antigens, as with sarcomas and melanomas,[4] acute leukaemias,[5] and Burkitt's lymphoma (Drs Magrath and Ziegler), often retain good immune reactivity to common antigens; thus the general immune status often fails to correlate with the host-tumour relationship. We assume that in these cases suppressor cells specifically inhibit tumour rejection mechanisms. For the demonstration of this activity suppression of lymphocyte blastogenesis to autologous tumour cells by a clone of lymphoid cells added to the mixture of reactor and tumour cells would be necessary.

This oversimplified view is in need of experimental support, but the basic techniques for the detection of human suppressor cells are already available.[1 2] Means of deactivating suppressor cells could develop into an innovative modality of tumour immunotherapy, whereas activation of suppressor cells (by administration of fetal antigens?) may be useful for organ transplant acceptance. The hormonal milieu of the immediate post-partum period, selective immunosuppression during tumour chemotherapy,[6] and ablative procedures (splenectomy, lymphadenectomy, adult thymectomy) come to mind as possible procedures of deactivation and elimination of suppressor cells.

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Co-trimoxazole and cephalexin in urinary tract infection

SIR,—A plausible explanation is to hand for the superior performance of co-trimoxazole compared with cephalexin in the urinary tract infection trial of Drs P E Gower and P R W Tasker (20 March, p 684).

Contrary to their statement that "cephalexin readily induces spheroplast formation," it has repeatedly been shown[1-4] that the sole effect of therapeutically useful concentrations of cephalexin (and the related cephradine and cephaloglycin) is to cause filamentation of enterobacteria by inhibiting the division process. Because of this cephalexin is more slowly bactericidal than other β-lactam antibiotics and more bacteria are likely to survive in the urine, where, as Drs Gower and Tasker rightly point out, a twice-daily dosage may achieve only transient high levels.

In addition, all cephalosporins now available are somewhat susceptible to enterobacterial β-lactamases, including a slow-acting enzyme characteristic of ampicillin-sensitive *Escherichia coli* strains.[4] Consequently tests of sensitivity of enterobacteria to cephalosporins are affected by inoculum size; this is particularly marked with ampicillin-resistant strains.[4 5] Infected urine frequently contains more than 10^8 bacteria/ml. Concentrations of cephalosporins achievable in urine only transiently suppress such a bacterial population, recovery occurring as the antibiotic is broken down.[4 5] Disc sensitivity tests may give an over-optimistic view of the sensitivity of such strains to cephalosporins, even when conventional "high inocula" are used, as in the Bauer-Kirby test.[6]

Sulphonamides and trimethoprim are also susceptible to inoculum effects, but this is unrelated to degradation of the drugs.[7 8] In contrast to β-lactam antibiotics, the components of co-trimoxazole are excreted into the urine slowly and the antibacterial activity is maintained in support of intrinsic clearance mechanisms.

Evidence for the validity of these considerations has been provided by experiments employing an in-vitro model in which some important aspects of the treatment of bacterial cystitis can be simulated.[7 9-11] Such studies have shown that cephalosporins perform less well than penicillins (including benzyl- and phenoxymethyl-penicillin) in the dynamic conditions of the urinary bladder, using an initially dense, but ostensibly sensitive, bacterial population.[9] Tested against ampicillin-resistant strains judged "cephalosporin-sensitive" on the basis of disc tests, cephalosporins exhibited a further reduced capacity to suppress bacterial growth.[10] In both these studies cephalexin was the least effective cephalosporin tested. When sulphamethoxazole and trimethoprim were tested in the model their efficiency in clearing infection was rather better than predicted by conventional tests.[7]

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Preventing animal diseases

SIR,—In a letter (14 February, p 393) Sir James Howie referred to a statement by Mr M E Hugh-Jones, of the Central Veterinary Laboratory, Weybridge, which posed five basic questions to be answered before monitoring systems are introduced in the zoonoses field. In a leading article (p 355) you took up the challenge and focused attention on the particular problem of salmonella infection from poultry.

In a subsequent letter (28 February, p 521) Dr D J H Payne and Mr E Lowes referred to the formation of liaison groups with medical, veterinary, and environmental health representation. The Eastern Regional Medical-Veterinary Liaison Group is such a body and has been in existence for over three years. Throughout that time a major concern has been the recognition of numerous outbreaks of food poisoning in human populations where chicken or turkey has been the vehicle of infection and the evidence of enzootic salmonella infection in poultry establishments in the area covered by the professional groups represented on the committee. Following the references to the problem in the *BMJ* our committee has had further discussion and we wish to make two suggestions additional to those in your leading article on the information that is needed.

We recognise that many poultry establishments have voluntarily instituted their own monitoring programmes and that information on salmonella isolations resulting from such programmes is being made available through the implementation of the Zoonoses Order, 1975. However, we feel that there are large sections of the poultry industry from which no information is available and we would urge medical officers of environmental health/community medicine specialists and environmental health officers to introduce regular bacteriological monitoring at poultry processing plants and retail outlets. In this way a more complete picture of the size of the problem could be built up.

We also suggest that there is a need for a requirement upon the poultry trade that birds entering the market carry a batch number or other identification mark so that in the event of human infection or the discovery of a bacteriologically positive carcase the source of infection can be traced to the poultry unit concerned. Under the present system investigations can rarely be taken beyond the point where the particular brand has been established, and in the case of the very large poultry firms this information of itself is inadequate.

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Rheumatoid arthritis and ankylosing spondylitis occurring together

SIR,—The paper by Professor G H Fallet and others (3 April, p 804) documents a series of patients who had the clinical picture of both rheumatoid arthritis and ankylosing spondylitis. They describe as extremely unlikely the possibility that these patients represent the random occurrence of two separate disease entities. Critical examination of the argument shows that coincidental occurrence is certainly not ruled out.

An accurate estimate of the real prevalence of ankylosing spondylitis is not available. The study of West[1] which Professor Fallet and his colleagues quote gives an extremely unreliable estimate. They also quote de Blécourt *et al*,[2] but the frequency of ankylosing spondylitis in the controls used in that study is of no value in estimating the prevalence in the population as these controls were *families* of probands without spondylitis. The data of Lawrence[3] offer the best available estimate of the prevalence of ankylosing spondylitis, but even here there are methodological problems which suggest that his prevalence figures are only an approximation and probably an underestimate.

The figure given by the authors for the a priori probability that an individual will have both severe rheumatoid arthritis and ankylosing spondylitis is unreliable and probably too