The series described shows a small but definite alteration of the pattern of rejection of skin homografts applied to rabbits' ears after immunization with an azo-coupled extract of activated lymph nodes. A larger series with closer control of such variables as dose rates of immunant and routes of injection, together with histological examination of the regional nodes at the time of rejection, would be desirable.

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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 hæmolytic complement (C')[1] 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[2]. 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[3] 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[4] 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 fish 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[5].

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Correlation between Virulence and Phagocytosis of Genetic Recombinant between *Escherichia coli* and *Salmonella typhimurium*

The introduction of quantitative methods to measure the rate of phagocytosis of bacteria by the reticulo-endothelial system *in vivo* has revived interest in the examination of the relationship between bacterial virulence and phagocytosis[1-3]. Although recent investigations have shown no correlation between the rate of phagocytosis of different bacterial species and their virulence in mice[4,5], a rather different relationship has been noted for different bacteria of the same species. The rate of phagocytosis by reticulo-endothelial cells of an avirulent rough strain of *Salmonella typhi* is much greater than that of the virulent smooth form of the same micro-organism[6]. Jenkin and Rowley[7] have reported that virulent strains of *Salmonella* are phagocytosed by reticulo-endothelial system slower than non-virulent ones.

The findings of Baron *et al.*[8] on the hybridization of *Salmonella* species by mating with *Escherichia coli* offer material of exceptional interest for investigations in bacterial virulence.

The results presented here show that hybrids of the virulent *S. typhimurium* have lost their virulence as a consequence of recombination with the avirulent parental strain of *E. coli*. This loss of virulence is associated with a striking change in the rate of bacterial phagocytosis by the reticulo-endothelial system of mice. The strains of *E. coli Hfr* 3300, *S. typhimurium mutator* and *S. typhimurium recombinant* were provided by Dr. Changeux[9].

These micro-organisms were grown for 24 h on nutrient agar containing 30 μc. of ^{32}P/ml. ($Na_2{}^{32}PO_4$). They were then collected in Tyrode solution (*p*H 7·4), washed twice and resuspended in Tyrode solution at a final concentration of $2{\cdot}5 \times 10^9$ bacteria/ml. In other experiments, formalin-killed micro-organisms trace-labelled with iodine-131 (ref. 2) were used instead of living bacteria tagged with phosphorus-32. The results were identical in both cases. The bacterial suspension was injected intravenously in heparinized mice in a dose of 5×10^8/20 g. The rate of bacterial phagocytosis by reticulo-endothelial cells was determined from the kinetics of clearance of the blood radioactivity according to the method previously described for ^{131}I-labelled bacteria[2] and for ^{32}P-labelled bacteria[10]. The velocity of phagocytosis was expressed by the constant K calculated according to the equation:

$$\frac{\log C - \log C'}{t' - t} = K$$

C and C' are the concentrations of radioactivity in the blood at times t and t' respectively, time being expressed in minutes.

The virulence of the different bacterial strains was determined in mice inoculated intraperitoneally with in-