Farmer’s lung disease in Somerset

J. V. S. PETHER and F. B. GREATOREX
Public Health Laboratory, Musgrove Park Hospital, Taunton, Somerset

Pether, J. V. S. and Greatorex, F. B. (1976). British Journal of Industrial Medicine, 33, 265-268. Farmer’s lung disease in Somerset. A survey of laboratory records was made to assess the value of the precipitin test and isolation methods in the diagnosis of farmer’s lung disease and also to determine its prevalence in the farming population of Somerset. A link was established between the clinical diagnosis as written on the form that accompanied the specimen and the actual number of positive laboratory diagnoses made. Fifty (43%) of the clinically diagnosed patients were serologically positive for farmer’s lung during a four-year period. If the clinically diagnosed but serologically negative cases of farmer’s lung disease are added to this number, a prevalence of about 23 per 1000 of the farming population of Somerset is obtained.

Farmer’s lung disease is a form of extrinsic allergic alveolitis caused by inhaling antigenic material from mouldy hay (Pepys and Jenkins, 1965); it is characterized by dyspnoea and cough which appear some hours after exposure and occurs in farmers, their families, or hired workers who are continually exposed to spores from mouldy hay, grain, etc. Farmer’s lung was recognized and identified by Campbell (1932), but it was not until Pepys et al. (1963) identified the main thermophilic actinomycetes involved, now called Micropolyspora faeni and Thermoactinomyces vulgaris, that attention was drawn to this distressing and disabling disease. It was prescribed as an industrial disease by the Department of Health and Social Security in Statutory Instrument 1537 (1975). Since 1965 work has been concerned mainly with organisms identified as related to precipitins found in the sera of cases of farmer’s lung. Wenzel et al. (1974) identified a series of organisms and suggested that a battery of antigens be used to test blood for precipitins. Flockton and Cross (1975) pointed out that T. vulgaris does not conform readily to the type species concept. We have suggested (Greatorex and Pether, 1975) that what was originally described as a variant of T. vulgaris but is now identified as T. candidus (Cross and Unsworth, 1976) should also be used.

A negative precipitin reaction from a clinically diagnosed case of farmer’s lung disease is not uncommon and this makes it difficult to establish its prevalence. Surveys have been made of the prevalence of farmer’s lung in the United Kingdom. Grant et al. (1972) produced approximate figures, based only on cases that had positive serological results during a short period of time, of 43 per 1000 in the farming population of Orkney and 36 per 1000 for Ayrshire. They pointed out that had they included the clinically diagnosed but serologically negative cases their figures would have been much higher. Staines and Forman (1961), however, after a postal inquiry produced figures of 0.115 per 1000 for the south-east of England and 0.73 per 1000 for the south-west. However, many cases must have been overlooked by this method. Smyth et al. (1975) returned a figure for Devon of about 6 per 1000 of serologically positive cases, although Morgan et al. (1975) on the basis of moderate or strong positive results in serological testing during the winters of 1970-71 and 1971-72, found rates similar to those of Scotland (22/1000 in Devon and 54/1000 in Wales). They also emphasized the difficulties involved in estimating the prevalence of farmer’s lung. Somerset has a climate similar to that of Devon and farming methods are basically the same. It was decided to see if the prevalence of farmer’s lung in Somerset was similar to that of Devon using information
obtained from laboratory records. It was also decided to examine the value of the precipitin test in the diagnosis of farmer’s lung disease.

Materials and methods
Precipitation tests against Microsporospa faeni, Thermoactinomyces vulgaris, and Aspergillus fumigatus have been performed at this laboratory since August 1971. Tests are recorded in a bench book together with the patient’s name and clinical diagnosis, etc. The clinical material in this survey is taken from the bench book and from farmer’s lung disease inquiry forms supplied by the Mycology Reference Laboratory which were sent to some of the positive cases. The geographical area covered by this laboratory is the present county of Somerset. Specimens are received from three consultant chest physicians and from some general practitioners in the area. The total population of Somerset is 400,000, the farming population involved in this survey is some 5000 persons. The latter figure is made up of farmers, farm directors, their wives, and full-time hired hands; it was supplied by the local office of the Ministry of Agriculture. Part-time and seasonal workers were excluded.

Sera
Altogether 691 sera (657 patients) sent to the laboratory between August 1971 and August 1975 to investigate the possibility of farmer’s lung or aspergillosis were examined by immunodiffusion tests for precipitins to M. faeni, T. vulgaris, and A. fumigatus. Since February 1974 the sera had also been examined for precipitins to a Thermoactinomyces candidus (Kurup et al., 1975) isolated at this laboratory from the sputum of a patient suffering from farmer’s lung disease. The method for the immunodiffusion test is basically that of Ouchterlony and has been described in a previous paper (Greatorex and Pether, 1975). The M. faeni, T. vulgaris, and A. fumigatus antisera were supplied by the Mycology Reference Laboratory and used at the recommended dilutions; the T. candidus antiserum was identified at this laboratory and used neat.

Antigens
The M. faeni, T. vulgaris, and A. fumigatus antigens were supplied by the Mycology Reference Laboratory and used at the recommended dilutions. The T. candidus antigen was prepared at this laboratory; originally used at a dilution of 1/8 it is now used neat (Greatorex and Pether, 1975).

Sputa
From April 1974 until April 1975, 1300 sputa were cultured to demonstrate the presence of thermophilic actinomyces implicated in the pathogenesis of farmer’s lung disease. The method, selective for thermophilic organisms, is described in our previous paper (Greatorex and Pether, 1975).

Results
Table 1 gives the precipitin results when the 691 sera were split into three groups according to diagnosis. Group A is made up of sera from patients (farmers, wives, or farm workers) who had symptoms of farmer’s lung or were stated to have farmer’s lung, or were an ‘old case of farmer’s lung’. Group B comprises sera from patients whose symptoms had led the doctor to request for antibodies to Aspergillus species. Group C contains the bulk of the sera from patients whose symptoms did not fit the diagnosis of either farmer’s lung or aspergillosis. Of the sera from Group A 43% were positive to M. faeni and the other farmer’s lung antigens, approximately 50% of the Group B sera were positive to the A. fumigatus antigen, and only about 8% of the Group C sera were positive to any of the antigens used; these last precipitin reactions were mainly very weak. Thus, on the whole, there were few false positive reactions, although the patients in Group A showed a disappointingly low percentage of precipitins in their sera.

The Figure gives a graphic laboratory view of the incidence of farmer’s lung disease in Somerset. The step-chart is compiled from M. faeni antibody tests only but it clearly shows the greater incidence of farmer’s lung disease from December to April. The months of July, August, September, and October are relatively clear of the disease.

Table 2 shows the results of the isolation of thermophilic actinomyces from 1300 sputa. These organisms were isolated from the sputa of 20 patients, all of whom had clinically diagnosed farmer’s lung disease. M. faeni was isolated from the sputa of 16

<table>
<thead>
<tr>
<th>TABLE 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Precipitins to Thermophilic Actinomyces in 691 Sera; Number of Patients in Brackets</strong></td>
</tr>
<tr>
<td><strong>Group</strong></td>
</tr>
<tr>
<td>A</td>
</tr>
<tr>
<td>B</td>
</tr>
<tr>
<td>C</td>
</tr>
</tbody>
</table>

Group A: Sera from patients with diagnosed farmer’s lung disease or typical symptoms
Group B: Sera from patients suspected to be suffering from aspergillosis
Group C: Sera from patients with symptoms not typical of farmer’s lung disease
patients with farmer's lung; from the sputa of nine of these 16, Thermoactinomyces species was also isolated. T. vulgaris was the only isolate from the sputum of one patient and T. candidus was the only isolate from the sputa of a further three patients. Six of these isolates came from patients who had no precipitins in their sera to the appropriate antigens. The nine Thermoactinomyces species isolated in conjunction with M. faeni were identified as T. vulgaris (2), T. candidus (7).

Discussion
The diagnosis of farmer's lung disease is essentially clinical. The laboratory can help first to confirm the suspicions of the doctor and secondly it can investigate the epidemiology and pathogenesis of the disease. The results of this survey show that it is probable that the consultant or general practitioner miss very few cases; the laboratory results unfortunately are not so good. Of the sera examined from clinically diagnosed cases 57% were found to be serologically negative to the three farmer's lung disease antigens used. It is possible, however, that since a clinical diagnosis of farmer's lung has been made, advice in many cases is given to avoid fusty hay, with a consequent decrease in the number of antibodies to a level not detected by our method. The use of an additional antigen in the present survey has so far not increased the number of positive tests significantly but it has improved some results and possibly provided a better picture of the organisms involved in the disease. T. candidus seems to be involved in farmer's lung disease in Somerset more than the standard T. vulgaris strain.

The farming population covered by this survey is 5000, the number of serologically positive cases of clinical farmer's lung disease was 50, which gives a prevalence rate of 10 per thousand of the serologically positive disease among the farmers of Somerset. This compares with 6 per 1000 quoted by Smyth et al. (1975), or 22 per 1000 by Morgan et al. (1975) for Devon. If the cases that were serologically negative although diagnosed as farmer's lung disease on clinical grounds are added to our figure of 10 per 1000 then a figure of 23 per 1000 is produced. This is about half the appropriate figure quoted by Morgan et al. (1975) for Devon and Grant et al. (1972) for Scotland when they added serologically negative clinical cases to the serologically positive ones. While it is recognized that a prospective survey of whole communities would give a more accurate figure for the prevalence of farmer's lung, it is probable that the prevalence shown by this small survey of laboratory results comes to something near the actual figure.

Culture of the thermoactinomycetes may be a useful pointer to the disease; six of the clinically positive but serologically negative patients with farmer's lung had these organisms isolated from their sputa. We suggest that a search for other thermoactinomycetes should be made, precipitins to which may go some way to improve the serological diagnosis of farmer's lung. It is also suggested that isolation of the organisms from sputa is a useful confirmation of the diagnosis.

We wish to thank the many clinicians who provided data and specimens from their patients. The director, Dr D. W. R. Mackenzie, and staff of the Mycology Reference Laboratory are also thanked for their assistance and the provision of standard organisms and antisera.

References
Flockton, H. I. and Cross, T. (1975). Variability in
The August (1976) Issue

Predictions of mortality from mesothelial tumours in asbestos factory workers  M. L. Newhouse and G. Berry

Monitoring liver disorders in vinyl chloride monomer workers using greyscale ultrasonography  D. M. J. Williams, P. M. Smith, K. J. W. Taylor, I. R. Crossley, and B. W. Duck

Loss of pulmonary elastic recoil in workers formerly exposed to proteolytic enzyme (alcalase) in the detergent industry  A. W. Musk and B. Gandevia

Byssinosis: a follow-up study of cotton ginning workers in the Sudan  M. Khogali

Acute inhalation toxicity of cotton plant dusts  R. Rylander and M.-C. Snella


Urinary non-precipitable lead in lead workers  M. Kawai

Erythrocyte fluorescence and lead intoxication  K. G. A. Clark

Notes and miscellanea

Parosmia and hyposmia induced by solvent exposure  E. A. Emmett

Book reviews

Information section

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, London WC1H 9JR, Price £2.50 (U.S.A. $7.50).