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Symposium on 'Reducing cardiovascular disease risk: today's achievements, tomorrow's opportunities'

Modification of cardiovascular risk: lifestyle *v.* drugs

(The following are summaries by the authors of their presentations)

Cardiovascular disease risk: a round table approach. How do factors related to diet, obesity, activity and drugs contribute to a combined strategy for prevention?

Margaret Ashwell[1]*, Adrianne Hardman[2] and Michael Oliver[3]
[1]*Ashwell Associates, Ashwell Street, Ashwell SG7 5PZ, UK*
[2]*Department of Physical Education, Sports Science and Recreational Management, Loughborough University, Loughborough LE11 3TU, UK*
[3]*Cardiac Medicine, National Heart and Lung Institute, Imperial College School of Science Technology and Medicine, London SW3 6LY, UK*

The round table model has already been used to emphasize the role that many dietary components play in the prevention of cardiovascular disease (Ashwell, 1997). It allows the different components to be matched against the corresponding physiological risk factors which can then, in turn, be matched against the corresponding pathological event leading to a heart attack, i.e. injury to coronary arteries, atheroma and fibrous plaque formation and thrombosis. Fig. 1 shows an updated version of this model. A newly-identified physiological risk factor has been added, i.e. endothelial function; there is preliminary evidence that folate and the antioxidant vitamins can improve this function. New dietary components have also been added where there is some evidence that they beneficially affect previously-identified physiological risk factors, i.e. K (which can help prevent raised blood pressure), flavonoids and polyphenols (which help to protect against lipid oxidation), and soyabean protein phyto-oestrogens and plant sterol esters (which might improve the atherogenic lipid profile). Fig. 2 extends the principle of the round table model to show how the different obesity-related factors, i.e. total fat and visceral (central) fat, affect the same physiological risk factors and pathological events (for review, see Ashwell, 1996).

Physical activity is not a single entity and it can be categorized according to type (endurance, strength, speed or power) and amount (intensity, duration, frequency). The effects of exercise can be short-term (lasting minutes or hours after an exercise session) or long-term adaptations (persisting for days or weeks after a programme of regular exercise is discontinued). In Fig. 3, the long-term effects of physical activity have been portrayed with particular reference to the frequency and intensity of endurance-type exercise (for review, see Hardman, 1999).

Fig. 4 shows how the same model can be used to portray the role that various pharmacological agents such as statins, and anti-hypertension agents, can play in the prevention of cardiovascular disease. Recent guidelines for the prevention of CHD have been published by the British Cardiac Society, the British Hyperlipidaemia Association and the British Hypertension Society (Wood *et al*. 1998). Cessation of smoking is also included here.

It is important to realize that all the round table models are intended only as 'aide memoires'; the presence of a component in each model is based on a general consensus of opinion, but the weight of the evidence cannot be portrayed, and this evidence is much stronger for some relationships than others.

Our best strategy for preventing cardiovascular disease at the close of the second millennium appears to be a combination of the cessation of cigarette smoking, the avoidance of visceral obesity, a diet rich in fish, cereals and fruit and vegetables, frequent moderate exercise and control of hyperlipidaemia and hypertension.

***Corresponding author:** Dr Margaret Ashwell, fax +44 (0)1462 743166, email Ashwell@compuserve.com

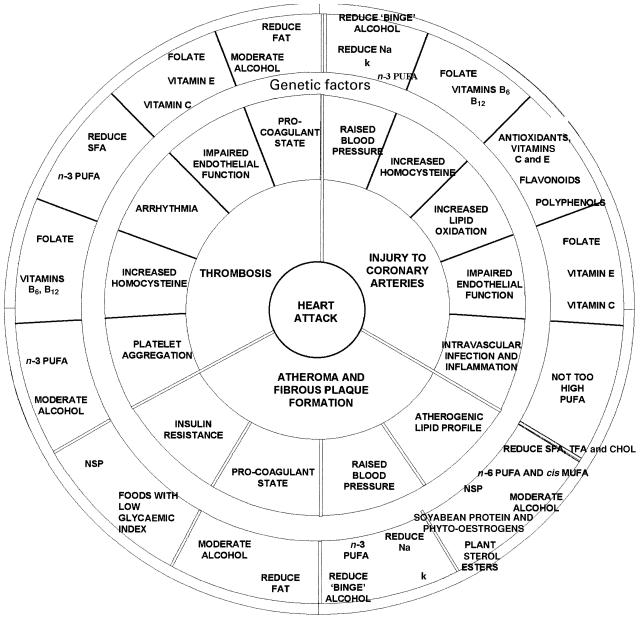

Fig. 1. A round table model of dietary factors in the prevention of coronary disease. CHOL, cholesterol; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids; TFA, trans fatty acids.

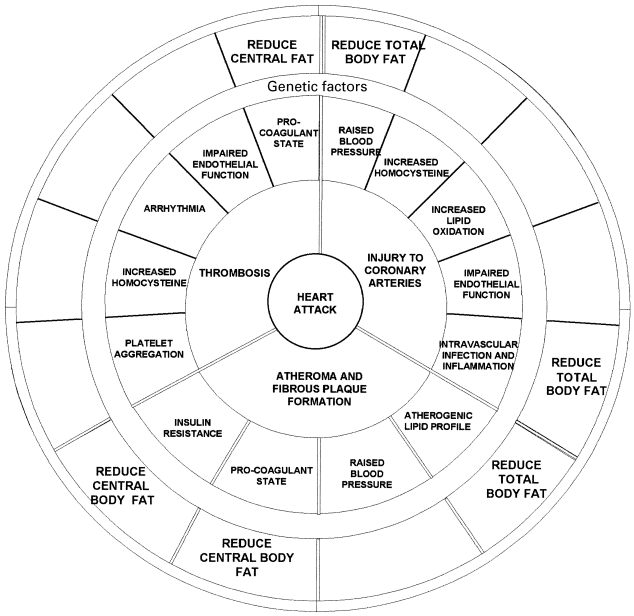

Fig. 2. A round table model of obesity-related factors in the prevention of coronary disease.

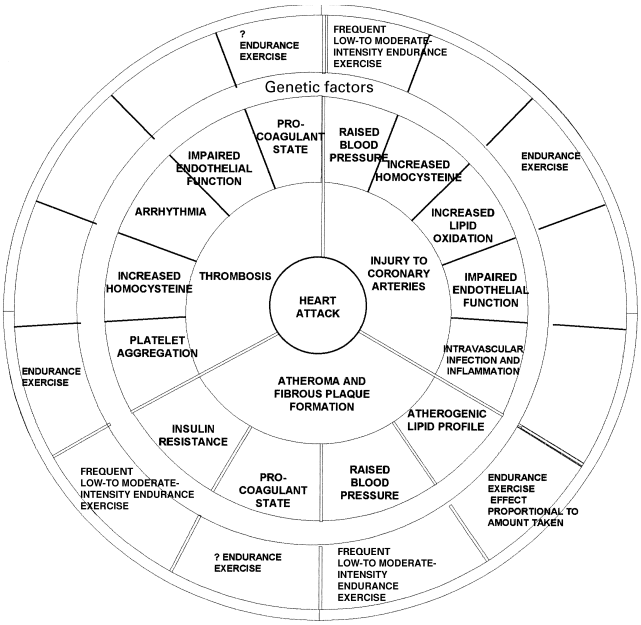

Fig. 3. A round table model of activity-related factors in the prevention of coronary disease.

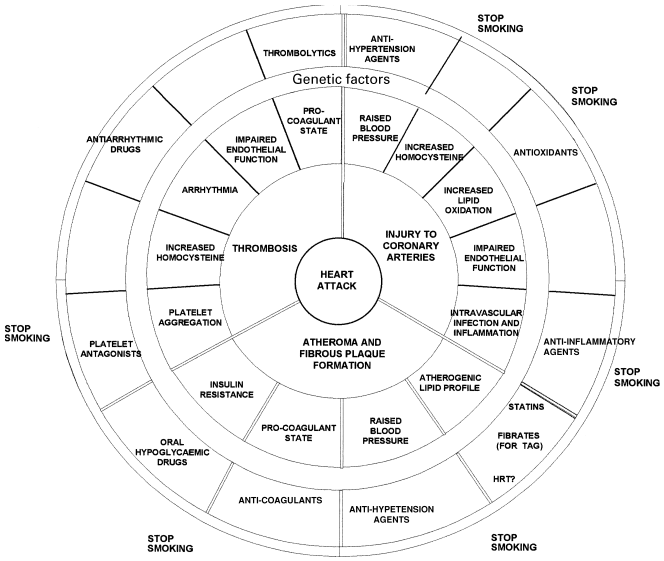

Fig. 4. A round table model of pharmacological factors in the prevention of coronary disease. TAG, triacylglycerol; HRT, hormone-replacement therapy.

Round Table model: Cardiovascular disease: Risk factors: Prevention strategy

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