Pulmonary epithelial integrity in children
– relationship to swimming pool attendance and ambient ozone exposure

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Objective

The aim of the present project was to measure lung function and possible changes in serum levels of Clara cell protein (CC16) in relation to ambient ozone exposure and other environmental factors. CC16 is a new biomarker of early adverse effects on the airways.

Subjects and Methods

Based on a larger study in November 2001, 57 healthy children (33 boys and 24 girls) with a mean age of 10.8 ± 0.4 years, and with no history of respiratory or kidney disease were reexamined in May 2002. Subjects who reported pollen allergy and/or who had a FEV1 <80% of the predicted value in November were not invited. All lung function tests were performed by the same nurse, and blood was sampled before and after 2 hrs light outdoor exercise Ozone exposure was estimated as the total exposure measured between 7 am until the second blood sample was taken. Mean exposure dose was estimated by assuming an exposure level of 50% of the outdoor concentration during time spent indoors. Information on exposure to chlorine in pools was obtained via a questionnaire.

Results

FEV1 was significantly higher after exercise than before both in children who had regularly attended chlorinated swimming pools and in children not swimming (Table 1). The FEV1 in swimmers before and after exercise was lower than in non-swimmers, p<0.05. The mean daytime outdoor level of ozone during the study ranged from 77–116 µg/m^3, and estimated individual exposure varied from 352–914 µg/m^{3}hour. The difference (D) in CC16 was defined as afternoon value - morning value. On average these values were two hours separate, range 1.5–3.0.

Regardless of inclusion of all children or only those not attending chlorinated swimming pools, there were no consistent differences between CC16 levels in serum before (S1) and after exercise (S2) and the difference was not statistically significant. However, among children regularly swimming in chlorinated swimming pools (n=21), there was considerably less difference (S2-S1) in CC16 levels than among non– swimmers (n=33), p<0.001, t- test). The average CC16 level both before and after exercise was lower than in non-swimmers, Wilcoxon W p<0.01 (Table 2). When the relationship between ozone exposure and CC16 levels were examined, no significant correlations were found. However, when S2 was considered, a correlation coefficient of r= 0.18 was found (p=0.20) when all individuals were included. If swimmers were excluded, r was =0.32 (p=0.07).

Conclusion

This study in Swedish school children did not show a statistically significant relationship between CC16 levels in serum and ambient ozone exposure. Lower CC16 levels and less variation in CC16 values before and after exercise among children regularly attending chlorinated swimming pools may be explained by a somewhat decreased pool of CC16 in the Clara cells in the lungs of these children. Such a decreased pool may be a result of repeated release of CC16 and other epithelial protective proteins as a result of toxic insult to the epithelium of the lung. Such an interpretation is supported by the observation (*Carbonnelle S et al, Bio-markers 2002;6:464-78*) that disturbances of the lung epithelial barrier may occur in children exposed to elevated levels of airborne NCl_3 when attending chlorinated swimming pools.

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Table 1. Forced expiratory volume during one second (FEV_1) before (S1) and after (S2) outdoor exercise in swimmers and non-swimmers.

Children	FEV_1 (S1)	FEV_1 (S2)	Paired t-test
Swimmers, n= 22	2.07 ± 0.25	2.11 ± 0.25	p<0.001
Non swimmers, n= 34	2.25 ± 0.32	2.29 ± 0.33	p<0.004
All, n=56	2.18 ± 0.31	2.22 ± 0.32	p<0.001

Table 2. CC16 µg/l in children before (S1) and after (S2) exercise.

Category	CC16 µg/l (S1)	CC16 µg/l (S2)
Swimmers, n= 21	5.7 ± 2.4	5.2 ± 1.8
Non swimmers, n= 33	8.2 ± 2.8	8.0 ± 2.6

Statistically significant difference between swimmers and non swimmers p<0.01