

Editorial

Obesity and allergic disease: closely related epidemics of the 21st century

I recently attended a very interesting symposium which brought together experts in obesity and allergy. This was a very opportune event because we have been receiving an increasing number of manuscripts presenting investigations of the putative association between obesity and atopic diseases. The modern allergy epidemic has followed a very similar demographic path to that of obesity and its associated metabolic syndrome (1–5). There is also a remarkable concordance of the early life influences on each problem suggesting that interventions which might be considered to affect the development of one might also change prevalence of the other.

Like allergic disease, obesity also has its origins in fetal life. Variations in maternal nutrition and fetal growth clearly impact on susceptibility allergy (6) but also obesity, insulin resistance and other features of the metabolic syndrome (7). Late intrauterine growth faltering is sometimes associated with rapid postnatal growth which in turn increases the risk of obesity, certainly in murine models (8). We have shown that rapid neonatal weight gain is also associated with impaired lung function at 4 wk of age and therefore a probable increased risk of wheezing in early life (9). One study has shown an association between rapid early postnatal weight gain in low-birth weight infants and both later obesity and asthma (10).

Most studies have reported associations between current obesity and asthma but there is less consistency about the relationship with other atopic diseases. Thus in Japanese school children high body mass index was positively associated with asthma prevalence (an effect seen most strongly in girls) and atopic eczema severity but negatively related to allergic rhinitis and conjunctivitis (11). In the last issue of *Pediatric Allergy and Immunology*, a study of Belgian school children reported strong associations between obesity and exercise-induced respiratory symptoms but not allergic sensitization, eczema or rhino-conjunctivitis (12).

There are potential mechanistic explanations for the association in that the peptide regulatory factors associated with obesity often described as adipokines also have a range of effects on inflammatory processes. However, the influences are confusing. Thus serum levels of resistin produced by adipose tissue were negatively associated with asthma and IgE levels but positively associated with bronchial hyper-responsiveness. Serum leptin and adiponectin on the other hand had no apparent effects (13). This conflicts with other studies showing positive associations between leptin levels and asthma as discussed in a recent review (14).

There has been an interesting focus on dietary fatty acids in relation to susceptibility to obesity. A shift in the ratio of intakes of omega-6 to omega-3 polyunsaturated fatty acids has been related to the increase in prevalence of obesity and of course the same associations have been seen in relation to allergy (15, 16). It will therefore be interesting to elaborate on whether studies of the effects of high fish intake or fish oil supplementation in pregnancy on allergic disease will also have an impact on obesity (17). Future intervention trials should harness the expertise of allergists, nutritionalists and endocrinologists.

There are clearly great benefits to be derived from bringing together experts investigating common conditions which are afflicting the world's population in the 21st century to pool knowledge and understanding which might in the end lead to the development of strategies which prevent disease and improve overall health.

In the meantime, a review of the state of knowledge would suggest that there are strong associations between obesity and asthma but this would appear to be independent of allergy. Peptide regulatory factors generated by adipose tissue certainly have the capacity to modify airway inflammation and this provides a credible mechanistic explanation for the association.

The key questions to answer are, which is the cart and which the horse, or is the association

based on a common pre-existing cause? A controlled trial of weight reduction in obese asthmatics which benefited asthma control might suggest that obesity and the resulting adipokines aggravate asthma (18). This would be supported by the association of an increase in wheezing amongst girls who become overweight (1). However, the common demographics and early life influences of the two epidemics of the 21st century suggest that the relationship is rather more complex.

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