RESEARCH SUMMARY

Stress-Asthma Relationship in Children

Lecture given by Dr Seija Sandberg, University College London,
"International Association for the Study of Attachment (IASA)" conference held at St John's College, Cambridge: August 2010.
(The web address for this presentation is cited at the end of this summary)

This work collects the evidence for a stress-asthma relationship in children. The presentation focuses on psycho-social stress experienced in early childhood for children who are genetically at risk from asthma. Asthma is an inflammatory disorder of the airways; it is a complex condition with numerous possible causes and a genetic susceptibility component. The disorder is characterised by the patient having periodic problems of breathlessness, coughs and wheezes. These signs are caused by bronco-constriction and over secretion of mucus. Asthma has now become the commonest chronic disease in childhood (11-14% of school-age children in Western Europe). Eczema is a related disorder, with a genetic susceptibility component, giving rise to chronic skin inflammation.

Stress needs to be defined. The cause of stress is termed a stressor; it can be thought of as any intrusion into a child's life that unbalances the natural physiological equilibrium. Stress is the change in this physiological balance that is caused by the stressor. The stress reaction is the manifestation of this intrusion on to the physiological status quo and can usually be measured and therefore quantified. An example of acute stress would be an accident (stressor) giving rise to serious injury; school exams (another stressor) can be seen as an episodic stress; chronic stress might be experienced in prolonged conflict situations (stressor), for example where a family is breaking up, loss of a parent, or an adverse environment of deprivation.

In our immune system there are special cells called T-helper cells. A special type, T-helper cell type 2, eventually leads to the production of antibodies against environmental proteins called allergens. In asthma there is an inappropriate response of these cells to allergens.

Acute stress affects the immune system through two main physiological systems: the Sympathetic Adrenal Medullary Axis (SAM) and the Hypothalamic-Pituitary Adrenal Axis (HPA). The activation of SAM prepares the body for a fight or flight response to the stress threat. SAM works rapidly through the release of adrenaline and noradrenaline from the adrenal medulla. The HPA is much slower in onset than SAM and is more concerned with restoring the body's response after trauma. HPA works slowly by the release of cortisol from the adrenal cortex.

Chronic stress also involves both SAM and HPA. Chronic stress stimulates the T-helper 2 response to produce a set of proteins called cytokines (in particular the

interleukins); unfortunately these cytokines adversely affect the immune system to worsen the allergy.

In early infancy, the baby becomes attached to its caregiver (usually one or both of its parents). If the caregiver-infant relationship causes the infant stress in the infant's first year, a child with a genetic risk of asthma shows an increased relative risk to asthma and manifests wheezing in its first year. If the stress continues, serum IgE (immunoglobulin E, a class of antibody that plays a role in allergy) is increased and frequent infections are noticed in the child. Parental stress in the early years of the child doubles the risk of asthma with children in the age range 6 – 8 years. A genetic predisposition to eczema has a prevalence of 6% in children with parents that are not divorced, however, following divorce it is raised to 18% at age 4 years. Noticeable behavioural problems occur in children with asthma that is induced by family stress. Finally, a severe life event, coupled with ongoing high chronic stress, enhances the probability of a new asthma attack occurring within the following 2 weeks.

The final section of the lecture asks the question: why does early life stress increase the risk of asthma? As yet there is no definitive answer. There is, however, epidemiological evidence to suggest that the critical time for establishing asthma is between conception and 3 years. Cortisol, the chemical associated with the HPA stress mechanism, together with corticotrophin releasing hormone, can pass from the mother, through the placenta, to the child. This entry of cortisol may affect the development of the immune system in the child. A more recently discovered, and perhaps more worrying, cause is the possibility that epigenetic changes may occur in gene expression without directly altering the DNA sequence; cytokine gene regulation is one candidate. This has led to speculation that there may be trans-generational inheritance of allergy; for example, a grandmother smoking whilst she is pregnant may increase the risk of asthma being passed through that child to her directly descended grandchild.

This presentation can be found at the following web address:
http://www.iasa-dmm.org/images/uploads/Seija%20Sandberg,%20Stress%20asthma%20relationship%20%20in%20children%20camb.pdf

Website: http://www.whataboutthechildren.org.uk **Tel:** 0845 602 7145
What About The Children? (***WATCh?***)**:** Company No. 5252973 - Reg. Charity No. 1108816