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Triclosan Exposure and Allergic Sensitization in Norwegian Children

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Abstract

Background—Exposure to the synthetic antimicrobial chemical, triclosan, used in personal care products, has been hypothesized to lead to allergic disease. We investigated whether triclosan exposure was associated with allergic sensitization and symptoms in 10-year old Norwegian children.

Methods—Urinary concentrations of triclosan were measured in one first morning void from 623 children, collected 2001–2004. Logistic regression models, controlling for urine specific gravity, parental allergic disease, maternal education, and household income, were fitted for allergic sensitization (either skin prick test positivity or serum specific IgE ≥ 0.35 kU/L to at least one of 15 evaluated inhalant and food allergens), current rhinitis, and current asthma (questionnaire and exercise challenge test).

Results—The adjusted odds ratio (aOR) for allergic sensitization among those in the fourth quartile of triclosan concentration was 2.0 (95% confidence interval (CI): 1.1, 3.4) compared with the reference group (< the limit of detection) and the aOR per log₁₀ unit increase in triclosan was 1.2 (95% CI: 1.0, 1.4). The aOR for current rhinitis was 1.9 (95% CI: 1.1, 3.4) for the fourth quartile and 1.2 (95% CI: 0.97, 1.4) per log₁₀ unit increase in triclosan.

Conclusion—Triclosan concentrations were associated with allergic sensitization, especially inhalant and seasonal allergens rather than food allergens. Current rhinitis was associated with the highest levels of triclosan, whereas no association was seen for current asthma. These results are consistent with recent findings in other studies and provide additional evidence for an association between triclosan and allergy.

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Conflict of interest We confirm that no authors have any conflict of interest to disclose

Keywords

allergy; antibacterial; asthma; rhinitis; triclosan

Triclosan is a synthetic, broad-spectrum antimicrobial agent used extensively for more than 20 years to inhibit bacterial growth on skin and other surfaces. It is used in a variety of consumer products, including toothpaste, mouthwash, deodorants, soap, sportswear, toys, and plastic kitchenware (1, 2). In the European Union, about 85% of the total volume of triclosan is used in personal care products, with the balance going into textiles (5%) and plastic and food contact materials (10%) (3). In Norway, Sweden, and Denmark the main source of exposure is believed to be toothpaste (1, 4, 5). In Norway in 2001, toothpaste, for which the upper limit value of triclosan is 0.5% by weight, accounted for 74% of the total amount of triclosan applied in personal care products (5). Triclosan has been found in human urine, blood, plasma, and milk (1, 8, 9), with median plasma and urinary half-lives of 19 and 11 hours, respectively. Triclosan is completely and rapidly absorbed in the gastrointestinal tract, while a lower rate of absorption occurs dermally (7). It is excreted in urine mostly in its conjugated form (10).

Triclosan works by blocking the active site of an enzyme that is essential for fatty acid synthesis in bacteria, enoyl-acyl carrier protein reductase (ENR) (11). Because humans lack this enzyme, triclosan was for many decades considered to be safe for human health. However, this has recently been questioned (12, 13) and human exposure to triclosan has been found to be within the range where significant *in vitro* bioactivity occurs (13). Concern has also been raised over triclosan's potential for endocrine disruption, as the antimicrobial had been shown to disrupt thyroid hormone homeostasis and possibly reproductive hormones (14).

Because triclosan is effective against many, but not all strains of bacteria (3), it may alter the bacterial flora of the skin, the oral mucosa, and the intestines (14, 15). Based on the “hygiene hypothesis” of allergy in childhood, excessive use of triclosan may alter the commensal microbiota flora leading to impairment of normal maturation of the immune system (16–19). Moreover, recently an experimental study found triclosan to enhance allergic responses in a mouse model of asthma (20) and urinary triclosan levels in children and adolescents from the US National Health and Nutrition Examination Surveys (NHANES) 2005–2006 were found to be positively associated with allergic sensitization (21).

We aimed to examine urinary concentrations of triclosan in relation to allergic sensitization by skin prick test and serum specific IgE (sIgE), current rhinitis and current asthma in Norwegian children.

Methods

Design and subjects

The present study was based on the 10-year follow-up of the Environment and Childhood Asthma study, described elsewhere (22) and in the online data supplement. Of the 1019 who participated in the 10-year follow-up, funds were available to analyze urine specimens for 623, who were included in the present cross-sectional study. Subjects who had current asthma at age 10 were preferentially sampled (Bertelsen et al, in revision, 2012), yielding an asthma-enriched population (Figure S1). The study was approved by the Regional Committee for Medical and Health Research Ethics and registered in the Norwegian Biobank Registry. Written informed consent was obtained from the parents.

Urine collection and analyses

Participants collected their first morning voids at home on the same day as the follow-up visit. The urine samples were frozen at -80°C in 2 mL polypropylene tubes until shipped on dry ice to CDC (Atlanta, USA), where the total (free + conjugated) concentration of triclosan was measured by a modification of the automated online solid phase extraction high performance liquid chromatography tandem mass spectrometry method reported in Ye et al, 2005 (23). The limit of detection (LOD) for triclosan was $2.3\text{ }\mu\text{g/L}$ and for values less than this, we used all machine-observed concentrations without any substitution of concentrations below the LOD (53% of the samples) to avoid introducing bias (24, 25). The urine samples were shipped back to the Norwegian Institute of Public Health, where urine specific gravity (SG) was measured by a handheld Atago refractometer (PAL 10-S, Bellevue, WA, USA). The storage and shipping procedures are unlikely to have affected the triclosan levels in the urine samples (online supplement).

Clinical examination and outcome definitions

Allergic sensitization against perennial allergens (*Dermatophagoides pteronyssinus*, *D. farinae*, German cockroach, dog, cat, and rabbit), seasonal allergens (birch, timothy, mugwort, *Cladosporium herbarium*, and *Alternaria alternata*), and food allergens (egg-white, milk, peanut, and codfish) was determined by skin prick tests and specific IgE in serum (online data supplement).

Current rhinitis was defined by a positive response to the question: “During the last 12 months, has the child had a problem with sneezing, or runny or blocked nose when he/she did not have a cold or the flu?”

The definition of *current asthma* at 10 years was defined by a parental report of history of asthma, plus at least one of the following: reported dyspnea, chest tightness, or wheezing during the previous 12 months; reported use of asthma medication during the previous 12 months; or a positive exercise challenge test (a decrease in forced expiratory volume in one sec (FEV₁) of 10% or more compared with baseline (26)) performed 2–7 days after the first visit (22).

Statistical analyses

Logistic regression models were used to evaluate the association between triclosan and allergic sensitization (any allergen and for different allergen classes), sIgE $>0.35\text{ kU/L}$, skin prick test positivity, current rhinitis, and current asthma. Models were fitted with triclosan as categorical and continuous variables separately (additional details in online data supplement). Parental history of allergic disease, maternal education level, household income, and urine specific gravity were used as covariates in the multivariate models, as determined by a directed acyclic graph (online data supplement). All statistics were performed with Statistical Package for Social Sciences (SPSS, Version 19.0; SPSS Inc., Chigaco, IL, USA).

Results

Participant characteristics

Compared to the 396 children from the 10-year follow-up who were *not* included in the present investigation, the 623 children with urinary triclosan measurements had a lower median age, a higher prevalence of current asthma and atopic eczema, and a slightly lower prevalence of allergic sensitization, but were otherwise comparable (Table S1). The median age of the 623 children (294 girls and 329 boys) was 10.7 years (range: 8.8–12.5). Skin prick test results were available for 621 and sIgE for 604 participants (602 participants with

both test results). Allergic sensitization was found in 210 (35%) children, 161 with at least one positive skin prick test and 196 with sIgE ≥ 0.35 kU/L to at least one allergen (Table 1). Overall, 193 children were sensitized to inhalant allergens, and 82% of the children sensitized to food allergens ($n=92$) were also sensitized to inhalant allergens. Boys were more likely than girls to be sensitized (41% versus 28%, $P=0.001$) and to have allergic rhinitis (27% versus 18%, $P=0.005$). Among the children with current rhinitis and current asthma, 73% and 57% were allergically sensitized, respectively.

Triclosan

The concentration of triclosan was above the LOD (2.3 $\mu\text{g/L}$) in 47% of the 623 urine samples, with a maximum of 3610 $\mu\text{g/L}$ (three children had triclosan concentrations above 1300 $\mu\text{g/L}$) (Figure 1). Among the 210 children with allergic sensitization, 52% had detectable triclosan concentrations, compared with 44% of the 392 children without allergic sensitization, $P<0.05$ (Table 1). The frequency of detectable triclosan concentrations was not statistically significantly different between children with and without current rhinitis (50% and 46%, respectively), with or without current asthma (both 47%), or according to other child characteristics (Table 1). Urine specific gravity was higher in boys (mean: 1.024, range 1.004–1.039) than in girls (1.023, range 1.006–1.036), $P<0.001$, and also higher for children with detectable triclosan concentrations (1.025, range 1.011, 1.039) compared to the children with concentrations $\leq$ LOD (1.023, range 1.004, 1.036), $P<0.001$.

Triclosan, allergy and asthma

The adjusted odds ratio of allergic sensitization increased with quartiles of triclosan concentration compared with the reference group (Table 2 and Figure 2). The P -value for trend was 0.03, and the aOR for allergic sensitization per one unit $\log_{10}$ increase in triclosan concentration was 1.2 (95% CI: 1.0, 1.4). Similar associations were observed for skin prick test positivity and sIgE ≥ 0.35 kU/L, though results for sIgE were slightly stronger (Table 2). For the different classes of allergens, we found triclosan to be associated with sensitization to inhalant allergens, in particular inhalant and seasonal allergens, whereas no association was observed for food allergen sensitization (Table 3).

For current rhinitis, the aOR was 1.9 (95% CI: 1.1, 3.4) for the highest triclosan concentration category compared with the reference group ($P=0.02$ for trend) and the aOR for current rhinitis per $\log_{10}$ unit increase in triclosan was 1.2 (95% CI: 0.97, 1.4) (Table 2). When we examined the association between triclosan and current rhinitis in a multinomial logistic regression model, taking allergic sensitization to inhalant allergens into account, there was some evidence of a stronger association between triclosan and current rhinitis in children who were allergically sensitized compared to children who were not allergically sensitized (Table S3).

No association was observed between triclosan concentration and current asthma for all participants (Table 2 and Figure 2), or when stratified by allergic sensitization; aOR for current asthma were 1.1 (95% CI: 0.86, 1.4) and 0.86 (95% CI: 0.64, 1.2) per unit $\log_{10}$ increase in triclosan for children *with* and *without* allergic sensitization, respectively. For parental reported history of asthma ($n=209$), the aOR was 0.89 (0.76, 1.03) per $\log_{10}$ unit increase in triclosan. Although the present study was enriched with children with current asthma, further adjustment for sampling group (current asthma) did not alter the results reported for allergic sensitization and current rhinitis in Table 2 (details given in the online supplement).

Additional adjustment for sex did not change the point estimates reported in Tables 2 and 3 (results not shown). No statistically significant interactions were observed between sex and

triclosan for any of the outcomes presented in tables 2 and 3 (all $p>0.2$). By stratifying the models for triclosan by sex (Table S2), the estimates were not notably different in boys and girls.

Discussion

Urinary concentrations of the antimicrobial compound triclosan were associated with allergic sensitization, in particular to inhalant allergens, as well as with current rhinitis among the children with the highest levels of triclosan. However, we found no association between triclosan levels and current asthma at 10 years of age.

Triclosan was less commonly detected in the Norwegian (47%) compared to the 6–11 year old US children in NHANES 2003–2004 (69%) and in NHANES 2004–2005 (80%) (27). However, the 95th percentile of triclosan in the Norwegian children was 444 $\mu\text{g/L}$ compared to 148 $\mu\text{g/L}$ in the NHANES 2003–2004 children (9). The difference in the percentage of urine samples with detectable levels of triclosan between the Norwegian and U.S. children indicate possible differences in use of triclosan-containing products. Due to concern about the emergence of antibiotic-resistant bacteria, the Norwegian authorities have since year 2000 encouraged retailers and consumers to avoid routine use of products declared as antibacterial (5). Such recommendations have to our knowledge not been issued in the U.S.

The association between triclosan, allergic sensitization, and current rhinitis is in line with findings from NHANES 2003–2006, where a positive association was reported between urinary triclosan concentrations and doctor-diagnosed allergy in children (28), and with allergic sensitization for 6–18 year old participants in NHANES 2005–2006 (21). In contrast to the present study, gender modified the association between triclosan and allergic sensitization to food allergens, with a positive association seen for boys, but not for girls in NHANES 2005–2006. The association between triclosan and allergic sensitization was robust in our various sensitivity analyses. We did, however, find no association between triclosan and current asthma. Among the participants in the present study, only 57% of the children with current asthma were allergically sensitized. Nevertheless, after stratification by allergic sensitization, triclosan was not statistically significantly associated with current asthma in children with allergic sensitization, which is also in line with the results reported from NHANES 2005–2006 (21).

The link between triclosan and allergic sensitization is difficult to explain given that little information is available from experimental data on the mechanism of triclosan in relation to allergic disease outcomes. However, in a mouse model of asthma, levels of specific IgE to ovalbumin (OVA) as well lung eosinophils were increased with co-exposure to triclosan given dermally (20). Further, triclosan administration produced enhanced airway hyperreactivity to OVA as well as metacholine compared with sensitization to OVA alone. Triclosan alone did not result in enhanced airway reactivity (20). Triclosan is also known to have a very low sensitizing potential even in high-risk patients affected by eczema (29), and together this points to an adjuvant effect of triclosan. To our knowledge, there are no experimental data available for triclosan and rhinitis specifically.

The possibility of reverse causation is particularly important to address in studies assessing associations between concurrent exposures and outcomes. However, we consider it unlikely that a family history of allergic disease or allergic disease present in the child would initiate the use of antibacterial products and thus lead to the observed association between triclosan and allergy. Triclosan has been shown to help alleviate the symptoms of atopic dermatitis, presumably due to reduced levels of skin microorganisms (30). If triclosan containing products are used for treatment of atopic eczema in children, one could anticipate higher

systemic levels of triclosan caused by the impaired skin barrier. However, use of triclosan containing products has never been advocated as treatment for atopic eczema in Norway. Overall, we found no association between triclosan concentrations and current atopic eczema (results not shown), and none of the 12 children with the highest levels of triclosan (above 1000 µg/L) had current atopic eczema. In general, a very limited amount of triclosan enters the human body dermally; only 6.3% of the applied triclosan dose penetrated the human skin in *in vitro* (7). Thus, the vast majority of triclosan exposure comes from oral sources, with toothpaste believed to be one of the most important contributors. In a selection of different brands of triclosan-containing toothpaste available on the Norwegian market in 2001, the triclosan concentration ranged from 0.23 to 0.53% of product weight (5) (Figure S2). After use of triclosan containing toothpaste, triclosan is retained in the saliva for several hours (6). It is, however, unlikely that toothpaste alone is responsible for the very high levels of triclosan observed for some of the children in our study, but we have no information on specific exposure sources in our population. We consider the use of oral sources of triclosan to be unlikely to differ by history of allergic disease. Nevertheless, we cannot exclude the possibility of lifestyle as a confounding factor, but we attempted to adjust for this by controlling for maternal education and household income in our model.

In the present study the biomarker of exposure to triclosan was based on one urine sample. One urine sample was, however, found to be predictive of the mean triclosan levels for U.S. children over a 6-month period during which repeated urine samples were collected (31). The intraclass correlation coefficient (ICC) for triclosan was 0.4 and one of the highest ICCs among the 19 evaluated compounds. Nevertheless, we cannot rule out the possibility that the children in our study were already allergically sensitized before they started using triclosan-containing products. However, when we restricted our models to include only the children *without* a previous doctor-diagnosis allergy as reported in the parental interview (116 children), the association between triclosan and allergic sensitization remained with an aOR of 1.2 (1.0, 1.5) per log₁₀ unit increase in triclosan.

Although both NHANES and the present study are cross-sectional, the consistency in the association between triclosan and allergic sensitization is interesting given that these studies are based on two such different populations of children. Although the Norwegian population are enriched with asthma cases, they are also more demographic similar than the NHANES children, which are representative of the noninstitutionalized US population, and therefore includes different ethnic groups and a broader age-range. None of these studies had the possibility to explore triclosan exposure in infancy during which priming of the immune system is believed to occur. However, exposure to enhancers of allergic disease, as indicated for triclosan, is likely to be relevant also during the preteenage years, which may be an important time for the onset of allergy. Nevertheless, in the future, longitudinal studies would be useful for evaluating the temporal association between triclosan and allergy development, and more experimental studies are needed to evaluate the mechanism of triclosan in relation to allergy development.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
Box-plot of triclosan concentration distribution categories (with median values)

Figure 2. OR and 95% CI for triclosan concentration categories (above LOD) as compared to the reference category < LOD for A) allergic sensitization by SPT or sIgE B) current rhinitis and C) current asthma

Table 1

Subject and parental characteristics of 623 participants and percentage within each category with urinary triclosan concentrations equal to or above the limit of detection (LOD = 2.3 (µg/L))

Characteristics	% of n=623*	Triclosan %	LOD
SUBJECTS			
Sex			
Girls	47	47	
Boys	53	47	
BMI [†]			
< 85 th percentile	84	45	
85 th percentile	16	55	
Firstborn			
No	53	47	
Yes	47	47	
Skin prick test (SPT) positive			
No	74	45	
Yes	26	51	
sIgE 0.35 kU/L			
No	67	44 [‡]	
Yes	33	53	
Either SPT positive or sIgE 0.35 kU/L			
No	65	44 [‡]	
Yes	35	52	
History of doctor-diagnosed allergy			
No	83	49	
Yes	17	46	
Current rhinitis			
No	74	46	
Yes	26	50	
History of doctor-diagnosed rhinitis			
No	88	46	
Yes	12	50	
Current asthma			
No	79	47	
Yes	21	47	
History of asthma			
No	66	49	
Yes	34	43	
Current atopic eczema			
No	77	46	
Yes	23	49	

Characteristics	% of n=623*	Triclosan %	LOD
PARENTS			
Asthma or rhinitis at child's birth			
No	64	46	
Yes	36	49	
Maternal education, years			
12	47	51 [‡]	
13–16	31	46	
17	20	40	
Annual household income (in 1000 NOK)			
<350	13	41	
> 350–560	28	46	
> 560 – 750	30	53	
> 750	30	43	

* Information was missing for SPT, (n=2), IgE (n=19), SPT and sIgE combined (n=21), history of doctor-diagnosed allergy (n=2), current rhinitis (n=1), history of doctor-diagnosed rhinitis (n=6), current asthma (n=1) household income (n=6), maternal education (n=2).

[†] BMI age and gender adjusted percentiles

[‡] Statistically significant difference between the groups by χ^2 test for proportions ($P < 0.05$)

Table 2

Crude odds ratio (OR) and adjusted odds ratio (aOR) for allergic sensitization, current rhinitis and current asthma according to level of urinary triclosan

	n, non-cases	n, cases	Crude OR (95% CI)	P for trend*	aOR [†] (95% CI)	P for trend*
Allergic sensitization (based on either SPT or sIgE)						
Triclosan category	< 2.3 (LOD)	100	1		1	
	2.3 – 4.3	50	0.94 (0.53, 1.6)		0.96 (0.54, 1.7)	
	4.4 – 11.3	41	1.5 (0.87, 2.6)		1.5 (0.86, 2.7)	
	11.4 – 121	43	1.5 (0.86, 2.5)		1.4 (0.83, 2.5)	
	> 121	38	2.0 (1.2, 3.4)	0.02	2.0 (1.1, 3.4)	0.03
Per 1 unit log ₁₀ increase	392	210	1.2 (1.1, 1.4)		1.2 (1.0, 1.4)	
Allergic sensitization defined by SPT positivity						
Triclosan category	< 2.3 (LOD)	80	1		1	
	2.3 – 4.3	56	1.1 (0.60, 1.9)		1.1 (0.62, 2.0)	
	4.4 – 11.3	50	1.3 (0.76, 2.4)		1.4 (0.77, 2.6)	
	11.4 – 121	55	1.0 (0.58, 1.9)		0.99 (0.54, 1.8)	
	> 121	46	1.8 (1.0, 3.1)	0.05	1.7 (0.96, 3.0)	0.09
Per 1 unit log ₁₀ increase	457	164	1.2 (1.0, 1.4)		1.1 (0.97, 1.3)	
Allergic sensitization defined by sIgE 0.35 kU/L						
Triclosan category	< 2.3 (LOD)	93	1		1	
	2.3 – 4.3	52	0.91 (0.51, 1.6)		0.94 (0.52, 1.7)	
	4.4 – 11.3	42	1.6 (0.92, 2.8)		1.6 (0.91, 2.8)	
	11.4 – 121	45	1.5 (0.86, 2.6)		1.4 (0.82, 2.5)	
	> 121	38	2.3 (1.4, 3.9)	0.003	2.2 (1.3, 3.8)	0.008
Per 1 unit log ₁₀ increase	406	198	1.3 (1.1, 1.5)		1.3 (1.1, 1.5)	
Current rhinitis						
Triclosan category	< 2.3 (LOD)	72	1		1	
	2.3 – 4.3	63	0.70 (0.36, 1.4)		0.72 (0.37, 1.4)	
	4.4 – 11.3	52	1.3 (0.72, 2.4)		1.4 (0.73, 2.5)	
	11.4 – 121	56	1.1 (0.62, 2.1)		1.1 (0.57, 2.0)	
	> 121	48	1.9 (1.1, 3.3)	0.02	1.9 (1.1, 3.4)	0.02
Per 1 unit log ₁₀ increase	479	143	1.2 (0.98, 1.4)		1.2 (0.97, 1.4)	

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	n, non-cases	n, cases	Crude OR (95% CI)	P for trend*	aOR [†] (95% CI)	P for trend*
Current asthma						
Triclosan category						
< 2.3 (LOD)	264	68	1		1	
2.3 – 4.3	59	16	1.1 (0.57, 1.9)		1.1 (0.60, 2.1)	
4.4 – 11.3	56	14	0.99 (0.52, 1.9)		0.99 (0.51, 1.9)	
11.4 – 121	56	17	1.2 (0.65, 2.2)		1.1 (0.59, 2.0)	
> 121	59	13	0.87 (0.45, 1.7)	0.6	0.89 (0.45, 1.8)	0.7
Per 1 unit log ₁₀ increase	494	128	1.1 (0.88, 1.3)		1.0 (0.88, 1.3)	

* Trend-test by median category values (0.3, 3.3, 6.4, 33.7, and 358 µg/L). All triclosan concentrations are in µg/L.

† Adjusted for urine specific gravity, parental history of allergic disease, household income and maternal education level. Additional adjustment for sex did not alter the estimates reported above.

Table 3

Crude odds ratio (OR) and adjusted odds ratio (aOR) for sensitization towards any inhalant allergen, seasonal, perennial and food allergens according to level of urinary triclosan

	n, non-cases	n, cases	Crude OR (95% CI)	P for trend*	aOR [†] (95% CI)	P for trend*
Sensitization to any inhalant allergen						
Triclosan category < 2.3 (LOD)	240	92	1		1	
2.3 – 4.3	53	22	1.1 (0.63, 1.9)		1.1 (0.64, 2.0)	
4.4 – 11.3	45	26	1.6 (0.90, 2.7)		1.7 (0.94, 3.0)	
11.4 – 121	47	26	1.5 (0.86, 2.5)		1.4 (0.83, 2.5)	
> 121	40	32	2.2 (1.3, 3.7)	0.01	2.1 (1.2, 3.6)	0.02
Per 1 unit log ₁₀ increase	425	198	1.3 (1.1, 1.5)		1.2 (1.1, 1.4)	
Sensitization to seasonal allergens						
Triclosan category < 2.3 (LOD)	255	77	1		1	
2.3 – 4.3	59	16	0.92 (0.50, 1.7)		0.94 (0.50, 1.7)	
4.4 – 11.3	50	21	1.5 (0.82, 2.6)		1.6 (0.85, 2.9)	
11.4 – 121	54	19	1.2 (0.68, 2.2)		1.2 (0.65, 2.2)	
> 121	44	28	2.2 (1.3, 3.8)	0.005	2.1 (1.2, 3.7)	0.02
Per 1 unit log ₁₀ increase	462	161	1.2 (1.0, 1.4)		1.2 (1.0, 1.4)	
Sensitization to perennial allergens						
Triclosan category < 2.3 (LOD)	265	67	1		1	
2.3 – 4.3	56	19	1.4 (0.75, 2.4)		1.4 (0.77, 2.5)	
4.4 – 11.3	52	19	1.5 (0.81, 2.7)		1.6 (0.84, 2.9)	
11.4 – 121	51	22	1.7 (0.96, 3.1)		1.6 (0.89, 2.9)	
> 121	52	20	1.5 (0.86, 2.8)	0.3	1.5 (0.82, 2.8)	0.4
Per 1 unit log ₁₀ increase	476	147	1.2 (1.0, 1.4)		1.2 (1.0, 1.4)	
Sensitization to food allergens						
Triclosan category < 2.3 (LOD)	270	50	1		1	
2.3 – 4.3	64	7	0.58 (0.25, 1.4)		0.59 (0.25, 1.4)	
4.4 – 11.3	56	12	1.1 (0.56, 2.3)		1.0 (0.49, 2.1)	
11.4 – 121	63	8	0.67 (0.30, 1.5)		0.63 (0.28, 1.4)	
> 121	57	15	1.4 (0.72, 2.6)	0.2	1.3 (0.64, 2.5)	0.3

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	n, non-cases	n, cases	Crude OR (95% CI)	P for trend*	aOR† (95% CI)	P for trend*
Per 1 unit log ₁₀ increase	510	92	1.0 (0.84, 1.2)		0.99 (0.81, 1.2)	

* Trend-test by median category values (0.3, 3.3, 6.4, 33.7, and 358 µg/L). All triclosan concentrations are in µg/L.

† Adjusted for urine specific gravity, parental history of allergic disease, household income and maternal education level.