ANIMAL PRODUCTS IN A HEALTHY DIET

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SUMMARY

Recent meta-analysis of 19 intervention studies on plasma cholesterol-disease iations have revealed a significant excess of non-cardiac deaths at low cholesterol s and no association between plasma cholesterol and risk of cardiovascular disease in ıen. Further recent work indicates that not all saturated fatty acids are equal in ng blood cholesterol and that trans unsaturated fatty acids from vegetable fats are ıgly and positively associated with the risk of death from heart disease. Adverse :eption of the nutritional value of animal products is increasingly leading to ıitional problems among women in particular. Aside from inadequate iron and ium intakes, recent work has shown how foetal-placental growth has a subsequent . profound effect on adult chronic disease. In devising dietary guidelines which take ɔunt of the real pattern of eating, a more imaginative approach is needed.

INTRODUCTION

It is easy for nutritionists to define or identify diets which are nutritionally ıdequate and which would in the medium to long term threaten health. Where intakes essential nutrients are adequate, defining a healthy diet is not especially easy, at least at e scientific level. There are a number of reasons why this is so and it is worthwhile flecting on them. Firstly our knowledge of risk factor-chronic disease associations is not ; strong as many advocates of change would have us believe. Secondly, our knowledge f how the balance of different nutrients influences risk factors for chronic disease is nanging very rapidly. Thirdly there is increasing recognition that our fixation with the utritional aspects of coronary heart disease has led to patterns of eating which onsiderably increase the risk of other diseases such as nutritional anaemias and ɔsteoporosis. Undoubtedly others will emerge. Finally, human nutritionists have lisplayed a remarkable ineptness in studying the complexity of human eating patterns ;uch that their dietary guidelines take little or no account of what people think, believe and so on in regard to healthy eating. Each of this will be discussed in turn in this paper.

Blood cholesterol and chronic disease

Ever since it became evident that hypercholesterolaemia was a risk factor for coronary heart disease (CHD), there has been a steady stream of primary and secondary intervention studies each attempting to provide definitive evidence that lowering blood cholesterol would lower CHD. Most have succeeded in providing supporting evidence but none has been definitive. Given the complexity of the experimental task, this should not be surprising. In a provocative review of the area, Oliver (1991) highlighted the niggling irritant of these studies : that while deaths from CHD fell with intervention to lower blood cholesterol total mortality did not. He concluded: "At present, available data indicate that total mortality is unchanged when hypercholesterolaemia is lowered : the fall in cardiac mortality is offset by an apparent increase in non-cardiac deaths. These findings can no longer be dismissed as a statistical quirk which will hopefully disappear

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when new trials are reported. The problem was first raised in 1978 and has been consistently observed since then".

People with stable but elevated levels of blood cholesterol maintain a homeostasis in sterol metabolism. Intervention to lower blood cholesterol will disturb this homeostasis to achieve a new equilibrium at a lower level. Whether the process of temporary disequilibrium or the stable sterol homeostasis at a lower level alters membrane function or neuronal integrity and thereby increases the risk of other diseases is a speculative explanation of excess non-cardiac death. An alternative approach is to ask whether an inherently stable low cholesterol level itself increases risk of non-cardiac deaths. The question became so urgent that the National Heart Lung and Blood Institute in the US convened a group to study the relationship between mortality trends and baseline plasma cholesterol levels in 19 trials carried out across the globe over a 20 year period. Deaths ocurring within 5 years of baseline were excluded because these could confound associations of blood cholesterol with subsequent disease. From the 19 trials, some 68,000 deaths were analysed for an association with baseline cholesterol. The results are summarized in Table 1 (Jacobs et al. 1992) and reveal a number of shocks. The first shock is that total mortality remains relatively unaltered across a broad range of blood cholesterol values with some tendency for an increased risk of mortality at the extremes of very low and very high levels The second shock is that among women, there is no association between blood cholesterol and cardiovascular disease. The third shock is that non-cardiac mortality is elevated at lower levels of blood cholesterol. The word "shock" is deliberately chosen because these findings should have profound reverberations among those seeking to drastically change our eating habits. Clearly, among men, very high levels of blood cholesterol increase the risk of CHD and efforts at dietary intervention in this group is fully justifiable and should continue as at present. However, there should be a re-think of the wisdom of altering blood cholesterol among all women and in men with moderately high or normal cholesterol levels. Nutritionists should insist that their epidemiological colleagues address this problem and, if possible, give clear reasons why the movement for mass dietary change should not now stop or at least be radically reconsidered.

Table 1 NHLBI conference on low blood cholesterol : mortality associations. Risk ratios for death > 5 years after baseline (160 - 199mg/dl cholesterol = 1)

	All causes		Disease		Cancer		Non-cancer, non-cardiovascular disease	
Blood chol. (mg/100ml)	M	F	M	F	M	F	M	F
< 160	1.17	1.10	1.04	0.96	1.18	1.05	1.32	1.41
160-199	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
200-239	1.02	0.94	1.16	0.95	0.95	1.01	0.89	0.92
> 240	1.14	0.97	1.48	1.09	0.95	0.97	0.87	0.82

Jacobs et al. (1992)

Dietary fats and risk factors for CHD

Work carried out in the 1960s at Harvard and Minnesota (Hegsted et al. 1965; Keys and Parlin 1965) on how dietary fats were to influence blood cholesterol dominated community nutrition programmes for the ensuing two decades. "Saturates" were bad.

yunsaturates" were good. "Monounsaturates" did not matter. It was all so simple, now, so wrong. The work of Hegsted et al. (1965) showed that not all saturated fatty s were equal in their cholesterol raising properties. Whilst this was not completely ›red in the ensuing twenty years, it was conveniently forgotten because public health grammes demanded simplicity. This area of the variable effects of different SFA on ɔd cholesterol has now been revisited and it is clear from recent studies that fats rich in ':0 - C16:0 are far more hypercholesterolaemic than fats rich in C18:0. Table 2 nmarizes the findings of Denke 7 Grundy (1991) in a study using formula diets ıtaining 40% energy from fat with varying amounts of the cholesterol-raising SFA 12:0 - Cl 6:0) and the non-cholesterol-raising SFA (C18:0). The role of SFA of 10 or less 'bon chains is unclear but the in vitro studies of Woollet et al. (1992) would not suggest ıt these fatty acids are a problem with respect to cholesterol. Clearly, not all dietary fats :h in SFA are the same in this context. In reality this is not of great significance in mmunity nutrition because stearic acid (C18:0) is a relatively minor component of the ıman diet. However, it is an issue for concern in the nutritional labelling of specific ·ods such as chocolate. A case for a re-think of the role of animal in CHD is also evident om the recent Caerphilly Cohort Study (Fehily et al. 1993). 2,423 men aged 45 - 59)llowed over several years yielded 148 cases of major CHD events. there were no ignificant differences in odds ratio for CHD across the five quintiles of animal fat intake e. from 22.3% to 36.2% energy, from animal fats. The association of animal fats with CHD will be considered later in this section under trans fatty acids.

Table 2 The effects of cholesterol-raising saturated fatty acids on plasma lipids in man

Diet	Saturated fatty acids (SFA) as % energy	C12:2 to 16:0 inclusive as energy	Plasma cholesterol (mmol / l) Total	LDL
Butter fat	25.4	17.5	5.7	4.2
Beef fat	18.8	11.2	5.5	4.0 *
Cocoa butter	23.2	10.0	5.3	3.8 *
Olive oil	8.0	5.8	5.1	3.4 *

* Significantly different form butter ($p < 0.05$).
Denke and Grundy (1991)

The findings of the studies of the 1960s that monounsaturated fatty acids (MUFA) are neutral in regard to their effects on blood cholesterol are now rejected. Since 1985, some 26 studies have consistently shown that MUFA lower blood cholesterol as effectively as polyunsaturated fatty acid (PUFA) and that this effect is confined to the detrimental low-density lipoprotein (LDL) cholesterol. In contrast to MUFA, high intakes of PUFA not only lower LDL cholesterol but also lower high-density lipoprotein (HDL) cholesterol. Since HDL cholesterol is protective against CHD, this presents a disadvantage to the use of high intakes of PUFA. Low-fat diets, universally regarded as nutritionally good, also lower HDL-cholesterol, a fact repeatedly ignored in the compilation of data for developing dietary guidelines. The relative effects of different dietary fat levels and fatty acid types on blood cholesterol has been reviewed elsewhere (Gibney 1992). All of this means, that for those willing to peruse the scientific data, our knowledge of the relationship of dietary fats and blood cholesterol has changed very dramatically in the last

five years. During all this time the issue of trans-fatty acids has played a minor role. Very recent evidence moves them to centre-stage.

Mensink and Katan (1990) were the first to show that high intakes of trans 18:1 (elaidic acid) elevated LDL cholesterol and lowered HDL cholesterol in healthy adult men. Their work was criticized on the ground of the very high levels of trans fatty acids used in the diet (10% of energy as opposed to an average normal intake of 6% of energy). However, subsequent studies have confirmed a basis for a serious concern about the health implications of trans fatty acids. In a cross-sectional study of 748 men aged 43 - 85 years, trans-fatty acid intakes were found to be direcetly related to LDL cholesterol and to be inversely related to HDL cholesterol (Troisi et al. 1992). Two independent studies confirmed that trans 18:1 significantly elevates Lp(a), a major indicator of risk of CHD, hitherto not found to be altered by dietary fat (Mensink et al. 1992; Nestel et al. 1992). Recently the database of the Nurses Cohort Study (Willett et al. 1993) has shown a truly remarkable association between the risk of coronary heart disease and intakes of trans fatty acids, even after adjustment for eleven lifestyle and dietary factors (eg smoking, weight, family history, SFA etc). The results are summarized in Table 3 for women with a stable margarine intake over 10 years and clearly show that trans-isomers of ruminant fats are to be utterly excluded from this remarkable result of increased risk of CHD with high intakes of trans fatty acids. The main sources of trans fatty acids are hydrogenated vegetable oils used in cereal products, confectionery products, in catering fats and in margarines. These fats show marked positional isomerisation within the configurational isomer fraction. Brisson (1991) has calculated that in hydrogenated vegetable oils, the distribution of the trans double bond is normally distributed across carbons 4 to 12 on the octadecanoyl acyl chain. In contrast, there is a 70 - 80% concentration of the trans double bond in position 7 (from methyl end) of the octadecanoyl acyl chain in ruminant fats. In looking to the future, it is conceivable that food technologists will attempt to look at the cholesterol raising properties of different positional isomers of trans C18:1 (elaidic acid) in order to develop "designer-fats" : ones which fulfil the technological need without adversely effecting blood cholesterol. In the mean time, hydrogenated vegetable oils are the new bête-noir of dietary fats.

Table 3 Relative risk of CHD (1980 - 1989) to energy-adjusted trans-fatty acid intake among 69,181 women without change in amrgarine consumption (1970 - 1980)

Quintile of trans isomer intake	Trans isomers		
	All fats	Vegetable fats	Ruminant fats
1	1.0	1.0	1.0
2	1.23	1.43	0.76
3	1.11	1.11	0.69
4	1.36	1.39	0.55
5	1.67	1.78	0.59

Willett et al. (1993)

Dietary fats : non-cholesterol risk factors

A considerable part of the evidence linking dietary fat to risk factors for coronary heart disease has been derived from epidemiological studies. Such studies require very large samples and do not lend themselves to in-depth metabolic studies. Cholesterol is

ɜily and cheaply measured on autoanalysers. Therefore it features as a dominant factor the area of epidemiological studies of CHD. That volume of representation in such ɪdies does not necessarily equate with its true proportionate role in the aetiology of ID. Several areas of fat metabolism have now emerged with varying strengths of ɔssible association with coronary heart disease. It is important that these be borne in ind lest in modifying fat intake in one direction to achieve one end we create a problem sewhere.

There is ample evidence to show that fish oils play a protective effect in CHD largely ɪrough a reduction in platelet aggregation. This area has been extensively reviewed by ɛveral authors (Kinsella et al. 1990) and the evidence to support an increased intake of ɪe n-3 polyunsaturated fatty acids is now very substantial. There is also growing vidence that anti-oxidant vitamins such as vitamin E, vitamin C and B-carotene, play an ctive and protective role in CHD. The mechanism involves reducing the susceptibility of .DL to peroxidative damage. Normally cells do not accumulate cholesterol and only do ɔo in the the pathogenesis of arterial disease. Uptake of LDL is tightly regulated by the .DL receptor pathway, the number of receptors increasing and decreasing as the cells ɪeed for membranogenesis dictates. Only oxidized or other chemically modified LDL is taken up in excess and this occurs through a poorly regulated scavenger pathway. Several studies have shown that feeding high PUFA levels will enrich LDL with oxidizable long chain polyensic fatty acids. In vitro, these LDL particles are readily oxidized and taken up in excess by cultured human macrophages. Indeed there is a direct linear correlation between % C18:2 in LDL and the susceptibility of LDL to oxidative damage (Reaven et al. 1993). On an epidemiological basis there is evidence that plasma vitamin E levels are inversely related to the risk of CHD and that this variation in plasma vitamin E explains for more of the cross-national variation in CHD than variation in plasma cholesterol (Gey et al. 1991). Low fat diets, may have the disadvantage of being low in vitamin E. Retzlaf et al. (1991) found a significant fall in vitamin E intake on low fat diet, with the degree of the fall increasing as the fat level decreases. Finally there is experimental evidence of a role of dietary fats in modulating the inflammatory response in rats (Mulrooney and Grimble 1993). Tumor necrosis factor - (TNF) is produced in response to inflammatory stimuli and has a marked effect on the redistribution of tissue nitrogen concommitant with a rise in hepatic output of acute phase proteins. Rats injected with TNF show marked alterations in protein synthesis. In the liver both corn oil and coconut oil allow normal to elevated responses in protein synthesis with intraperitioneal injection of TNF. No such elevation is found in rats fed butter or fish oil. The process of atherogenesis involves many of the cells of the inflammatory process and we should be aware that dietary fat influences their function is a manner not necessarily in parallel with their influence on blood cholesterol.

Adverse nutritional effects of reduced intake of animal foods

The majority of the world is vegetarian and copes quite well without meat. Their traditional diets have evolved over centuries and pare perfectly balanced. In the developed world, foods from the animal kingdom are being increasingly shunned not always with proper care to ensure a balanced intake of nutrients. McGowan and Gibney (1993) in a case control study of nutrient intakes among adults pursuing a self-prescribed milk-free diet for the treatment of self-diagnosed food allergy, found calcium intakes to be grossly inadequate. Even allowing for the use of calcium supplements, some 25% of the subjects avoiding milk had calcium intakes below the Lower Reference Nutrient Intake (LRNI). Intakes below the LRNI are by definition “almost certainly inadequate for most

individuals. The poor perception of the nutritional value of dairy products has produced a generation of young females with calcium intakes well below that required to sustain optimal bone density. A major survey of the diets of British Schoolchildren showed that 57% of 15 year old girls were consuming less than the then recommended daily allowance (RDA) of 700mg/d (DHSS 1989). Some 17% were consuming less than 500mg of calcium per day. Recent revisions of the UK RDA has increased this value for calcium among mid-teenage girls to 100mg/d and set an LRNI of 480mg (DHSS 1991). It would thus appear that about one in five of these young women are increasing their chances of post-menopausal osteoporosis.

The most common nutritional deficiency in the developed world is that of iron in women. In the UK, National Diet and Nutritional survey, some 33% of women had serum ferritin levels below 25µg/l indicative of depliected iron stores (Gregory et al. 1990). These would be typical for European women. Heavy menstrual blood losses are likely to be a major determinant of iron deficiency anaemia in women. However, adequate iron intakes through the consumption of red meat, rich in highly bioavailable iron, is likely to reduce the extent of inadequate iron stores. Recent data shows that young women put on low-meat diets and given 12 week exercise programme of moderate aerobics, show a significant fall in plasma haemoglobin and ferritin (Lyle et al. 1992). Iron supplements redressed this but the most effective route was without any iron supplements and with an adequate intake of iron from red meat. Some 28% of UK women do not eat meat (Gregory et al. 1990). This is difficult to understand from the nutritional viewpoint since several studies have clearly shown that hypercholesterolaemia is very readily treated with red meat included in the diet (Watts et al. 1988; Kestin et al. 1989; O'Dea et al. 1990).

By far the most disturbing data on nutritional issues for women comes from Barker (1991). In a review of several studies from his group, this author provides powerful evidence that intra-uterine undernutrition programmes the developing foetus for subsequent onset of chronic disease. Table 4 provides data on the relationship between adult hypertension and placental weight and birthweight. The lower the placental weight and the higher the birthweight, the lower is mean systolic pressure. At the opposite end of the scale (highest placenta and lowest birthweight) blood pressure in adulthood is 50% higher. In essence, our obsession with fat, cholesterol and obesity may distract us from more pressing issues and may, in itself, create problems yet to become manifest.

Table 4 Mean systolic blood pressure (mmHg) of men and women aged 46 - 54 years according to birthweight and placental weight

	Placental weight (lb)			
Birthweight (lb)	- 1.0	- 1.25	- 1.5	> 1.5
- 5.5	152	154	153	206
- 6.5	147	151	150	166
- 7.5	144	148	145	160
> 7.5	133	148	147	154

Barker (1991)

Animal products and healthy eating advice

In most developed countries, meat and dairy products provide about 60% of dietary fat. It is therefore understandable that animal products have been targeted in the

nulation of dietary advice. However, this formulation has tended to be embarassingly erficial. For example, in four northern EC states for which data exists (Ireland, UK, ımark and the Netherlands) meat and dairy products contribute 50% of dietary fat ıke in subsets of the population with either high or low total fat intakes (% of energy). effect, most patterns of meat and dairy product intake do not, per se, distinguish ween those e who get it right and those who get it wrong with regard to dietary fat els (Gibney 1991). Table 5 gives data from the Northern Ireland dietary survey (Barker al. 1992) in which the contribution of foods to fat intake, adjusted for variation in ergy intake, is compared for subjects above or below 35% of energy from fat. Only two ɔd categories successfully discriminate between high- and low-fat diets for both sexes, real products (breads, cakes, pastries, biscuits) and spreadable fat. Similar such studies ross several EC countries show the same pattern or lack of it (Gibney 1991). In effect, to ɪive dietary guidelines which take account of the real world more sophisticated studies e needed to decide on which foods truly pose a problem and for whom. The several ɪisting studies are themsevles not capable of resolving this issue. They do serve to ● strate the naivety of the present approach.

able 5 Mean fat contributions (g/10MJ) of various food groups in low- and high-fat consumers in Norther Ireland

	Males			Females		
	Low-fat	High-fat		Low-fat	High-fat	
Fat energy %	_ 35	> 35		_ 35	> 35	
number	45	213		53	281	
% of sample	17	83		16	84	
Cereal products	15.7	20.1	**	17.0	22.1	**
Milk and milk products	12.5	13.8		11.6	14.9	**
Egg and egg products	3.1	5.4	**	3.6	5.5	**
Meat and meat products	17.3	26.1	***	20.6	22.8	
Chips	9.1	10.4		5.0	10.8	***
Spreadable fats	15.2	19.0	*	14.2	18.9	**

Significantly different from low-fat group : * $p < 0.05$; ** $p < 01$; *** $p < 0.001$
Barker et al. (1992)

CONCLUSIONS

In the last five years, a log-jam in the hetherto unscholarly study of the role of diet in chronic disease has been broken. The role of animal products in a healthy diet must be considered in light of new data particularly in light of new data on antioxidant vitamins, hydrogenated fats and materno-foetal nutrition.

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