EDITORIAL

FETAL PROGRAMMING AND FUTURE DISEASE

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Recent epidemiological studies have demonstrated a close relationship between low birth weight and a high predisposition to arterial hypertension, hyperlipidemia and non-insulin-dependent diabetes in adulthood. These findings may coexist as Syndrome X, more recently named as Metabolic Syndrome, and understood as precursory of coronary heart disease and cerebral stroke. It is conceivable that fetal adaptation to the adverse conditions of intrauterine environment can determine permanent metabolic alterations that could bring repercussions in the adult life.

Intrauterine programming or Barker's Theory established a basis for population and experimental research, new possible causal factors of cardiovascular disease in adult life in addition to the well known genetic and environmental factors.

The earliest evidence showing the relation between Intrauterine Growth Restriction and occurrence of adult cardiovascular diseases stems from retrospective epidemiological studies.

This line of study began with an analysis of the birth registry in England and Wales. Geographic areas with a high recent incidence of coronary heart disease match areas showing a high rate of infant mortality in the beginning of the twentieth century. These findings present a paradox, because such diseases have always been associated with the wealthy population. Therefore, research was conducted to find a relationship between fetal under nutrition and future occurrence of hypertension or coronary heart disease in those adults.

These studies gave origin to the intrauterine programming theory, based on sample evaluation of the English population. Research centered on 16.000 births in Hertfordshire, between 1911 and 1930 show that death rate from ischemic cardiac disease was higher in adults that had presented birth weights less than 2,500 grams. Another study carried out in Sheffield which included birth weight and gestational age confirmed the Hertfordshire study, but showed no relationship between prematurity and adult hypertension. Similar associations were observed in the United States and in the South of India.

The hypothesis that certain adult diseases originate in fetal life is reasonable. However, much of the evidence is inadequately supported by faulty data interpretation or statistical errors.

The cause effect relationship between low birth weight and hypertension needs more accurate population growth analysis, including fetal biology, follow-up during childhood and adolescence, and determination of adult body mass index. These adjustments become necessary due to a perceived change of growth pattern that may occur during the development process.

On the other hand, it must be emphasized that the Intrauterine Growth Restriction is different around the world, asymmetrical (i.e., nutritional restriction in late pregnancy) in developed countries and symmetrical (i.e., nutritional restriction throughout pregnancy) in developing countries. In this context Barker's Theory is more especially relevant to symmetrical growth restriction. Therefore, such factors must be considered in the analysis of incidence of future diseases.

These findings are polemic and must be subject to careful analysis. The theme led to the development of experimental and clinical research, presenting results against or in favor of Barker's Theory. These considerations are important to understand the biology of Fetal Programming. Future research and Public Health interventions should concentrate on the mechanisms underlying this relationship.

In this issue Mion et al. report the incidence of hypertension in a sample of adults. A follow-up study looking at the birth weight of the population included in this sample would be highly interesting in the light of Barker's theory.

REFERENCES

1. Barker DPJ, Bull AR, Osmond C, Simmonds SJ. Fetal and placental size and risk of hypertension in adult life. Br Med J 1990;301:259-62.

2. Barker DPJ, Hales CN, Fall CHD, Osmond C, Phipps K, Clark PMS. Non insulin dependent diabetes, hypertension and hyperlipidemia (Sindrome X): relation to reduced fetal growth. Diabetologia 1993;36:62-7.

3. Mion Jr. D, Pierin AMG, Bambirra AP, Assunção JH, Monteiro JM, ChineN RY et al. Hypertension in employees of a University General Hospital. Rev. Hosp. Clín. Fac. Med. S. Paulo 2004;6:329-336.