Although there will have and model therefore available survival may be difficult and in improving figures, which will be unduly pessimistic, when we are counseling caretakers or prospective parents of children with cystic fibrosis.

The predicted improvements in survival do have implications for population screening. The identification of carriers (heterozygotes) and consequent prenatal screening may be difficult to justify as parents may not feel that termination is appropriate for a fetus with a potential median survival of 40 years. In contrast, our data do not have any implications for neonatal screening, which may contribute to improvements in survival by identifying children with cystic fibrosis early in life.

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Invasive aspergillosis in a patient with idiopathic thrombocytopenic purpura without underlying lung disease

Dr L B Palmer and others (January 1991;46: 15–20) describe six patients with invasive aspergillosis whose only impairment of defense comprised underlying lung disease and corticosteroid treatment. They pointed out two features—firstly, that invasive aspergillosis can also be observed in non-immunosuppressed patients and, secondly, that the role of chronic lung disease as a facilitating factor for its development. We report a patient with idiopathic thrombocytopenic purpura—a condition in which the immunology of the next decade may not be immediately: receiving corticosteroids, who developed disseminated aspergillosis without any previous lung disease.

A 63 year old man diagnosed two months earlier as having idiopathic thrombocytopenic purpura presented with fever and progressive dyspnoea. He was having high dose corticosteroids (60–100 mg prednisone daily) and had been diabetic for 10 years. On admission a chest radiograph showed bilateral pulmonary infiltrates. The white blood cell count was 12.3 x 10^9/l (90% neutrophils), haemoglobin 14 g/dl, and platelet count 65 x 10^9/l. He required intubation and mechanical ventilation. Bronchoalveolar lavage showed Pneumocystis carinii. Co-trimoxazole and corticosteroid treatment was started on the fifth day. The patient was sereogeneous for the human immune deficiency virus. He improved during the next 10 days but then deteriorated and empirical antibiotic treatment with erythromycin, cepazidime, amikacin, and amphotericin B was started. A radiograph showed a cavity in the right middle lobe, a right pneumothorax, and pneumomediastinum. Culture of bronchial secretions from a second lavage was positive for Aspergillus fumigatus but no P carinii was identified this time. Despite full dose treatment with amphotericin B, the patient deteriorated and died a week later after invasive aspergillosis was diagnosed and at necropsy A fumigatus was recovered from multiple abscesses in lungs, kidneys, thyroid gland, and heart. The fungus was also found in mitral valve vegetations and in an embolus in the right pulmonary artery.

There are several interesting findings in this patient. Firstly, serious opportunistic infections developed without a major cause of immunosuppression (that is, neoplasia or HIV infection), much as in patients 1 and 6 in the series of Dr Palmer and colleagues. Immune disorders caused by diabetes might have played a part along with corticosteroid treatment in the development of this fulminating illness. In recent years the range of underlying diseases associated with aspergillosis has expanded substantially and diabetic patients have developed invasive infections. Secondly, the infection was extensive, as invasive pulmonary aspergillosis was rapidly followed by fulminating dissemination to the kidneys, heart, thyroid gland, and probably brain. The disseminating pattern differs from that reported by Dr Palmer and colleagues, in which aspergillosis was confined to the lungs. Finally, the presence of A fumagatus endocarditis, an exceptional finding in thrombocytopenic patients, is probably related to platelet malfunction and the low counts. Taken together these confirm that the spectrum of invasive aspergillosis is broader than was originally considered. This infection should be suspected even in patients with minimally compromised immunity.

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European conference on aerosols

A European conference on aerosols, organised jointly by the Aerosol Society and its German equivalent, Gesellschaft für Aerosolforschung e V, will be held at Gresham College, Oxford, on 6–11 September 1992. The main topics will be aerosols in the environment, health aspects of aerosols, characterisation of aerosols, industrial aerosols, and fundamental properties of aerosols. This conference will include one on drug delivery. Further information may be obtained from the conference coordinator, A L Cussen (tel 0275 37787, fax 0275 847303, or Aerosol Society, PO Box 34, Portishead, Bristol BS20 SNR).