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ABSTRACTS IN CARDIOLOGY

A new marker for atherosclerosis?

Heat shock or stress proteins are generally thought to be cardioprotective. Several, including hsp65, show increased expression in the cells of human atherosclerotic plaques, where they may be acting as molecular chaperones.

However, Xu *et al* have shown that antibodies to hsp65 are independently associated with carotid atherosclerosis in the over 60s. What are the origins of these antibodies and do they play a part in the pathogenesis of atherosclerosis? It may be that the antibodies are produced in response to human hsp65 expressed by stressed cells during the early stages of atherosclerotic plaque formation.

This would suggest that atherogenesis has an autoimmune component. In individuals with atherosclerosis antibodies to hsp65 might represent a physiological immune regulatory phenomenon, or the antibodies may be pathological—directed against the plaque cells. There is a further possibility. Because the stress proteins are phylogenetically well preserved, the antibodies may have arisen against bacterial hsp65, implying an infective aetiology for atherosclerosis.

Whatever their origin and function, such antibodies may turn out to be a useful marker of atherosclerosis.

S A THORNE

Association of serum antibodies to heat-shock protein 65 with carotid atherosclerosis

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Abstract

Arteriosclerotic lesions can be induced in normocholesterolaemic rabbits by immunisation with heat-shock protein (hsp) 65, a stress protein expressed in high concentrations in human atherosclerotic lesions. If an immune reaction to hsp65 also plays a part in human atherogenesis, it should be possible to detect anti-hsp65 antibodies in patients with atherosclerotic lesions.

To study the possible relation between immune reaction to hsp65 and atherosclerosis, 867 normal inhabitants of South Tyrol, aged 40-79 years, were selected randomly for determination of serum antibodies against hsp65, simultaneous sonographic assessment of carotid atherosclerotic lesions, and evaluation of established risk factors—ie, blood cholesterol, hypertension, smoking, diabetes

mellitus, and obesity. Autoantibodies to nuclear antigens, thyroid antigens, and rheumatoid factors were also measured. Serum anti-hsp65 antibodies were significantly ($p < 0.05$) increased in subjects aged 60-79 years with carotid atherosclerosis compared with those without lesions, and increased antibody concentration was independent of age, sex, and other established risk factors. On the other hand, the incidence and titres of autoantibodies did not correlate with carotid atherosclerotic lesions.

Our data provide the first evidence of a strong correlation between anti-hsp65 antibodies and carotid atherosclerosis, suggesting that hsp65 might be involved in the pathogenesis of atherosclerosis. (*Lancet* 1993;341:255-59.)