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1: [Growth Horm IGF Res.](#) 2003 Aug;13 Suppl A:S72-84.

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Testosterone and atherosclerosis.

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Hypoandrogenemia in men and hyperandrogenemia in women are associated with increased risk of coronary artery disease but also with visceral obesity, insulin resistance, low high-density lipoprotein (HDL) cholesterol, elevated triglycerides, low-density lipoprotein (LDL) cholesterol and plasminogen activator inhibitor (PAI-1). These gender differences and confounders render the precise role of endogenous androgens in atherosclerosis unclear. Exogenous androgens, on the other hand, induce both apparently beneficial and deleterious effects on cardiovascular risk factors by decreasing serum levels of HDL-C, PAI-1 (apparently deleterious), Lp(a), fibrinogen, insulin, leptin and visceral fat mass (apparently beneficial) in men as well as women. However, androgen-induced declines in circulating HDL-C should not automatically be assumed to be pro-atherogenic, since it may reflect accelerated reverse cholesterol transport instead. Short-term application of supraphysiological doses of exogenous T can reduce the severity and frequency of angina pectoris and improve the electrocardiographic signs of myocardial ischaemia; long-term effects have not been investigated. Nonetheless, interpretations of the effects of pharmacological doses of androgens on arterial compliance and flow-mediated dilatation in particular must be treated with circumspection also because at physiological concentrations, beneficial, neutral, and detrimental effects on vascular reactivity can be observed. Testosterone exerts 'pro-atherogenic' effects on macrophage function by facilitating the uptake of modified lipoproteins and an 'anti-atherogenic' effect by stimulating efflux of cellular cholesterol to HDL. In the majority of animal experiments, exogenous testosterone exerted neutral or beneficial effects on the development of atherosclerosis. In conclusion, the overall effect of administration of testosterone on cardiovascular-disease risk is difficult to assess because androgens have such an extraordinary array of effects in vivo. When dealing with a complex multifactorial condition such as CAD, it is premature to assume that clinical benefits can be derived from manipulation of the sex steroid milieu - even when these assumptions are based on biologically plausible mechanisms or, indeed, on cross-sectional risk-factor observational data. Neither needs the therapeutic use of testosterone in men be restricted by concerns regarding cardiovascular side effects.

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