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Complement Levels in Experimental Allergic Encephalomyelitis

During the course of investigations designed primarily for the characterization of mammalian tissue antibodies, experimental allergic encephalomyelitis was produced in guinea pigs by subcutaneous inoculation with an emulsion of rabbit brain in complete Freund’s adjuvant. The principles of laboratory animal care as promulgated by the National Society for Medical Research were observed. In a representative experiment, encephalomyelitis was clinically manifested by paralysis in 6 of 8 animals. Circulating hemolytic complement (C') levels, however, were unaltered after the injection of brain and during the course of illness, even though the paralysis usually terminated in death. The failure to detect changes of C'-levels in this condition lends support to Waksman’s generalization that changes in C' have not proved to be very useful as indices for characterizing the pathological mechanisms of certain human diseases. In allergic encephalomyelitis, however, the blood-brain barrier may interpose a particularly severe restriction as far as changes in C'-levels may be concerned.

Although C'-levels were unaltered, C'-fixing antibodies against homogenates of guinea pig brain regularly occurred in high titre (27 or greater) in guinea pigs paralysed as a result of the rabbit brain–Freund’s adjuvant injection. Comparable anti-guinea pig brain antibody levels were also produced in guinea pigs injected with a white fish (Coregonous sp.) brain–Freund’s adjuvant emulsion. In addition, albino rats receiving the rabbit brain–Freund’s adjuvant inoculum showed similar antibody levels in C'-fixation tests with albino rat brain antigen. Neither of the two latter groups of animals, however, developed clinical signs of disease (that is, paralysis) and on this basis ‘auto-antibodies’ per se apparently do not play a causative part in experimental allergic encephalomyelitis. On the contrary, recent evidence indicates that serum containing high levels of anti-brain C'-fixing antibodies may exert a protective effect and thus prevent development of disease in animals injected with such serum. It would be of interest, therefore, to determine whether the brain, lacking the capacity to induce encephalomyelitis in the guinea pig, contains ‘protective’ antigen. Conceivably, antiserum to fish brain might neutralize the encephalitogenic activity of mammalian brain in a manner similar...
to the inhibition of enzymes by antibody that does not combine with the site of enzyme action.

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