\text{(0.289 L/kg-d)}}$$

= 46 rounded to **50 µg/L**

* MDH utilizes the EPA Exposure Decision Tree (EPA 2000) to select appropriate RSCs. Given the significant potential non-water sources of exposure (EPA 2008 b.e) an RSC of 0.2 is selected.

Reference Dose/Concentration:	0.067 mg/kg-d (male Wistar rats PND 23-54)
Source of toxicity value:	MDH 2014
Point of Departure (POD):	7.23 mg/kg-d (BMDL for decreased total thyroxine (tTf) from Zorilla et al. 2009 based on a benchmark response of 20%)
Human Equivalent Dose (MDH 2011):	7.23 mg/kg-d x 0.28 = 2.0 mg/kg-d
Total uncertainty factor:	30
Uncertainty factor allocation:	3 for interspecies differences (for toxicodynamics) and 10 for intraspecies variability
Critical effect(s):	Decreased serum total thyroxine (tT4)
Co-critical effect(s):	Increased liver weights in pregnant animals, decreased fetal body weight, decreased serum estradiol, decreased tT4
Additivity endpoint(s):	Developmental; Female reproductive system (E); Hepatic (liver) system; Thyroid (E)

Subchronic Non-Cancer Health Risk Limit ($nHRL_{Subchronic}$) = $nHRL_{Short\text{-}term}$ = 50 µg/L

$$\frac{\text{(Reference Dose, mg/kg/d) x (Relative Source Contribution) x (Conversion Factor)}}{\text{(Subchronic intake rate, L/kg-d)}}$$

$$= \frac{(0.033 \text{ mg/kg/d}) \times (0.2) \times (1000 \text{ µg/mg})}{(0.077 \text{ L/kg-d})}$$

= 85.7 rounded to 90 µg/L

Reference Dose/Concentration:	0.033 mg/kg-d (CD-1 mice)
Source of toxicity value:	MDH 2014
Point of Departure (POD):	25 mg/kg-d (LOAEL,13 week study, MRID 43022605 aci EPA 2008a)
Human Equivalent Dose (MDH 2011):	25 x 0.13 = 3.3 mg/kg-d
Total uncertainty factor:	100
Uncertainty factor allocation:	3 for interspecies differences (for toxicodynamics), 10 for intraspecies variability, and 3 for extrapolating from a LOAEL to a NOAEL
Critical effect(s):	Liver enzyme changes indicative of liver damage
Co-critical effect(s):	Decreased serum tT4 levels
Additivity endpoint(s):	Hepatic (liver) system; Thyroid (E)

The Subchronic nHRL must be protective of the shorter exposure durations that occur within the subchronic period and therefore, the Subchronic nHRL is set equal to the Short-term, nHRL of 50 µg/L. Additivity Endpoints: Developmental; Female reproductive system (E); Hepatic (liver) system; Thyroid (E).

Chronic Non-Cancer Health Risk Limit ($nHRL_{Chronic}$) = $nHRL_{Short\text{-}term}$ = 50 µg/L

$$\frac{\text{(Reference Dose, mg/kg/d)} \times \text{(Relative Source Contribution)} \times \text{(Conversion Factor)}}{\text{(Chronic intake rate, L/kg-d)}}$$

$$= \frac{(0.047 \text{ mg/kg/d}) \times (0.2) \times (1000 \text{ µg/mg})}{(0.043 \text{L/kg-d})}$$

= 219 rounded to 200 µg/L

Reference Dose/Concentration:	0.047 mg/kg-d (CD-1 mice)
Source of toxicity value:	MDH 2014
Point of Departure (POD):	10 mg/kg-d (NOAEL,18 month dietary study, See 1996 aci EPA 2008a, SCCP 2009 and Rodricks et al 2010
Human Equivalent Dose (MDH 2011):	10 x 0.14 = 1.4 mg/kg-d
Total uncertainty factor:	30
Uncertainty factor allocation:	3 for interspecies differences (for toxicodynamics), 10 for intraspecies variability
Critical effect(s):	Changes in hematological (blood) parameters (e.g., decreased hemoglobin, hematocrit); hepatocellular hypertrophy, increased liver weight
Co-critical effect(s):	Changes in hematological (blood) parameters; increased incidence or severity of histological changes in the liver; decreased serum tT4 levels
Additivity endpoint(s):	Hematological (blood) system; Hepatic (liver) system; Thyroid (E)

The Chronic nHRL must be protective of the short-term, and subchronic exposures that occur within the chronic period and therefore, the Chronic nHRL is set equal to the Short-term nHRL of 50 µg/L. Addivity Endpoints: Developmental; Female reproductive system (E); Hepatic (liver) system; Thyroid (E).

Cancer Health Risk Limit (cHRL) = "Not Applicable"

Cancer Classification: "Not likely to be carcinogenic in Human"
Source: EPA 2008a
Slope factor: NA

Volatile: No

Summary of Guidance Value History:
A noncancer Health-Based Value (nHBV) of 50 µg/L was derived in 2010 for short-term, subchronic and chronic exposure durations. An Acute nHBV of 200 µg/L was also derived in 2010. The re-evaluation in 2014 incorporated more recent toxicity information and the HED methodology. An Acute value was not derived because it could not be substantiated that the effects were due to acute (< 1 day) of exposure. The re-evaluation did not result in a change to the final short-term, subchronic and chronic nHBV values which remain at 50 µg/L. The 2014 HBVs were adopted into rule as HRLs in 2015.

Summary of toxicity testing for health effects identified in the Health Standards Statute:

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Tested?	Yes	Yes	Yes	Yes	Yes
Effects?	Yes[1]	Yes[2]	Yes[3]	Yes[4]	Yes[5]

Note: Even if testing for a specific health effect was not conducted for this chemical, information about that effect might be available from studies conducted for other purposes. Most chemicals have been subject to multiple studies in which researchers identify a dose where no effects were observed, and the lowest dose that caused one or more effects. A toxicity value based on the effect observed at the lowest dose across all available studies is considered protective of all other effects that occur at higher doses.

Comments on extent of testing or effects:

[1] Dose-related decreases in serum levels of a variety of hormones (thyroxine (T4), estradiol, testosterone) have been reported. Alterations in thyroxine and estradiol levels have been identified as critical/co-critical effects and form the basis for the short-term HRL. Triclosan has also been evaluated for estrogenic activity using the sensitive utertrophic screening assay. When administered alone triclosan did not exhibit activity. When co-administered with ethinyl estradiol (E2) triclosan potentiated the estrogenic response. Using a range of E2 doses the authors demonstrated that at lower E2 doses high doses of triclosan were needed to cause potentiation. The lowest dose of E2 tested was within the range of doses women on contraceptives or hormone therapy may be exposed to. However potentiation at this E2 dose required triclosan human equivalent doses that were >70 times higher than the short-term, subchronic and chronic RfDs.

Decreases in testes weight and testosterone levels have been observed but the dose levels at which these effects have occurred has been inconsistent. Decreases in male reproductive organ weights were reported at dose levels similar in magnitude to the short-term point of departure by Kumar et al (2009). However, these observations are not consistent with other studies and there are concerns

regarding the purity of triclosan used in this study. Given these uncertainties MDH has chosen not to include the results from Kumar study in the derivation of the RfD.

Under *in vitro* conditions triclosan has exhibited antagonistic activity in both estrogen and androgen responsive bioassays.

[2] Skin sensitizing potential of triclosan has been extensively studied in multiple species, including humans, and resulted in no evidence of skin sensitization. A limited number of epidemiological studies have reported positive associations between exposure to triclosan (as measured by urinary triclosan levels) and increased allergic sensitization to inhalant and food allergens. These associations have not been consistent across studies. Study limitations include cross-sectional design, lack of clinical confirmation and exposed to multiple chemicals. In an animal model of asthma, dermal administration of triclosan did not result in airway reactivity. However, when dermally administered in conjunction with an injected allergen triclosan produced enhanced airway hyperreactivity; however this indicator of asthma in laboratory animals is inconsistent with the epidemiology studies that found no association between triclosan and asthma in humans.

The association between triclosan and allergic sensitization is difficult to explain since triclosan itself has been shown to have no sensitizing potential and little if any information is available regarding potential mechanism of triclosan in relation to allergic disease. More experimental studies are needed to determine triclosan's potential role in allergen sensitization.

[3] Decreased pup weight with accompanying developmental delays in ossification have been reported at human equivalent dose levels > 100 times higher than the short-term, subchronic or chronic RfDs.

[4] A 2 generation study has been conducted in rats. No effects on fertility indices were reported at human equivalent dose levels 500 times higher than the short-term, subchronic or chronic RfDs. The impact of triclosan exposure on puberty has been evaluated in both males and females. No effects on reproductive development were observed at human equivalent dose levels >500 times higher than the short-term, subchronic or chronic RfDs.

[5] A single 14 day neurotoxicity study has been performed. Inhibition of movement, decreased muscular tone, polydypsia and polyuria were observed at human equivalent dose levels nearly 1000 times higher than the short-term, subchronic or chronic RfDs. No change in brain weight, histological alterations or peripheral nerve changes were reported.

References:

ATSDR (Agency for Toxic Substances and Disease Registry). Minimal Risk Levels. http://www.atsdr.cdc.gov/mrls.html and Toxicological Profiles - http://www.atsdr.cdc.gov/toxpro2.html

Ahn KC, B Zhao, J Chen, G Cherednichenko, E Sanmarti, MS Denison, B Lasley, IN Pessah, D Kultz, DPY Chang, SJ Gee, BD Hammock. 2008. In Vitro Biologic Activities of the Antimicrobials Triclocarban, Its Analogs, and Triclosan in Bioassay Screens: Receptor-Based Bioassay Screens. EHP 116(9)1203-1210.

Allmyr M, J Adolfsson-Erici, MS McLachlan, G Sandborgh-Englund. 2006. Triclosan in plasma and milk from Swedish nursing mothers and their exposure via personal care products. Sci Total Env 372:87-93.

Allmyr M, F Harden, LML Toms, JF Mueller, MS McLachlan, M Adolfsson-Erici, G Sandbrogh-Englund. 2008. The influence of age and gender on triclosan concentrations in Australian human blood serum. Sci Total Env 393:162-167.

American Water Works Association (AWWA) Research Foundation 2008. Toxicological Relevance of EDCs and Pharmaceuticals in Drinking Water.

Anderson SE, J Franko, ML Kashon, KL Anderson, AF Hubbs, E Lukomska, BJ Meade. 2013. Exposure to Triclosan Auments the Allergic Response to Ovalbumin in a Mouse Model of Asthma. Tox Sco 132(1):96-106.

Australian Guidelines- Natural Resource Management Ministerial Council; Environmental Protection and Heritage Council; and National Health and Medical Research Council (2008). "Augmentation of Drinking Water Supplies." from http://www.environment.gov.au/system/files/resources/9e4c2a10-fcee-48ab-a655-c4c045a615d0/files/water-recycling-guidelines-augmentation-drinking-22.pdf .

Axelstad, Marts, Boberg, Julie, Vinggard, Anna Marie, Christiansen, Sofie, and Hass, Ulla. 2013. Triclosen exposure reduces thyroxine levels in pregnant and lactating rat dams and in directly exposed offspring. Food and Chemical Toxicology. 59: 534-540.

Aylward LL, Hays SM. 2011. Consideration of dosimetry in evaluation of ToxCast™ data. J Appl Toxicol. Nov; 31(8):741-51.

Bedoux G, B Roig, L Thomas. 2012, Occurrence and toxicity of antimicrobial triclosan and by-products in the environment. Environ Sci Pollut Res Int. May; 19(4):1044-65.

Bertelsen, RJ, Longnecker, MP, Lovik, M, Calafat, AM, Carlsen, KH, London, SJ, and Lodrup Carlsen, KC. 2013. Triclosan exposure and allergic sensitization in Norwegian children. Allergy. 68: 84-91.

Calafat A, X Ye, LY Wong, JA Reidy, LL Needham. 2008. Urinary Concentrations of Triclosan in the U.S. Population: 2003-2004. EHP 116(3)303-307.

California Environmental Protection Agency (CalEPA), OEHHA Toxicity Criteria Database. http://www.oehha.ca.gov/risk/ChemicalDB/index.asp and http://www.oehha.ca.gov/risk/pdf/cancerpotalpha81005.pdf

CalEPA 2007. Department of Pesticide Regulation, Medical Toxicology Branch. Summary of Toxicology Data 2-Chloro-2-(2,4,-Dichloro-phenoxy)Phenol. Original Date: September 10, 2003. Revised 1/25/05, 7/24/07.

California Water Resources Control Board http://www.waterboards.ca.gov/water_issues/programs/water_quality_goals/

Cherednichenko G, R Zhang, RA Bannister, V Timofeyev, N Li, EB Fritsch, W Feng, GC Barrientos, NH Schebb, BD Hammock, KG Beam, N Chiamvimonvat, IN Pessah. 2012. Triclosan impairs excitation-contraction coupling and Ca2+ dynamics in striated muscle. Proc Natl Acad Sci USA. Aug 28; 109(35):14158-63

Clayton EM, Todd M, Dowd JB, Aiello AE. 2011. The impact of bisphenol A and triclosan on immune parameters in the U.S. population, NHANES 2003-2006. Environ Health Perspect. Mar; 119(3):390-6.

Colgate-Palmolive 2008. Product Monograph. Colgate Total and Colgate Total Advanced Health.

Cosmetic Ingredient Review. 2010. Scientific Literature Review: Safety of Triclosan as a Preservative in Cosmetics. April 9, 2010. http://www.cir-safety.org/staff_files/triclo042010litreview.pdf

Crofton KM, KB Paul, MJ De Vito, JM Hedge. 2007. Short-term in vivo exposure to the water contaminant triclosan: evidence for disruption of thyroxine. Env Tox Pharm. 24:194-197.

Crofton KM. 2008. Review Article: Thyroid disrupting chemicals: mechanisms and mixtures. International Journal of Andrology 31, 209–223.

Dann AB and A Hontela. 2011. Triclosan: environmental exposure, toxicity and mechanism of action. Journal of Applied Toxicology 31:285-311.

Dayan AD. 2007. Risk Assessment of triclosan [Irgasan] in human breast milk. Food Chem Tox 45:125-129.

EPA Integrated Risk Information System (IRIS) http://www.epa.gov/iris/subst/index.html

EPA National Center for Environmental Assessment
http://cfpub.epa.gov/ncea/cfm/archive_whatsnew.cfm

EPA Office of Drinking Water http://www.epa.gov/waterscience/criteria/drinking/dwstandards.pdf

EPA 2000. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health. EPA-822-B-00-004. October 2000.

EPA 2008a. Office of Pesticide Programs. 5-Chloro-2-(2,4-dichlorophenoxy)phenol (Triclosan): Toxicology Chapter for the Reregistration Eligibility Decision (RED) Document. Case No. 2340. August 29, 2008.

EPA 2008b. Office of Pesticide Programs. Reregistration Eligibility Decision (RED) for Triclosan. Case No.2340. EPA 739-RO-8009. September 2008.

EPA 2008c. Office of Pesticide Programs. Triclosan: Report of the Cancer Assessment Review Committee (CARC). January 4, 2008.

EPA 2008d. Office of Pesticide Programs. Triclosan: Revised Report of the Hazard Identification Assessment Review Committee and Antimicrobial Division Toxicity Endpoint Committee. August 29, 2008.

EPA 2008e. Office of Pesticide Programs. Triclosan: Occupational and Residential Exposure Assessment. September 11, 2008.

EPA 2010. OPP RED Triclosan Factsheet. March 2010.
http://www.epa.gov/pesticides/reregistration/REDs/factsheets/triclosan_fs.htm

European Union Pesticides Database
http://ec.europa.eu/food/plant/protection/evaluation/database_act_subs_en.htm

Fang JL, L RL Stingley, FA Beland, W Harrouk, DL Lumpkins, P Howard. 2010. Occurrence, Efficacy, Metabolism, and Toxicity of Triclosan. J Env Sci Hlth, Part C 28:147-171.

Gee RH, A Charles, N Taylor, PD Darbre. 2007. Oestrogenic and androgenic activity of triclosan in breast cancer cells. J Appl Tox 28:78-91.

Geens T, L Roosens, H Neels, A Covaci. 2009. Assessment of human exposure to Bisphenol-A, Triclosan and Tetrabromobisphenol-A through indoor dust intake in Belgium. Chemosphere 76:755-760.

Guo LW, Q Wu, B Green, G Nolen, L Shi, J LoSurdo, H Deng, S Bauer, JL Fang, B Ning. 2012.Cytotoxicity and inhibitory effects of low-concentration triclosan on adipogenic differentiation of human mesenchymal stem cells. Toxicol Appl Pharmacol. Jul 15; 262(2):117-23.

Health Canada Existing Substances - Priority Substances Assessment Program and Screening Assessment Reports: http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/index-eng.php#existsub

Health Canada and Environment Canada. Preliminary Assessment: Triclosan (CAS Number 3380-34-5). March 2012. http://www.ec.gc.ca/ese-ees/6EF68BEC-5620-4435-8729-9B91C57A9FD2/Triclosan_EN.pdf

Honkisz E, D Zieba-Przybylska, AK Wojtowicz. 2012. The effect of triclosan on hormone secretion and viability of human choriocarcinoma JEG-3 cells. - Reprod Toxicol. Nov; 34(3):385-92.

International Agency for Research on Cancer (IARC). Agents Reviewed by the IARC http://monographs.iarc.fr/ENG/Classification/index.php

International Programme on Chemical Safety http://www.who.int/ipcs/assessment/en/

International Toxicity Estimates for Risk (ITER) http://iter.ctcnet.net/publicurl/pub_search_list.cfm

James MO, W Li, DP Summerlot, R Rowland-Faux, CE Wood. 2009. Triclosan is a potent inhibitor of estradiol and estrone sulfonation in sheep placenta. Env Int doi:10.1016/j.envint.2009.02.004

Jianlin WU, Y Hao, C Zongwei. 2009. Investigation on metabolism and pharmacokinetics of triclosan in rat plasma by using UPLC-triple quadrupole MS. Chinese Journal of Chroma 27(5)724-730.

Jung EM, An BS, Choi KC, Jeung EB. 2012. Potential estrogenic activity of triclosan in the uterus of immature rats and rat pituitary GH3 cells. Toxicol Lett. Jan 25; 208(2):142-8.

Koeppe, Erika S., Ferguson, Kelly K., Colacino, Justin A., Meeker, John D. Relationship between urinary triclosan and paraben concentrations and serum thyroid measures in NHANES 2007-2008. 2013. Science of the Total Environment. 445-446: 299-305.

Krishnan K, M Gagne, A Nong, LL Aylward, SM Hays. 2010. Biomonitoring Equivalent for Triclosan. Reg Tox Pharm doi:10.1016/j.yrtph.2010.06.004.

Kumar V, C Balomajumder, P Roy. 2008. Disruption of LH-induced testosterone biosynthesis in testicular Leydig cells by triclosan: Probable mechanism of action. Toxicology 250:124-131.

Kumar V, A Chakraborty, MR Kural, P Roy. 2009. Alteration of testicular steriodogenesis and histopathology of reproductive system in male rats treated with triclosan. Repro Tox 27:177-185.

Lan, Zhou, Kim, Tae Hyung, Bi, Kai Shun, Chen, Xiao Hui, Kim, Hyung Sik. 2013. Triclosan exhibits a tendency to accumulate in the epididymis and shows sperm toxicity in male Sprague-Dawley rats. Environ Toxicol. doi: 10.1002/tox.21897.

Lankester, Joanna, Patel, CHirag, Cullen, Mark R., Ley, Catherine, Parsonnet, Julie. 2013. Urinary triclosan is associated with elevated body mass index in NHANES. PLoS One. 8:11.

Lee HR, KA Hwand, KH Nam, HC Kim, KC Choi. 2014. Progression of Breast Cancer Cells was Enhanced by Endocrine-Disrupting Chemicals, Triclosan and Octylphenol, via an Estrogen Receptor-Dependent Signaling Pathway in Cellular and Mouse Xenograft Models. 2014. Chem Res Tox 27:834-842.

Louis GW, DR hallinger, TE Stoker. 2013. The effect of triclosan on the uterotrophic response to extended doses of ethinyl estradiol in the weanling rat. Repro Tox 36:71-77.

Minnesota Department of Health (MDH). (2011). "MDH Health Risk Assessment Methods to Incorporate Human Equivalent Dose Calculations into Derivation of Oral Reference Doses." from http://www.health.state.mn.us/divs/eh/risk/guidance/hedrefguide.pdf .

Morisseau C, O Merzlikin, A Lin, G He, W Feng, I Padilla, MS Denison, IN Pessah, BD Hammock. 2009. Toxicology in the fast lane: application of high-throughput bioassays to detect modulation of key enzymes and receptors. Env Health Perspectives 117(12)1867-1872.

National Toxicology Program http://ntp-server.niehs.nih.gov/

Oak Ridge National Laboratory. Screening Levels for Chemical Contaminants. http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/index.htm

Paul KB, JM Hedge, MJ De Vito, KM Crofton. 2010a. Developmental triclosan exposure decreases maternal and neonatal thyroxine in rats. Env Tox and Chemistry 29(12): 2840-2844.

Paul KB, JM Hedge, MJ De Vito, KM Crofton. 2010b. Short-term exposure to triclosan decreases thyroxine in vivo via upregulation of hepatic catabolism in young Long-Evans rats. Tox Sci 113(2)367-379.

Paul KB, JM Hedge, R Bansal, RT Zoeller, R Peter, MJ De Vito, KM Crofton. 2012. Developmental triclosan exposure decreases maternal, fetal and early neonatal thyroxine: A dynamic and kinetic evaluation of a putative mode-of-action. Toxicology 300(2012): 31-45.

Paul KB, JT Thompson, SO Simmons, JP Vanden Heuvel, KM Crofton. 2013. Evidence for triclosan-induced activation of human and rodent xenobiotic nuclear receptors. Toxicology In Vitro 27:2049-2060.

Paul KB, JM Hedge, DM Rotroff, MW Hornung, KM Crofton, SO Simmons. 2014. Development of a Thyroperoxidase Inhibition Assay for High-Throughput Screening. Chem Res Tox 27:387-399.

Pearce EN and LW Braverman. 2009. Environmental pollutants and the thyroid. Best Practice & Research Clinical Endocrinology & Metabolism 23:801-813.

Queckenberg C, J Meins, B Wachall, O Doroshyenko, D Tomalik-Scharte, B Bastian, M Abdel-Tawab, W Fuhr. 2010. Absorption, Pharmacokinetics and Safety of Triclosan after Dermal Administration. Antimicrobial Agents and Chemotherapy Jan. 2010, pages 570-572.

Rodericks JV, JA Swenberg, JF Borzelleca, RR Maronport, AM Shipp. 2010. Triclosan: A critical review of the experimental data and development of margins of safety for consumer products. Crit Rev Tox 40(5):422-484.

Rodriquez PEA, MS Sanchez. 2010. Maternal exposure to triclosan impairs homeostasis and female pubertal development in wistar rat offspring. J Tox Env Hlth, Part A, 73: 1678-1688.

Sandborgh-Englund G, M Adolfsson-Erici, G Odham, J Ekstrand. 2006. Pharmacokinetics of triclosan following oral ingestion in humans. J Tox Env Hlth, Part A 69:1861-1873.

Sankoda K, Matsuo H, Ito M, Nomiyama K, Arizono K, Shinohara R. 2011. Identification of triclosan intermediates produced by oxidative degradation using TiO2 in pure water and their endocrine disrupting activities. . Bull Environ Contam Toxicol. May; 86(5):470-5.

Savage JH, EC Matsui, RA Wood, CA Keet. 2012. Increased food sensitization and aeroallergen sensitization. J Allergy Clin Immunol 130:453-460.

Scientific Committee on Consumer Products (SCCP) 2009. Opinion of Triclosan. Jan 21, 2009. European Commission

Stoker TE, EK Gibson, LM Zorrilla. 2010. Triclosan exposure modulates estrogen-dependent responses in the female Wistar rat. Tox Sci. Sep; 117(1):45-53.

Syracuse Research PhysProp Database. http://www.syrres.com/esc/physdemo.htm

TOXNET search http://toxnet.nlm.nih.gov/

WHO Recommended Classification of Pesticides by Hazard. 2004. http://www.who.int/ipcs/publications/pesticides_hazard_rev_3.pdf

World Health Organization: http://www.who.int/water_sanitation_health/dwq/gdwq3rev/en/index.html (search Chapter 8 Chemical Aspects and Chapter 12 Chemical Fact Sheets for chemical name)

Zorrilla LM, EK Gibson, SC Jeffay, KM Crofton, WR Setzer, RL Cooper, TE Stoker. 2009. The effects of triclosan on puberty and thyroid hormones in male Wistar rats. Tox Sci 107(1)56-64.