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Infant Swimming Practice, Pulmonary Epithelium Integrity, and the Risk of Allergic and Respiratory Diseases Later in Childhood

Alfred Bernard, PhD, Sylviane Carbonnelle, MD, Xavier Dumont, BSc, Marc Nickmilder, PhD

Unit of Toxicology, Department of Public Health, Faculty of Medicine, Catholic University of Louvain, Brussels, Belgium

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

ABSTRACT

OBJECTIVE. Irritant gases and aerosols contaminating the air of indoor swimming pools can affect the lung epithelium and increase asthma risk in children. We evaluated the impact of infant swimming practice on allergic status and respiratory health later in childhood.

METHODS. Clara cell protein, surfactant-associated protein D, and total and aeroallergen-specific immunoglobulin E were measured in the serum of 341 schoolchildren aged 10 to 13 years, among whom 43 had followed an infant swimming program. Asthma was defined as doctor-diagnosed asthma and/or positive exercise-induced bronchoconstriction (15% decrease in postexercise forced expiratory volume).

RESULTS. There were no significant differences between the infant swimming group and the other children regarding the levels of exhaled nitric oxide and total or aeroallergen-specific serum immunoglobulin E. Children who swam as infants showed, by contrast, a significant decrease of serum Clara cell protein and of the serum Clara cell protein/surfactant-associated protein D ratio integrating Clara cell damage and permeability changes of the lung epithelial barrier. These effects were associated with higher risks of asthma and of recurrent bronchitis. Passive exposure to tobacco alone had no effect on these outcomes but seemed to interact with infant swimming practice to increase the risk of asthma or of recurrent bronchitis.

CONCLUSIONS. Our data suggest that infant swimming practice in chlorinated indoor swimming pools is associated with airways changes that, along with other factors, seem to predispose children to the development of asthma and recurrent bronchitis.

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Key Words
chlorine, trichloramine, nitrogen trichloride, baby swimming, Clara cell protein, CC16, childhood asthma, recurrent bronchitis

Abbreviations
NO—nitric oxide
eNO—exhaled nitric oxide
EIB—exercise-induced bronchoconstriction
FEV1—forced expiratory volume in 1 second
CC16—Clara cell protein
SP-D—surfactant-associated protein D
IgE—immunoglobulin E
OR—odds ratio
CI—confidence interval

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Address for correspondence to Alfred Bernard, PhD, Unit of Toxicology, Faculty of Medicine, Catholic University of Louvain, Avenue E Mounier 53, Box 53.02, B-1200 Brussels, Belgium. E-mail: alfred.bernard@uclouvain.be

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275). Copyright © 2007 by the American Academy of Pediatrics
PUBLIC SWIMMING POOLS need to be disinfected, and usually this is done by water chlorination using sodium or calcium hypochlorite, chlorine gas, or chloroisocyanurates. These chlorine-based disinfectants, loosely referred to as “chlorine,” are added in water to release hypochlorous acid, a powerful oxidant that is the active biocide. When reacting with nitrogenous compounds originating from sweat, saliva, or urine brought by swimmers, hypochlorous acid generates a complex mixture of harmful byproducts, among which the most irritant are the chloramines. Monochloramine and dichloramine (NH₂Cl and NHCl₂, respectively) are water soluble, and the sum of their concentrations in water are referred to as combined chlorine. By contrast, the trichloramine, also called nitrogen trichloride (NCl₃), is a water-insoluble gas that, once formed, is immediately released in the air, which gives indoor pools their distinctive “chlorine” smell. Depending on the bather’s hygiene and the pool ventilation, mean levels of trichloramine in community indoor pools in Europe are in the range of 300 and 800 µg/m³, which makes this gas one of the most concentrated air pollutants to which children of developed countries are regularly exposed.¹-³ The air of swimming pools, particularly just above the water’s surface, is also contaminated by mist or aerosols laden with hypochlorous acid, combined chlorine, and other water-soluble chlorination byproducts, all increasing the burden of oxidants actively inhaled by swimmers.

The acute toxicity of chlorine-based disinfectants has been known for a long time. Case reports regularly describe lung injuries after community accidents in indoor swimming pools.⁴-⁷ Inhalation of chlorine gas is usually responsible for the acute lung damage that fortunately is usually transient with recovery of the lung function within a period of a few weeks. The possibility that the gaseous and aerosolized chlorination products building up in pool air can cause chronic respiratory effects in swimmers has, however, been acknowledged only recently. Our investigations on children attending indoor chlorinated swimming pools have shown that trichloramine, together probably aerosolized hypochlorous acid and chloramines, can damage the lung epithelium⁸ and promote the development of asthma, especially in atopic children.⁹-¹¹ These effects might lie behind the strong ecological associations that we have recently evidenced across Europe between childhood asthma prevalence and the availability of indoor chlorinated swimming pool.¹² Studies by other researchers confirmed the detrimental effects of these chemicals on the airways of recreational swimmers,¹³ including asthmatics,¹⁴ while providing additional evidence that swimming pool attendance during infancy might contribute to the development of allergic diseases.¹⁵

One of the most critical factors in determining the risks of chlorination products for children seems to be the timing of exposure. The risk of developing asthma or of lung inflammation as assessed on the basis of exhaled nitric oxide (eNO) seems to culminate when children regularly attend indoor pools before the age of 7 years.⁹,¹¹ A likely explanation for this higher sensitivity of young children is that they cannot really swim before the age of 6 or 7 years and, therefore, have to attend the small heavily polluted pool. When playing or learning to swim, young children probably also inhale and swallow more aerosols and water droplets containing hypochlorous acid and soluble chloramines. Another likely explanation is that the lungs of very young children are still developing, thus they are presumably more vulnerable to the irritating effects of chloride and its derivatives.¹⁶

These findings unavoidably raise the question of the safety of infant swimming, especially because the higher water temperature and the greater organic pollution in swimming pools attended by young children are conditions favoring the formation of chlorination byproducts. In the United States, it is estimated that between 5 and 10 million infants and preschool children participate in formal swimming instructions programs, and among them there are probably several million individuals who have learned swimming as infants.¹⁷ Surprisingly, most industrialized countries have popularized this practice in the absence of reliable data concerning the possible consequences of exposing infants to the toxic gases and aerosols building up in the air of indoor pools. The only study having addressed this issue is that of Nystad et al.,¹⁸ who found that infant swimming practice was associated with an increased risk of recurrent respiratory tract infections and otitis media in the first year of life. This study, however, did not report data on the levels of chlorine in the pools attended by the infants, nor did it assess the effects of infant swimming on the development of asthma and allergy later in childhood.

In this cross-sectional study, we compared the respiratory health, allergic status, and pulmonary epithelium integrity of school-aged children who took part in an infant swimming program with that of their peers who did not. Whenever possible, objective outcome measures were used in addition to the traditional indicators of allergic and respiratory diseases assessed by questionnaire.

MATERIALS AND METHODS

Forty-three children having taken part in an infant swimming program were identified in a survey that involved a total of 341 schoolchildren 10 to 13 years of age. These children were recruited in 10 primary schools in southwestern Brussels. Levels of active and combined chlorine in the public swimming pool attended by swimming infants were within recommended limits at that time (<1.5 and 2 mg/L, respectively). Concentrations of trichloramine in pool air, available from 2001 when the systematic survey of this gas started in Brussels, ranged from 170 to 540 µg/m³ (mean: 325 µg/m³; n = 7).
Levels of trichloramine were probably in the same range some 10 years ago because operating conditions and recommended limits of chlorine had remained unchanged since the 1980s. All children were examined in their school between March 28 and May 29, 2002, thus outside main periods of pollination in Belgium. The protocol for examining children was described in detail elsewhere. Briefly, after their parents had given written informed consent, the children underwent a medical examination that included measurement of height and weight, collection of 1 blood sample (7 mL) after application of an anesthetic cream (EMLA, AstraZeneca, Karlskargo, Sweden). Asthma was screened by using the exercise-induced bronchoconstriction (EIB) test (on the basis of a 15% decrease of forced expiratory volume [FEV₁] after a 6-minute roundabout indoors with submaximal effort), a noninvasive test that has been found to be predictive of clinical asthma or asthma symptoms in several studies. The examination also included the measurement of eNO and serum Clara cell protein (CC16) and surfactant-associated protein D (SP-D) markers of the deep lung epithelium integrity. Total immunoglobulin E (IgE) and IgE against the 12 most common aeroallergens were also measured in serum (Immulite Total and AlatTOP; Diagnostic Products Corp, Los Angeles, CA). Information about the respiratory health of children (respiratory symptoms during the previous 12 months, doctor-diagnosed asthma, and recurrent bronchitis) and their exposure to risk factors of allergic diseases, maternal smoking during pregnancy, as well as of children having access to a backyard chlorinated pool, or who had been exposed to tobacco smoke during pregnancy, as well as of children having access to a backyard chlorinated pool, were, however, noticeably greater among swimming infant children. These children also had a significantly greater cumulated attendance at indoor chlorinated swimming pools.

The allergic status and the markers of lung inflammation and epithelial integrity of children who swam as infants did not differ from their peers with respect to age, gender, ethnicity, BMI, and family history of asthma or hay fever. Children in the swimming infant group were also not significantly different from their peers regarding birth weight, number of siblings, housing density, or proportions of children who were breastfed, attended day care, or lived with pets since birth. The proportions of children exposed to tobacco smoke at home or who had been exposed to tobacco smoke during pregnancy, as well as of children having access to a backyard chlorinated pool were, however, noticeably greater among swimming infant children. These children also had a significantly greater cumulated attendance at indoor chlorinated swimming pools.

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RESULTS

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ethnicity (partial r: 0.087; P = .024). Passive exposure to tobacco smoke during pregnancy or at home as well as the access to a backyard pool had thus no influence on the serum levels of CC16 or on the serum CC16/SP-D ratio. Analysis of the effects of infant swimming and passive smoking on pneumoproteins by 2-way analysis of variance confirmed the decrease of serum CC16 and CC16/SP-D ratio in children in the infant swimming group and found no interaction between infant swimming and passive smoking (all P > .15).

As shown in Table 3, changes observed in serum pneumoproteins were associated with poorer respiratory health. Children who swam as infants showed an increased risk of chest tightness. There were no statistically significant differences in the other respiratory symptoms, which tended to be more prevalent in the children in the infant swimming group. These children were also ~3 times more likely to be positive in the EIB test, to have doctor diagnosed and/or EIB test-screened asthma, and to suffer from recurrent bronchitis. Of note, the lack of statistical significance in the increased risk of doctor-diagnosed asthma with infant swimming was because of the influence of backyard pool (OR: 4.27; 95% CI: 1.05–17.4). Indeed, removal of the backyard pool factor from the list of predictors increased the OR for doctor-diagnosed asthma associated with infant swimming to a level that was significantly >1 (OR: 2.96; 95% CI: 1.08–8.11). No significant association emerged between any of these outcomes or passive exposure to tobacco smoke at home or during pregnancy.

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Characteristics of Children Who Swam as Infants and Their Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swimming Infants (N = 43)</td>
<td>Other Children (N = 298)</td>
</tr>
<tr>
<td>Age, mean (SD), yr</td>
<td>11.5 (0.6)</td>
</tr>
<tr>
<td>Boys, n (%)</td>
<td>22 (51.1)</td>
</tr>
<tr>
<td>White, n (%)</td>
<td>35 (81.4)</td>
</tr>
<tr>
<td>BMI, mean (SD), kg/m²</td>
<td>18.5 (2.7)</td>
</tr>
<tr>
<td>Mother and/or father with asthma, n (%)</td>
<td>6 (14.0)</td>
</tr>
<tr>
<td>Mother and/or father with hay fever, n (%)</td>
<td>13 (30.2)</td>
</tr>
<tr>
<td>Birth weight, mean (SD), kg</td>
<td>3.19 (0.57)</td>
</tr>
<tr>
<td>No. of siblings, mean (SD)</td>
<td>2.56 (1.30)</td>
</tr>
<tr>
<td>Housing density, mean (SD), persons per room</td>
<td>0.83 (0.36)</td>
</tr>
<tr>
<td>Breastfeeding, n (%)</td>
<td>9 (20.9)</td>
</tr>
<tr>
<td>Exposure to pets since birth, n (%)</td>
<td>9 (20.9)</td>
</tr>
<tr>
<td>Child care attendance n (%)</td>
<td>24 (55.8)</td>
</tr>
<tr>
<td>Passive smoking at home, n (%)</td>
<td>18 (41.8)</td>
</tr>
<tr>
<td>Maternal smoking during pregnancy, n (%)</td>
<td>16 (37.2)</td>
</tr>
<tr>
<td>Backyard pool, n (%)</td>
<td>6 (14.0)</td>
</tr>
<tr>
<td>Cumulated indoor pool attendance, median (interquartile range)</td>
<td>146 (88–281)</td>
</tr>
</tbody>
</table>

* By 2-sided unpaired t test.
* By χ² test.
* By 2-sided Mann-Whitney U test.

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Total and Aeroallergen-Specific Serum IgE, eNO, and Serum Pneumoproteins in Children Who Swam as Infants and Their Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swimming Infants (N = 43)</td>
<td>Other Children (N = 298)</td>
</tr>
<tr>
<td>Total IgE, median (interquartile range), kIU/L</td>
<td>54.7 (24.6–162)</td>
</tr>
<tr>
<td>Aeroallergen-specific IgE</td>
<td></td>
</tr>
<tr>
<td>Panel of 12 aeroallergens, n(%)</td>
<td>13 (30.2)</td>
</tr>
<tr>
<td>House dust mite, n(%)</td>
<td>6 (14)</td>
</tr>
<tr>
<td>Cat, n(%)</td>
<td>2 (4.7)</td>
</tr>
<tr>
<td>Dog, n(%)</td>
<td>4 (9.3)</td>
</tr>
<tr>
<td>Pollen, n(%)</td>
<td>7 (16.3)</td>
</tr>
<tr>
<td>eNO Median (interquartile range), ppb</td>
<td>10.2 (7.15–14.1)</td>
</tr>
<tr>
<td>&gt;30 ppb, n(%)</td>
<td>3 (7.0)</td>
</tr>
<tr>
<td>Pneumoproteins</td>
<td></td>
</tr>
<tr>
<td>CC16, mean (SD), µg/L</td>
<td>8.0 (3.3)</td>
</tr>
<tr>
<td>SP-D, mean (SD), µg/L</td>
<td>113 (42)</td>
</tr>
<tr>
<td>CC16/SP-D ratio, median (interquartile range)</td>
<td>0.07 (0.05–0.12)</td>
</tr>
</tbody>
</table>

* By 2-sided Mann-Whitney U test.
* By χ² test.
* By 2-sided unpaired t test.
* Statistically significant after application of the Bonferroni’s correction to the multiple comparisons of the lung epithelium markers (critical P = .05/3).
except for the risk of wheezing that was increased by maternal smoking during pregnancy (OR: 2.75; 95% CI: 1.04–6.61). Passive exposure to tobacco smoke and infant swimming seemed, however, to potentiate the detrimental effects of infant swimming practice. The OR for asthma (doctor-diagnosed and/or screened with the EIB test) associated with infant swimming was indeed >2 times greater when children were also exposed to parental smoking (OR: 6.54; 95% CI: 1.18–36.4 vs OR: 2.52; 95% CI: 0.86–7.39) although the odds for recurrent bronchitis associated with infant swimming was >4 times greater among children who had been exposed to maternal smoking during pregnancy (OR: 6.99; 95% CI: 1.60–30.5 vs OR: 1.61; 95% CI: 0.71–3.65).

To determine whether changes in serum pneumoproteins were primarily because of the infant swimming practice and not the mere consequences of asthma or recurrent bronchitis that were more frequent in the children in the swimming infant group, we assessed by a 2-way analysis of variance the effects of infant swimming with the EIB test) that parents were not aware of when filling the questionnaires. The parents were also blinded to the tested hypothesis because initially the study was not designed to look specifically at the effects of infant swimming practice. In addition, the tested hypothesis of adverse respiratory effects associated with infant swimming is far from being common in the community. Actually, this hypothesis is just the opposite of the belief that the parents probably have when they decide to take their infant to swimming pool. The possibility that they

**DISCUSSION**

Our findings show that the infant swimming practice is associated with lung epithelium alterations that seem to predispose children to the development of asthma and recurrent bronchitis. These effects cannot be explained by inherited differences in the genetic disposition to develop these diseases, because children who swim as infants were well matched with the others with respect to the family history of respiratory disease, as well as to the serum levels of total IgE. Our findings cannot be explained either by the fact that on average the children in the infant swimming group were more exposed to parental smoking or to chlorine from backyard pools. In our study, we found no evidence that infant swimming increased the risk of respiratory allergy. There was also no increase in the risk of airways inflammation as assessed by the eNO test, a finding that is not surprising given the very the close relationship between eNO and allergic sensitization observed by us and also reported by recent studies.28,29

As with any epidemiologic study using data from self-administered questionnaires, our study can be prone to recall bias. We believe, however, that it is unlikely that our observations were generated or distorted by a systematic bias in the parental responses to the questions about their child’s health or swimming practice. The strongest argument against that possibility is that the most statistically significant observations were made with objective outcome measures (serum pneumoproteins and EIB test) that parents were not aware of when filling the questionnaires. The parents were also blinded to the tested hypothesis because initially the study was not designed to look specifically at the effects of infant swimming practice. In addition, the tested hypothesis of adverse respiratory effects associated with infant swimming is far from being common in the community. Actually, this hypothesis is just the opposite of the belief that the parents probably have when they decide to take their infant to swimming pool. The possibility that they
could have biased their responses in favor of adverse effects seems thus unlikely.

Our data suggest that the poorer respiratory health of children who swim as infants could be linked to distal airways damage detected by the assay of serum pneumo-proteins. The concentration of serum CC16 is a well-validated marker of the lung epithelium barrier integrity, reflecting either the number of Clara cells lining terminal airways or the permeability of the alveolar-capillary barrier.\(^\text{26,27}\) When adjusted for the level of a surfactant-associated protein, such as SP-D, the concentration of CC16 proves to be an even more sensitive marker, probably because this ratio integrates both the damage to Clara cells and the increased leakiness of the alveolar-blood barrier.\(^\text{30}\) A decrease in serum CC16 reflecting a parallel loss of Clara cells was demonstrated in humans and in rodents acutely or chronically exposed to a variety of lung toxicants.\(^\text{31}\) For instance, active smoking leads to a 20% to 30% decrease of serum CC16, mirroring a parallel decrease in the number of Clara cells.\(^\text{30–34}\) A similar decrease of serum CC16 was observed after occupational exposures to crystalline silica\(^\text{35}\) or firesmoke.\(^\text{36}\) The decrease of serum CC16 in children who swim as infants averaged 20%. Thus, it is almost of the same magnitude as that caused in adults by tobacco smoke or industrial chemicals. If one refers to the clinical consequences of active smoking or of occupational exposures to crystalline silica and other lung irritants, it would not be surprising that similar alterations of the respiratory epithelium in young children could make them more prone to develop some respiratory diseases. This interpretation is also consistent with the antiinflammatory properties of CC16,\(^\text{37,38}\) as well as with the finding that asthma\(^\text{39}\) and other respiratory diseases\(^\text{40}\) are associated with lower intrapulmonary pools of CC16 because of Clara cell damage or the intravascular leakage of the protein.

Given the lack of data concerning the toxicity of

![Figure 1](https://www.pediatrics.org/)

**FIGURE 1**

Effects of total asthma (A), recurrent bronchitis (B), and swimming infant practice (A and B) and of their possible interactions on the serum concentrations of CC16 and SP-D and the serum CC16/SP-D ratio. Results were assessed by 2-way analysis of variance followed by Dunett’s multiple-comparison test. Mean (with SE) values that are significantly different from that of controls: \(*P < .05; \*P < .01.\)
swimming pool chemicals for the respiratory tract of infants and young children, the pool factor responsible for these airways alterations is difficult to identify. We strongly suspect, however, that the chlorination products that infants inhale as gases, aerosols, or even water repeatedly damage the airway epithelium. One culprit might be trichloramine, the highly volatile and reactive gas formed when chlorine reacts with organic matter brought by swimmers. This gas that gives indoor swimming pools their characteristic chlorine smell was, indeed, found to cause asthma and pulmonary epithelium damage in lifeguards and recreational swimmers.6–11 Because their lungs are still developing, infants could be particularly sensitive to this irritant and be affected despite the rather limited time they spend in pools (usually 20–30 minutes per session). The levels of trichloramine in the studied pool were on average below the provisional 2-hour air quality guideline of 500 μg/m³ recently recommended by the World Health Organization.12 If trichloramine is responsible for the respiratory effects observed in our study, this would mean that the World Health Organization guideline is too high and should be lowered to be more in accordance with studies6,9 showing that trichloramine can disrupt the lung epithelium barrier of swimmers at concentrations ranging from 355 to 490 μg/m³. Another important route of exposure that might cause significant damage to infant’s lungs is the inhalation of aerosols or of small volumes of chlorinated water when the infants actively play or have their head under water. Damage to the respiratory tract caused by the inhalation of heavily chlorinated water seems especially plausible because the maximum recommended levels for chlorine in the Brussels pool that our children attended when they were infants were relatively high. For instance, the recommended limit for combined chlorine was 2 ppm, a concentration twice higher than the current standard (0.8 ppm) and 10 times higher than the standard applied in Germany (0.2 ppm).13 Because infants cannot control their breathing as well as when they get older, the infant swimming practice is considered to be safe because of the laryngeal or gag reflex that is triggered when water gets into infant’s mouth. By closing off the larynx with the epiglottis, this reflex is supposed to keep pool water from entering the lungs.42 However, the gag reflex, even if very effective, cannot prevent small amounts of chlorinated water deposited or trapped in the upper respiratory tract to be conducted more deeply in the lungs when the infant surfaces to breathe. Cases of hyponatremic described after dunking infants in swimming pools43,44 attest to the amount of water that an infant can ingest and possibly inhale when being under water. Last, one cannot exclude the possibility that the inhalation of hypotonic water also causes some epithelial changes aggravating the effects of chlorination products.

Although we did not specifically interview the parents about this issue, our study provides some interesting insights into the reasons why parents take their infant to swimming programs. As one could expect, prevention of drowning seems to be an important reason because there were 5 times more children having access to a backyard pool among the infant swimming group than among the other children. Although drowning is a leading cause of unintentional injury and death in children, frequently involving backyard pools, it should be noted that the protection offered by infant swimming programs is much debated. According to the American Academy of Pediatrics, there is indeed no clearcut scientific evidence that the likelihood of drowning is reduced by the participation to such programs.17 The American Academy of Pediatrics argues that until the age of 4 years, infants are not developmentally ready for formal swimming lessons and that the participation in such programs could give to parents a false sense of security about their child’s skill in water. The American Academy of Pediatrics did not consider chemical hazards, but if, as suggested by our findings, chlorine used to disinfect pools poses some threat to infants’ health, this would certainly further justify a critical appraisal of infant swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participate to swimming infant programs. Intriguingly, the infant swimming group included a much greater proportion of infants than the children of swimming programs. There might be, however, an even more debatable reason encouraging parents to participant
group were more frequently exposed. Having access to a backyard chlorinated pool emerged, indeed, as a significant predictor of doctor-diagnosed asthma, which slightly weakened the association emerging with infant swimming. This is clearly another unexplored area that warrants additional research, especially because residential pools usually have higher levels of chlorine than public pools and offer children almost unlimited opportunities for swimming during the hot season.

CONCLUSIONS
Our study shows that the infant swimming practice in indoor chlorinated pools can be associated with airways changes that predispose children to asthma and recurrent bronchitis later in childhood. Given the increasing popularity of swimming pools, there is a definitive need to assess the effects of chlorination products on the respiratory tract of very young children. In the meantime, because in most countries these products are not as strictly regulated and monitored as indicators of microbial risks, we can only recommend caution before regularly taking infants to poorly maintained swimming pools with excessive levels of chlorine in the water and in the air.

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REFERENCES
34. Shijubo N, Itoh Y, Yamaguchi T, et al. Serum and BAL Clara cell 10 kDa protein (CC10) levels and CC10-positive bronchial cells are decreased in smokers. *Eur Respir J.* 1997;10:1108–1114
42. Freedman FB. *Water Babies.* London, United Kingdom: Lorenz Books; 2003

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**THE MOMMY TRACK**

“Why do women fall off academia’s science track at a faster clip than men? The cause is not innate sex differences, a new study suggests, but neither is it a simple matter of gender discrimination. If a problem exists, the authors conclude, it’s about motherhood, not women in general. Using the 1973–2001 Survey of Doctorate Recipients, the study found that while women are less likely than men to enter tenure-track positions in the sciences, the difference is explained completely by ‘fertility decisions.’ Single, childless women are between 11 percent (in the life sciences) and 21 percent (in the physical sciences) more likely to have a tenure-track job within five years of finishing their doctorate than single, childless men. . . . Children can hurt a woman’s chances significantly: Having a child at pre-kindergarten age took 8 percentage points off a woman’s chance of getting that tenure-track job.”

Ginther DK. *Atlantic.* March 2007
Noted by JFL, MD