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Gestation, Growth and Old Age Vulnerability

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D. J. P. Barker, The fetal origins of diseases of old age. *European Journal of Clinical Nutrition*, **46**, (1992), Suppl. 3, S3–S9.

In this paper Professor Barker reviews the evidence that important aspects of disease in middle and later life are determined by events during fetal life and early infancy. His group has been pursuing this hypothesis for some years following the observation that regional patterns of mortality for diseases such as stroke and coronary heart disease in adult life matched the patterns of infant mortality of 50 years or more before. Barker and colleagues have been able to discover datasets that permit the direct linkage on an individual basis of information about early childhood development, birth weight and even placental weight with the mortality of the individual concerned in middle age. The practicalities of the research mean that so far links have been completed for more men than women because most women change their name on marriage.

The hypothesis is interesting at a biological level as it links with experiments in animals indicating that patterns of anatomy and of metabolism may be permanently affected by experience in early life. Many body organs become fully developed during fetal or early childhood development and are incapable of further development or adaptation thereafter. In animal studies, nutritional patterns may affect the development of the pancreas in such a way as to make its possessor more liable to diabetes in later life. Baboons exposed to certain types of diet in early life develop permanent changes in their pattern of cholesterol metabolism.

Among the results of Barker's work has been a link between birth weight and placental size and later blood pressure. Using a set of obstetric records concerning normal-gestation births of men and women now in their fifties, it was found that there was a fall in mean blood pressure in middle life with increasing birth weight. It was also found that there was a direct relation of midlife blood pressure to placental weight. In other words, blood pressures tended to be highest

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of all in those middle aged people who at birth had been small in relation to their placental weight. In these studies the range of blood pressure differences increases with age, a finding which may reflect an underlying tendency for higher blood pressures to rise faster under the influence of whatever are the environmental factors in western populations that cause blood pressure to rise with age.

Barker postulates that some of the environmental factors causing small birth weight, which may include aspects of maternal deprivation, are causative of degenerative diseases in later life. He points out that there are two other general ways in which his findings might be explained. It is possible that the link is purely genetic in the sense, for example, that whatever genes determine birth and placental weight also directly cause high blood pressure in later life. This seems unlikely as birth weight seems to be affected more by conditions of pregnancy than by inheritance. The second relates broadly to the 'cycle of deprivation' model, and postulates that the children of mothers who were sufficiently socially and nutritionally deprived to produce small babies are likely themselves to live lives of social and nutritional deprivation. Barker attempts to refute this idea by demonstrating that birth weight was unrelated to social class and that the relation of birth weight to blood pressure was demonstrable within each class. This finding does however raise the question of whether the differences in birth weight are in fact due to maternal deprivation, since if that were so one would expect a social class gradient in birth weight.

A general problem with epidemiological studies of this kind are the statistical difficulties in 'correcting' findings for what are thought to be interfering variables. Most forms of correction involve regression models and these depend on the assumption that the independent variable (usually the one for which correction is being made) is measured without error. If this assumption is incorrect the regression line is biassed downwards and the applied correction is inadequate. Adjustment for cigarette smoking is particularly problematic as smokers often underestimate how much they smoke when answering research questionnaires.

The Barker hypothesis impinges on the concept of 'thrifty genes' formulated by J. V. Neel thirty years ago (Neel 1962). If one takes in more calories in food than one uses, the excess is either burnt off in the process of thermogenesis or laid down in fat. Neel postulated that since fat people can be expected to survive longer than thin in times of privation, populations who had passed through periods of intense deprivation would come selectively to contain a high proportion of people with a genetically determined pattern of metabolism that tends to lay down fat. Neel suggested that if such people came to live in

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circumstances of constant over-provision of food they might develop, in addition to obesity, metabolic side effects of their 'thrifty' genes in the form of diabetes and coronary heart disease. Possible examples of thrifty gene syndromes have been identified in studies among Asians in South Africa, Amerindians and Polynesians who, through economic development or migration, have come to live under conditions of abundant food and limited exercise. More recently a further example has been suggested in a syndrome of central obesity and diabetes leading to cardiovascular disease among people from South Asia living in London (McKeigue *et al.* 1991). If Barker is right some instances of thrifty gene syndrome may reflect not a genetically determined response to environmental factors acting in adult life but the deprived circumstances of the victims' mothers during pregnancy.

Kuh and Davey (1993) have drawn attention to the policy implications of the Barker hypothesis in an historical context. In recent years research and policy emphasis for the prevention of cardiovascular and other chronic diseases of middle life has been on lifestyle and environment in adult life. Earlier in the century it was assumed that effort should be concentrated on mothers and infants on the assumption, based perhaps on observations of social class, that healthy children would make healthy adults. Do the Barker findings suggest that public health policy should move back to focus on pregnancy and antenatal care?

Barker writes of a process of 'programming' underlying the way in which fetal and early childhood experience leads to disease in middle and old age. This is an unfortunate term that may mislead. It may seem to imply that following particular early life events, middle age disease becomes inevitable and the adult lifestyle irrelevant. This may not be so. On biological grounds one might suspect that the changes induced by early life experience are in an evolutionary context adaptive. In other words a deprived fetus is being prepared for a deprived life. It is only if it encounters unexpected affluence and abundance of food that the undesirable consequences of its metabolic pattern will assert itself. In other words, Neel's thrifty genes might work by providing metabolic switches that determine their bearers' subsequent susceptibility to environmental circumstances. If this is so, lifestyle will be an important aspect of the aetiology of disease in later life, and particularly in a subgroup of the population whose susceptibility is determined at birth and shortly after. This might be the explanation of a number of observations including the fact that adult lifestyle factors, although clearly implicated at the population level in the aetiology of cardiovascular disease, account for so little of the total variance in incidence. The answer will emerge (if not before) among the subsequent

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generations of the South Asians of London, in whether they continue to show a genetically determined pattern of cardiovascular disease and diabetes or, being born of more affluent mothers, in whether their disease incidence comes to conform to that of other racial groups born in Britain.

More work needs to be done, and particularly in countries free of the confounding effects of the social class structure of early twentieth century Britain, before the Barker hypothesis can achieve universal acceptance. It certainly should not be allowed to justify a fatalistic attitude to lifestyle improvements in adult life. If correct, however, it may lead to better targeting of lifestyle interventions, particularly in terms of diet, to those members of the population for whom they are crucial – while the rest of us will be able to gorge ourselves in peace.

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Social Policy — Lorna Warren

Preparing these abstracts in 1993, I approached potential articles for what they might reveal about the position of older people in society and about their relationship with other generations. The three articles which I have chosen are linked by the themes of needs, resources and empowerment.

> Andrew Bebbington and Bleddyn Davies, Efficient targeting of community care: the case of the home help service, *Journal of Social Policy*, **2**, 3 (1993) 373–391.

1993 was not only the European Year of Older People and Solidarity Between the Generations, it also saw the full implementation of the 1990 National Health Service and Community Care Act. As a group, older people constitute one of the major users of community care, of

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