mortality was reduced towards zero (e.g., a reduction of 0.26% coronary heart disease risk per 1 g/day \(^{-1}\) of arginine, \(P=0.53\), adjusted for saturated fatty acids and smoking; additional adjustment for flavonoids was hampered by multicollinearity). These results indicate that arginine is not clearly associated with coronary heart disease mortality at the population level. This is in agreement with our findings at the individual level\(^4\).

In both study designs, arginine intake was strongly associated with the intake of energy and other nutrients through its widespread presence in animal and vegetable foods. The potential impact of arginine, therefore difficult to disentangle. Additional observational and animal experiments.

of arginine is therefore difficult to cultivate important. It's widespread presence in animal and vegetable foods. The potential impact of arginine, therefore difficult to disentangle. Additional observational and animal experiments.

**Men with coronary artery disease have lower levels of androgens than men with normal coronary angiograms**

We read with great interest the paper by English et al.\(^1\) which demonstrated lower levels of androgens in men with coronary artery disease compared to those with normal coronary angiograms. We believe that this paper raises important questions on the still unresolved issue related to the effects of sex hormones on coronary atherosclerosis. We would like to make a few comments with regard to the results of this study, which we think are of special importance.

The subjects included in the study of English et al. differed in respect of age and body mass index, which can affect sex hormone levels. In support of this, they found no difference in total and free testosterone levels between either group after correcting for age and body mass index. However, the difference in bioavailable testosterone and free androgen index remained statistically significant, raising the question of low androgens as a risk factor in male atherosclerosis. We conducted a similar angiographic study of 337 age- and body mass index matched men, which showed no significant difference with respect to oestrogen, free and total testosterone levels in patients with coronary artery disease compared to those with normal coronaries\(^5\). We believe that the relationship between male sex hormones and cardiovascular disease is still a matter of debate. The results of several studies related to the causative role of male sex hormones in coronary artery disease are conflicting and do not provide consistent evidence. The finding of low levels of endogenous testosterone in coronary artery disease has been shown mostly in case-control studies\(^6\)-\(^9\). However, they are not proven with prospective studies, which indicated that baseline serum levels of testosterone were unrelated to the risk of subsequent coronary artery disease\(^7\)-\(^9\).

English et al. showed decreased levels of oestrogens in patients with coronary artery disease, a result which is contradictory to the results of most published studies, including ours which reported either normal or moderately increased oestrogen concentrations in patients with coronary artery disease\(^5\)-\(^9\). We believe that their result is mostly due to methodological problems.

If low androgen status were a predisposing factor for coronary atherosclerosis, we would expect the lowest androgen levels to be in patients with the most extensive disease. This finding was inconsistent in some recent studies\(^6\)-\(^9\). On the contrary English et al. showed no association between plasma androgen levels and the degree of coronary atherosclerosis with regard to the number of diseased vessels. In our study, the extent of coronary artery disease was graded with the Gensini score\(^5\). However, similar to English et al. we found no correlation between age-adjusted sex hormone levels and the Gensini score in patients with coronary artery disease.

We believe that the role of sex steroids in male atherosclerosis, both the presence and extent, needs to be further clarified with large prospective studies before low testosterone levels can be claimed as a risk factor.

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