mice to infection with group A Streptococcus, suppressed the streptococcicidal activity of mouse blood, and inhibited formation of antibody to Streptococcus. These effects of the α-globulin were greatest when the protein was injected one day before bleeding the animals or bacterial challenge. These effects were dose dependent. The doses of α -globulin that significantly suppressed production of antibody to streptococci were ineffective in increasing the susceptibility of mice to infection and in depressing the bactericidal activity of the blood of mice. It is concluded that lymphocytes and macrophages are susceptible to different concentrations of α -globulin. A high incidence of myocardial, pericardial, and hepatic lesions was found in animals treated with α-globulin and challenged with streptococci, both virulent and nonvirulent for mice. Alpha globulin alone did not cause any similar effects. It is suggested that the depression of phagocytosis by α -globulin enhanced the proliferation of streptococci and allowed the in-vivo production of larger amounts of tissue-damaging toxins.

An a-globulin fraction of normal human plasma increased the susceptibility of