with disseminated carcinoma to respond to the LAK process, and indicates the requirement for techniques to increase lymphocyte cellular renewal in such patients.

Lymphokine activated killer source cannot simply be explained on the basis of cell type, but surely represents a functional disruption of a down regulated networks when performed outside the body. This results in large numbers of effector lymphocytes derived from those cell types, both T-cell and NK cell, in which anti-tumour activity can be augmented.

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Reply

SIR,—We were pleased to receive Mr O'Byrne's interesting reflections on our paper (*Gut* 1987; **28:** 1420–5). Mr O'Byrne's points are well taken. We do indeed recognise the differences which may exist between the antitumour cytotoxic responses present in peripheral blood, and those which may be elicited from tumour infiltrating lymphocytes. Our point in studying peripheral blood was that certainly at present, and for the foreseeable future, lymphokine activated killer cells for therapeutic purposes would continue to be generated from peripheral blood until techniques for the reliable expansion of tumour infiltrating lymphocytes have been perfected. Our viewpoint was therefore a practical one rather than a theoretical one.

In order to invoke the idiotype/anti-idiotype network as a cause of suppression of a cytotoxic cellular immune response, however, specificity of that response has to be assumed. Unfortunately for Mr O'Byrne's argument, the very characteristic which separates LAK cells and NK cells from T-cell mediated cytotoxic responses is their very lack of specificity. Thus LAK cells from cancer patients and from normal controls are capable of killing tumour cells of a very wide variety of origins, indeed under certain circumstances, even normal cells.

There is much evidence to suggest that the tumour mediated immunosuppression present in cancer patients has a far more simple explanation. We have found tumour cells to actively suppress the generation of LAK cells *in vitro* (Guillou, Ramsden, and Sedman – in preparation). Because there is ample evidence that tumour cells secrete autocrine growth factors, which themselves regulate such events as natural killer cell activity, it may well be that these are a more likely candidate for mediating the suppression than idiotype/anti-idiotype mechanisms.

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An epidemic of pseudomembraneous colitis or simply a nosocomial case clustering

SIR,—In their paper (*Gut* 1987; **28:** 1467–73) Dr Nolan and colleagues report on the occurrence of an 'epidemic of pseudomembraneous colitis' (PMC) in three hospitals involving 23 patients in 10 months. Evidence in support of an epidemic caused by nosocomial transmission of *Clostridium difficile* should, however, rely on distinctive antimicrobial resistance patterns, in addition to agarose- or polyacrylamide gel electrophoresis, crossed immunoelectrophoresis and/or phage-typing.[1–6] The retrospective nature of the above study, in which cross infection was supported only in 16 patients by a positive culture and in no case by use of bacterial typing techniques, makes the assumption of an 'importance of person to person spread' purely speculative. Furthermore, there is hardly convincing evidence that all 23 patients included actually developed PMC, because endoscopic and histological proof of PMC were missing in five other patients. Also we are aware of negative cultivations of *Clostridium difficile* from patients with PMC and cytotoxin production, but in this study only a single patient was assayed for the cytotoxin. Although *Clostridium difficile* is the most abundant bacteria in this condition also *Staphylococcus aureus* and *Clostridium perfringens* have been reported to be responsible for PMC, but were not even looked for. A final open question concerning this paper relates to the 20 patients without clinical symptoms, in whom *Clostridium difficile* was identified. Why were these patients not regarded as nosocomially infected? In our ward we have had a clustering of antibiotic associated diarrhoea and PMC in six patients in two months, similarly suggestive of a nosocomial spread.