Psittacosis

Bedsoniae, the agents of psittacosis, are micro-organisms that have a place somewhere between viruses and bacteria. Like bacteria they contain both RNA and DNA as nucleic acids, whereas viruses have only one or the other. Bedsoniae have a cell wall containing muramic acid, a feature of bacteria but not of viruses, nor of mycoplasmas, which in many other respects closely resemble bedsoniae. The presence of such a cell wall is of more than theoretical interest, for it is on the cell wall that penicillin acts, and bedsoniae like some bacteria are sensitive to penicillin, while viruses and mycoplasmas are not. Bedsoniae depend for survival on parasitism within a host cell, and they may be on the verge of biological independence, for they can produce metabolites on their own, and there is some tentative experimental evidence that they may replicate on enucleate cytoplasmic fragments.¹

Over 70 members of the psittacine or parrot family have been shown to be infected with bedsoniae. The list includes macaws, cockatoos, parakeets, and budgerigars. Bedsoniae have been isolated from at least 26 different types of finches, including canaries, bullfinches, goldfinches, and sparrows, as well as from crossbills, siskins, tits, and many other common birds. Pigeons, both wild and domesticated, are often heavily infected, and bedsoniae have been found in pheasants, egrets, seagulls, puffins, and petrels. Infection is common in poultry—hens, ducks, geese, and turkeys. The birds usually have no symptoms, but sometimes the infection causes severe economic losses in the flocks.

With all this infection in birds, what is the danger to man? In the United States only 36 cases of human psittacosis were reported with onset in 1970, and the highest number in any one year from 1961 onwards was 102.² Some cases may not have been reported, and others may not have been diagnosed because of their mildness, but obviously the incidence in man compared with the incidence in birds is very low in the United States. In Britain it appears to be so too. Indeed, news of a confirmed case, because of its rarity, is likely to hit the daily headlines. The reason for the low infectivity in man is probably the nature of the infection in birds, low-grade or latent rather than overt. Pathological changes occur in the abdominal organs of birds much more often than in the respiratory tracts, and many infected birds have no symptoms at all. But sometimes the infection flares up, especially at breeding time or when birds are kept in crowded, insanitary conditions. Then many birds die, especially the young ones, while others may have infectious discharges from their beaks and diarrhoea, and excrete the organism in their faeces and urine. Feathers becomes grossly contaminated and dust in the environment may be loaded with bedsoniae. In such conditions bedsoniae may be inhaled by the aviarist and he gets psittacosis from his neglected birds. But instances are on record where momentary contact with a bird has led to human infection, as when all of 26 persons who passed through a room containing two apparently healthy parrots were infected, or when 12 actors on a stage were infected by the thirteenth member of the cast, a parrot.³

Railway guards have been infected from racing pigeons in crates in their vans,⁴ and as many as 500 cases of human psittacosis, 19 of them fatal, may have been caused by the breeding and transport of racing pigeons.⁵ Yet the pigeon strain of bedsoniae is probably not very virulent for man, and there is no evidence that tourists become infected in San Marco or Trafalgar Squares, though they may well be breathing bedsoniae in aerosol concentration.⁶ The parrot and especially the turkey strains of bedsoniae seem much more virulent for man, and psittacosis is an occupational hazard for those who have to handle infected turkey carcases. The same is true for laboratory workers handling bedsoniae cultures. But British budgerigars seem rarely to cause human infections.

The illness in man is usually mild and often undiagnosed. Serological surveys have disclosed evidence of infection in from 0·3% to 18·5% of sample populations, but little evidence of illness other than sometimes mild upper respiratory symptoms.⁷ ⁸ Patients ill enough to reach hospital, however, have a sharp febrile illness, with generalized aches and pains and usually cough. Radiography shows areas of consolidation much greater than physical signs would suggest, and these shadows may take several weeks to disappear. Most patients are on the way to recovery within a week, but sometimes convalescence is prolonged. Bedsoniae may be cultured from sputum or blood, and acute and convalescent sera show a rise in titre in the complement-fixation test. The organism often becomes quickly resistant to penicillin, so that a tetracycline is a better drug to use.

Some patients are much more ill, with involvement of the respiratory, cardiovascular, and central nervous systems.
Circulatory collapse, respiratory insufficiency, or disorientation and semiconsciousness may quickly appear, and for such patients emergency medical treatment of shock, dehydration, and respiratory failure is required. Fatality rates of between 15% and 20% or higher have been reported, mainly in the pre-antibiotic era. More accurate diagnosis now uncovers milder cases, and modern treatment reduces mortality, so that the fatality rate of diagnosed cases is now below 5%. In one series of 550 cases, diagnosed and treated early, it was 0-5%. Most of the deaths occur in older people, but the disease is occasionally fatal in infants and children. Two fatal cases in adults with associated endocarditis have recently been reported.

People who keep birds, especially in considerable numbers, should know of the dangers and be encouraged through their clubs to maintain a high standard in their aviaries. Birds should be bought only from shops which obtain their stocks from reliable commercial breeders. Mass-produced birds are sometimes treated prophylactically with antibiotics which apparently ensure freedom from food poisoning. It is not so easy to free poultry from infection. The risk in poultry-processing plants can be reduced by using wet instead of dry methods during plucking, but the danger from handling viscera remains, and one must therefore carefully supervise the day-to-day health of workers. The importing of birds was at one time prohibited, but the prohibition was revoked by the Parrots and Miscellaneous Birds (Prohibition of Importation) (Revocation) Order 1966. The original Order aimed at the prevention of foul pest as well as psittacosis, but it became obvious that imported birds were of little importance in the spread of either.


The Tropical Intestine

Some attacks of acute or chronic diarrhoea in hot climates can be attributed to bacterial pathogens or intestinal parasites, but often a causative organism is not isolated. Tropical sprue is one such syndrome of unknown aetiology which may even have become in some countries a blanket diagnosis covering any chronic malabsorption state. The clinical picture is broad. Diarrhoea and steatorrhoea with associated abdominal symptoms may follow an influenza-like illness. Absorption of xylose, fat, or vitamin B12 are usually impaired, and barium follow-through radiographs and biopsy of intestinal mucosa show non-specific abnormalities. Signs of secondary nutritional deficiency appear later, particularly hypoproteinaemic oedema and megaloblastic anaemia. The condition often responds to treatment with folic acid or broadspectrum antibiotics. Thus the clinical features overlap with those of both adult coeliac disease and the stagnant loop syndrome.

Tropical sprue has now been reported from most parts of the tropics but its prevalence is often unknown. It seems to be rare in Africa. Originally described in European colonists, the disease is now known also to affect the indigenous population. Occasionally it develops after a person has moved to a temperate climate. H. Sprinz and his colleagues, working in Thailand in 1962, found that the jejunal mucosa of apparently healthy adults was structurally abnormal when compared with normal subjects in the U.S.A. or Europe, and also their xylose absorption was impaired. The villi were shorter, broader, and thicker (leaf forms) and many had fused together to form short ridges. Convulsions were rare, and a flat mucosa devoid of villi was never seen. There were an increased number of chronic inflammatory cells in the lamina propria.

These findings have since been confirmed by studies from many other countries. One suggestion is that tropical sprue (symptomatic tropical malabsorption) is just the tip of a vast iceberg of subclinical (asymptomatic) tropical jejunitis, and another that the two conditions are separate entities with some overlapping features. Any definition of a normal intestinal mucosa must now take geographical variations into account, and it is important that these changes should not be attributed to a concurrent tropical disease such as cholera or parasitic infestation.

We know less about the prevalence of asymptomatic malabsorption in the tropics. Subnormal xylose tolerance may be found in up to 66% of subjects, but the test is notoriously unreliable. There have been no comparable studies of vitamin B12 or fat absorption, but probably malabsorption of these substances is less common and it is difficult to correlate evidence of abnormal structure with function. Knowledge of the natural history of the intestinal changes was first obtained by studying American soldiers in Vietnam and Peace Corps workers in Pakistan. The subjects were not asymptomatic, since many suffered from recurrent, mild diarrhoea. About 33-50% developed structural and functional changes in the small intestine which were less severe than in the indigenous population and which usually disappeared within a few weeks or months of returning home.

These observations have now been extended by studying residents of tropical areas who have migrated to temperate climates. Asymptomatic West Indians recently moved to New York showed intestinal abnormalities similar to those found in their country of origin, but they usually regressed rapidly. C. Gerson and his colleagues have now reported reversal of abnormalities in Indians and Pakистanis living in New York and have exonerated curried foods and intestinal parasites as causes. Since the lesions are not present in the fetal intestine, appear rapidly after birth, and are reversible a genetic cause is unlikely. Current aetiological hypotheses favour repeated contamination by bacteria or viruses and malnutrition.

J. M. Faisley has confirmed that subclinical abnormalities of intestinal mucosal structure and function may occur in Nigeria, where tropical sprue is not endemic, and has related some of them to protein malnutrition. A reversible intestinal lesion is known to occur in children with kwashiorkor and has been reported in adults but its role in adults is uncertain. In areas where tropical sprue is endemic the patients may have been suffering from protein deficiency secondary to sprue. In non-endemic areas the patients were given folic acid as well as protein, and this