

Correspondence

Alcohol consumption in pregnancy

Sir,

We would like to draw attention to the following points in the paper by Davis, Partridge, and Storrs¹ in which they conclude that 'there is no safe level of alcohol consumption in pregnancy'. In this paper 973 women were followed up after filling in questionnaires on alcohol consumption in the antenatal period. Of 479 births to women who claimed to drink no alcohol, there were 11 abortions, 5 perinatal deaths, and no infants with major malformations. Of the 494 infants born to women who drank at least some alcohol, there were 11 abortions, 7 perinatal deaths, and 6 infants with major malformations. Thus, although there was no association with mortality, there was said to be significant association at the 1% level with major malformations. In fact the difference in incidence between drinkers and non-drinkers is only just significant at the 5% level with Yates's continuity correction applied ($\chi^2 = 4.04$, $P = 0.044$). A 2 tailed exact test leads to a similar result ($P = 0.034$).

In fact, the 6 cases of malformation all occurred among the 359 pregnancies of mothers with low average daily intakes of alcohol (1-10 ml), and it may be that the authors have tested this group against all others. It is not clear whether all 8 groups were used, but if so then the large number of very small expected frequencies would invalidate the use of the χ^2 test. It is, in addition, statistically frowned on to examine the data and then decide which groups to test against the others. In this particular case it is illogical as well as unsound to take the moderate drinkers in isolation. Thus, both the marginal significance of this single result and the observation that it is due to the low abnormality rate for the non-drinkers, rather than a high rate for the drinkers compared with the area population, leads to doubt concerning the general conclusion.

In addition, the authors analysed data on the birthweights and head circumferences of all infants, according to the gestational age at delivery. There were no significant differences between the mean birthweights or mean head circumferences at each gestational age between the infants of all drinkers and those of non-drinkers. The authors, however, stated that infants of mothers drinking over 20 ml of alcohol per day did have smaller head circumferences.

This general conclusion was reached on the basis of 3 selected subgroups. At gestational ages 35, 38, and 39 weeks the average head circumference of children of heavy drinkers was claimed to be significantly smaller (presumably relative to non-drinkers). No such differences were found for this outcome at any other gestational age. In addition, the results of the *t*-test were based on samples of only 1, 4, and 5 cases respectively. They are unlikely to be representative of the relevant populations.

In summary, while the few positive results are open to doubt, most of the comparisons made between the drinking groups give no evidence to reject the null hypothesis (of no drinking effect). Most of the defects in this study were based on the dubious method of searching for significant effects among small subgroups. The basic design of the study, however, is admirable, and a repeat of the survey with larger numbers should help to clarify the problem of whether moderate drinking has a harmful effect on the fetus.

Reference

- ¹ Davis PJM, Partridge JW, Storrs CN. Alcohol consumption in pregnancy. How much is safe? *Arch Dis Child* 1982; 57: 940-3.

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Sir,

We would like to suggest that the methods and results of the study by Davis *et al*¹ are subject to several important methodological and statistical flaws.

(1) Of 1120 initial invitations to participate there was a combined non-response and unknown outcome rate of 13.1%. There is no mention of any attempt to check whether non-respondents and those lost to follow up differ in any important way from the achieved sample with respect to certain standard criteria such as age, parity, social class, and smoking behaviour (presumably this information would have been available from antenatal records). Clearly, bias in response or outcome follow up (such as heavier drinking among non-respondents or higher rates of fetal loss among those lost to follow up) may have seriously compromised the results of the study.

(2) No standardisation is made in the results for the variability in gestational age at recruitment to the study, despite suggestions in the published reports that the effects of alcohol on the fetus may vary during pregnancy.²

(3) The authors obtain a significant result for the difference between the proportion of congenital abnormalities among babies of non-drinkers in their sample and the population of South Warwickshire.

However, the proportions (%) of congenital abnormalities among the groups are actually as follows:

	Sample	South Warwickshire
	Non-drinkers	Drinkers
	0.0	1.23
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	0.62	
		1.64

It can be seen that the proportion among the study population is in fact less than half that for South Warwickshire (0.62% as opposed to 1.64%, $P < 0.01$). The obvious