British Journal of Nutrition (2006), **96**, 1170

Nutrition Discussion Forum

Fatal flaw in the fetal argument

The fetal-origins hypothesis, in its initial form, postulated that maternal and fetal undernutrition is associated with an increase in an individual's propensity towards the development of CHD and its allied risk factors in adult life (Barker, 1993), including hypertension, dyslipidaemia and Type 2 diabetes. Indeed, an inverse association between birth weight and blood pressure has been considered to provide the strongest evidence in support of the fetal origins hypothesis (Robinson, 2001). However, recent systematic reviews of the large number of studies in this area have shown little support for two of the major components of the hypothesis, namely inverse associations between birth weight and blood pressure (Huxley *et al.* 2002) and with dyslipidaemia (Huxley *et al.* 2004), flaws, which if not fatal, are at the very least problematic, for the fetal-origins hypothesis (Huxley, 2006).

Evidence from animal studies has been used to provide support in favour of the fetal-origins hypothesis, but as Professor Cohen implies, extrapolating these findings to human populations is fraught with difficulty. In humans, there are limited data as to the effects of maternal nutrition on offspring's adult health, but what data there are would tend to suggest that maternal undernutrition is not strongly associated with either higher blood pressure or cholesterol levels in offspring (Stanner *et al.* 1997; Roseboom *et al.* 1999).

But, even allowing for a small inverse association between birth weight and CHD risk factors, from a public health perspective, strategies aimed at reducing the burden of chronic disease through the modification of adult lifestyle factors such as cigarette smoking, diet and physical activity are likely to be far more achievable and have a much greater impact than interventions aimed at reducing the burden of disease through increases in birth weight (Huxley *et al.* 2002, 2004).

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DOI: 10.1017/BJN20061888

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