Fetal growth and cardiovascular risk factors in an Australian Cohort

Vivienne Moore

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Department of Public Health
Faculty of Medicine
University of Adelaide
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Abstract

There is an extensive history to the idea that experiences during early life have long lasting effects on health. The fetal origins theory, which is more specific than its predecessors, was proposed and developed from the late 1980s by Barker and his colleagues.

According to the fetal origins theory, growth before birth is a potent influence on the risk of cardiovascular disease, as well as other chronic diseases, in later life. Poor fetal growth, manifest in the weight and shape of the baby at birth, is thought to be related to adult cardiovascular disease events through enduring physiological changes which accompany impaired growth.

A number of cohort studies, primarily from Britain and the United States, have demonstrated an inverse association between birth weight and risk of incipient or fatal cardiovascular disease. Internationally, more than 20 studies have now found an inverse relationship between birth weight and later blood pressure, which is hypothesised to amplify with age. Far fewer studies have considered blood lipids; relationships between birth weight and this outcome are inconsistent.

More recently Barker has identified certain patterns of disproportionate fetal growth, thought to result from undernutrition in different periods of gestation, as additional markers for elevated risk of cardiovascular disease. The disproportions are: thinness, possibly accompanied by a small head; and shortness with a large head circumference compared to chest size. Discrepancies between the weight of the baby and its placenta are also considered to indicate increased risk of cardiovascular disease. These specific propositions are based on work undertaken mainly in Britain by Barker and his colleagues. Relevant international evidence lacks coherence and is limited, due in part to detailed information about birth dimensions being rarely available.
Barker’s interpretation of associations with birth weight has been contested. Critics have argued most strongly that the results may be explained by adverse socio-economic circumstances which contribute to low birth weight initially and thereafter, through established behavioural risk factors, to increased likelihood of clinical risk factors and cardiovascular disease. Studies which take into account the socio-economic environment at birth and during childhood as well as contemporary circumstances have been called for.

In the present study relationships between fetal growth, as manifest in size and shape at birth, and later blood pressure and blood lipids were investigated in an Australian cohort. Data on these outcomes for cohort members at age 8 years were available from a previous study. Birth details (body weight, placental weight, head circumference, chest circumference and length) were abstracted from hospital records. In addition, a follow up of cohort members was undertaken to collect new data pertaining to the two cardiovascular risk factors at 20 years of age. Socio-economic circumstances were characterised at birth, age 8 and age 20.

The cohort comprised 856 individuals born in the Queen Victoria Hospital in Adelaide, South Australia, during 1975-76. Members were enrolled in the cohort through participation in a study of cardiovascular risk factors at age 8 years. By the time of the follow up conducted 12 years later, around one per cent of cohort members had withdrawn or died and only 2 per cent of the remaining members could not be traced. Thus, almost all cohort members were invited to take part in the follow up and over 70 per cent eventually did so.

At age 8, for boys and girls together, relationships between birth weight and blood pressure were weak and not statistically significant. There was some evidence of simultaneous effects of birth weight and placental weight, the latter variable being positively associated with blood pressure in childhood.
At age 20, among males, there was little evidence of relationships between birth dimensions and current blood pressure. In contrast, among females at age 20, a one kilogram increase in full-term birth weight was associated with a decrease in systolic pressure of 4.4 mm Hg (95% CI 2.1 to 6.7), after adjustment for current weight and height. In females, amplification of the relationship between birth weight and blood pressure had occurred between age 8 and age 20. Furthermore, there was evidence that each of the birth disproportions specified by Barker were linked to elevated blood pressure in early adulthood.

Concerning blood lipids, both thinness and shortness at birth were associated with poor lipid profiles at age 8, for all children born at term, after adjustment for sex, current weight and age. At age 20, among males born at term, shortness and thinness at birth were again associated with elevated concentrations of total cholesterol and low density lipoprotein cholesterol, after adjustment for current weight and age. However, these patterns were largely unseen in women at age 20. Birth weight was not related to lipid profiles at either age.

Associations between birth dimensions and later cardiovascular risk factors that emerged in the present study did not appear to the product of behaviours that affect these risk factors, nor did they appear to be a consequence of socio-economic variation. This study offers some insight with regard to the lack of coherence in results from other studies.

Overall the findings were mixed and the present study does not give unequivocal support to the fetal origins theory. Nevertheless, there was evidence that poor fetal growth was associated with later cardiovascular risk factors.