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- *All letters must be typed with double spacing and signed by all authors.*
- *No letter should be more than 400 words.*
- *For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the BMJ.*
- *We do not routinely acknowledge letters. Please send a stamped addressed envelope if you would like an acknowledgment.*
- *Because we receive many more letters than we can publish we may shorten those we do print, particularly when we receive several on the same subject.*

Heroin addicts, AIDS, and aflatoxins

SIR,—Dr S M Lamplugh and others (2 April, p 968) show that in Africa many children are continuously or intermittently exposed to aflatoxins, and this is supported by other studies.[1,2] Aflatoxins have oncogenic and immunosuppressive effects that may influence the pattern of infection with the human immunodeficiency virus (HIV), which seems to be more prevalent and aggressive in African children than would be predicted from experience elsewhere.

In Britain HIV infection in heroin addicts is a cause for concern. Heroin arrives here by circuitous and clandestine routes after storage and packaging in various unconventional ways. We considered it likely that heroin on sale in the United Kingdom would be naturally contaminated before the dealers added their various contaminants. We contacted the Merseyside drug squad to obtain samples of heroin for analysis to determine whether it showed evidence of contamination with aflatoxin.

Eleven samples were supplied to us and analysed under police surveillance (at our request) in our laboratories.

The first six were contaminated heavily by undetermined substances and required complicated extraction procedures before they could be analysed for their aflatoxin content. Two of these samples were positive for aflatoxin B_1 and contained 507 and 1346 pg/g of heroin respectively. Because of the clean up procedures needed we are confident that the amount of aflatoxin recovered was an underestimate of the true content of the samples. The second batch of five samples was purer and required less preparation before analysis. One of these samples contained 9616 pg of aflatoxin B_1 in each gram.

Intravenous heroin users are probably intermittently injecting aflatoxin B_1 into their systemic circulation. An important difference between this kind of exposure and that from food contamination is that after absorption from the gut some or all of the aflatoxin is detoxified by the liver. Intravenous heroin users thus risk direct systemic exposure to aflatoxin B_1. One consequence of this is suppression of cell mediated immunity.

In view of the unusual epidemiology and aggressive behaviour of HIV infection in children in Africa, who are highly exposed to aflatoxins, and the serious problem of HIV infection in drug abusers in Britain, we think that the interaction of HIV infection and aflatoxin exposure demands urgent further investigation. We have delayed reporting these results pending analysis of urine samples from heroin addicts to detect aflatoxin excretion. Unfortunately, almost a year after our first approaches to colleagues working with drug addicts we are still awaiting specimens. We would be pleased to examine urine for aflatoxin content from any drug users currently injecting heroin.

R G HENDRICKSE
S M MAXWELL

Liverpool School of Tropical Medicine,
Liverpool L3 5QA

1 Hendrickse RG, Coulter JBS, Lamplugh SM, *et al.* Aflatoxins and kwashiorkor: a study in Sudanese children. *Br Med J* 1982;285:843-6.
2 Coulter JBS, Hendrickse RG, Lamplugh SML, *et al.* Aflatoxins and kwashiorkor: clinical studies in Sudanese children. *Trans R Soc Trop Med Hyg* 1986;80:945-51.

β Blockers, lipids, and coronary atherosclerosis

SIR,—We fully agree with Dr Robin J Northcote (12 March, p 731) that, though there is convincing evidence that β blockers alter the plasma lipoprotein profile,[1-3] it would not be advisable to abandon their use in patients with hypertension or coronary artery disease. There is compelling evidence from several studies[4-6] that β blockers administered long term after myocardial infarction reduce both mortality (by 26-39%) and the incidence of non-fatal infarction, the favourable effect persisting at least for three years. In the β blocker heart attack trial propranolol decreased the incidence of sudden death by 13% in patients without heart failure and by 47% in patients who had prior heart failure by improving ischaemia and preventing reinfarction.[7]

We estimated serum total cholesterol, triglyceride, lipoprotein cholesterol subfractions (high density (HDL), low density, and very low density), apo A, apo B, and ratios obtained from these variables in 56 patients with coronary artery disease. Based on their smoking habit and intake of propranolol (120 to 240 mg daily for over four weeks) the patients were subgrouped as follows: (*a*) non-smokers not taking propranolol (n=10); (*b*) smokers taking propranolol (n=12); and (*c*) non-smokers taking propranolol (n=10). Significantly lower values ($p<0{\cdot}01$) of HDL cholesterol,