Emmy Wittig, who kindly carried out the nitrogen analyses. The experiments were made with equipment purchased by the Rockefeller Foundation, to which we are much in debt. I thank also Dr. Hodgson, who provided the mixture of labelled amino-acids.

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Hydroxyproline and the Shrinkage Temperature of Collagen

Dr. B. J. Rigby and Dr. J. D. Spikes[1] have quoted a value of 8·3 per cent for the hydroxyproline content of fibrinoid tissue as being taken from a paper by Bowes, Elliott and Moss[2]. The figure given in this paper is in fact 7·6 for the hydroxyproline-nitrogen expressed as per cent of the total nitrogen of the gelatin solution obtained on autoclaving fibrinoid tissue. Assuming a total nitrogen content of 18 per cent, this corresponds to 12·8 per cent hydroxyproline on protein weight which, according to Fig. 1 of Rigby and Spikes, should give a T_s of 60° or more. Their suggestion that rheumatic effects are the result of a structural breakdown due to temperature effects only is, therefore, not supported.

We would also like to direct the attention of the authors to certain exceptions to the argument that the T_s of collagen is related to its hydroxyproline content, for example, invertebrate collagens[3–5].

In the absence of any evidence that the preparation is homogeneous it seems more likely that variation in the hydroxyproline content of mammalian tissues is an indication of the collagen content, rather than of differences in the composition of the collagen itself.

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WE regret the error in our original publication[1], and thank Drs. Bowes and Moss for the correction. As they point out, the T_s corresponding to the correct value for the hydroxyproline in the example quoted is about 60° C. However, as was stated in the original article of Bowes, Elliott and Moss[2], fibrinoid tissue from patients suffering from rheumatic fever and rheumatoid arthritis contains less hydroxyproline than normal tissue, so that our statement was qualitatively correct. We reiterate our main theme, that since the denaturation and shrinkage of collagen is a time-dependent process[3], collagen will denature at temperatures below the instantaneous shrinkage temperature given a sufficiently long time, and that this time will be reduced (at any fixed temperature) if the percentage of hydroxyproline is also reduced. Gustavson[4] has presented experimental evidence along these lines showing that calf-skin begins to denature in water at 45° C. after one week. Stress relaxation studies[5] also show that rat-tail tendon slightly extended in 0·9 per cent saline begins to denature at about 40° C., although there is no tendency for the tendon to shrink when unloaded. We consider this to be the beginning of the structural breakdown which, however, only takes place on a large enough scale to cause instantaneous shrinkage at the higher temperature known as the shrinkage temperature.

With reference to invertebrate collagens we point out that our speculations applied only to vertebrates and were applicable only because of the similar chemical nature of these collagens. With invertebrate collagen the greater variability in chemical composition[6] means that differences in chemical properties cannot be related to one particular constituent.

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HÆMATOLOGY

Possible Origins of the Anti-Gm Sera

SINCE Grubb and Laurell discovered the Gm factor in 1956 (ref. 1), all those who have worked in this field have carried out the tests with sera from patients suffering from rheumatoid-arthritic diseases. Thus Harboe and Lundevall[2] and then Harboe[3] found and described the Gmx and Gmb factors in 1959. Also using a rheumatoid-arthritic serum, Steinberg[4] was able to demonstrate in the sera of Negroes the presence of a Gm-like factor, a factor which is not found in the white population. In 1959, however, Grubb found an anti-Gm factor in the serum of a young girl who was suffering from hepatitis with plasma cells[5].

Investigation of the Gm factor is based on the fact that a certain percentage of normal sera has the property of inhibiting the agglutinating power of some rheumatoid-arthritic sera. It is characteristic of this agglutinating power that the sera agglutinate O CDE red cells sensitized by an incomplete anti-D. Such agglutinating powers have been found and described by Milgrom *et al.* in the sera of clinical patients and by Unger *et al.* in the sera of healthy persons. But these authors have also noticed the non-inhibition of these agglutinating powers by the adjunction of human γ-globulins or normal sera.

From examination of more than 12,000 blood donors from the population of the Seine-Maritime, France[6,7], and also patients from different departments of our hospital, we have found this agglutinating power in some sera. But these sera could be inhibited by the adjunction of other normal sera[8]. Further investigations have shown that these sera were anti-Gm.

Hence, at the present time, we have been able to demonstrate the presence of anti-Gma in the sera of