DR. BERRY This is a very small series but there was a very nice correlation with the degree of lymphocyte infiltration which might tie in with the idea that it is in fact showing a cellular immune mechanism.

DR. P. FOWLER (*Macclesfield*) A very few rheumatoid patients develop parotid gland enlargement when treated with Pyrazoles. It is interesting to speculate that these patients might be the ones in your rheumatoid group with sensitivity to parotid antigen, but have never produced any clinical signs of this until they have been treated with this group of drugs. It would be very interesting if you could look into this.

DR. BERRY I should be very interested in doing something along these lines.

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Proteolytic Activity of *Mycoplasma arthritidis*. By J. W. CZEKALOWSKI, D. A. HALL and P. WOOLCOCK (*University of Leeds*)

It has been claimed that mycoplasmas are associated with certain symptoms of arthritis, both in man and in animals. One of the reasons for the development of arthritic lesions could be that changes may take place in the protein-polysaccharide complexes of joint tissue. Bearing this in mind, we have explored the effects of various mycoplasmas and their metabolic products on proteins which are likely to be encountered in joints either in native or modified forms. Of several mycoplasmas examined, only two strains, namely PG6 and PG27, of *Mycoplasma arthritidis* showed liquefying effect on gelatin, a property examined in our screening programme, and the factors responsible for this activity appeared both intra- and extracellularly.

Two enzymes one of high and the other of low molecular weight, have been isolated by processes of ammonium sulphate and acetone precipitation and gel filtration of the filtered growth medium. Although both enzymes attack gelatin, neither is active against intact undenatured collagen, and hence they should be classified as gelatinases rather than collagenases. However, since neither of them has any effect on other soluble proteins such as casein, they must be differentiated from enzymes displaying generalized proteolytic activity such as trypsin and chymotrypsin, which also attack gelatin. Both enzymes release free amino groups and oligopeptides from gelatin, but only the heavier molecule does this with the simultaneous liberation of free hydroxyproline. It may,

therefore, be assumed that the two enzymes attack different parts of the gelatin molecule and, in view of their high specificity of action, we consider that they may represent a new class of enzyme.

The association of these enzymes with strains of *Mycoplasma arthritidis* offers a suggestion that these organisms may participate in the further degradation of joint collagen after its initial denaturation by other so-far unknown factors.

Discussion

DR. A. G. S. HILL (*Stoke Mandeville*) You mentioned that PG27 came from a human case. I don't think you were very specific about a case of what?

DR. CZEKALOWSKI PG6 was isolated from a rat and PG27 from an arthritic patient, but not from the arthritic lesion. Over the years, either PG27 has become contaminated with a rat strain, as may happen in laboratories, or antigenic degradation has taken place during propagation. Serologically they are very close to each other, but it is still possible to show the difference between PG27, the original human strain, and PG6, the rat strain.

PROF. D. A. WILLOUGHBY (*London*) What is the pH activity of the enzymes that attack gelatin? They were tested at pH 6; what is the shape of the curve of activity of the enzymes?

DR. HALL They both appear to have a rather broad range of activity stretching from pH 6 to 9.

DR. M. WILLIAMS (*Liverpool*) A word about the confusion between *Mycoplasma arthritidis* and *Mycoplasma hominis* type 2: the latter was originally isolated from the female urethra and it was not associated with human rheumatoid arthritis until Jansson (1971) published her paper on the subject. The original confusion probably arose through PG27—the human strain of PG6—being isolated in separate laboratories and being passed from one laboratory to another. I do not think there is now very much doubt that the two strains are immunologically and serologically identical. With regard to the pathogenesis of mycoplasmas, they do appear to be quite 'host-specific' in terms of the pathology that they can produce. I have isolated *Mycoplasma fermentans* from human early effusions but have been unable to induce a similar arthritis with this organism in any other sort of experimental animal. From that point of view, it is comforting to know that *Mycoplasma arthritidis* is the only species which has been shown to produce gelatinases, since this organism appears to be pathogenic only for the rat.

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