

Sex hormone response to alcohol

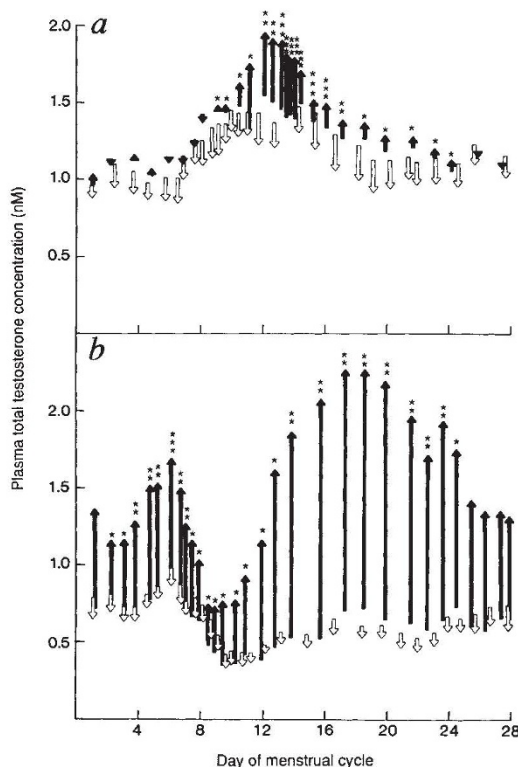
SIR — It is well known that acute alcohol increases subjective feelings of sexual arousal, excitement and desire¹. The controversial suggestion has been made that the sex steroid testosterone could be one factor involved in the basic regulation of these feelings and behaviour^{2,3}. It has been shown, however, that acute alcohol ingestion, if anything, tends to decrease blood testosterone levels in normal healthy men⁴. This makes it unlikely that heightened sexual arousal would be mediated by acute testosterone changes induced by alcohol intake in men, and, so far, testosterone has not been linked to the increased sexual excitement observed in women. But such a linkage could be possible, as we have found that alcohol, even at a very low dose, increases blood plasma total testosterone concentrations very quickly in women but not at all in men.

We gave healthy premenopausal women a low dose (0.34 g kg⁻¹) of alcohol, or placebo juice, on two separate occasions starting at 6 p.m. Testosterone levels decreased slightly during placebo conditions, but alcohol increased the testosterone concentrations (see figure). In the subjects not using contraceptives, the testosterone changes were most pronounced during the ovulatory phase, when basal testosterone was also at its maximal concentration. In the group using oral contraceptives, and thus having substantially lower (average 50%) basal levels than non-users, marked elevations were observed after alcohol intake, with the maximal changes appearing around days 17–20. During these days, the testosterone levels after alcohol exceeded those during corresponding days in subjects not using contraceptives. The steep decrease of basal testosterone levels during days 6–10 was probably due to the start of the daily ingestion of contraceptive pills after menstruation.

In corresponding experiments on 48 young healthy men, no effects on testosterone were observed as a function of alcohol. In another study, the effects of the alcohol dose (0.34–1.02 g kg⁻¹) and time after drinking (40–150 min) were investigated in 10 women using oral contraceptives. Alcohol markedly increased testosterone levels but no dose or time effects were observed.

Acute alcohol-induced elevations of testosterone levels in women have not

been reported previously. This could be due to the fact that the effect is particularly clear only in women taking oral contraceptives and in non-users during the ovulatory phase. Moreover, previous reports have included fewer than 10 subjects per study^{5–7}, and the present work is, to our knowledge, the first major investigation on this topic. Further, detection of the clear effects of alcohol was contingent



Average placebo (open arrows) and alcohol-induced (closed arrows) testosterone changes during different days of the menstrual cycle. *a*, Women not taking the contraceptive pill; *b*, women taking the pill. Each arrow marks the mean ($n=7$) of the average changes during 1 and 2 hours after the start of drinking. The diagram is arranged so that each arrow shares 6 data points with arrows adjacent to it, 5 with arrows 1 position removed, 4 with arrows 2 positions away, and so on, and none with arrows 7 or more positions away from it. Matched *t*-tests were used to calculate the significance (* $P<0.05$, ** $P<0.01$, *** $P<0.001$) between the placebo and alcohol-induced changes. Further experimental details are available from the authors by request.

on subjects receiving a placebo to control for confounding circadian effects, and on the phase of the menstrual cycle and the use of oral contraceptives. Nevertheless, the present results are consistent with one earlier study in which no testosterone changes occurred after alcohol, despite a decrease after the placebo, during days 21–22 of the cycle⁷.

The cause of the alcohol-mediated testosterone elevation in women remains unknown. The fact that no clear dose or

time effects were observed may suggest that alcohol metabolism is involved in testosterone increase.

Although the levels of free testosterone and/or its metabolites are thought to be the active component, we measured total testosterone levels in the present study because the effects in the initial studies were so clear in women. Free testosterone and sex-hormone-binding globulin levels should also be assessed in future, especially in men, because of the failure to find any significant changes in their total testosterone concentrations after they had taken low doses of alcohol.

Our results, demonstrating a testosterone elevation in women but not in men, may provide a physiological explanation for subjective discrepancies^{8–10} in sexual excitement reported by men and women after drinking. While subjective excitement co-varies with penile tumescence in men, both effects being suppressed at higher alcohol concentrations⁹, elevated sexual arousal remained in conjunction with increasing estimates of intoxication in women, although vaginal responsiveness was also reduced^{8,10}. There is as yet no convincing explanation for these gender differences. The alcohol-mediated testosterone elevation discovered here may provide at least a partial answer, assuming that testosterone and/or its active metabolite(s) could trigger feelings of sexual excitement.

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1. Crowe, L. C. & George, W. H. *Psychol. Bull.* **105**, 374–386 (1989).
2. Bancroft, J. J. *Br. med. Bull.* **37**, 153–158 (1981).
3. Alexander, G. M. & Sherwin, B. B. *Psychoneuroendocrinology* **18**, 91–102 (1993).
4. Välimäki, M. & Ylikahri, R. *Alcohol Alcohol.* **18**, 313–320 (1983).
5. McNamee, B. *et al. Br. J. Addict.* **74**, 316–317 (1979).
6. Becker, U. *et al. Drug Alcohol Depend.* **22**, 141 (1988).
7. Välimäki, M., Härkönen, M. & Ylikahri, R. *Alcohol. clin. exp. Res.* **7**, 289–293 (1983).
8. Wilson, G. T. & Lawson, D. M. *J. abnorm. Psychol.* **85**, 489–497 (1976).
9. Wilson, G. T. & Lawson, D. M. *J. abnorm. Psychol.* **85**, 587–594 (1976).
10. Wilson, G. T. & Lawson, D. M. *J. abnorm. Psychol.* **87**, 358–367 (1978).

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