molecule of the protein probably binds seven copper ions in a protein of molecular weight 132000, giving 0·33% of metal in the protein. In other words the serum caeruloplasmin found in the neonates should bind 300 μg copper and in the patients with Wilson's disease 330 μg. These figues closely approximate to the 5·0 μmol/l (315 μg/l) shown in the figure. In other words there is no "free copper" present in either of the two groups shown in this series, albeit the numbers are small. This might seem surprising for patients with Wilson's disease, though they may have received long term treatment with penicillamine and have been effectively "decoppered"—but this explanation would seem to be improbable for the neonates.

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RIFAMPICIN AND ANTIBIOTIC-ASSOCIATED COLITIS

SIR,—We were interested in the case-reports of rifampicin (rifampin) associated pseudomembranous colitis published in *The Lancet* of Nov. 29.[1,2] We tested 20 isolates of *Clostridium difficile* and found all to be very susceptible to rifampicin, with minimum inhibitory concentrations (MIC) ⩽0·2 μg/ml. We then did experiments to see if rifampicin would induce colitis in the Golden Syrian hamster, an animal in which antibiotic-associated colitis (AAC) is readily induced by many antimicrobial agents. When hamsters were given single doses of rifampicin, orogastrically or subcutaneously, none acquired enterocolitis. Next, to see if rifampicin could prevent AAC caused by other antibiotics, it was given subcutaneously to hamsters before they were given subcutaneous clindamycin. In these hamsters colitis sometimes developed, but they lived longer than those receiving clindamycin alone. *C. difficile* isolates from hamsters with enterocolitis were very susceptible to rifampicin.

After six months of experiments with rifampicin in these quarters, large numbers of animals died unexpectedly with caecitis after receiving rifampicin only. *C. difficile* isolates from the caecal contents of these animals were resistant to rifampin (MIC >100 μg/ml).

Thus, rifampicin at first protected against AAC and the *C. difficile* isolates were susceptible to it. However, typical clinical and morphological changes compatible with AAC were induced with rifampicin after the organisms had acquired resistance to rifampicin following frequent exposure of the hamster colony to the drug. Development of rifampicin resistance in *C. difficile* might be most likely to happen in people on long-term rifampicin (e.g., for tuberculosis). Thus, it would not be surprising if rifampicin were to be implicated as a cause of AAC in man. Our experiments also indicate the importance of cross-infection with *C. difficile* in animal quarters.

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COMPUTERS AND CONFIDENTIALITY

SIR,—Letters from the chairmen of the Child Health Computing Committee (Jan. 3, p. 47) and of the Central Ethical Committee of the B.M.A. (Feb. 7, p. 327), and a *Lancet* editorial (Dec. 6) on computerised child health records, claim that there is a shortage of facts for debate and that facts are hard to come by. This is not so. The report of Ontario's Royal Commission on the Confidentiality of Health Records[3] runs to three volumes, summarising three years' investigation by an Ontario Supreme Court judge. It catalogues widespread, frequent, routine, illegal abuse of medical records by the national security service (on one occasion for political blackmail), by the local police, by private detectives, by insurance companies, and by immigration authorities in the province of Ontario. For example, the Immigration Department made 60–70 requests monthly for information from health records and Ministry of Health officials withheld that fact from the provincial Minister of Health (the supposed representative of the people). With the frequently changing immigration laws in the U.K., it is not difficult to see that the U.K. immigration authorities would similarly have great recourse to information from computerised health records. With regard to child health Justice Krever comments that existing laws aimed at health care workers do not "provide adequate assurance that the confidentiality of school children's health records will be protected".

Justice Krever made 170 recommendations for changes to (Ontario) law and, with medical computer specialist advice, made copious recommendations to promote privacy and security of computer records.

I, for one, find 1662 pages of evidence and closely reasoned argument sufficient facts for the current debate.

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CYCLOPHOSPHAMIDE AND SUPPRESSOR CELL FUNCTION

SIR,—Dr Raube and colleagues (Jan. 31, p. 235) describe long-term impairment of suppressor cell function in patients who were treated with cyclosphosphamide because of minimal change nephropathy. For several reasons their results are debatable.

Their assay for T suppressor cell function was unusual. Responder cells were pre-incubated for 24 h at 37°C before suppressor cells were added, which in itself influences suppressor cell function, since short-living suppressor cells might be lost during pre-incubation.[1] Their assay is further obscured by their not removing concanavalin A from the activated suppressor cells by treatment with α-methylglucoside[2] and not separating concanavalin A activated T cells from monocytes, since monocytes also may function as suppresor cells in this system.[3] The doubled cell density in the reconstituted culture of suppressor and responder cells compared with the cell densities in the separate cultures of these cells, might by itself be responsible for the observed "suppression". Although Raube et al. noted differences between several groups of patients, I am not at all convinced that they have detected impairment of suppressor-cell function.

These workers do not give their criteria for treating some patients with cyclophosphamide. Obviously, patients who have not responded to corticosteroids differ from the remainder before cyclophosphamide treatment begins. Since suppressor-cell function was not measured before treatment, altered lymphocyte reactivity several years after the last dose cannot be attributed to cyclophosphamide.

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COAGULASE-NEGATIVE STAPHYLOCOCCI

SIR,—Your Jan. 17 editorial well summarises the importance of the opportunistic pathogen *Staphylococcus epidermidis*, although you under-rate the importance of *Staph. saprophyticus (Micrococcus* sp). However, on the choice of antibiotic treatment for *Staph. epidermidis* infections you are too gloomy. You record the conclusion of Forse et

1 Melanger M, et al. Pseudomembranous colitis and rifampin. *Lancet* 1980; ii: 1192,
2. Bommelaer G, et al Pseudomembranous colitis and rifampin. *Lancet* 1980; ii: 1192.
3 Report of the Commission of Inquiry into the Confidentiality of Health Records (chairman Justice Horace Krever). Government of Province of Ontario, Canada, 1980.

1. Bresnihan B, Jasin HE. Suppressor function of peripheral blood mononuclear cells in normal individuals and in patients with systemic lupus erythematosus *J Clin Invest* 1977; **59:** 106
2. De Gast GC, The TH, Ponds E, Kallenberg CGM Suppression of DNA synthesis by Con A activated human lymphocytes: Stimulation by Con A bound to non-T cells unless removed after activation *Clin Exp Immunol* 1977, **30:** 457.
3. Kallenberg CGM, De Gast GC, The TH. Suppression of DNA synthesis by Con A-activated human lymphocytes Role of monocytes in Con A-induced suppression. *Clin Exp Immunol* 1980; **41:** 583.