Correspondence

Death railway and Weary-Dunlop Boonpong Fellowship Program

Sir,

I read with interest the article by Robson and colleagues.\(^1\) The authors state that the death railway was a track from Boon Pong in Thailand to Thanbyuzayat in Burma. I disagree. It should be Ban Pong, rather than Boon Pong.\(^2\) However, Mr Boonpong Sirivejjabhandu was a Thai businessman during the World War II. He provided support to many then prisoners who were captured by the Imperial Japanese Army to build the death railway. Sir Ernest Edward ‘Weary’ Dunlop, a well-known Australian surgeon also received such help while captured. Even both of them passed away, the friendship between Boonpong and Dunlop exists until nowadays. Weary-Dunlop Boonpong Fellowship Program is a scholarship for Thai surgeons to study surgery in Australia.

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References


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Why the overstated beneficial effects of statins do not resolve the cholesterol controversy

Sir,

Atherosclerosis is believed to be a chronic inflammatory disease affecting the intima of arteries.\(^1\) Controversy continues to surround its etiology and pathogenesis, particularly with respect to cholesterol and lipids.\(^2\) However, in his review,\(^3\) Thompson asserted that the cholesterol controversy should be considered overtaken by the discovery and development of statin drugs. We suggest that hypercholesterolemia does not represent a ‘causa sine qua non’ for the development of atherosclerosis and that the beneficial effects of statins are overstated and do not resolve the cholesterol controversy.

It is well known that patients may present with coronary heart disease (CHD) events despite low low-density lipoprotein (LDL) levels, which fall well within guideline-recommended targets. Indeed, it has been shown\(^4\) that half the patients hospitalized with CHD had admission LDL <100 mg/dl, and LDL <70 mg/dl was observed in 17.6% of patients; less than one-quarter of patients had an admission LDL >130 mg/dl.\(^4\)

Not surprisingly, accelerated severe atherosclerosis and its complications can be produced experimentally in herbivores with serum cholesterol levels below 100 mg/dl under conditions analogous to those prevailing in humans.\(^2\) Furthermore, lipid and cholesterol crystals are found as a nonspecific pathological change in many chronic degenerative and inflammatory diseases and cannot be assumed to be causal. In particular, the histological features of tuberculosis resemble what has been described in atherosclerosis;\(^5\) hypercholesterolemia is not a prerequisite for such pathogenetic events. On the contrary, hypocholesterolemia, beyond being a common finding in different forms of pleuropulmonary tuberculosis, may represent a deleterious factor for the host in its fight against mycobacteria.\(^6\)
On the other hand, in primary prevention, statin therapy reduces the risks of CHD events for men but not for women, without reducing total mortality for either men or women. Furthermore, there is reason to believe that the beneficial action of statin drugs is mediated in spite of their cholesterol-lowering effects. Indeed, statins have an effect only when patients have a heightened inflammatory state. Finally, it has been shown that low total cholesterol is an independent and robust predictor of future external-cause mortality, and this association could not be explained by other obvious interrelated risk factors.

As Thompson stated, the cholesterol controversy in Britain dates back to 1950. Changing our current practice pattern could take other 50 or more years, but we may one day prescribe cholesterol-raising medications to certain patients.

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History of the cholesterol hypothesis in Britain
Sir,
‘The statins trials resolved the controversy’ is an error of scientific thought, and it is sad that so many respected clinical scientists such as Prof. G.R. Thompson fail to appreciate this.

Yes, there has been a controversy concerning cholesterol, which is clearly a risk indicator, not for just coronary heart disease (CHD) but for all deaths, but only in men of working age. We know from Framingham studies that cholesterol is neutral in women and older men and we know from other studies that a high blood cholesterol is a survival advantage in elderly people. The question following the MRFIT and Whitehall studies is whether the elevated level of cholesterol is a risk indicator on a causative pathway or whether it is a reflection of existing sub-clinical CHD. Subsequent studies have shown that it matches the inflammatory marker C-reactive protein very closely.