oestrogens to be statistically significantly better than placebo at relieving hot flushes. Sound and well-argued criticism of any scientific work is valid but the gross misrepresentation of our studies by Dr Mulley and Professor Mitchell is inexcusable.

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Campylobacter enteritis

SIR,—We have followed with great interest the correspondence on campylobacter enteritis since the original article by Dr M B Skirrow (2 July, p 9). During a three-month period, from the end of June 1977, we have examined 563 faecal samples for potential intestinal pathogens. Of these, 280 were from patients with recent acute gastrointestinal symptoms, 117 from contacts of these cases, and 156 from asymptomatic patients. The table below summarises our results.

Campylobacters of the *C jejuni-C coli* group were isolated on the selective blood agar medium incubated at 43°C under microaerophilic conditions as described by Dr Skirrow. Our isolation rate of 14% in patients with symptoms is higher than previously reported in your columns. We found that 38% of our patients with campylobacters were children under 10 years of age; five patients had another intestinal pathogen isolated in association with the campylobacter. In addition, over the same period examination of 63 chicken carcases from a common source yielded 39 (62%) positives for the *C jejuni-C coli* group by direct culture technique; enrichment methods were not employed. Similarly, 167 caecal samples from apparently previously healthy poultry gave 114 (68%) positive isolates. Antibiotic disc diffusion tests of the human and avian strains were identical for aminoglycosides and erythromycin (all sensitive), and trimethoprim and cephaloridine (all resistant); but they differed in the case of ampicillin (25 μg disc). Here we consistently found that the avian strains were resistant, while 29 of 33 human strains were sensitive.

The epidemiology of campylobacter enteritis remains uncertain, although chickens have been considered as a primary source, and indeed serum from one of our patients with symptoms agglutinated both human and avian strains, thus suggesting a common antigen. Our results show poultry are a reservoir for campylobacter, but until a reliable scheme of strain identification is developed the place of avian strains in the epidemiology of human campylobacter enteritis may not be easily established. Our high isolation rate may be explained by the fact that Herefordshire is predominantly a rural area and people may more frequently be exposed to campylobacters from a variety of potential animal reservoirs, or indeed from the environment.

Potential pathogen isolated	No (%) positive samples from		
	280 patients with symptoms	117 contact patients	156 asymptomatic patients
Campylobacter sp	39 (13·9)	2 (1·7)	1 (0·6)
Salmonella sp	12 (4·3)	2 (1·7)	0
Shigella sonnei	11 (3·9)	14 (12·0)	1 (0·6)
Enteropathogenic *E coli*	5 (1·8)	0	2 (1·3)
Parasites	8 (2·9)	0	0

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SIR,—While campylobacters are increasingly reported as a cause of gastroenteritis the epidemiology remains unclear, though the finger of suspicion is pointed regularly at chickens and other birds as sources of the organisms.[1 2] We investigated a series of cases of gastroenteritis due to campylobacter occurring after a wedding reception. So far as we are aware this is the first report of a common-source outbreak.

Of 29 people from different parts of the country who attended a wedding breakfast, five fell ill with colicky abdominal pain and profuse watery diarrhoea. Three of them were also pyrexial and had rigors. The diarrhoea persisted for about three days. The incubation periods ranged from 48 to 84 h, with a mean of 62 h. A campylobacter was isolated from one patient at the height of her illness, but by the time the extent of the outbreak had become apparent all other patients had fully recovered and the organism was not isolated from their stools. Blood taken one month after the illness from four of those affected, including the patient from whom a campylobacter had been isolated, was tested for agglutinating antibodies. The index case showed a titre of 1/320, as did one of the others, while the other two had titres of more than 1/640, suggesting that these patients had all been infected by the same organism.

The wedding breakfast consisted of several types of cold cooked meats, salmon, salad, cheesecake, fruit, and sweets. The meats were chicken, ham, beef, tongue, and Scotch eggs. No food was available for examination and no single dish was common to all sufferers. The chicken was supplied as uncooked portions from a large wholesaler on the day before the reception, refrigerated overnight, cooked early the following morning, and allowed to cool before serving. It had not been deep-frozen. A similar pack of chicken from the same manufacturer received a month later was opened on arrival at the restaurant and a portion taken for culture. A campylobacter was isolated from the chicken skin but not from the meat. Agglutinins to this organism were not detected in the serum of our patients, but it is likely that there is wide distribution of serotypes in the chicken population[3] and the human sera were therefore tested against four other isolates of campylobacter obtained from local chickens. With two of the chick isolates titres were just as high as against the human strains; with the others no cross-reaction was demonstrable. The evidence for the involvement of chickens in the outbreak, though presumptive, is strong.

Cooking should kill these organisms and it is unlikely that the chicken was itself the sole final vehicle for infection in this outbreak, as one of those taken ill had not eaten any. Before being cooked the chicken pieces had been skinned on a working surface upon which other cooked meats were later prepared. It therefore seems likely that in this case cross-contamination had occurred between uncooked and cooked food and several different dishes may have finally become contaminated.

Henceforth campylobacter must be considered as a possible cause of food poisoning.

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SI units and acidity

SIR,—I hope that many correspondents have already suggested the most obvious solution of the problem described by Dr P J Tomlin (24 September, p 833)—the discrepancies that are found when the same laboratory measurement is expressed in different units and then the mean and standard deviation are used in determining clinically "normal" values. The appropriate method of presenting the measurements is the old but often neglected percentile technique, which "is a simple way of obtaining understandable information from any sample of measurements or counts."[1] It requires only the arrangement of the observed values in rank order, which would be the same whether the SI or the "old" units had been used, and it does not depend on any information or assumption regarding the shape (Gaussian or other) of the frequency distribution.

Readers who are not familiar with the applications and potentialities of the method could find much discussion in the textbook just cited and in three more recent publications[2-4] which discuss clinical norms. Those who find it difficult to transfer their allegiance from the mean and standard deviation to the median and percentiles may be helped by knowing how the mean-SD allegiance probably came about. Dr Feinstein[2] suggests that it arose because the mean and variance (or its square root, SD) are fundamental to the *t*-test and related tests of "statistical significance." Having been involved in medical statistics since 1928 and at one time a worshipper of *t* and its relatives, I agree with Dr Feinstein's diagnosis. Indeed, I think it is possible to detect a time of change in the writings of Sir Ronald Fisher. An article[5] that was published in 1921 discussed the variation among individual measurements without invoking Gaussian-curve assumptions, but in 1925 there appeared the first edition of the book[6] that was the starting-point for the wide