of the four who believed strongly in iridology initially (belief >90%) also did not change their opinion. Most respondents, however, were less decided before they read the report. Thirty eight (20 of papers in the *BMJ*, 11 of papers in *J R Coll Gen Pract*, and seven of papers in the journals of alternative medicine) were uncertain (belief 40-60%) before reading the report, but two thirds of these (14, nine, and three respectively) did not believe in iridology (belief <10%) after reading it.

Four authors indicated that their belief before reading the report was 0% and one that his belief after reading it was 100%. The paper's impact on the 73 other authors was expressed as a likelihood ratio, which was calculated by dividing the odds of belief after reading the report by that before reading it. For example, if a reader's belief changed from 60% to 10% the likelihood ratio = $(0.1/0.9)/(0.6/0.4) = 0.07$. The median likelihood ratio was 0.07, with the second quartile = 0.03 and the fourth quartile = 0.28. The curved lines in the figure that represent these likelihood ratios fitted reasonably well with the data, especially at beliefs below 60% before the report was read. The median likelihood ratios for authors of papers in the *BMJ*, *J R Coll Gen Pract*, and the journals of alternative medicine were 0.06, 0.06, and 0.17 respectively.

Comment

I restricted the inquiry to authors in medical journals as they may be better able to judge empirical evidence. The respondents' beliefs and their propensities to change them may have been different from those of the authors who did not respond. Regression to the mean was not a problem, especially among the authors who reported a previous belief in iridology of about 50%:

Belief in iridology as a diagnostic aid among 78 authors of papers in "*BMJ*," "*J R Coll Gen Pract*," and three journals of alternative medicine before and after reading a report that gave empirical evidence against its validity. Curved lines indicate median, second quartile (P_{25}) and fourth quartile (P_{75}) likelihood ratios

their change to strong disbelief was impressive. Participation was lowest among doctors of alternative medicine. The report's impact on such doctors was large but less pronounced than that on other doctors (likelihood ratio closer to one). In general the likelihood ratio seemed to describe the results reasonably; its usefulness for measuring the longer term impact of a publication needs further study.

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Aflatoxins and heroin

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Aflatoxins are oncogenic and immunosuppressive.¹ In Africa, where many children are exposed to aflatoxins,^{2,3} infection with HIV seems to be more prevalent and aggressive than might be predicted. In Britain users of intravenous heroin are prone to infection, particularly with hepatitis B virus and HIV, which seem peculiarly aggressive in these drug abusers. Heroin is produced in subtropical countries from plants and may be susceptible to contamination by aflatoxins. We investigated whether aflatoxins play a part in the pathogenesis of disease in heroin abusers.

Subjects, methods, and results

The Merseyside drug squad supplied 13 samples of heroin that had been seized. These were analysed for aflatoxins. Urine samples were collected from 133 heroin abusers who were attending clinics in Amsterdam (60), Merseyside (61), and London (12); the samples were rendered anonymous. Urine specimens were also obtained from 99 male volunteers of a similar age (17-40) in Merseyside, who served as controls. All samples were extracted in chloroform and passed through a silica column.⁴ Duplicate samples of heroin were extracted by the method of Haladay and Lansden.⁵ Aflatoxin concentrations were determined by high performance liquid chromatography with detection of fluorescence.

Four of the 13 samples of heroin tested contained aflatoxin B₁ (in concentrations of 1.63, 4.31, 5.23, 30.82 nmol/kg). Aflatoxins were detected in 27 (20%) of the 133 urine specimens from the drug users and were more common in the specimens from Amsterdam (19/60) than from Merseyside (8/61). No specimen from London contained aflatoxins. Two of the 99 control urine specimens contained aflatoxins. The table shows the aflatoxins that were detected and their concentrations.

Results of analysis of urine for aflatoxins in heroin addicts and normal controls

Subjects	No tested	No (%) positive	Aflatoxins detected (nmol/l)
Heroin addicts Amsterdam	133	27 (20)	B ₁ , 8 patients (2.02-25.80) M ₁ , 6 patients (1.03-29.09) M ₂ , 6 patients (0.4-1.88) B ₂ , 2 patients (0.09, 0.13) Aflatoxinol, 1 patient (0.32)
Merseyside	61	8 (13)	B ₁ , 1 patient (0.73) M ₁ , 6 patients (0.12-1.46) B ₂ , 6 patients (0.13-1.53)
London Controls	12	0	
Merseyside Controls	99	2 (2)	B ₂ , 2 controls (0.13-0.24)

Comment

These results show that a hitherto unsuspected group of people are exposed to aflatoxins, which are injected intravenously. An important difference between this kind of exposure and that from contaminated food is that after absorption from the gut some or all of the aflatoxin in food is detoxified by the liver. Intravenous heroin users thus risk direct systemic exposure

to aflatoxin B₁; one consequence of this might be suppression of cell mediated immunity.

Aflatoxins were detected more commonly in urine from users in The Netherlands than from those in Merseyside. Aflatoxin B₁, the most toxic aflatoxin, was detected in eight specimens from The Netherlands but only one from Merseyside; the most common aflatoxin detected in the specimens from Merseyside was aflatoxin M₁, a less toxic derivative of B₁. These variations may be due to differences in the quantity or quality, or both, of heroin or to a difference in the timing of the urine collection after the last heroin injection. Another possibility is that heroin addicts in The Netherlands have greater dietary exposure to aflatoxins than their counterparts in Merseyside. Control urine specimens were not available from The Netherlands.

Because of the recognised susceptibility of drug addicts to infections like hepatitis and HIV infections and the immunosuppressive effects of aflatoxins in

animals¹ our results indicate the need for further study of exposure to aflatoxins in heroin addicts.

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Is abdominal aortic aneurysm familial?

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Abdominal aortic aneurysm is largely a disease of men and is rare before the age of 50. Only a small proportion of aneurysms are diagnosed during life; most are not fatal, and death from ruptured aneurysm may not be detected unless necropsy is performed. In addition, abdominal aortic aneurysm is not familiar to the public, and people who have the disease are more likely to be aware of affected relatives than those who do not. For these reasons the prevalence of abdominal aortic aneurysm in first degree relatives has not been established.

Patients, methods, and results

The probands were 108 patients (102 men, six women) admitted to hospital with a primary diagnosis of abdominal aortic aneurysm. We asked them about all first degree relatives except their children. Ages and remembered causes of death of first degree relatives who had died and names and addresses of parents and siblings still alive were recorded. All brothers aged 50-79 and sisters aged 55-79 thought likely to attend were invited to this hospital for ultrasound scanning of the abdominal aorta.

Five fathers and one mother were unknown. One father and four mothers were still alive but aged over 80. Of 301 siblings, 136 were known to be dead (77 men and 59 women). Thus 179 fathers or brothers were dead, of whom six (3%) were thought to have died from a ruptured abdominal aortic aneurysm. Two (1%) of the 162 mothers or sisters who had died were believed to have had a ruptured abdominal aortic aneurysm.

Altogether 143 siblings were within the age ranges defining eligibility for ultrasound scanning. We could not scan 56 because they lived overseas, their addresses were unknown, or they were too ill to travel. Thus we

invited for examination 87 siblings living in England or Wales; 31 attended. One man declined the invitation because he was soon to have an abdominal aortic aneurysm resected in his local hospital. We detected abdominal aortic aneurysms in four of the 16 men who attended but none in the 15 women. Thus of 17 brothers examined by ultrasound, five (29%), who were related to four probands, had an abdominal aortic aneurysm.

Comment

The occurrence of abdominal aortic aneurysm in members of the same family has been reported,^{1,2} and the incidence has been estimated at three³ and eight times⁴ the incidence in the control population. Ruptured abdominal aortic aneurysm causes 0.8% of deaths of men and 0.2% of deaths of women each year in England and Wales. In our study the incidence of death from ruptured aneurysms in male first degree relatives was 3% and therefore about four times that expected. This result, and the findings of both previous studies,^{3,4} relied on the accuracy of certification of cause of death and the relatives' understanding and subsequent memory of it.

In no previous study have siblings of patients with abdominal aortic aneurysm been examined with a reliable screening test for the disease. Screening unselected men aged 65-74 yields a prevalence of abdominal aortic aneurysm of 5.4%.⁵ The prevalence in men over 50 is not known but is probably less than 2%. This compares with the 29% found on ultrasonography in brothers of patients with abdominal aortic aneurysm. Our results support the hypothesis that abdominal aortic aneurysm is a familial disease.

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