POINT OF VIEW

Pulmonary health and healthy diet

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There is evidence that a high level of unsaturated fatty acid dietary intake is related to an increase in prevalence of asthma and chronic obstructive pulmonary disease (COPD)[1]. It has been hypothesized that the increase in asthma may, in part, be a consequence of changing diet, and early life dietary exposures might be particularly important in the development of childhood asthma[2,3]. It is well known that changes in dietary habits, such as increasing salt intake, and decreasing intake of fruit and vegetables, contribute to the rise in asthma and COPD mortality and morbidity[1].

It is also important to underline that maternal well-being has important implications for the future health of the baby and classical epidemiological studies of Forsdahl suggested a causative link between early life environmental factors and subsequent disease[4]. Barker et al. showed the correlation between low birth weight and subsequent adult cardiovascular disease (CVD), which led to the hypothesis that adverse environmental factors in early life cause disruption of normal growth and development, leading to a more susceptible adult phenotype, prone to CVD[5].

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Early life (from conception to 2 years) dietary exposure might be particularly important in the development of childhood asthma. The timing of any nutritional intervention during pregnancy is likely to be important because early intervention (even before 15 weeks gestation) has the potential to influence both the airway and immunological components of asthma whereas later nutritional manipulation may only influence the immunological component of asthma[2].

Breast feeding has been reported to reduce the risk of asthma among toddlers diagnosed before 2 years of age and also in pre-school children[7]. Kull et al., using a cohort of 4089 newborn infants, showed a significant preventive impact of breast feeding on the development of asthma; suspected allergic rhinitis; atopic dermatitis; and wheezing[7]. The risk reduction was clearest in relation to the duration of exclusive breast feeding[8].

In addition, breast feeding has been proposed as a protective factor for childhood overweight. The dramatic increase in overweight and obese children has a positive relationship with the rise in childhood asthma during the last three decades[9]. There is also strong evidence for a positive association between overweight and obesity and asthma in children[9]. In line with this, Mai et al[10], for the first time, assessed breast feeding as a common environmental influence on overweight and asthma in susceptible children and found that overweight as a consequence of short duration of breast feeding is associated with asthma.

Programming of obesity can arise from environmental influences occurring in the embryo through to neonatal life and early childhood. Studies suggested that a host of different hormonal and dietary insults *in utero* and in early postnatal life seem to converge in a common phenotype of hyperphagia, obesity with

altered adipocyte function due to fatty acids deregulation[11]. Despite the programming of obesity being a multifactorial process, the diversity of models with a common end-point might suggest some common pathways[11]. There is a role for altered adipocyte development and glucocorticoid signalling, but we might speculate that the plasticity of the hypothalamus in late pregnancy and early life is key to the programming of the metabolism towards establishing an elevated body weight set point[11]. If obesity during pregnancy increased risk for the pregnancy outcome and also risks for the future health of the child, obesity per se influences airway inflammation, increases leptin secretion which influences signals satiety of the hypothalamus, stimulates Th1, and suppresses Th2. Also obese individuals influence a breathing pattern with high frequency and lower tidal volume, shorter muscle length, with consequent reduced bronchodilator effect of tidal stretch in airways[9]. Ultimately obesity increases oestrogen secretion with the result of early *menarche* in girls and subsequent higher risk of new onset asthma[9].

We could speculate at this point on a correlation between lung development and adipocyte development with a strict correlation in reduced balance and primitive damage of both systems that could develop asthma in one way and in another way overweight and obesity. It could be fascinating to have such a simple conclusion, but several other factors play roles in these multifactor situations.

Obesity and asthma may be determined by common genetic mechanisms. It was found that the covariation between obesity and asthma could be caused by shared genetic risk factors for both conditions. Candidate genes have been identified which are associated with both obesity and asthma, in particular the glucocorticoid receptor has been postulated to be involved in the inflammatory responses associated with both obesity and asthma[12].

Several prospective studies suggested that obesity increases the risk of subsequent asthma. Possible mechanisms for this relationship include airway inflammation; mechanical changes associated with obesity; changes in airway hyper/responsiveness; and changes in physical activity and diet[12]. This hypothesis is that high body weight, and spending a lot of time watching television, independently increase the risk of asthma symptoms in children[12].

Therefore the paradigm regarding the relationship between respiratory disease and diet gained in evidence. Sodium intake showed an unfavourable association in asthmatics, while the intake of fruit and vegetables and fish is more likely to be beneficial[1]. The protective effects of fruit, antioxidant, and other micronutrients may be mediated in the respiratory tract lining fluids and cells. Antioxidant vitamins C and E in the respiratory tract are thought to prevent or limit the inflammatory response by reducing reactive oxygen species and inhibiting lipid peroxidation. The anti-inflammatory effects of n-3 and n-6 fatty acids may be due to their integration into cell membranes of the respiratory epithelium, and modulation of the inflammatory cascade. In addition to maternal intake of vitamin E, vitamin D and zinc during pregnancy, Willers et al.[13] found that maternal consumption of apples and fish during pregnancy may reduce the risk of children developing asthma and atopic disease.

While significant positive association between overweight and asthma is well known, recently Tsai et al.[14] showed that overweight and greater TV-watching time increased respiratory symptoms with the occurrences of asthma. On the contrary, physical activity decreases the risk of respiratory symptoms in schoolchildren.

Much work remains to be done to elucidate the relationship of obesity and asthma. In particular using animal models it could be fundamental to test the interaction between asthma and diet controlling environmental exposures (e.g., allergen, infection), host factors (e.g., hormones), and airway inflammation. On the other hand, ongoing research into the genetic basis of asthma and obesity and increased efforts at relating these genes to specific asthma phenotypes should begin to enhance our understanding of the common genetic basis of these "disorders".

REMARKS

Several remarks could be pointed out in order to understand and establish the relationship between obesity and asthma:

– Children and adolescents often have bad dietary behaviour and dietary intake of fundamental nutrients below levels recommended for health promotion. This may affect the attainment of optimal lung function.

– Chronic cough and wheeze associated with low dietary micronutrients intake, may lead to airway remodelling.

– Promoting fruit and fish consumption in addition to vitamin supplementation could ensure the adequate intake of antioxidants and n-3 fatty acids protecting respiratory health in rapid growing subjects.

– Obesity as increased deposition of fat in the visceral compartment, in muscle and liver, is associated with an adverse metabolic profile leads to promoting healthful child-feeding strategies and healthful weight among children.

TAKE HOME MESSAGE

All efforts should be made to prevent intrauterine insults that may perturb lung development:[15]

– no maternal smoking during pregnancy
– no maternal under-nutrition and over-nutrition
– no maternal hypertension which may cause placenta hypoxia
– management of maternal obesity and gestational diabetes, and specially the control of maternal hyperglycaemia, hyper-insulinaemia and hyper-leptinaemia before and during pregnancy
– no oligohydramnios

In children these may be of help[15]:

– reduction of allergen exposure in early life
– regular consumption of fresh fruit and vegetables
– reduction of indoor and outdoor pollution
– regular physical activity

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