realize that they are denying them the benefit of the art of medicine which they are pledged to practise—I am, etc.,

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Gut Flora after Cephalexin

SIR,—In their paper "Changes in Gut Flora after Cephalexin Treatment" (12 September, p. 624) Dr. H. Gaya and colleagues confirm earlier reports from the same hospital of a high faecal carriage rate of *Ps. aeruginosa* in patients who swallow it in food or other substances.[1] They attempt to relate this to treatment with cephalexin and to compare cephalexin with other antibiotics as a factor in the acquisition of *Ps. aeruginosa* and colonization of the bowel.

In support of their opinion they say it has been found that colonization is aided by ampicillin, but in the paper to which they refer as a source of this statement the authors, who studied the effects of ampicillin in a single volunteer fed measured doses of *Ps. aeruginosa*, conclude that antibiotics (not only ampicillin) *may be* of importance in establishing colonization. They continue "However, it is known that colonization may occur in the absence of antibiotics. Whether the establishment of colonization is due to a particular property of the strain of organisms or to some unidentified characteristic of the carrier is unknown."[2] On the basis of this work and their own data Dr. Gaya and his colleagues speculate not only on the influence but the comparative influence of cephalexin and ampicillin on colonization.

We question whether they are justified in their conclusions. In an earlier publication Dr. Gaya and others report in a study, apparently carried out in patients from the same medical wards as those involved in their recent trial, that 44% of patients fed from the diet kitchen and 23% fed from the main kitchen became carriers of *Ps. aeruginosa*. They also state that *Ps. aeruginosa* was "irregularly and inconstantly present in particular situations" and discuss the role of the patients' disease and antibiotic therapy in acquisition of the organism.[1]

In the light of this experience it would seem to be essential to have patients matched not only for length but also dates of stay in hospital, for disease states, and source of food in order to make a comparison between different groups. The numbers of patients in their comparative groups are unequal and so it would appear that this was not done. It is also difficult to understand why the data on acquisition are reported in only 12 of the original group of 18 patients, although the authors suggest that stool specimens were collected from all of them. It would also be of interest to know how many of their patients acquired pseudomonas or proteus at some time during their hospital stay—for example, before starting treatment with cephalexin—and what were the three unspecified "other antibiotics" in one of the control groups. Their statement that "a less marked trend [that is, acquisition rate] was seen with proteus" is surely misleading in view of a P value of 0.08.

With regard to the assay results carried out in our laboratories, we would not feel justified from our considerable experience of antibiotic assays in comparing them with data from a much better controlled study[3] as Dr. Gaya and his colleagues have done. Some of the specimens were kept for as long as four weeks before we received and assayed them and arrived in an unfrozen state. The stability of cephalexin, especially in urine samples from patients suffering from a number of different diseases, perhaps on other drugs, and kept for that length of time is uncertain, and could account for misleading results. Some of the results are difficult to explain except on grounds of stability, unless there was a failure to administer the dose or collect urine specimens. For example, a patient with one of the highest peak levels (43·6 μg./ml.) at one hour after a 1 g. dose showed nil at the same interval after a second dose and only 29% of the dose administered was recovered in a 24-hour specimen of urine.

Finally, there are some errors in the paper according to information given to us by the authors at the time of the assays. The figures for serum samples are incorrect. Only 13 patients were studied, not 18 (at 1 hour) and 15 (at 4 hours). Some of the samples were repeats from the same patients. One of the curves in Figure 1A is also wrong, both for the number of samples and their timing. The statement in the text "Peak blood levels occurred at half hour to two hours" should read "Peak blood levels occurred at one to three hours (Fig. 1A)". The strain of *Sarcina lutea* used as test organism in the assay was N.C.I.B. 8553, and not 8533 as stated.—We are, etc.,

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Bib into Apron

SIR,—As Mr. R. J. McNeill Love (26 September, p. 767) so aptly points out, it is the lack of any omentum, for all practical purposes, that is a major factor in the high mortality of appendicitis in infancy.

This is well illustrated by quoting an eminent Guy's surgeon who said "though the omentum is an apron of fat in the adult, it is a mere bib in young children."—I am, etc.,

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Diabetic Acidosis

SIR,—It was of interest to note that in their paper on organic acid production in diabetic ketosis (12 September, p. 610) Dr. P. Z. Zimmet and colleagues found a mean acetoacetate level on admission of 4·5 mEq/l. The technique they have used for ketone body measurement depends on the staged conversion of acetoacetate and 3-hydroxybutyrate to acetone, which is measured colorimetrically with 2-4 diphenylhydrazine. Shaw *et al.*,[1] using an essentially similar method (but with salicylaldehyde as colour reagent), found a mean acetoacetate level of 9·45 mEq/l. Both these methods assume that negligible amounts of preformed acetone are present in diabetic ketosis.

Our observations on 41 patients in ketosis showed a blood acetoacetate range of 1·5 to 5·4 mEq/l. with a mean of 3·02 mEq/l.[2] These determinations were made using the AutoAnalyzer technique of Salway.[3] However, acetone is by no means a quantitatively minor ketone body and its levels far exceed those of acetoacetate in ketoacidosis.[2] This explains the high acetoacetate figures of Shaw *et al.* because they include preformed acetone. In Dr. Zimmet's series the levels seem too high for true acetoacetate determinations yet too low for a combined acetoacetate-acetone reading. There is no apparent reason for the discrepancy, but it does not detract from the arguments advanced in their paper.—I am, etc.,

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House-dust and Asthma

SIR,—With reference to the correspondence initiated by Dr. B. J. Freedman (18 July, p. 166) one of the surprising features of allergy to house dust is that in spite of its ambiguity the active agents (mites) have so long been overlooked. We should also be careful not to concentrate too narrowly on *Dermatophagoides pteronyssinus* as the causal agent.

In a preliminary study of allergy in Barbados W.I., where asthma is common, a number of samples of bedroom dust freshly examined under a dissecting microscope have shown the presence of mites in exceptionally large numbers. Skin reactions to house-dust extracts, *D. pteronyssinus* extracts, and *D. farinae* extracts are positive in a high proportion of cases.

Six varieties of mites have been identified in four samples of dust by one of us (A.M.H.) as follows: *Dermatophagoides pteronyssinus; Glycyphagus domesticus Chyletus* spp; *Chortoglyphus* spp; *Blomia* spp; and family *Glycyphagidae*, undetermined genus and sp.

Most *Dermatophagoides* though present in large numbers were dead on arrival in England. Mites belonging to the species *Glycyphagus domesticus* were found only in small numbers. The remaining ones apparently multiplied for at least two months in a plastic container with no medium other than the dust in which they were found.

It seems possible that all or some of these mites are responsible for house-dust allergy, as well as *D. pteronyssinus*. They may induce specific immune responses in asthmatic patients or more probably possess antigenic properties in common with *D. pteronyssinus*. Further work is being undertaken to establish their importance in causing asthma.—We are, etc.,

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