Organisation's projections suggest that 1 in every 1000 people in India will be infected by 2000.[2] An Indian news magazine recently highlighted the problem of cross border trafficking by prostitutes from Nepal.[3] Commercial sex is an important route of transmission of HIV in Asian countries.

At the heart of this problem is the complacency shown by the central government in New Delhi. It is astonishing that, while the numbers of cases of HIV infection and AIDS are rising, the programme budget remains underspent.[1] There is no coherent national strategy on conducting safe sex campaigns. Government departments shy away from giving explicit information about the spread of HIV. Campaigns through leaflets will have no role in rural areas owing to low literacy rates. But it is not too late to reverse the rising number of cases. Satellite television is now reaching the most remote parts of India. Both the government and voluntary organisations should be keen on using this in their fight against HIV infection and AIDS.

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Ciprofloxacin in general practice

EDITOR,—The Lesson of the week by Roland J Körner and colleagues highlighted the dangers of fluoroquinolones.[1] We have also noted that patients with chest infections have been treated with ciprofloxacin in general practice; the slower resolution of their *Streptococcus pneumoniae* infection required a change of antimicrobial agent, and one patient died.

We understand, however, that ciprofloxacin is not promoted as first line treatment in general practice for community acquired chest infections, but some general practitioners in our area have provided promotional material that does. This material contains data showing that 14·4% of bacterial pathogens in acute exacerbations of chronic bronchitis are *S pneumoniae*.

Bantz and colleagues (some of whom worked for Bayer) are cited in Bayer's promotional leaflet for Ciproxin; they mentioned a >95% resolution rate but compared only doxycycline with ciprofloxacin and made no reference to bacterial pathogens. In the same supplement to the *American Journal of Medicine*, however, other papers gave less favourable views—for example, "the activity of ciprofloxacin against Streptococci and Enterococci is marginal, at best."[3]

The only other reference concerning efficacy that is cited in Bayer's promotional leaflet is a study of just 34 patients.[4] Those with *Haemophilus influenzae* rapidly recovered, and these organisms were not culturable beyond three days. Of those with *S pneumoniae* infection, five still had positive results after three days, five after 11 days, and one after 25 days. Five patients had a relapse and were then treated with either amoxycillin or cotrimoxazole and clinically recovered. Six patients acquired *S pneumoniae* infection during or after treatment, and three required treatment. Two patients had rising minimum inhibitory concentrations to ciprofloxacin in *S pneumoniae*, with organisms being isolated further into ciprofloxacin treatment.

In the same issue of the *Journal of Antimicrobial Chemotherapy* a leading article on quinolones in chest infections, concludes that there is little reason for optimism about the role of quinolones in chest infections mainly because of problems with resistance, recurrence, and reinfection with *Pseudomonas aeruginosa* and *S pneumoniae*.[5]

Clearly, ciprofloxacin is not a suitable agent for use in general practice for the blind initial treatment of chest infections and should not be so promoted.

We believe that there are major discrepancies between the promoted image and the clinically interpreted usefulness of ciprofloxacin. We hope that this sort of problem is not widespread in the pharmaceutical industry but wonder how extensive it is considering the gamut of drugs being actively promoted, often to those without the time or resources to easily and critically interpret the data presented to them.

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Fluoroquinolones in chronic obstructive pulmonary disease

EDITOR,—In their editorial Harold Hosker and colleagues suggest that fluoroquinolones are suitable second line antibiotics for infective exacerbations of chronic obstructive pulmonary disease.[1] We report two cases that illustrate our reservations about this suggestion.

Case 1—A 75 year old woman was admitted complaining of severe shortness of breath and right sided pleuritic chest pain. She had been admitted several times before with exacerbation of chronic obstructive pulmonary disease. Before this admission she had taken ciprofloxacin 500 mg twice daily and prednisolone for six days. On examination she had signs of right lower lobe pneumonia, which was confirmed on chest radiography. She was hypoxic (oxygen pressure 6·8 kPa, carbon dioxide pressure 5·21 kPa) and had a white cell count of 23×10^9/l, with 84% neutrophils. *Streptococcus pneumoniae* capsular type 4 was isolated from blood cultures, with a minimum inhibitory concentration for ciprofloxacin of 4 mg/l and for benzylpenicillin of 0·125 mg/l. She responded to amoxycillin and was discharged after seven days.

Case 2—A 72 year old man with a 30 year history of chronic obstructive pulmonary disease was admitted with severe shortness of breath. Before admission he had been treated with ciprofloxacin 250 mg twice daily and prednisolone. On examination he was afebrile, breathless, and cyanosed. There were widespread crackles and wheezes in the chest but no focal abnormality in the chest radiograph. *S pneumoniae* capsular type 15 was isolated from sputum taken after admission. The minimum inhibitory concentration for ciprofloxacin was >8 mg/l and for penicillin 0·125 mg/l. He was treated with amoxycillin and erythromycin and discharged after 22 days.

S pneumoniae is the pathogen most commonly identified in pneumonia acquired in the community and in lower respiratory tract infection seen in general practice[2]; it is frequently isolated in chronic obstructive pulmonary disease.[3] Fluoroquinolones such as ciprofloxacin and ofloxacin are not particularly active against it. Serum bactericidal concentrations after oral or intravenous dosage are minimal,[4] which may lead to antimicrobial resistance. Fluoroquinolones are highly active against *Haemophilus influenzae* and some of the bacteria causing atypical chest infections, which may contribute to infective exacerbations of chronic obstructive pulmonary disease.

Failure of treatment with fluoroquinolones in respiratory tract infection has been reported.[5] The datasheets for fluoroquinolones indicate that the drugs are not predictably active against *S pneumoniae*, yet some are heavily promoted for the treatment of chronic bronchitis in general practice. We believe that fluoroquinolones should not be used alone for blind treatment of respiratory tract infections, including exacerbations of chronic obstructive pulmonary disease. An agent effective against pneumococcus should always be included until a microbiological diagnosis is made.

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Safety of thalidomide

EDITOR,—Use of thalidomide only on a named patient basis does not necessarily mean that patients are protected from its side effects, as implied by Baroness Cumberlege, the junior health minister.[1] According to Sir William Asscher, the former chairman of the Committee on the Safety of Medicines, "the companies who supply a drug for named patient use are not required by law to provide warnings about the use of the drug" and, furthermore, "the information provided with the supplies of thalidomide [to patients with leprosy in the United Kingdom] does not specifically mention peripheral neuropathy as a side effect" (personal communication). Until 1979 patients with leprosy were not warned that the drug could cause loss of sensation in the extremities (M F R Waters, personal communication). Requests to find out whether they have now been warned have not been answered by Sir William. Early recognition of thalidomide neuropathy with immediate withdrawal offers the best chance of recovery, but this option is being denied to patients with leprosy. Even worse, the patients will assume that any loss of sensation occurring during treatment will be due to the disease.

Electrophysiological studies, particularly the recording of sural nerve action potentials, have proved valuable in detecting thalidomide neuropathy at an early stage,[2] but these have not been