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Pediatrics 2009;124;1110-1118; originally published online Sep 14, 2009;
DOI: 10.1542/peds.2009-0032

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American Academy of Pediatrics

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Impact of Chlorinated Swimming Pool Attendance on the Respiratory Health of Adolescents



WHAT'S KNOWN ON THIS SUBJECT: Outdoor or indoor swimming pools can increase asthma risks, but their overall influence on allergic diseases has not been evaluated.



WHAT THIS STUDY ADDS: This study shows that attendance at chlorinated swimming pools exerts a strong adjuvant effect that contributes significantly to the burden of asthma, hay fever, and allergic rhinitis during adolescence.

abstract

OBJECTIVE: The goal was to estimate the burden of allergic diseases associated with chlorinated pool exposure among adolescents.

METHODS: We examined 847 students, 13 to 18 years of age, who had attended outdoor or indoor chlorinated pools at various rates. Of them, 114 had attended mainly a copper-silver pool and served as a reference group. We measured total and aeroallergen-specific immunoglobulin E (IgE) levels in serum and screened for exercise-induced bronchoconstriction. Outcomes were respiratory symptoms, hay fever, allergic rhinitis, and asthma that had been diagnosed at any time (ever asthma) or was being treated with medication and/or was associated with exercise-induced bronchoconstriction (current asthma).

RESULTS: Among adolescents with atopy with serum IgE levels of >30 kIU/L or aeroallergen-specific IgE, the odds ratios (ORs) for asthma symptoms and for ever or current asthma increased with the lifetime number of hours spent in chlorinated pools, reaching values of 7.1 to 14.9 when chlorinated pool attendance exceeded 1000 hours. Adolescents with atopy with chlorinated pool attendance of >100 hours had greater risk of hay fever (OR: 3.3-6.6), and those with attendance of >1000 hours had greater risk of allergic rhinitis (OR: 2.2-3.5). Such associations were not found among adolescents without atopy or with copper-silver pool attendance. The population attributable risks for chlorinated pool-related ever-diagnosed asthma, hay fever, and allergic rhinitis were 63.4%, 62.1%, and 35.0%, respectively.

CONCLUSION: Chlorinated pool exposure exerts an adjuvant effect on atopy that seems to contribute significantly to the burden of asthma and respiratory allergies among adolescents. *Pediatrics* 2009;124:1110-1118

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KEY WORDS

chlorine, swimming pool, childhood asthma, atopy, aeroallergens, hay fever, rhinitis

ABBREVIATIONS

CPA—chlorinated pool attendance

OR—odds ratio

PAR—population attributable risk

FEV₁—forced expiratory volume in 1 second

EIB—exercise-induced bronchoconstriction

IgE—immunoglobulin E

www.pediatrics.org/cgi/doi/10.1542/peds.2009-0032

doi:10.1542/peds.2009-0032

Accepted for publication May 28, 2009

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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

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FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

Swimming pools commonly are disinfected through water chlorination with hypochlorite, chlorine gas, or chloroisocyanurates. These chlorine-based disinfectants, loosely referred to as chlorine, release in water hypochlorous acid, a nonselective biocide that oxidizes all forms of organic matter in pool water, which has advantages but also some disadvantages. Because of its strong oxidizing potential, chlorine can inactivate a wide spectrum of waterborne pathogens, but it also reacts with organs of swimmers in contact with pool water or air, causing irritation of the skin, eyes, and upper respiratory tract. In addition, when oxidizing organic matter from swimmers or other sources, chlorine releases a mixture of harmful byproducts, including irritants such as chloramines and haloacetic acids.¹⁻⁴

Regulatory bodies and health authorities long have regarded these irritating effects of pool chlorine as a mere source of discomfort for swimmers, arguing that the dangers of chlorine, if any, must be weighed against the risks of inadequate disinfection and the health benefits of swimming.^{1,5} This way of reasoning, which has dominated swimming pool management for several decades, is being increasingly challenged by reports of health problems among swimmers.⁴ For several years, it has been known that elite swimmers have a higher prevalence of respiratory symptoms, asthma, and airway inflammation than do other athletes.⁴ It was assumed that this poorer respiratory health of swimmers was the consequence of a selection bias attributable to the lower asthmagenicity of indoor swimming, compared with other sports. Researchers increasingly acknowledge that these respiratory problems may be attributable, at least in part, to chlorine used to disinfect pool water.⁶⁻⁸

Public concern about the dangers of pool chlorine, however, was particularly aroused when it was found that indoor chlorinated pools may be detrimental to the airways of children, causing epithelial damage and increasing asthma risk.⁹⁻¹² Studies by other investigators confirmed these respiratory effects of pool chlorine¹³⁻¹⁵ and provided additional evidence that chlorinated pools may contribute to the development of allergic diseases.¹⁶

Initially, the respiratory problems of swimmers were attributed to the irritating effects of trichloramine, the gas that gives indoor pools their typical smell and can cause asthma in life-guards.^{17,18} However, the recent finding that asthma risk is increased similarly with attendance at outdoor pools demonstrates that trichloramine cannot be the main cause of respiratory effects in swimmers, because this highly volatile gas is quickly dispersed into the atmosphere when it is released outdoors.¹⁹ Substances causing asthma and breathing difficulties in swimmers must be sought among the chlorination products present in pool water or floating at the surface of the pool as aerosols or vapors. If this reasoning is correct, then it means that the respiratory impact of chlorinated pools can be assessed correctly only by taking into account total exposure to chlorinated pools, regardless of the type of pool and the conditions of attendance.

The aim of the present study was to assess for the first time the overall impact of chlorinated pool exposure on the respiratory health of adolescents, by considering the total time spent in indoor or outdoor chlorinated pools. The study took advantage of the existence in Belgium of copper-silver pools, which enabled us to identify a reference population of swimmers with no or minimal exposure to chlorination products.

METHODS

The ethics committee of the Faculty of Medicine of the Catholic University of Louvain approved the study protocol. We recruited adolescents in 3 secondary schools in the southern part of Belgium, in the cities of Louvain-la-Neuve, Bastogne, and Lessines. Participation rates were similar among the 3 schools, as well as between girls and boys (between 70.6% and 72.1%).

Students in Louvain-la-Neuve had access to an indoor pool sanitized through the copper-silver method, whereas students at the other 2 schools could visit only indoor pools disinfected with chlorine. According to the Belgian legislation, each swimming pool is required to check the microbial and chemical qualities of water regularly by measuring several parameters, including active (0.5–1.5 ppm) and combined (<2 ppm) chlorine. In 2003, the legislation was enforced through a lowering of the standard for combined chlorine (<0.8 ppm) and the setting of a standard for trichloramine levels in pool air (<500 $\mu\text{g}/\text{m}^3$ in air sampled 1.5 m above the pool surface). The concentrations of trichloramine in air ranged from 300 to 500 $\mu\text{g}/\text{m}^3$. There were occasional exceedances of active or combined chlorine levels but without attaining levels that might pose a risk to swimmers, according to international guidelines (concentrations of active and combined chlorine never exceeded 4 ppm). The water of the copper-silver pool was sanitized with concentrations of copper (0.6–1.2 mg/L) and silver (2–10 $\mu\text{g}/\text{L}$) that were lower than drinking water guidelines.²⁰

The protocol for examining students was described in detail elsewhere.¹⁹ Briefly, parents completed a questionnaire inquiring about the health of their child and risk factors for asthma and allergic diseases. The questions

about respiratory symptoms and allergic diseases were those of the International Study of Asthma and Allergy in Childhood.²¹ The questionnaire also included questions intended to estimate the total time the child had spent in indoor or outdoor chlorinated pools. Students were asked not to visit a chlorinated pool during the 48 hours preceding the study. The examination of students, which was performed in schools, included measurements of height and body weight, an interview with the adolescent about respiratory symptoms, collection of a blood sample, and screening for exercise-induced bronchoconstriction (EIB). The EIB test consisted of measuring the decrease in forced expiratory volume in 1 second (FEV₁) after 6 minutes of indoor running with submaximal effort, and results were considered positive when the exercise caused a decrease in FEV₁ of $\geq 10\%$.²² Asthma medication use was not discontinued before the test. Asthma was defined either as "ever asthma," corresponding to asthma diagnosed at any time by a physician, or as "current asthma," corresponding to physician-diagnosed asthma that, at the time of the study, was being treated with medication (bronchodilators and/or inhaled corticosteroids) and/or was associated with positive EIB test results. Because the examination of adolescents in schools precluded any allergic provocation test, we screened for allergies by measuring total and aeroallergen-specific immunoglobulin E (IgE) concentrations in serum, using the Immulite IgE kit (Diagnostic Products, Los Angeles, CA). Sensitization against specific aeroallergens was defined as serum concentrations of specific IgE of >0.35 kIU/L.

We assessed associations between outcomes and cumulative chlorinated pool attendance (CPA) stratified into 4 categories, that is, <100 hours, 100 to

500 hours, 500 to 1000 hours, or >1000 hours. Adjusted odds ratios (ORs) for the outcomes in these categories were calculated with backward logistic regression models, using as the reference level the occurrence of the outcome among adolescents with CPA of <100 hours. Backward selection started with a model including all potential control variables and each step was performed by deleting the least-significant predictor, until the model contained only variables with $P < .20$. We tested a total of 26 potential predictors, including CPA, gender, total and aeroallergen-specific IgE levels, parental asthma or allergy, and maternal smoking during pregnancy. Population attributable risks (PARs) were calculated by using the formula $P(OR - 1)/[P(OR - 1) + 1]$, where P is the prevalence of the exposure and OR is the adjusted OR attributable to the exposure. The level of statistical significance was set at $P < .05$.

RESULTS

Table 1 shows the characteristics of participants. The 2 genders were similarly represented in the 3 schools except for the school in Bastogne, which included more girls. There were no or little differences between the 3 schools with respect to age (15 years, on average), ethnicity, and indicators of respiratory health except for the prevalence of hay fever and FEV₁ values, which were higher in Bastogne and Lessines, respectively. Students in Louvain-la-Neuve had higher socioeconomic status than their peers, as reflected by parental education levels and several lifestyle factors, such as breastfeeding, exposure to tobacco smoke, and access to a backyard pool. Because they had access to an indoor copper-silver pool, students in Louvain-la-Neuve had much lower CPA levels than did those in Bastogne and Lessines. Among them, 118 had a lifetime CPA value of <100 hours.

Those students were selected to constitute the reference group.

We first assessed associations between chlorinated pools and outcomes through stratification of data into categories of increasing CPA (Table 2). The rate of sensitization to major aeroallergens did not vary with CPA, whereas the total serum IgE concentrations showed, if anything, a tendency to decrease. There also was little variation in FEV₁ values, which showed only a very modest increase (2%, on average) between the lowest and highest CPA categories. Prevalences of wheezing and EIB, although increased in all groups with CPA values of >100 hours, did not show any significant exposure-related trend. In contrast, the prevalences and odds of ever asthma, current asthma, cough, and shortness of breath increased almost linearly with the time spent in chlorinated pools. These exposure-related increases persisted when students in Bastogne and Lessines, who visited only chlorinated pools, and those in Louvain-la-Neuve, who also had access to the copper-silver pool, were examined separately (eg, P values for trend were .02 and .07 for ever asthma and .02 and .04 for current asthma, respectively).

For cough and shortness of breath, these associations even persisted when adolescents with a diagnosis of asthma were excluded (P for trend of .004 and .05, respectively). The risk of hay fever was increased in all groups with CPA values of >100 hours, whereas the risk of allergic rhinitis was increased only in the group with the highest CPA value (>1000 hours).

Risks of respiratory symptoms (Table 3) and asthma (Table 4) associated with CPA were strongly influenced by atopic status. CPA increased the risks of wheezing, cough, shortness of breath, ever asthma, and current asthma only among adolescents with

TABLE 1 Characteristics of Adolescents

Characteristics	Louvain-la-Neuve (<i>N</i> = 357)	Bastogne (<i>N</i> = 349)	Lessines (<i>N</i> = 141)	<i>P</i>
Adolescents				
Boys, <i>n</i> (%)	167 (46.8)	130 (37.2)	72 (51.1)	.006
Age, mean $\pm$ SD, y	15.4 $\pm$ 0.81	15.5 $\pm$ 0.83	15.5 $\pm$ 0.87	.54
White, <i>n</i> (%)	343 (96.1)	344 (98.6)	137 (97.2)	.13
BMI, mean $\pm$ SD, kg/m ²	20.1 $\pm$ 2.3	21.0 $\pm$ 3.3	21.0 $\pm$ 3.2	<.001
Parents				
Asthma, <i>n</i> (%)	49 (13.7)	39 (11.2)	25 (17.7)	.15
Hay fever, <i>n</i> (%)	101 (28.3)	71 (20.3)	36 (25.5)	.05
Allergic rhinitis, <i>n</i> (%)	60 (16.8)	52 (14.9)	21 (14.9)	.75
Education, <i>n</i> (%)	278 (77.9)	98 (28.1)	28 (19.9)	<.001
Early life				
Birth weight, mean $\pm$ SD, kg	3.32 $\pm$ 0.55	3.33 $\pm$ 0.58	3.25 $\pm$ 0.61	.35
Breastfeeding, <i>n</i> (%)	305 (85.4)	191 (54.7)	76 (53.9)	<.001
Child care attendance, <i>n</i> (%)	241 (67.5)	111 (31.8)	43 (30.5)	<.001
Exposure to tobacco smoke				
Active smoking, <i>n</i> (%)	20 (5.6)	14 (4.0)	16 (11.3)	.007
Smoking during pregnancy, <i>n</i> (%)	25 (7.0)	55 (15.8)	25 (17.7)	<.001
Passive smoking at home, <i>n</i> (%)	20 (5.6)	14 (4.0)	16 (11.3)	.007
Environment				
No. of older siblings, mean $\pm$ SD	0.93 $\pm$ 0.98	0.98 $\pm$ 1.02	0.85 $\pm$ 0.99	.43
Mold on bedroom walls, <i>n</i> (%)	30 (8.4)	19 (5.4)	10 (7.1)	.30
Housecleaning with bleach, <i>n</i> (%)	77 (21.6)	91 (26.1)	60 (42.6)	<.001
Living close to busy road, <i>n</i> (%)	55 (15.4)	56 (16.0)	43 (30.5)	<.001
Exposure to pets since birth, <i>n</i> (%)	51 (14.4)	47 (13.5)	17 (12.1)	.79
Swimming pool attendance				
Indoor				
<i>n</i> (%)	243 (68.1)	348 (99.7)	138 (97.9)	<.001
CPA, median (IQR), h	126 (48–286)	400 (255–657)	407 (217–724)	<.001
Outdoor				
<i>n</i> (%)	270 (75.6)	183 (52.4)	61 (43.3)	<.001
CPA, median (IQR), h	229 (70–477)	147 (57–336)	308 (134–599)	<.001
Indoor and outdoor				
<i>n</i> (%)	324 (90.8)	348 (99.7)	138 (97.9)	<.001
CPA, median (IQR), h	312 (126–649)	524 (300–895)	522 (280–1002)	<.001
Aeroallergen-specific serum IgE, <i>n</i> (%)				
Dust mite	103 (28.9)	103 (28.9)	32 (22.7)	.34
Dog	15 (4.2)	15 (4.2)	10 (7.1)	.40
Cat	49 (13.7)	49 (13.7)	14 (9.9)	.50
Pollen	65 (18.2)	65 (18.2)	19 (13.5)	.002
Mold	11 (3.1)	11 (3.1)	7 (5.0)	.14
≥ 1 aeroallergen	136 (38.1)	136 (38.1)	43 (30.5)	.19
Total serum IgE level, median (IQR), kIU/L	60.0 (22.1–173)	60.0 (22.1–173)	44.9 (13.8–147)	.004
Respiratory function and diseases				
Proportion of predicted FEV ₁ , mean $\pm$ SD, %	99.0 $\pm$ 13.9	100.9 $\pm$ 13.9	104.0 $\pm$ 15.1	.001
Positive EIB test results, <i>n</i> (%)	51 (14.3)	63 (18.0)	22 (14.6)	.39
Ever asthma, <i>n</i> (%)	38 (10.6)	36 (10.3)	14 (9.9)	.97
Current asthma, <i>n</i> (%) ^a	21 (5.9)	21 (6.0)	10 (7.1)	.87
Hay fever, <i>n</i> (%)	45 (12.6)	67 (19.2)	19 (13.5)	.04
Allergic rhinitis, <i>n</i> (%)	60 (16.8)	58 (16.6)	23 (16.3)	.99

P values indicate the level of statistical significance in $[\chi^2]$ tests (prevalences) or Kruskal-Wallis analyses of variance (mean or median values). IQR indicates interquartile range.

^a Includes 52 subjects with ever physician-diagnosed asthma who, at the time of the study, were receiving asthma medication (*n* = 37) and/or had positive EIB test results (*n* = 24).

higher total serum IgE levels and had no influence on these outcomes among the other adolescents. For the risks of ever or current asthma, the same patterns of interactions between CPA and atopy, defined as sensitization

to any aeroallergen, emerged. An interaction with the risk of EIB also emerged and was significantly increased only among sensitized adolescents. For respiratory symptoms, however, the interaction between CPA and

aeroallergen sensitization was less clear cut, with CPA increasing the risk of cough for adolescents without and with atopy and the risk of shortness of breath only for adolescents without atopy (Table 4).

TABLE 2 Total and Any Aeroallergen-Specific Serum IgE Levels, FEV₁, and Risks of Respiratory Symptoms, EIB, and Physician-Diagnosed Allergic Diseases Among Adolescents With Increasing Cumulative CPA

	CPA				<i>P</i>
	<100 h	100–500 h	500–1000 h	>1000 h	
<i>N</i>	114	369	221	143	
CPA, median (IQR), h	24 (0–56)	288 (194–378)	693 (600–825)	1483 (1165–1967)	
Boys, <i>n</i> (%)	63 (55.3)	147 (39.8)	99 (44.8)	60 (42.0)	.03
Age, mean ± SD, y	15.5 ± 0.88	15.5 ± 0.84	15.5 ± 0.85	15.5 ± 0.86	.88
Total serum IgE level, median (IQR), kIU/L	58.6 (22.8–152)	53.1 (19.8–157)	42.3 (16.1–126)	44.9 (15.4–149)	.10
Aeroallergen-specific serum IgE, <i>n</i> (%)	44 (38.6)	142 (38.5)	62 (37.1)	53 (37.0)	.90
Proportion of predicted FEV ₁ , mean ± SD, %	100.2 ± 14.7	99.7 ± 14.0	100.9 ± 14.4	103.1 ± 13.8	.11
Wheezing					
<i>n</i> (%)	9 (8.8)	46 (12.5)	24 (10.9)	23 (16.1)	.12
OR (95% CI) ^a	1.00 (1.00–1.00)	1.40 (0.65–3.00)	1.32 (0.58–2.00)	2.09 (0.90–4.84)	
Cough					
<i>n</i> (%)	11 (9.6)	62 (16.8)	32 (14.5)	39 (27.3)	<.001
OR (95% CI) ^b	1.00 (1.00–1.00)	1.39 (0.68–2.85)	1.32 (0.62–2.82)	3.00 (1.41–6.38)	
Chest tightness					
<i>n</i> (%)	3 (2.6)	16 (4.3)	7 (3.2)	10 (7.0)	.37
OR (95% CI) ^c	1.00 (1.00–1.00)	1.25 (0.34–4.52)	1.02 (0.25–4.17)	2.78 (0.72–10.7)	
Shortness of breath					
<i>n</i> (%)	4 (3.5)	23 (6.2)	17 (7.7)	16 (11.2)	.01
OR (95% CI) ^d	1.00 (1.00–1.00)	1.49 (0.49–4.55)	2.15 (0.68–6.78)	3.25 (1.01–10.5)	
Positive EIB test results					
<i>n</i> (%)	13 (11.4)	66 (17.9)	33 (14.9)	24 (16.8)	.68
OR (95% CI) ^e	1.00 (1.00–1.00)	1.61 (0.84–3.11)	1.28 (0.64–2.61)	1.56 (0.74–3.28)	
Ever asthma					
<i>n</i> (%)	6 (5.3)	36 (9.8)	23 (10.4)	23 (16.1)	.007
OR (95% CI) ^f	1.00 (1.00–1.00)	1.80 (0.71–4.57)	2.08 (0.79–5.48)	3.74 (1.40–9.93)	
Current asthma					
<i>n</i> (%)	2 (1.8)	22 (6.0)	14 (6.4)	17 (11.9)	.002
OR (95% CI) ^g	1.00 (1.00–1.00)	2.99 (0.66–13.5)	3.77 (0.81–17.5)	8.05 (1.74–37.2)	
Hay fever					
<i>n</i> (%)	8 (7.0)	63 (17.1)	35 (15.9)	25 (17.4)	.10
OR (95% CI) ^h	1.00 (1.00–1.00)	3.04 (1.34–6.87)	2.64 (1.12–6.23)	3.49 (1.42–8.57)	
Allergic rhinitis					
<i>n</i> (%)	12 (10.5)	65 (17.9)	38 (17.3)	26 (18.2)	.21
OR (95% CI) ⁱ	1.00 (1.00–1.00)	2.00 (0.99–4.03)	1.96 (0.93–4.11)	2.38 (1.08–5.22)	

P values indicate the level of statistical significance in χ^2 tests for trend. CI indicates confidence interval; IQR, interquartile range.

^a Adjusted for parental allergy, total serum IgE level, BMI, birth weight, maternal smoking during pregnancy, active smoking, pacifier use, house with double glazing, and living <100 m from a busy road.

^b Adjusted for parental asthma, gender, older siblings, parental smoking, air fresheners, house with double glazing, and living <100 m from a busy road.

^c Adjusted for gender, birth weight, pacifier use, smoking, mold on bedroom walls, and living <100 m from a busy road.

^d Adjusted for gender, maternal smoking during pregnancy, breastfeeding, smoking, air fresheners, mold on bedroom walls, and living <100 m from a busy road.

^e Adjusted for parental asthma, gender, total serum IgE level, sensitization to any aeroallergen, maternal smoking during pregnancy, older siblings, house cleaning with bleach, and mold on bedroom walls.

^f Adjusted for sensitization to any aeroallergen, parental smoking, birth weight, breastfeeding, pacifier use, and living <100 m from a busy road.

^g Adjusted for parental asthma, gender, sensitization to any aeroallergen, maternal smoking during pregnancy, and house cleaning with bleach.

^h Adjusted for parental allergy, BMI, sensitization to any aeroallergen, day care attendance, and air fresheners.

ⁱ Adjusted for parental allergy, BMI, total serum IgE level, sensitization to any aeroallergen, birth weight, and maternal smoking during pregnancy.

CPA also interacted with aeroallergen sensitization, and specifically pollen and dust mite sensitization, to increase the risks of hay fever and allergic rhinitis (Table 5). The exposure-response relationships for hay fever and allergic rhinitis were noticeably different, and they also were different from the relationships observed for asthma. Although the odds for allergic

rhinitis were increased only among sensitized adolescents with the highest CPA values (>1000 hours), the odds for hay fever were increased significantly among sensitized adolescents with CPA values of >100 hours and above this threshold tended to plateau. This nonlinear relationship suggests an early increase of hay fever risk toward a plateau that seems to be

reached when CPA exceeds 500 hours. To test this hypothesis, we further assessed the dose-response relationships by stratifying data for adolescents with CPA values of <500 hours. As shown in Fig 1, the prevalence of hay fever among these adolescents increased linearly with CPA, whether all subjects were considered or boys and girls were considered separately, and

TABLE 3 Risks of Wheezing, Cough, and Shortness of Breath With Increasing CPA for Adolescents Classified as Nonatopic or Atopic on the Basis of Total or Aeroallergen-Specific Serum IgE Levels

Symptom and CPA	Total IgE Level		Aeroallergen-Specific IgE Level	
	<30 kIU/L (N = 315)	>30 kIU/L (N = 532)	<0.35 kIU/L (N = 526)	>0.35 kIU/L (N = 321)
Wheezing				
OR (95% CI)				
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
100–500 h	0.95 (0.28–3.32)	2.00 (0.72–5.55)	1.10 (0.39–3.11)	1.95 (0.60–6.37)
500–1000 h	0.89 (0.25–3.20)	1.73 (0.57–5.24)	1.16 (0.39–3.50)	1.52 (0.93–5.37)
>1000 h	1.04 (0.27–4.10)	3.36 (1.11–10.2)	1.49 (0.48–4.65)	3.45 (0.96–12.4)
P for trend	.81	.07	.31	.21
Cough				
OR (95% CI)				
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
100–500 h	0.76 (0.27–2.16)	2.09 (0.75–5.79)	1.09 (0.48–2.48)	2.74 (0.58–13.0)
500–1000 h	0.49 (0.16–1.52)	2.89 (0.97–8.18)	0.90 (0.37–2.20)	3.61 (0.73–18.0)
>1000 h	2.19 (0.74–6.50)	3.89 (1.31–11.6)	2.40 (1.00–5.78)	5.65 (1.12–28.6)
P for trend	.07	.008	.03	.02
Shortness of breath				
OR (95% CI)				
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
100–500 h	0.86 (0.19–3.86)	3.38 (0.42–27.0)	0.90 (0.23–3.55)	3.32 (0.39–28.4)
500–1000 h	1.03 (0.21–4.99)	5.25 (0.64–43.3)	1.34 (0.33–5.49)	4.85 (0.55–43.0)
>1000 h	1.10 (0.21–5.63)	9.58 (1.16–79.3)	2.15 (0.52–8.93)	6.03 (0.64–58.8)
P for trend	.65	.005	.04	.13

Numbers in the 4 CPA categories were as follows: IgE level of <30 kIU/L, 35, 128, 95, and 57; IgE level of >30 kIU/L, 79, 241, 126, and 86; level of IgE against any aeroallergen of <0.35 kIU/L, 70, 227, 139, and 90; level of IgE against any aeroallergen of >0.35 kIU/L, 43, 142, 82, and 53. ORs were adjusted for the same predictors as in Table 2. P values indicate the level of statistical significance in χ^2 tests for trend. CI indicates confidence interval.

TABLE 4 Risks of Ever Asthma, Current Asthma, and EIB With Increasing CPA for Adolescents Classified as Nonatopic or Atopic on the Basis of Total or Aeroallergen-Specific Serum IgE Levels

	Total IgE Level		Aeroallergen-Specific IgE Level	
	<30 kIU/L (N = 315)	>30 kIU/L (N = 532)	<0.35 kIU/L (N = 526)	>0.35 kIU/L (N = 321)
Ever asthma				
OR (95% CI)				
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
100–500 h	0.62 (0.15–2.64)	3.14 (0.88–11.2)	1.05 (0.27–3.98)	2.77 (0.76–10.1)
500–1000 h	0.81 (0.18–3.62)	3.75 (1.01–13.9)	1.27 (0.32–5.08)	2.88 (0.75–11.0)
>1000 h	0.60 (0.11–3.35)	7.82 (2.11–29.1)	1.42 (0.33–6.09)	7.11 (1.83–27.5)
P for trend	.70	.0004	.44	.003
Current asthma				
OR (95% CI)				
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
100–500 h	0.55 (0.05–6.59)	5.44 (0.69–43.0)	0.92 (0.10–8.81)	5.39 (0.67–43.1)
500–1000 h	0.39 (0.02–6.68)	8.19 (1.02–65.8)	0.79 (0.07–9.29)	7.32 (0.80–59.6)
>1000 h	1.33 (0.11–16.8)	14.9 (1.85–120)	3.02 (0.33–28.0)	12.6 (1.52–105)
P for trend	.76	.0005	.09	.006
Positive EIB test results				
OR (95% CI)				
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
100–500 h	2.32 (0.67–7.99)	1.46 (0.66–3.23)	0.95 (0.43–2.10)	4.19 (1.18–14.9)
500–1000 h	0.95 (0.25–3.52)	1.38 (0.59–3.26)	0.66 (0.27–1.60)	3.83 (1.04–14.1)
>1000 h	1.42 (0.35–5.80)	1.71 (0.70–4.20)	1.03 (0.42–2.58)	3.67 (0.93–14.6)
P for trend	.49	.24	.71	.26

Numbers in the 4 CPA categories stratified according to total serum IgE levels or aeroallergen-specific serum IgE levels were the same as in Table 3. ORs were adjusted for the same predictors as in Table 2. P values indicate the level of statistical significance in χ^2 tests for trend. CI indicates confidence interval.

even when only adolescents without parental hay fever were considered. Quite remarkably, this early increase was found only among subjects who were sensitized to aeroallergens or pollen.

The PARs for atopic diseases associated with CPA were calculated by considering as exposed the students with atopy who had spent >100 hours in chlorinated swimming pools. When atopy was defined on the basis of total serum IgE levels, these calculations yielded PARs of 63.4% for ever asthma, 79.2% for current asthma, 62.1% for hay fever, and 35.0% for allergic rhinitis. We obtained similar PAR estimates when atopy was defined as sensitization to any aeroallergen (ever asthma: 46.2%; current asthma: 67.3%; hay fever: 49.8%; allergic rhinitis: 29.6%). By comparison, the PARs for ever and current asthma attributable to maternal smoking during pregnancy, the only other lifestyle risk factor remaining in the model with P values of <.20 (P = .09 and P = .11, respectively), were 8.3% and 10.1%, respectively.

DISCUSSION

Our findings show that CPA during childhood interacts with atopic status to increase the risk of asthma, hay fever, and allergic rhinitis. Although one could evoke the possibility of reverse causation to explain the associations between asthma and indoor pool attendance on the basis that indoor swimming is recommended frequently for individuals with asthma, this explanation does not hold for associations with hay fever and allergic rhinitis or for associations with respiratory symptoms (cough and shortness of breath) observed in the absence of a diagnosis of asthma. The hypothesis of reverse causation seems even more unlikely because it implies that only individuals with atopy would be encouraged to swim, a possibility refuted by

TABLE 5 Risks of Hay Fever and Allergic Rhinitis With Increasing CPA for Adolescents Classified as Nonatopic or Atopic on the Basis of Total or Aeroallergen-Specific Serum IgE Levels

	Total IgE Level		Aeroallergen-Specific IgE Level		Pollen-Specific IgE Level		Dermatophagoides-Specific IgE Level	
	<30 kIU/L (N = 315)	>30 kIU/L (N = 532)	<0.35 kIU/L (N = 526)	>0.35 kIU/L (N = 321)	<0.35 kIU/L (N = 671)	>0.35 kIU/L (N = 176)	<0.35 kIU/L (N = 622)	>0.35 kIU/L (N = 225)
Hay fever								
OR (95% CI)								
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)		
100–500 h	0.61 (0.11–3.27)	4.50 (1.73–11.7)	1.31 (0.36–4.80)	4.68 (1.66–13.2)	2.02 (0.66–6.14)	4.13 (1.22–14.0)		
500–1000 h	0.66 (0.12–3.69)	3.72 (1.36–10.2)	1.46 (0.38–5.69)	3.27 (1.10–9.72)	1.79 (0.55–5.76)	4.38 (1.18–16.2)		
>1000 h	0.52 (0.08–3.55)	5.39 (1.89–15.4)	1.36 (0.32–5.81)	5.70 (1.82–17.9)	2.30 (0.69–7.70)	6.56 (1.61–26.9)		
P for trend	.99	.02	.65	.049	.30	.03		
Allergic rhinitis								
OR (95% CI)								
<100 h	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)			1.00 (1.00–1.00)	1.00 (1.00–1.00)
100–500 h	4.31 (0.53–35.1)	1.88 (0.87–4.03)	1.70 (0.56–5.21)	2.04 (0.83–5.04)			1.98 (0.78–5.01)	2.00 (0.69–5.78)
500–1000 h	3.52 (0.41–30.0)	2.01 (0.88–4.59)	1.63 (0.50–5.28)	1.93 (0.74–5.05)			1.48 (0.55–3.97)	2.65 (0.84–8.31)
>1000 h	3.54 (0.39–32.5)	2.22 (0.93–5.31)	1.44 (0.39–5.23)	3.54 (1.27–9.84)			1.78 (0.62–4.90)	3.46 (1.04–11.5)
P for trend	.65	.11	.84	.10			.66	.012

Numbers in the 4 CPA categories stratified according to total serum IgE or aeroallergen-specific serum IgE levels were the same as in Table 3. Numbers in the other CPA categories were as follows: pollen-specific IgE level of <0.35 kIU/L, 92, 283, 180, and 116; pollen-specific IgE level of >0.35 kIU/L, 22, 86, 41, and 27; Dermatophagoides-specific IgE level of <0.35 kIU/L, 83, 267, 169, and 103; level of IgE against any aeroallergen of >0.35 kIU/L, 31, 102, 52, and 40. ORs were adjusted for the same predictors as in Table 2. P values indicate the level of statistical significance in χ^2 tests for trend. CI indicates confidence interval.

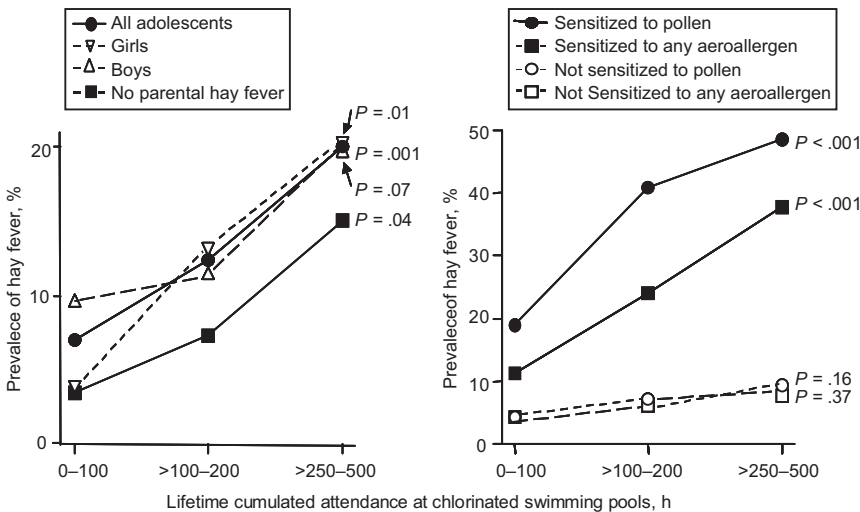


FIGURE 1 Prevalences of hay fever among adolescents according to their attendance at indoor or outdoor chlorinated swimming pools, for all subjects, for girls and boys separately, for adolescents without parental hay fever, and for adolescents sensitized or not sensitized against pollen or any aeroallergen. P values correspond to χ^2 tests for trend.

the fact that the proportion of individuals with atopy showed a tendency to decrease with lifetime pool attendance. These associations cannot be ascribed to swimming itself, because none of the studied outcomes was influenced by attendance at the copper-silver pool. The only plausible explanation is that the chlorine-based oxidants

in water or air floating at the pool surface cause some airway changes and promote the development of allergic diseases.

The present study confirms the pool chlorine-atopy interaction in asthma development that we found previously among schoolchildren who attended

indoor chlorinated pools.¹¹ Although they were based on different populations of swimmers, 2 Scandinavian studies reported observations that also suggest the existence of interactive effects of atopy and exposure to chlorinated pools. Among competitive swimmers, Helenius et al⁶ found that asthma risk was >10 times greater among elite swimmers who had atopy than among those who did not. Investigating the effects of infant swimming on the respiratory health of children, Nystad et al²³ reported that infant swimming increased the risk of wheezing among children whose parents had atopy but not among those whose parents did not. Because children of parents with atopy are more likely to have atopy themselves, this study points indirectly to an interaction between atopic status and CPA in the development of childhood asthma.

Probably the most important finding of our study is that the pool chlorine-atopy interaction described initially for asthma may extend to other common allergic diseases, such as hay fever and allergic rhinitis. Exposure-response relationships seemed to

differ according to the type of respiratory allergy, which is not surprising, given the variable deposition of inhalants along the respiratory tract. In particular, the interaction between chlorine and atopy in the risk of hay fever was triggered by much lower CPA levels, compared with asthma and allergic rhinitis. These differences in exposure-response relationships, as well as probably some differences in chlorine levels in swimming pools, might explain some conflicting results from other studies. In a retrospective analysis of data on medical history and swimming pool attendance of a cohort of adults in Germany, Kohlhammer et al¹⁶ found that early school pool attendance was associated with increased risk of hay fever but no association with asthma emerged, in contrast to our findings. We think that the explanation for this discrepancy might lie in the fact that the German cohort was composed of adults who, during their childhood, had been much less exposed to chlorinated pools than are present-day children. It is possible that, when they were children, these adults had attended chlorinated pools enough to exceed the CPA threshold for inducing hay fever but not enough to attain the CPA threshold for inducing asthma. More recently, the same German team further explored the relationship between swimming during infancy and the development of aller-

gies by analyzing 6-year follow-up data for a prospective birth cohort study.²⁴ The authors could not detect clear associations between swimming and the development of allergic diseases. The authors explained the findings on the basis of the fact that the German chlorine standards at the time those children attended pools had already been decreased to levels that were much lower (by factors of 2 and 10, respectively) than the standards in effect in the former German study²⁴ and our studies.^{10–12} Another possible explanation would be that the population studied by Schoefer et al²⁴ was still too young for detection of associations with asthma and other atopic diseases, conditions that usually are diagnosed correctly later in childhood.²⁵

Disruption of epithelial barriers increasingly seems to be a basic mechanism in allergic sensitization.²⁶ Most potent allergens, such as dust mites, cat dander, and pollen, display a proteolytic activity that allows them to open tight junctions, to cross epithelial barriers more easily, and from there to interact with immune cells.^{27–29} Chlorination products such as hypochlorous acid and chloramines are membrane-permeant oxidants that also can breach tight junctions and can facilitate the transepithelial delivery of allergens. These chemicals might thus promote allergic diseases through a mechanism similar to aller-

gens displaying proteolytic activity. Because there is no reason to think that chlorine-based oxidants would react differently with the epithelia of the upper and lower airways, we think that differences in exposure-response relationships between hay fever and asthma might reflect primarily differences in the doses of chlorination products deposited along the respiratory tract. If the risk of hay fever increases significantly with much lower CPA levels than asthma, then it might be simply because the upper airways receive most of the burden of chlorine that swimmers inhale actively at the surface of the pool.³⁰

CONCLUSIONS

Our study shows that CPA exerts a strong adjuvant effect on the development of asthma, hay fever, and allergic rhinitis. These findings reinforce the need to pursue research in this area and to enforce regulations concerning the levels of these chemicals in water and air of swimming pools.

ACKNOWLEDGMENTS

This work was supported by the National Fund for Scientific Research in Belgium, the Agency for Environmental and Occupational Health Safety in France, the governments of the Walloon Region and the French Community of Belgium, and the European Union (project INTARESE; coordinator: David Briggs).

REFERENCES

1. World Health Organization. *Guidelines for Safe Recreational Waters, Vol 2: Swimming Pools and Similar Recreational-Water Environments*. Geneva, Switzerland: World Health Organization; 2006
2. Erdinger L, Kirsch F, Sonntag HG. Irritating effects of disinfection by-products in swimming pools. *Zentralbl Hyg Umweltmed*. 1998;200(5–6):491–503
3. Kim H, Shim J, Lee S. Formation of disinfection by-products in chlorinated swimming pool water. *Chemosphere*. 2002;46(1):123–130
4. Bernard A. Chlorination products: emerging links with allergic diseases. *Curr Med Chem*. 2007;14(16):1771–1782
5. Byrne D. Chlorine in swimming pools. *Official J Eur Union*. 2001;44(11):C318E/036
6. Helenius I, Ryttilä P, Metso T, Haahtela T, Venge P, Tikkanen HO. Respiratory symptoms, bronchial responsiveness, and cellular characteristics of induced sputum in elite swimmers. *Allergy*. 1998;53(4):346–352

7. Helenius I, Hahtela T. Allergy and asthma in elite summer sport athletes. *J Allergy Clin Immunol*. 2000;106(3):444–452
8. Helenius I, Ryttilä P, Sarna S, et al. Effect of continuing or finishing high-level sports on airway inflammation, bronchial hyperresponsiveness, and asthma: a 5-year prospective follow-up study of 42 highly trained swimmers. *J Allergy Clin Immunol*. 2002;109(6):962–968
9. Carbonnelle S, Francaux M, Doyle I, et al. Changes in serum pneumoproteins caused by short-term exposures to nitrogen trichloride in indoor chlorinated swimming pools. *Biomarkers*. 2002;7(6):464–478
10. Bernard A, Carbonnelle S, Michel O, et al. Lung hyperpermeability and asthma prevalence in schoolchildren: unexpected associations with the attendance of indoor chlorinated pools. *Occup Environ Med*. 2003;60(6):385–394
11. Bernard A, Carbonnelle S, De Burbure C, Michel O, Nickmilder M. Chlorinated pool attendance, atopy and the risk of asthma during childhood. *Environ Health Perspect*. 2006;114(10):1567–1573
12. Bernard A, Carbonnelle S, Dumont X, Nickmilder M, Nickmilder M. Infant swimming, pulmonary epithelium integrity and the risk of allergic and respiratory diseases later in childhood. *Pediatrics*. 2007;119(6):1095–1103
13. Lagerkvist B, Bernard A, Blomberg A, et al. Pulmonary epithelial integrity in children: relationship to ambient ozone exposure and swimming pool attendance. *Environ Health Perspect*. 2004;112(17):1768–1771
14. Stav D, Stav M. Asthma and whirlpool baths. *N Engl J Med*. 2005;353(15):1635–1636
15. Lévesque B, Duchesne JF, Gingras S, et al. The determinants of prevalence of health complaints among young competitive swimmers. *Int Arch Occup Environ Health*. 2006;80(1):32–39
16. Kohlhammer Y, Doring A, Schafer T, Wichmann H, Heinrich J; KORA Study Group. Swimming pool attendance and hay fever rates later in life. *Allergy*. 2006;61(11):1305–1309
17. Thickett K, McCoach J, Gerber J, Sadhra S, Burge P. Occupational asthma caused by chloramines in indoor swimming-pool air. *Eur Respir J*. 2002;19(5):827–832
18. Jacobs JH, Spaan S, van Rooy GB, et al. Exposure to trichloramine and respiratory symptoms in indoor swimming pool workers. *Eur Respir J*. 2007;29(4):690–698
19. Bernard A, Nickmilder M, Voisin C. Outdoor swimming pools and the risks of asthma and allergies during adolescence. *Eur Respir J*. 2008;32(4):979–988
20. World Health Organization. *Guidelines for Drinking-Water Quality, Vol 2: Health Criteria and Supporting Information*. 2nd ed. Geneva, Switzerland: World Health Organization; 1996
21. International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. *Lancet*. 1998;351(9111):1225–1232
22. Dickinson JW, Whyte GP, McConnell AK, Nevill AM, Harries MG. Mid-expiratory flow versus FEV₁ measurements in the diagnosis of exercise induced asthma in elite athletes. *Thorax*. 2006;61(2):111–121
23. Nystad W, Håberg SE, London SJ, Nafstad P, Magnus P. Baby swimming and respiratory health. *Acta Paediatr*. 2008;97(5):657–662
24. Schoefer Y, Zutavern A, Brockow I, et al. Health risks of early swimming pool attendance. *Int J Hyg Environ Health*. 2008;211(3–4):367–373
25. Reed CE. The natural history of asthma. *J Allergy Clin Immunol*. 2006;118(3):543–548
26. Holgate ST. Epithelium dysfunction in asthma. *J Allergy Clin Immunol*. 2007;120(6):1233–1244
27. Wan H, Winton HL, Soeller C, et al. Der p 1 facilitates transepithelial allergen delivery by disruption of tight junctions. *J Clin Invest*. 1999;104(1):123–133
28. Sehgal N, Custovic A, Woodcock A. Potential roles in rhinitis for protease and other enzymatic activities of allergens. *Curr Allergy Asthma Rep*. 2005;5(3):221–226
29. Runswick S, Mitchell T, Davies P, Robinson C, Garrod DR. Pollen proteolytic enzymes degrade tight junctions. *Respirology*. 2007;12(6):834–842
30. Drobnic F, Freixa A, Casan P, Sanchis J, Guardino X. Assessment of chlorine exposure in swimmers during training. *Med Sci Sports Exerc*. 1996;28(2):271–274

Impact of Chlorinated Swimming Pool Attendance on the Respiratory Health of Adolescents

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Pediatrics 2009;124;1110-1118; originally published online Sep 14, 2009;
DOI: 10.1542/peds.2009-0032

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