

specificity of these proteins as antigens, but some experiments have been published in which the original biological specificity of the proteins also remains.²

It would seem possible, therefore, to alter chemically with the same methods such substances as display a specific biological activity without destroying this latter quality. This process appeared to us of special interest in the case of animal proteins, which play an important rôle in pathology because they carry the immune properties of the animal body, namely, the antibodies. The antibodies have a specific affinity towards their antigens (pathologic bacteria) but they usually do not destroy them or do not even lower their resistance enough to permit them to be phagocytized. We thought that in some instances a chemical alteration of the type mentioned above might increase the destructive effect of antibodies on pathogenic antigens and convert these antibodies into a quasi specific disinfectant or chemotherapeutic agent. We are attempting to obtain such an effect by introducing groups to change the physical properties of the immunebody-carrier proteins, or groups which are known to possess disinfecting or chemotherapeutic activity, or known to be apt to increase the disinfecting power of organic disinfectants.

Experimental work along this line was started in these laboratories some months ago, and without knowledge of the somewhat similar experiments and results which were recently published by Bronfenbrenner.³ In view of the accordance of our findings (with respect to the fact that chemical alteration, if carefully conducted, does not destroy immune properties) with his (presumably using different agents) a preliminary report upon some phases of our work would seem to be in order.⁴

In certain of our experiments para-aminophenylarsonic acid (atoxyl) was used for diazo-coupling because of its activity as a chemotherapeutic agent in certain protozoan diseases. The antibody protein was a Type I and II pneumococcus antibody, for this can be prepared in a comparatively highly purified state and its strength can be measured more easily than that of any immune serum produced against a protozoan parasite. The diazotization was carried out in the usual way. However, the pH was not allowed to change during the whole process of coupling more than from 5.0 to 7.5, approximately. A product

resulted which was almost insoluble around its isoelectric point at pH 6, and soluble to a dark brown solution at neutral or alkaline reaction, soluble with a light yellow color on the acid side of the isoelectric point. This "antibody-dye" could easily be reprecipitated by dialysis and adjustment of pH. On carrying out the process in the same way a second time, identical products were apparently obtained, the $\text{As}_2\text{O}_3/\text{N}$ ratio being in one case 0.028, in the other 0.027. If the products were taken up in the same volume of physiological saline as the original, agglutination was observed up to the same dilution as with the original antibody preparation (1/320). A very marked prezone was found, which was not present in the original preparation. Mice infected with 100,000 lethal doses of virulent pneumococci could be protected fully, *i.e.*, cured with 0.2 cc of the preparation containing respectively 6.6 mg N and 0.18 mg As_2O_3 , and 7.5 mg N and 0.2 mg As_2O_3 per cc, when injected intravenously, simultaneously with the infection, or 4 hours after, or 20 hours after the infection. Normal horse serum-globulin coupled with atoxyl had almost no effect. It is also interesting to note that 0.5 cc of the original antibody solution killed mice almost instantly when injected intravenously, whereas the same amount of the coupled product had no effect.

Finally, we also note that the introduction of the easily detectable arsenic into the antibody-carrier protein is helpful for quantitative study of the degree of purification of antibodies, as well as quantitative study of the antibody reaction. Further experiments are in progress.

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BOOKS RECEIVED

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² A. Klopstock and C. E. Selter, *Zeitschrift für Immunität*, 55: 118, 1928; M. Heidelberger and F. E. Kendall, *Proc. Soc. Exp. Biol. Med.*, 26: 482, 1929.

³ Bronfenbrenner, *Proc. Soc. Exp. Biol. Med.*, 28: 734, 1930.

⁴ Bronfenbrenner's object in starting his experiments was different from the above. He wanted to deprive immune sera of their original biological specificity in order to avoid anaphylactic shock at repeated injections of sera derived from the same species.