EPITHEORESE KLINIKES FARMAKOLOGIAS KAI FARMAKOKINETIKES, INTERNATIONAL EDITION 15: 149-150 (2001}

Fetal Growth and Environmental Pollution

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Accepted for publication·. 1 August 2001

There are two types of intra-uterine growth retardation (IUGR) defined.

(a) Type I IUGR occurs early in pregnancy, mainly in the second trimester, and the fetus appears capable of down-regulating so that the growth retardation affects all systems symmetrically.

(b) In Type II IUGR, which occurs in the third trimester, the fetus appears to lose the ability to down-regulate and a system of hierarchical sacrifice comes into play. Thus these children are born too long for their weight, hence the term asymmetric IUGR. This variety of IUGR is often associated with placental insufficiency but toxic aetiologies also have to be considered, maternal smoking being the most common.

Our research group has been investigating the effects of Type II IUGR on the development of a number of organ systems, including the kidney, lung and nervous system, having hypothesised that IUGR infants are physiologically compromised and have a shallow homeostatic reserve (1). This correlates well with the known fact that Sudden Infant Death Syndrome (SIDS) -"cot death"- is 10 times more common among IUGR children than normal birthweight children. We have shown that children suffering from IUGR and victims of SIDS have decreased numbers of renal nephrons (2,3); in addition, victims of SIDS have decreased numbers of terminal airways in the bronchial tree (4,5). A major point is that none of these changes are detectable to the "naked eye" but that they have to be objectively measured using stereological techniques (6). These changes are also thought to have consequences for adult disease (7).

Many ubiquitous environmental pollutants are known to be able to cause IUGR. Much of this knowledge has been gained either from high exposure incidences such as Miyamata disease (mercury exposure) and the Yu Cheng, Yusho epidemics, or from animal toxicology. Other epidemiological investigations show effects from lower exposure levels, such as the Swedish fishermen's family study, which found that the mean birthweight of children born to families living on the East coast of Sweden was significantly lower than on the West coast. The reason for this is not known but it is hypothesised that the East coast families were eating more organo-chlorine pollutants in fish from the Baltic Sea than families on the West coast, where the fish was from the North Sea. It is hypothesised that this was causally related to the effect (8). However, a major point is that the graphs show that the majority of the children fall within the normal range of birthweight. Therefore it is not possible to decide in any one individual case whether that individual has been affected or not. Thus changes can only be measured at the population level.

'Summary of talk for Greek Pathology Society 15.4.2000

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Most people have a mixture of 300 to 500 persistent pollutants in the fat stores in their bodies. In addition to this, there are appreciable levels of transient pollutants such as organo-phosphorous pesticides. The effect of this chronic low-dose exposure to maternally donated pollutants throughout fetal life remains largely unknown. Data from recent studies will be presented.

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