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REVIEW

Mechanisms of Cell and Tissue Injury Induced by Group A Streptococci: Relation to Poststreptococcal Sequelae

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I. Introduction

Despite the voluminous literature available on the role played by group A streptococci in the pathogenesis of human disease, our knowledge of the mechanisms involved in the initiation of the tissue lesions characteristic of the sequelae after streptococcal infection in man is far from complete.

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Numerous attempts have been made over the years to induce in laboratory animals tissue lesions that will simulate those in humans who develop rheumatic fever, arthritis, and nephritis. A variety of streptococcal species that have been isolated from human patients have been inoculated via different routes into a variety of animal species. Since, however, the principle of differentiation of hemolytic streptococci into serologic types was described only at the end of the 1920s [1], the nature and biological properties of many of the streptococcal strains employed by earlier investigators have not been definitely established. The pathologic lesions that developed in the experimental animals ranged from septicemic lesions to granulomatous inflammation, which in some cases resembled those seen in humans suffering from