streptokinase, proteinase precursor or erythrogenic toxin failed to affect lysosomes or mitochondria; activated proteinase or erythrogenic toxin caused transient arthritis. The results indicate that minute quantities of streptolysins, especially SLS, can release hydrolytic enzymes from lysosomes. The subsequent denaturation of native tissue components may be a primary event leading to auto-immunity.

Effect of Ascorbic Acid on Rheumatoid Factor

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Thiol compounds can depolymerize rheumatoid factor (RF) in vitro by reducing disulfide bridges. One of these compounds, penicillamine, has in vivo produced a slight lowering of serum RF. Ascorbic acid (AA) is a potent reducing substance that can be given to humans in large amounts without toxicity. The euglobulin fraction of rheumatoid sera was dissolved in glycine saline buffer (pH 8.2), sterilized by passage through a 0.22 μ Millipore filter, and incubated with AA (1500 γ/ml.) at 37 C. After 42 hours incubation, the latex titer of the euglobulin fraction had been reduced from 1:5120 to $<$1:20. A 22 S component (i.e., RF + 7 S γ globulin) present in the rheumatoid euglobulin fraction could no longer be detected by analytical ultracentrifugation after this fraction had been incubated with AA. The way in which AA destroys the serologic activity of RF is not clear, but it may be due to reduction of disulfide bridges in RF by AA. The effect of AA on serum RF is being studied in 5 patients with rheumatoid arthritis. One has been given AA (4 Gm./day) for 6 weeks. The latex titer has decreased from 1:5120 to 1:320. Four patients have received AA for only 2 weeks. In 2 of these the latex titer has decreased slightly. These studies indicate that AA is capable of lowering serum RF in some patients with rheumatoid arthritis.

The Role of the Protein Moiety in the Antigenicity of Chondromucoprotein

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Chondromucoprotein was isolated from human articular cartilage and fractionated into two proteinpolysaccharides, PP-L and PP-H. PP-L was the purer fraction, containing about 25 per cent protein, but no collagen. PP-L was mixed with Freund's adjuvant and injected into rabbits. Four weeks after the initial injection the rabbits developed positive skin tests when challenged with PP-L. Biopsy showed changes consistent with an Arthus reaction. If the PP-L was first digested with a proteolytic enzyme (i.e., trypsin) skin tests were negative. After 4½ weeks booster injections of PP-L were given, and at 6 weeks potent antisera (AS) were obtained. Antibodies to PP-L were demonstrated by agar gel immunoelectrophoresis. After electrophoresis of PP-L at pH 8.6, AS absorbed with excess human serum were placed in the troughs. A prominent precipitin arc developed on the anodal side of the origin. This arc stained metachromatically. If AS was absorbed with PP-L, this arc was no longer obtained. If PP-L was digested with trypsin prior to electrophoresis, no arc was obtained. Antibodies to PP-L were also detected by tanned cell agglutination tests. Titers as high as 1:5120 were obtained. These studies indicate that human PP-L is antigenic in the rabbit and that the protein moiety of PP-L is necessary for the antigenicity. These findings suggest that the antigenic sites of human PP-L are either in or closely associated with its protein moiety.

Anticytoplasmic Antibodies in Patients with Systemic Lupus Erythematosus (SLE) and Infectious Hepatitis

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Antibodies against cytoplasmic constituents of the liver have been described in the serum of patients with SLE and infectious hepatitis by many authors. This study was performed in order to characterize these antibodies with respect to their im-