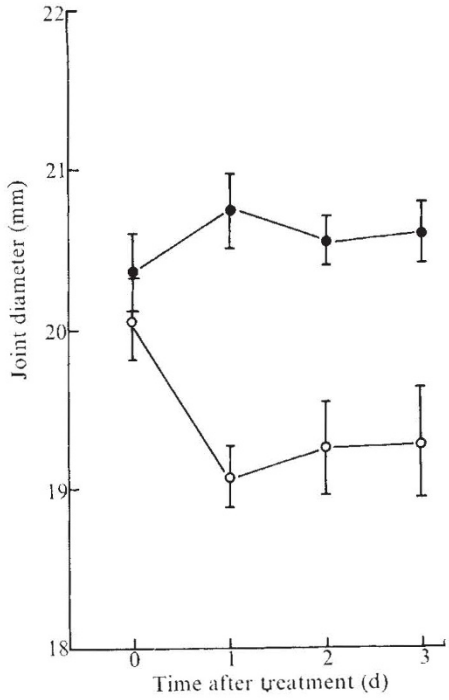

Fig. 2 Effect of liposomes containing cortisol palmitate on joint diameter. Maximum joint diameter was measured using a Baty spring-loaded micrometer. Values are mean ± s.e.m. of 6 animals. ○, Treated joint; ●, untreated joint.

The demonstration that an injection of cortisol palmitate incorporated into a liposome preparation has a much greater anti-inflammatory activity than an equivalent dose of microcrystalline cortisol acetate, a standard anti-inflammatory steroid ester, validates the use of liposome preparations for local delivery of steroids.

This work was carried out at Strangeways Research Laboratory, Cambridge, and at ICI Pharmaceuticals Division, Alderley Park, Cheshire. Similar results were obtained at both centres.

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Relationship between haptoglobin and *Streptococcus pyogenes* T4 antigens

During a survey of agglutinating activity of human saliva against streptococci[1] we found that saliva from individuals of blood group 0 agglutinated a particular strain of group G streptococci more often than did serum from those with blood group A or B. When we tested 833 sera we found a dimorphism for antibody-like substances against group G streptococci—sera could be divided into two groups according to their agglutination titre against group G streptococci. The first group had low titres, not rising above 1 : 4 and more usually 1 : 1; the second group had much higher titres ranging from 1 : 200 to 1 : 3,200. There was no overlap between the two groups of sera. We thought that this might be related to a genetic system, probably diallelic. We now report a relationship between the haptoglobin genotype of serum and the streptococcal antigens with which it reacts.

We tested our samples for a wide range of polymorphic blood groups (ABO, MNSs, P_1/P_2, rhesus, K/k, Le^a/Le^b, Duffy and Kidd), serum groups (Gm 1, Gm 2, Gm 4, Hp, Gc), enzyme groups (acP, PGM, ADA, AK, GPT and EsD) and HLA groups (factors of loci Sd^1 and Sd^2). All sera without agglutinating activity or with low titres against a particular strain of group G streptococci were of the haptoglobin (Hp) type Hp 1-1. All sera with high titres, however, were of Hp types 2-1 or 2-2. There was no effect due to sex or age.

Further studies showed that sera with the Hp^2 gene product reacted with *Streptococcus pyogenes* strains carrying the T antigen complex T4/24. This antigen may also be present in some C and G group streptococci and it was only these strains that were agglutinated with high titres by Hp 2-2 and Hp 2-1 sera. In our series 1.4 % of group C streptococci and 6.4 % of group G streptococci carried the T4/24 antigen. A relationship between the T4/24 antigen of streptococci and haptoglobin types has not been described before.

The mechanism of the reaction is unknown but the reacting substance in all human sera is haptoglobin itself. This was demonstrated in cooperation with Dr Uhlenbruck in Cologne, using a purified haptoglobin preparation (98 % purity, Behringwerke, Marburg) which agglutinated the streptococci in the same way. Further evidence was given by a survey of human cord blood samples. If agglutination was negative, haptoglobin could not be detected. Animal sera with Hp 1-1-like patterns (rhesus monkey, rabbit, pig and sheep) did not agglutinate the T4/24 streptococci or did so only to low titres.

Finally, we found that the agglutinating activity of Hp 2-2 or Hp 2-1 sera could be inhibited by the addition of Hp 1-1 serum of human or animal origin before the test. We believe that Hp 1-1 protein represents a form of 'blocking antibody' for the appropriate streptococci.

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