Early origin of coronary heart disease

Maternal nutrition in early pregnancy may affect placental ratio

EDITOR,—In their editorial discussing the early origins of coronary heart disease Nigel Paneth and Mervyn Susser emphasise the importance of further investigation of the influences on the ratio of placental to fetal weight.[1] Godfrey *et al* have proposed that iron deficiency has a role since low maternal haemoglobin concentrations and a decrease in mean red cell volume during pregnancy are associated with an increase in the ratio.[2] In contrast, Perry *et al* found no relation between maternal haemoglobin concentrations in early pregnancy and placental ratio at delivery[3] but confirmed the positive association between the ratio and maternal body mass index.[2]

Timing is another important consideration when the influence of the maternal environment on the developing fetus and placenta is studied. Many women start pregnancy with low iron stores, and we hypothesised that placental size would be increased by mid-pregnancy in those with low ferritin and haemoglobin concentrations. To investigate this we developed a method of measuring placental volume during the routine scan at around 18 weeks of pregnancy. In a study of 568 white women we found that low haemoglobin and ferritin concentrations at 14 weeks' gestation were associated with increased placental volume.[4] Placental volume was also greater in taller women, in multiparous women, and in those who smoked heavily at the time of their last period. We found no relation between maternal weight or body mass index and placental volume.

We have since examined the factors affecting the ratio of placental to fetal size in mid-pregnancy by estimating fetal weight using Shepard *et al*'s algorithm.[5] The relations between maternal characteristics and the ratio of placental volume to estimated fetal weight were examined by multiple regression analysis (table). The ratio was increased

Simultaneous effects of maternal characteristics on ratio of placental volume to estimated fetal weight (ml/g) at 18 weeks' gestation

Maternal factor	Regression coefficient (95% confidence interval)	P value
Gestation at which scan obtained (days)	−0·013 (−1·018 to −0·009)	<0·0001
Haemoglobin (g/l)	−0·002 (−0·0004 to 0·0)	0·05
Ferritin (square root μg/l)	−0·014 (−0·022 to −0·006)	0·001
Smoking at last menstrual period	0·027 (0·006 to 0·048)	0·012
Previous deliveries	0·007 (0·0004 to 0·014)	0·038

in scans obtained at earlier gestations, with lower maternal haemoglobin and ferritin concentrations, with increasing parity, and in mothers who smoked. None of these factors except gestation affected the estimated fetal weight, and the change in ratio was therefore principally due to changes in placental volume.

Perry *et al* concluded that their findings did not support a link between placental ratio and maternal nutrition at birth.[3] In contrast, our study suggests that the ratio is affected early in pregnancy by the mother's nutrition as shown by the haemoglobin and ferritin concentrations. The evidence that maternal nutrition affects the placental ratio is further strengthened by experiments in sheep, which have shown that restricted maternal food intake results in an increase in placental size.[6]

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The influence of parental somatic features on childhood weight has been extensively studied

EDITOR,—In their editorial on the early origin of coronary heart disease (the "Barker hypothesis") Nigel Paneth and Mervyn Susser overlooked my contribution.[1] In 1982 I suggested that some heavier children were relatively immune to non-insulin dependent diabetes and coronary heart disease in adult life and that the decline in rates of coronary heart disease that was occurring in some communities was linked to the increasing average weight of children in those communities.[2] I was also the first to publish this hypothesis in major North American journals.[3,4] In 1989 Barker and his colleagues put forward similar ideas.

I have had 11 contributions to the discussion on this subject published, including constructive appraisals of Barker *et al*'s early papers. For example, I advised against generalised postnatal nutritional intervention to promote heavier infants, suggested that the importance of placental weight was as a marker for maternal obesity, and warned that the prenatal nutrition theory was not consistent with the existing data on twins or on prenatal exposure to famine. To date, studies have vindicated these concerns.

There were differences in approach, whereas Barker *et al* focused on maternal nutrient intake during pregnancy I focused on the influence of parental somatic features on weight in childhood. For example, birth weight reflects both maternal adiposity and maternal muscle mass.[5] Adult somatic features, in turn, are determined by both hereditary and environmental factors.

Primary care doctors do not have the resources of major research teams. Nevertheless, our contributions should be acknowledged. Thus it seems fairer to describe the Barker hypothesis as the "Barker-Bradley hypothesis."

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Earlier published work supports the "Barker hypothesis"

EDITOR,—Nigel Paneth and Mervyn Susser discuss the fetal and early life origin of coronary heart disease, referring to the "Barker hypothesis."[1] We acknowledge the important contributions of Barker and his coworkers. More strongly than previous authors, they have pinpointed the relevance of fetal nourishment to later coronary risk. Readers of the *BMJ* should also be aware, however, of the earlier publications by Anders Forsdahl of the University of Tromsø. In 1973, in a paper published in Norwegian with an English summary, he advanced "the theory that extremely poor social conditions during childhood and adolescence can be considered as risk factors for cardiovascular diseases."[2] This was more than 10 years before the paper by Barker and Osmond in 1986.[3] Since 1977 Forsdahl has published several studies supporting his hypothesis in international journals.[4-6]

Thus we believe that if one person alone is to be given the honour for the theory that experiences in early life are of major importance for the risk of cardiovascular diseases several decades later it should probably be Anders Forsdahl.

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A definition of sexual health

EDITOR,—No seminar or conference on the subject of sexual health seems complete without a presentation or debate rhetorically titled "What is sexual health?" Those who have discovered the subject since the launch of *Health of the Nation* face a daunting choice of definitions. Most definitions, coming from a political[1] or health promotion[2] background, extol in lengthy prose the positive personal benefits of sexual relationships for self esteem and fulfilment, apparently ignoring the medical perspective; the medical perspective appears as a more or less comprehensive list of serious health problems associated with sexual activity, giving an entirely negative impression.

A concise definition of sexual health that unites both benefits and deficits in one sentence of plain English is clearly needed. It must be simple enough to be understood and remembered easily; applicable to a wide range of social, religious, and sexual cultures; yet be robust enough to stand critical analysis and sufficiently unambiguous to be capable of expansion without significant distortion. Such a sentence is: Sexual health is the enjoyment of sexual activity of one's choice, without causing or suffering physical or mental harm.[3]

This definition allows the term "enjoyment," in its widest interpretation, to be expanded to the lengths of previous statements without prescribing the type of sexual relationship—be it heterosexual, gay or lesbian, monogamous, or otherwise—provided it is by mutual consent without coercion or damage. It can also promote two of the safest options, masturbation and abstinence, as positively healthy choices. It is consistent with the traditional humanist medical principle of first do