PostScript

LETTERS

Lung function decline in laboratory animal workers

In their recent paper Portengen and colleagues' have made an important contribution to our understanding of laboratory animal allergy. However, they have omitted to draw attention to an observation of clinical importance to occupational physicians.

They have suggested that the lack of decline in lung function in “experienced” workers may be due to the healthy worker effect. Their suggestion is not supported by their own data: the decline in lung function over two years among newly exposed workers without symptoms of LAA was not significant and surprisingly there was a significant increase in function among the symptomatic experienced workers. This being the case there seems little reason to conclude that the loss of symptomatic workers (due to a healthy worker effect) would adequately explain the absence of a decline in function. An equally valid conclusion is that the effect observed in newly exposed workers is small and may not be sustained in the long term.

Physicians are wise not to preclude sensitised workers and those with symptoms of LAA from work with animals solely on the basis of concern that this may have a deleterious effect on health. Portengen et al have provided new evidence that supports this.

I agree that the results and conclusions should be interpreted with caution and that further work is needed. However, this is a reassuring study, with important implications for current animal workers and their health providers.

R M Preece
UK Occupational Health, AstraZeneca, Mereside, Alderley Park, Macclesfield SK10 4TF, UK; richard.preece@astraZeneca.com
doi: 10.1136/oem.2003.011767

Asthma and swimming pools: statistical issues

Bernard and colleagues’ presented results from several studies investigating childhood asthma in relation to swimming pool use. Though the studies were generally well conducted, there are some respects in which the statistical analysis and interpretation are misleading.

The study of asthma prevalence in relation to swimming pool use was essentially an ecologic design—the unit of analysis was the school. Though the study of correlations between asthma prevalence and indexes of pool use respects this (the p values are appropriate), the logistic regression analysis does not. The analysis is carried out as if there were 1881 independent observations of asthma and swimming pool use. In fact, the observations are not independent—there is “clustering” of asthma by school—even after allowing for effects of swimming pool use and other covariates. The extremely low p values in fig 6 therefore cannot be relied on.

The analysis applied to the study of chronic effects on lung epithelium is also limited in not allowing for possible clustering by school. Again, the p values presented overestimate the strength of evidence for an association.

Caution is also required in interpreting the correlations and p values in figs 5C and 5D. These do not test the association of asthma with pool use, but with a composite index of pool use, pets, and passive smoking. It is not possible from the results presented to distinguish the contribution of each. The authors acknowledge this, but reader confusion may arise because the term “adjusted” is more usually used in epidemiology to describe the adjustment for confounding of one effect by another—this is not the case here.

Finally, the correlations shown in figs 5A and 5B are selected from a wider range of measures of pool attendance, as shown in fig 4. Among these non-independent indices of exposure, the authors have chosen the one showing the strongest correlation with asthma prevalence. For this reason, the “significance” of the p value in fig 5B should be interpreted as suggestive rather than definitive.

We conclude that the epidemiological evidence for an association of asthma with swimming pool use is not as strong as claimed by the authors.

B Armstrong
Public and Environmental Health Unit, London School of Hygiene and Tropical Medicine, Keppel St, London WC1E 7HT, UK; ben.armstrong@lshtm.ac.uk

D Strachan
Department of Public Health Sciences, St George’s Hospital Medical School, Cranmer Terrace, London SW17 0RE, UK

doi: 10.1136/oem.2003.011205

References
Authors’ reply

Although we appreciate the interest of Dr Armstrong and Dr Strachan for our paper on the pool chlorine/asthma risk, we cannot really take on board their reasoning concerning the statistical analyses. When questioning the strength of the associations found in our studies, they seem indeed to attribute much importance to the p values of the associations emerging between cumulated pool attendance and indicators of asthma and lung epithelium permeability. The p values, however, are not reliable indicators to judge of the strength of associations found in epidemiology since they are highly dependent on the number of observations. Our assessment of the strength of these associations was therefore based more on the values of r² and on the fact that the associations found with pool chlorine exposure were much stronger than those emerging (and a fortiori not emerging) with other variables classically presented as possible contributors to asthma and lung damage in children (for example, environmental tobacco smoke, pets, outdoor pollution).

With regard to the third study linking asthma prevalence and pool attendance, we agree of course that this is a retrospective ecological study carried out by aggregating data from each school, which was made possible by the fact that pool attendance is a compulsory activity in Belgian primary schools. However, since this study was not specifically designed to assess the effects of pool chlorine, in our opinion, its major weakness lies in this school based aggregation than in the fact that we could not quantify the cumulated pool chlorine exposure of these children on an individual basis, some of them having certainly attended a chlorinated pool with their parents (recreational, baby swimming, etc) or as part of a sport activity. This is the reason why we cautiously concluded our paper by recommending further studies to test this chlorine hypothesis. We have now just published a second paper by recommending further studies to test this chlorine hypothesis. We have now just published a second paper by recommending further studies to test this chlorine hypothesis. We have now just published a second paper by recommending further studies to test this chlorine hypothesis.

The book by Armstrong and Dr Strachan on measurement error in epidemiology and exposure assessment. Development of new techniques is dealt with in an appropriate level of detail, is certain to be useful, particularly the area of environmental exposures, chlorination by-products, pesticides, and radio frequency exposures in relation to cancer. Although we appreciate the interest of Dr Armstrong and Dr Strachan for our paper on the pool chlorine/asthma risk, we cannot really take on board their reasoning concerning the statistical analyses. When questioning the strength of the associations found in our studies, they seem indeed to attribute much importance to the p values of the associations emerging between cumulated pool attendance and indicators of asthma and lung epithelium permeability. The p values, however, are not reliable indicators to judge of the strength of associations found in epidemiology since they are highly dependent on the number of observations.

Our assessment of the strength of these associations was therefore based more on the values of r² and on the fact that the associations found with pool chlorine exposure were much stronger than those emerging (and a fortiori not emerging) with other variables classically presented as possible contributors to asthma and lung damage in children (for example, environmental tobacco smoke, pets, outdoor pollution).

With regard to the third study linking asthma prevalence and pool attendance, we agree of course that this is a retrospective ecological study carried out by aggregating data from each school, which was made possible by the fact that pool attendance is a compulsory activity in Belgian primary schools. However, since this study was not specifically designed to assess the effects of pool chlorine, in our opinion, its major weakness lies in this school based aggregation than in the fact that we could not quantify the cumulated pool chlorine exposure of these children on an individual basis, some of them having certainly attended a chlorinated pool with their parents (recreational, baby swimming, etc) or as part of a sport activity. This is the reason why we cautiously concluded our paper by recommending further studies to test this chlorine hypothesis. We have now just published a second paper by recommending further studies to test this chlorine hypothesis.

The book by Armstrong and Dr Strachan on measurement error in epidemiology and exposure assessment. Development of new techniques is dealt with in an appropriate level of detail, is certain to be useful, particularly the area of environmental exposures, chlorination by-products, pesticides, and radio frequency exposures in relation to cancer. Although we appreciate the interest of Dr Armstrong and Dr Strachan for our paper on the pool chlorine/asthma risk, we cannot really take on board their reasoning concerning the statistical analyses. When questioning the strength of the associations found in our studies, they seem indeed to attribute much importance to the p values of the associations emerging between cumulated pool attendance and indicators of asthma and lung epithelium permeability. The p values, however, are not reliable indicators to judge of the strength of associations found in epidemiology since they are highly dependent on the number of observations.

Our assessment of the strength of these associations was therefore based more on the values of r² and on the fact that the associations found with pool chlorine exposure were much stronger than those emerging (and a fortiori not emerging) with other variables classically presented as possible contributors to asthma and lung damage in children (for example, environmental tobacco smoke, pets, outdoor pollution).

With regard to the third study linking asthma prevalence and pool attendance, we agree of course that this is a retrospective ecological study carried out by aggregating data from each school, which was made possible by the fact that pool attendance is a compulsory activity in Belgian primary schools. However, since this study was not specifically designed to assess the effects of pool chlorine, in our opinion, its major weakness lies in this school based aggregation than in the fact that we could not quantify the cumulated pool chlorine exposure of these children on an individual basis, some of them having certainly attended a chlorinated pool with their parents (recreational, baby swimming, etc) or as part of a sport activity. This is the reason why we cautiously concluded our paper by recommending further studies to test this chlorine hypothesis. We have now just published a second paper by recommending further studies to test this chlorine hypothesis.

The book by Armstrong and Dr Strachan on measurement error in epidemiology and exposure assessment. Development of new techniques is dealt with in an appropriate level of detail, is certain to be useful, particularly the area of environmental exposures, chlorination by-products, pesticides, and radio frequency exposures in relation to cancer. Although we appreciate the interest of Dr Armstrong and Dr Strachan for our paper on the pool chlorine/asthma risk, we cannot really take on board their reasoning concerning the statistical analyses. When questioning the strength of the associations found in our studies, they seem indeed to attribute much importance to the p values of the associations emerging between cumulated pool attendance and indicators of asthma and lung epithelium permeability. The p values, however, are not reliable indicators to judge of the strength of associations found in epidemiology since they are highly dependent on the number of observations.

Our assessment of the strength of these associations was therefore based more on the values of r² and on the fact that the associations found with pool chlorine exposure were much stronger than those emerging (and a fortiori not emerging) with other variables classically presented as possible contributors to asthma and lung damage in children (for example, environmental tobacco smoke, pets, outdoor pollution).

With regard to the third study linking asthma prevalence and pool attendance, we agree of course that this is a retrospective ecological study carried out by aggregating data from each school, which was made possible by the fact that pool attendance is a compulsory activity in Belgian primary schools. However, since this study was not specifically designed to assess the effects of pool chlorine, in our opinion, its major weakness lies in this school based aggregation than in the fact that we could not quantify the cumulated pool chlorine exposure of these children on an individual basis, some of them having certainly attended a chlorinated pool with their parents (recreational, baby swimming, etc) or as part of a sport activity. This is the reason why we cautiously concluded our paper by recommending further studies to test this chlorine hypothesis. We have now just published a second paper by recommending further studies to test this chlorine hypothesis.