Department of Preventive Cardiology,
21 Buccleuch Place,
EDINBURGH EH8 9LN

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Professor Henry Blackburn,
University of Minnesota,
Laboratory of Physiological Hygiene,
School of Public Health,
Stadium Gate 27,
611 Beacon Road,
Minneapolis, Minn. 55455

pull list + my last letter please (for copies folder) Check what reprints were sent him recently. Send last ms Houston-Rome.

Dear Henry,

Following up my recent letter, I woke during the night and, as sometimes happens, thought out a bit of the story and put it on tape by my bed.

The thoughts may not be in a strictly logical order, but perhaps may stimulate some response because I really do feel that we might be the best combination to spell out some of the points to which you referred. I am no expert in essential fatty acids and do not want to go further than prostacyclin-thromboxane, and not as far as that, ie further than 'prostaglandin' in writing for general physicians and the medical profession as a whole.

Optimal blood lipids for populations would ensure freedom from atherosclerosis and hence epidemic CHD.

Without atherosclerosis there would be no susceptibility to CHD.

Without hypercholesterolaemia (LDL-C) there would be no atherosclerosis - but this is not the whole story.

A vital fact is that similar blood cholesterol may be associated with very different incidences of CHD.

The answer is likely to be not hypercholesterolaemia, but habitual diet.

Diet is the only invariable necessary factor, and the fat story is central.

Other reasonable goals would be optimal fatty acid composition of the diet which would be reflected in that of adipose tissue, and hence in that of free fatty acids, cholesteryl esters, phospholipids in cell membranes (particularly platelets) and the endothelial barrier

It would ensure optimal platelet activity, particularly as regards the stearic-linoleic ratio.

Another reasonable goal would be optimal balance of prostaglandin activity, but this must be translated into practical terms of food and not with a view to giving pharmacological doses of, for example, eicosapentanoic acid, although this might be relevant to people with familial hyperlipidaemias.

Finally, optimal fibrinolysis could be included, and this likewise is related to the fat story.

The fact of widely varying individual susceptibility applies to virtually everything and, owing to the different varying effects of different genes, the story could be infinitely complex for those obsessed with mechanisms.

Understanding mechanisms provides confidence in the giving of advice, but is not necessary for effective preventive medicine.

The answer to the coronary problem lies in public health and not clinicians and their patients, however important this may be to individuals.

True primary prevention against atherosclerosis and the risk factors which lead to it means optimal diet in children.

Why not make this a goal?

The answer is unlikely to be in secondary and tertiary prevention owing to sudden death,

incidence /

incidence of atherosclerosis, etc.

Adults may be reluctant to change, but children are adaptable.

Atherosclerosis starts young.

Developing tissues are those with most susceptibility to injury, including relative EFA deficiency from excess saturated fat and cholesterol in the diet.

Eating habits, started early, are those most likely to persist into adult life, and they could be healthier ones.

Optimal diet therefore is the primary goal and other goals follow.

All other coronary risk factors are contributory, and often important, but they are not fundamental.

True primary prevention is likely to be much easier than secondary and tertiary prevention.

The atheromatous diet is fundamentally one rich in saturated fat and cholesterol with relative EFA deficiency, and, since 80% of saturated fat and all cholesterol is of animal origin, the subject of Cows and Coronaries is a reasonable one to be desensitised.

Essential amino acids can be equally well obtained from animal or mixed vegetable sources, but by any reasonable standards meat is a second-class source of protein.

This is because of the saturated fat and cholesterol and (thirdly) absence of fibre.

Renaud has clearly shown the importance of stearic acid in platelet membranes.

Stearic acid is thrombogenic, but not atherogenic.

The principal sources are meat and dairy fat.

Dairy fat is also high in myristic acid and experimentally it has been shown to be hypercholesterolaemic, atherogenic and thrombogenic at the same time.

Stearic acid then is an important theme.

This means both sides of the cow, ie milk (dairy fat) and meat fat, but also in part the apparently lean since animals are fattened for the market with infiltration of saturated fat into the lean where by competition they fight the EFA in the lean.

A better balance of fatty acids in adipose tissue would influence that in cholesteryl esters and hence regression (and progression) of atherosclerosis. There is abundant presumptive evidence on this, but of course no literal proof.

The fatty acid composition of free fatty acids has long been recognised.

That of phospholipids in cell membranes has been relatively neglected.

Competition between the two EFA series is likewise important.

Hence the advice to eat more fish and less meat, and more dark green vegetables.

All this is entirely rational at least in the UK where the consumption of meat has gone up and that of fish has gone down, and also that of vegetables.

The problem in the USA is beef much more than milk (dairy fat).

Consequently there should be a separate paper on this subject published in, say, the New England Medical Journal.

The largest single source of saturated fat in the UK is dairy fat (50% of the saturated fat).

Meat (principally beef) comes second.

Another pending paper then is on the remarkable habits of the Scots, but this is a separate subject at the moment.

The second characteristic of phospholipids in platelet membranes is the low concentration of linoleic acid.

Hence the importance of the fat switch.

The fat switch is not quite so important in true primary prevention, but vital (literally) for secondary and tertiary prevention.

First /

First, a more prudent diet has to be acceptable and a very low total fat diet would be acceptable to most people.

Secondly, in high incidence populations most already have atherosclerosis and the tai is arrest and regression and prevention of thrombo-embolic complications.

This is very different from primary prevention when the target is not to go Westernis

Thirdly, habitual diet in high incidence Western countries is very rich in saturated f which is almost unavoidable for those habitually consuming what is around.

Freedom of choice is largely a myth, at least in the UK owing to interference by govern ments, subsidies, inadequate labelling, excessive inappropriate promotion by powerful vested interests.

Optimal prostaglandin balance islikely to be achieved by correcting the diet to a more balanced one, as judged by evolutionary standards.

Our present diet was not planned or tested for health.

Why not change it, because change amounts in nutritional principle to dietary correction?

The im-prudent diet ~~and~~ is platelet function is being underestimated in importance and should be upgraded.

Diet is also related to fibrinolysis, but the mechanisms are less well understood.

The final goal should be optimal (reduced) postprandial lipaemia and the chylomicron story may be making a comeback.

True primary prevention trials are taking place all the time in healthy, well-nourished populations without a CHD problem.

The only advice they need is not to change in a Westernised direction.

The US and the UK need advice to change to a Mediterranean-Japanese-more vegetarian-Polynesian diet.

A little bit of each plus a little bit of what you fancy at home would make a splendid diet without effort or undue expense.

One focus then is stearic acid.

Another is to explain competition between fatty acids for the same enzyme systems for carbon chain elongation and further desaturation to more active derivatives.

Fish must be upgraded.

Describe the fat switch.

Describe the protein switch.

Describe the carbohydrate switch.

Describe the energy switch.

There ~~are~~ is no suggestions of prohibitions.

There is no suggestion of telling people what to eat, but many now enquire and more would like to know without having to ask.

The medical profession as a whole is profoundly ignorant in the UK, and some 20 years behind the USA.

They hardly know the alphabet of nutrition.

The problem howmuch legislation is reasonable has to be considered for the Conference.

It is unlikely that health education will alone suffice in socio-educational groups 4 and 5 in the foreseeable future.

Governments have no objection to legislating in favour of food producers.

Freedom of choice is largely a myth, but the public need tobe protected against interference, including price supports.

The future of the cow for the yar 2000 must be spelled out indetail.

The /

The world population will double in 30 years, mainly in the undernourished.

The economics of cereal feed rather than food must be spelled out.

Morality comes in.

Compassion of animals comes in.

There is much to support the desirable nutritional trend on other grounds which will appeal to varying groups.

Vegetarianism is a matter of personal choice, usually not made on nutritional grounds.

A return to a more vegetarian life on nutritional grounds is very sound advice, and almost common sense, and certainly common sense plus common science provides most answers.

If you consider there are any thoughts worth considering here, could you ponder on them and see how much, if any, they apply to the USA and this planet ingeneral, and the 1983 Conference inparticular.

The more papers than can be written in the next 12-18 months, the better.

They could strengthen the Conference and much could be set out in advance.

International papers are particularly desirable for an international conference and, if people have time to think about them in advance, so much the better.

The concept of primary, secondary and tertiary prevention is relevant to any talk on the P/S ratio.

Although it is the fatty acid composition rather than the fat content of diet which is most important, content, ie concentration, must clearly be considered inrelation to the ratio.

The ratio really doesn't matter if consumption of fat is sufficiently low because there is bound to be enough EFA and there is no competition from SAFA.

Cholesterol of course, like saturated fat, increases requirements for EFA.

Developing tissues of children require maximal EFA and minimal competition therefore.

This is quite enough for one letter.

With kind regards,
Yours sincerely,

Richard

R. W. D. TURNER

PS

If you work it out, everything boils down to the prudent diet now widely agreed, both qualitatively and quantitively, but with varying emphasis according to circumstances in populations and individuals.

The answer to the comment, 'All CHD cannot be explained by known risk factors' can surely largely be explained in the above letter, - and that couldbe another paper. There are about six I think.

True primary prevention of postoperative thrombo-embolism is likely to be habitual consumption of a more prudent diet.