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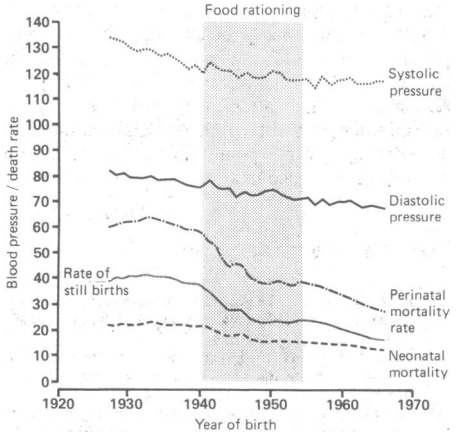
SIR,—Professor D J P Barker and colleagues suggest that the prevention of hypertension may depend on improving maternal nutrition.[1] Food rationing introduced in Britain during 1940-1 resulted in a more equitable distribution of food, and expectant mothers received free supplements of milk, eggs, and vitamins.[2] These changes were accompanied by an abrupt decline in perinatal mortality, particularly in the rate of stillbirths.[3] We investigated whether the blood pressure adjusted for age of British adults born during food rationing differed from that of those born before rationing was introduced.

During 1984-5 as part of the health and lifestyle survey the blood pressure of a random sample of British adults was measured at home by automated sphygmomanometry.[4] We analysed data on 5383 people born during 1927-66 (figure). Each year's data are based on at least 90 people. We found no discernible step around 1941-5 for either systolic or diastolic pressure.



Trends in mean unadjusted blood pressure (mm Hg) and perinatal mortality in England and Wales (per 1000 births)

Each person's blood pressure was modelled by multiple regression as a function of age (linear and quadratic terms) and the perinatal mortality rate in the year of birth. After adjustment for curvilinear effects related to age a decline in perinatal mortality rate of 10 per 1000 was associated with the following changes in blood pressure: systolic −0·3 (95% confidence interval −1·4 to 0·8) mm Hg, diastolic −0·6 (−1·4 to 0·2) mm Hg.

Whether or not the reduction in perinatal mortality during the 1940s was a direct result of wartime nutritional policy, it suggests more favourable intrauterine circumstances for babies born during the war. We found no evidence that this improvement had substantially influenced the blood pressures of today's middle aged adults.

Our analyses relate only to maternal nutrition during pregnancy and cannot assess the indirect effects of nutrition during the mother's own childhood. Nevertheless, we would be interested to know if Professor Barker and colleagues find any evidence in their data of a change in the distribution of birth weight, placental weight, or the ratio of the two among births from 1941 onwards.

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Lowering cholesterol concentrations and mortality

SIR,—Dr Matthew F Muldoon and colleagues suggest that low serum cholesterol concentration may lead to behaviour that increases the risk of violent death.[1] We examined whether plasma cholesterol concentration was related to death from violent causes in a large cohort study.

In the Whitehall study of London civil servants plasma cholesterol concentrations were measured in 17 718 men aged 40-64 during 1967-9.[2] Follow up to 31 January 1987 showed that 90 had died because of accidents, violence, or suicide (ICD codes E800-E999, 8th revision), of whom 36 had committed suicide (ICD codes E950-E959). Including the 10 violent deaths for which no responsibility could be attributed (ICD E980-E989) in the suicide group did not appreciably alter the results.

We found no significant association between plasma cholesterol concentration and all violent deaths or death by suicide (table). Proportional hazards analyses were performed with age and plasma cholesterol concentration as continuous variables. For all the violent deaths the relative rate associated with a 1 mmol/l increase in plasma cholesterol concentration was 0·98 (95% confidence interval 0·83 to 1·16). For suicide the corresponding relative rate was 0·87 (0·66 to 1·15). Adjustment for employment grade, a marker of socioeconomic state, did not materially alter these results.

Other prospective studies have not found a consistent relation between cholesterol concentration and violent death.[3,4] The size of our study means that it is unlikely that a large true effect has been missed. A direct behavioural effect of low cholesterol concentration might be expected particularly to affect suicide. Though the lowest rate was seen in the fifth quintile, the highest rate was in the fourth quintile and there was no consistent trend in the risk of suicide with cholesterol concentration.

Dr Muldoon and colleagues suggest that associations between serum cholesterol concentration, neuronal function, and behaviour could explain the relation between cholesterol concentration and deaths due to violence. We examined the association between serum cholesterol concentration and hostility in a second cohort of London civil servants. Hostility was indexed by the Cook-Medley hostility scale,[5] a component of the Minnesota multiphasic personality inventory. This scale has predicted mortality due to violent causes in some[6,7] but not all[8] studies. The correlations between serum cholesterol concentration and hostility score were negligible, being 0·016 (p=0·3, n=4246) for men and −0·005 (p=0·8, n=1742) for women. Examination of hostility score by decile of cholesterol concentration showed that, even at the lowest concentrations, there was no association with hostility. Furthermore, there was no relation between hostility and concentrations of apolipoprotein A or apolipoprotein B. These results were independent of employment grade.

Though some forms of aggression may be missed by using the Cook-Medley scale, our data do not support the notion that low serum cholesterol concentration has a direct effect on behaviour and in turn increases the risk of violent death. It may be more rewarding to investigate the influence of dietary changes or drugs used in the intervention studies than to focus on a possible direct effect of low serum cholesterol concentration.

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SIR,—Dr Matthew F Muldoon and colleagues have been too cautious in interpreting their results.[1] The results showed a higher total mortality during interventional treatment (albeit not significant). This was made up of a 14% reduction in mortality from coronary heart disease and a 44% increase in mortality from cancer. In addition, there was a 78% increase in mortality from causes unrelated to disease. Despite these findings they conclude that a reduction in cholesterol concentration tended to be associated with a reduction in mortality from coronary heart disease (p=0·06) and that there was "no consistent relation" between reduced cholesterol concentration and mortality from cancer (although the crude change in mortality from cancer was significant; p=0·01). They went on to "launder" the results until they found a significant effect on mortality from coronary heart disease and had eradicated the effect on mortality from cancer.

The first duty of authors is to report their results, however unpalatable they may be. Manipulation of data to explain what the authors construe as aberrant findings or exceptions should be the province of the discussion, and such discussion cannot be used as an excuse to report the principal findings as the exact opposite of what the results have shown.

In this case the authors were highly selective

Age standardised mortality/1000 person years (number of deaths) by quintile of plasma cholesterol concentration

	Plasma cholesterol (mmol/l)					χ^2 For trend (p value)
	≤4·11	−4·73	−5·27	−6·05	>6·05	
All violent deaths	0·29 (16)	0·34 (21)	0·27 (15)	0·33 (20)	0·31 (18)	0·00 (0·96)
Suicides	0·12 (7)	0·14 (9)	0·11 (6)	0·17 (10)	0·07 (4)	0·50 (0·48)