

COMEAP STATEMENT ON SWIMMING POOLS AND ASTHMA.

ASTHMA AND EXPOSURE TO CHLORINE AND ASSOCIATED REACTION PRODUCTS AT SWIMMING POOLS.

Background

1. In a statement published in August 2003 Members agreed to review again publications suggesting an association between the prevalence of childhood asthma and the use of indoor swimming pools using chlorination as a disinfection technique. The underlying hypothesis of such work is that exposure to chlorination by-products leads to damage to the lung and thus increases the likelihood of development of asthma: the greater the use of such pools, the greater the likelihood of asthma developing.
2. In November 2006 Howard Gosling (Chairman, Pool Water Treatment Advisory Group), wrote to Sir William Stewart (Chairman, Health Protection Agency) asking for advice on this issue. Dr Phil Penny (Medical Adviser to the Pool Water Treatment Advisory Group) and a representative from the then Department for Education and Skills (DfES) were asked to attend the COMEAP meeting of 23 February 2007 and did so.
3. A literature search was undertaken and Members were provided with recent papers. Members examined these and Professor David Strachan and Dr Ben Armstrong (Members with special expertise in epidemiological methods and statistical techniques) commented in detail.

The evidence

4. A series of papers, largely from Professor A Bernard's Department of Public Health at Louvain, Belgium has suggested that a consistent association between the prevalence of childhood asthma and the use of indoor (chlorinated) swimming pools exists across a number of European countries. The authors argue:
 - a) that the association is explained by exposure to by-products of chlorination, such as trichloramine.
 - b) that an increase in the permeability of the alveolar blood-air barrier and damage to Clara cells, found generally in the smaller airways, underlies this effect.
 - c) that the view that Clara cells are damaged is supported by a decreased level of the Clara cell marker protein CC16 in serum.
 - d) that the view that the permeability of the alveolar barrier is increased is supported by an increased level of the surfactant-associated protein SP-D in serum.
 - e) that high serum levels of CC16 indicate acute rather than chronic damage to Clara cells.

- f) that expired air nitric oxide levels (a marker of asthmatic inflammation) are associated with pool use.
 - g) that an increase in the risk of asthma associated with swimming pool usage was seen only in those with serum IgE levels in excess of 100kIU/l.
5. Members were aware that occupational exposures to chloramines had, in certain case studies, been associated with the development of asthma and that one challenge study had demonstrated a positive airway response to chloramines at levels similar to those found in swimming pools.

Members' opinions

6. It was noted that the design of the study by Nickmilder and Bernard (2007)(1) was ecological¹ and that control for confounding at an individual level had not been attempted.
7. It was noted that GDP/capita at a country level rather than at a local level had been used as a basis for adjustment for socio-economic confounding. It was argued that adjustment for possible socio-economic confounding at a more local level would be useful.
8. Clustering of outcomes was noted and this caused some concern regarding the independent error assumption of regression analyses. Allowance for within-country clustering was suggested as possible but this seemed not to have been undertaken.
9. It was not possible to determine from the evidence available (2,3), whether the changes in CC16 and CC16/SP-D ratios reported represented the effects of recent use or continuous long-term use or use of indoor (chlorinated) swimming pools that had occurred at a much earlier time. It was suggested that an analysis that included data on how recently individuals had used such facilities be done.
10. Members were unconvinced that changes in serum CC16 levels and serum CC16/SP-D ratios were reliable biomarkers of Clara cell or epithelial cell damage², nor that they were indicative of the likelihood of the development of asthma.
11. A number of other points were raised. These may be found in the minutes on this subject placed on the COMEAP website: <http://www.advisorybodies.doh.gov.uk/comeap>.

¹ Ecological studies in this context are studies using groups as the unit of observation rather than individuals (e.g. using information based on the whole population of a country).

² As opposed to, for example, an increase in serum CC16 being explained by stimulation of Clara cell secretion of CC16.

Conclusions

12. Members agreed that the evidence considered was suggestive of an association between the use of indoor (chlorinated) swimming pools and levels of childhood asthma. Members felt that the evidence was insufficient to give strong support to the idea that the use of indoor swimming pools *causes* healthy children to become asthmatic.

13. Members agreed that the likelihood of use of indoor (chlorinated) swimming pools being a major cause of the high levels of childhood asthma in the UK was low.

14. Members agreed that levels of chlorination of indoor swimming pools should be kept as low as is consistent with adequate anti-microbial activity.

15. Members agreed that further research into the possible but unproven effects of by-products of chlorination on the induction of asthma should be strongly encouraged.

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References

1. Nickmilder M. and Bernard A. (2007) Ecological association between childhood asthma and availability of indoor chlorinated swimming pools in Europe. *Occ. Environ. Med.* **64**, 37-46.
2. Bernard A., Carbonelle S., de Barbure C., Michel, O and Nickmilder M. (2006) Chlorinated Pool Attendance, Atopy and the Risk of Asthma during Childhood. *Environ. Health Perspect.* **114**, 1567-1573.
3. Bernard A. and Nickmilder M. (2006) Respiratory Health and Baby Swimming (Letter). *Arch. Dis. Childhood* **91**, 620-621.