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Testosterone and cardiovascular health after menopause, a benefit or risk?

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Testosterone (T) is an essential precursor for estrogen biosynthesis, primarily in non-gonadal tissues in postmenopausal women. At physiological concentrations T is a vasodilator, and has favourable effects on endothelial function and peripheral vascular resistance. T enhances endothelium-dependent (flow mediated) and endothelium-independent arterial vasodilation. Whether endogenous T protects against CVD (ischaemic heart disease and ischaemic stroke) and death is unclear. Most, but not all studies, have shown that low total and free T is associated with greater CVD risk. However available studies have significant limitations including small sample size, inclusion of convenience, clinic-based samples, case-control design and long intervals between the time of blood draw and CV events.

The association between T and CVD risk cannot be interpreted in isolation from the effects of sex hormone binding globulin (SHBG). SHBG binds T with high affinity, and taking into account binding to albumin, only 1-2% of T circulates unbound (free). In postmenopausal women, as in women with PCOS, low SHBG, not elevated total T, is significantly associated with a more adverse lipid profile (higher levels of triglycerides and lower HDL-cholesterol) 1, visceral fat accumulation 2 and increased risk of diabetes 3. Studies of postmenopausal women have reported that the free androgen index (FAI) but not total T is associated with the metabolic syndrome and CVD risk 4, 5. These studies indicate that SHBG, not T, may be driving the association between the FAI and CVD risk in both PCOS and the postmenopausal milieu.

In summary there is a need to clarify the associations between androgens, and CV events and total mortality in women. The associations between SHBG and CVD risk, CVD events and total mortality need to be clearly ascertained, as SHBG may prove to be an important CVD risk biomarker in women.

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