area that concerned our local research ethics committee (LREC). Our proposal was not helped when we attended an LREC meeting along with a commercial collaborator, shortly after Dolly the Sheep had first been shown to the world. The LREC allow for extraction of RNA and DNA but not for cellular immortalisation or cloning. In order to monitor this, our legal contract requires the commercial organisation to provide research protocols before we supply tissue to them.

We have heard expressed the fear that one day a genetic research finding might have far reaching clinical and ethical consequences for an individual donor patient. The Statement provides useful guidance on this and recommends treating genetic discoveries in the same way as ordinary research findings, although the "Notes reflecting UK law and practice" point out some of the potential ethical risks of DNA databanks to society as a whole.

A section on confidentiality emphasises the importance of system safeguards in preventing the leak of patient data. According to the working definitions in this section, the samples we provide to commercial companies are, strictly speaking, "linked" rather than anonymised. However, in our tissue bank, "linkage" can only be made by the medical intermediary and it is difficult to conceive a situation where we would agree to do this. In order to do so we would need the consent of the patient. In practical terms, therefore, the tissue we provide to commercial firms is "anonymised".

The final section on consent further reinforces our views.[1] We would argue that a general agreement to donate tissue as part of a signed consent-to-treatment form does not constitute proper informed consent which, if done properly, places an additional burden of explanation on the surgeon, whose role in adhering to the latest GMC guidelines[3] is difficult and time consuming enough. Furthermore, the "Notes reflecting UK law and practice" mention a European Directive, due to be implemented by 2000, which implies that informed consent will be required if ever a patent application is filed—the dream of many a commercial research company.

At Peterborough, we now employ two research nurses to obtain consent and counsel patients before operation. The nurses have redesigned the consent forms, produced a patient information pack, and are able to spend time with patients answering questions. This experience was presented to the British Association of Tissue Banks (BATB) in March 1999 and we hope to publish the results in due course.

There have also been important developments in the "whole body donation project".[1] What began as an informal arrangement with the North East Thames National Blood Service Tissue Services (NBSTS) is now covered by a formal written agreement. Donors who have expressed a wish to give more tissues than are currently routinely banked by NBSTS, and others identified as being unsuitable for the transplant programme by the transplant coordinators, are referred to the Peterborough Tissue Bank for postmortem tissue collection. Compared to surplus surgical material in hospital, the consent procedures undertaken by the transplant coordinators are more complicated. This is largely because of the issues that need to be covered when tissue is to be transplanted. Donors, where possible, and relatives understand the cost recovery, commercial nature of the Peterborough Tissue Bank.

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1 Gray N, Womack C, Jack SJ. Supplying commercial biomedical companies from a human tissue bank in an NHS hospital—a view from personal experience. *J Clin Pathol* 1999;**52**:254–6.
2 Working Party of the Royal College of Pathologists and the Institute of Biomedical Science. *Consensus statement of recommended policies for uses of human tissue in research education and quality control.* London: Royal College of Pathologists, 1999.
3 General Medical Council. *Seeking patients' consent: the ethical issues.* London: General Medical Council, 1998.

Increasing rates of ciprofloxacin resistant campylobacter

We read with interest the recent correspondence concerning ciprofloxacin resistance in campylobacter species.[1] Campylobacter enteritis is a self limiting disease in most individuals, and should not require antimicrobial treatment. If treated, the commonly used antibiotics are fluoroquinolones, or erythromycin, particularly for children. We reviewed the rates of resistance to ciprofloxacin and erythromycin in campylobacters isolated in our laboratory from 1995 to 1998 (table).

Rates of antibiotic resistance in Campylobacter species isolated 1995–8

Year	Total No of isolates	No (%) of ciprofloxacin resistant isolates*	No (%) of ciprofloxacin resistant cases with recent foreign travel†	No (%) of erythromycin resistant isolates†
1995	351	37 (10.5)	20 (54.1)	3 (0.9)
1996	344	37 (10.8)	16 (43.2)	6 (1.7)
1997	416	68 (16.3)	31 (45.6)	4 (1.0)
1998	495	89 (18.0)	43 (48.3)	5 (1.0)

*χ^2 = 14.4, 3 degrees of freedom, $p<0.01$.
†Trend not significant.

There was significantly increasing resistance to ciprofloxacin over the four year period. Ciprofloxacin resistance in this area is now higher than recently reported rates from Northumberland (6.7%) and the Laboratory of Enteric Pathogens (12%).[1] This suggests wide variation in resistance within the United Kingdom.

Considerably higher levels of ciprofloxacin resistance have been found in other countries, such as Spain (45%) and Thailand (84%).[2 3] However, we found that only around half of resistant strains were acquired abroad, with no significant increase in the proportion over the four years, suggesting that resistance is now well established within this area. If this is true for the United Kingdom as a whole, the data add further weight to the calls for more controlled quinolone usage both in veterinary and human medicine.[4]

As resistance to ciprofloxacin rises, erythromycin becomes a more important therapeutic option. Our data show that we have not found erythromycin resistance to be a significant problem for several years.

Knowing that local rates of quinolone resistance are rising may help influence prescribing habits. It would seem reasonable to restrict quinolone treatment to patients with severe illness or risk factors for poor outcome,[5] to limit further promotion of resistance to these valuable antimicrobial agents.

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1 Galloway A, Dickinson G, Harrison M. Ciprofloxacin resistant campylobacter [letter]. *J Clin Pathol* 1998;**51**:487.
2 Reina J, Ros MJ, Fernandez-Baca V. Resistance to erythromycin in fluoroquinolone-resistant *Campylobacter jejuni* strains isolated from human faeces [letter]. *J Antimicrob Chemother* 1995;**35**:351–2.
3 Hoge CW, Gambel JM. Trends in antibiotic resistance among diarrhoeal pathogens isolated in Thailand over 15 years. *Clin Infect Dis* 1998; **26**:341–5.
4 Piddock LJV. Fluoroquinolone resistance. *BMJ* 1998;**317**:1029–30.
5 Farthing M, Feldman R, Finch R, *et al.* The management of infective gastroenteritis in adults—a consensus statement by an expert panel convened by the British Society for the Study of Infection. *J Infect* 1996;**33**:143–52.

Book reviews

Molecular Biology of the Lung, Vol 1: Emphysema and Infection; Vol 2: Asthma and Cancer. Edited by D Raeburn and M Giembycz. (DM 528.00.) Birkhauser, 1998. ISBN 3 7643 5969 2.

These two volumes of this series represent a timely addition to an expanding field. The style, content, structure, and illustrations in both volumes are excellent. The information contained in them is accessible enough for the non-specialist while being detailed enough to be of interest to those working in the field.

The first volume deals with emphysema and infections. There is an interesting chapter on the use of transgenic mice which sets the scene nicely for the ensuing chapters. Emphysema is well covered, with several areas being highlighted including α1 antitrypsin deficiency, recombinant SLPI elastase inhibitors, proteases, and connective tissue genes. The chapters on infection, particularly those dealing with cystic fibrosis, are excellent.

In volume 2, leading lights in the field take us through the genetics of asthma including transcription factors, cytokine gene clusters, β adrenoceptors, and control of eosinophil migration. The section on cancer, while smaller, gives an excellent insight into gene expression in lung cancer and potential targets for genetic therapy. These volumes are essential reading for cell biologists with an interest in respiratory disease. They will provide a useful reference source for clinical scientists with an interest in other fields.

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In-Situ Hybridization—Principles and Practice, 2nd ed. Edited by J M Polak and J O'D McGee. (£39.95.) Oxford Medical Publications, 1999. ISBN 0 19 854880 X.

The first edition of this book sits on the bookshelf in our laboratory. It dates from 1989, almost a geological age ago in molecular biological terms. The new second edition