however, no cross reaction with the specific blood group A substance occurs, even in the cold. This is further evidence of the greater specificity of the antibodies produced by the rabbit, as compared with those formed by the horse, and would seem to offer an explanation for the observation made by Finland and Curnen that Type XIV antipneumococcus horse sera agglutinate human erythrocytes in high titre, whereas Type XIV antipneumococcus rabbit sera do not.

The relationship of the pneumococcus Type XIV specific polysaccharide to the blood group specific substances is further substantiated by the fact that absorption of Type XIV antipneumococcal horse serum with homologous polysaccharide removes the hemagglutinins for all blood groups.

Further studies on the chemical nature of the Type XIV pneumococcus polysaccharide and its chemo-immunological relationship to the blood group substances are in progress.

In conclusion the authors wish to express their thanks to Dr. Karl Landsteiner for his generous counsel.

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ULTRA-VIOLET ABSORPTION SPECTRUM OF CATALASE

THE absorption spectrum of catalase obtained from horse liver shows maxima at 4090, 5050, 5400 and 6220 Å.[1] The preparation in the air-driven quantity-ultracentrifuge[2] of solutions of catalase which appear to be homogeneous both upon ultracentrifugal[2] and electrophoretic[3] analysis has made possible the measurement of the absorption spectrum of the enzyme in the ultra-violet region.

The curve shown in Fig. 1 was obtained with a small Hilger quartz spectrograph and a Spekker spectrophotometer. The light source was a tungsten steel spark. The curve shows maxima at about 2,750 and 4,050 Å. The peak at 2,750 Å is due to the protein carrier of the enzyme, while the maximum at 4,050 Å is caused primarily by the hemin residue. A maximum in the ultra-violet region has previously been found in less highly purified catalase preparations.[4]

The values of the extinction coefficients at the two maxima, as calculated by the formula $\varepsilon_{c=1 \text{ mM Fe per liter}} = \frac{D}{d.c}$,[5] where D represents the observed optical density (see Fig. 1), d the thickness of the absorption cell (1 cm) and c the concentration of the enzyme in terms of mM Fe per liter, are $\varepsilon_{2,750\text{Å}} = 185$ and $\varepsilon_{4,050\text{Å}} = 80.5$.[6]

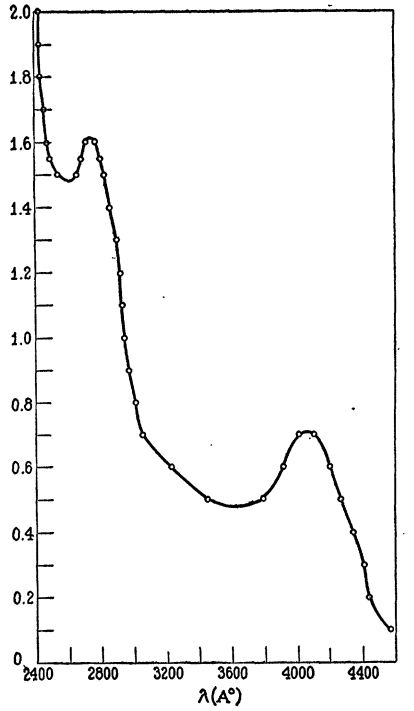

FIG. 1. Ultra-violet absorption curve of horse liver catalase. Solvent, 4.10-3 M. Phosphate, pH 8. Concentration of enzyme, 0.6 mg. per cc; of porphyrin-bound iron, 8.7 · 10-6 M. Abscissa, wavelength in Ångström units; ordinate, optical density $D = \log I_o/I$).

In contrast to other hemin-containing proteins, such as hemoglobin, chlorocruorin and the respiratory ferment, the value of the extinction coefficient at 2,750 Å, which is due to the protein component, is greater than that at 4,050 Å. This reversal in magnitude is probably due to the fact that in hemoglobin the mass ratio of hemin to protein is 2,600/68,000, while in catalase it is 2,600/250,000,[2] thus diminishing the quantitative contribution to the light absorption made by the four porphyrin groups present in these molecules. The underlying assumption is that the total content in aromatic amino acids in these conjugated proteins is of the same order of magnitude.

The visual examination of photographic plates obtained with the aid of a hydrogen discharge tube and a small Hilger quartz spectrograph reveals a band structure[7] typical for a globulin. There appear to be present bands due to tryptophane, tyrosine and phenyl-

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[2] K. G. Stern and R. W. G. Wyckoff, SCIENCE, 87: 18, 1938, *Jour. Biol. Chem.*, 124: 573, 1938.
[3] Unpublished.
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[5] D. L. Drabkin and J. H. Austin, *Jour. Biol. Chem.*, 112: 51, 1935–36.
[6] This value is lower than that previously reported on the basis of photo-electric spectrophotometric records obtained from other enzyme preparations (see note 1).
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alanine. The verification of this observation by chemical analysis will have to await the preparation of sufficient amounts of homogeneous catalase.

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THE ANTIGENIC STABILITY OF WESTERN EQUINE ENCEPHALOMYELITIS VIRUS

IT has recently[1] been shown that an especially potent vaccine against equine encephalomyelitis can be prepared from formalinized chicken embryos. The solid immunity that can be conveyed both to guinea pigs and horses[2] with such a vaccine provides a simple way of studying many properties of the virus responsible for this disease.

It is possible to determine in this fashion whether the virus remains the same from year to year and whether it is greatly altered by passage through different animals' hosts. Such information is essential whenever we seek to protect by vaccination, either with live or killed virus vaccines. It is especially important in the case of encephalomyelitis, because two varieties are already known to exist in the United States. One is active east, the other west of the Appalachian mountains. By all tests these two strains are as unrelated antigenically as two different diseases; an animal immunized to one has not lost any of its susceptibility to the other. We have been studying the homogeneity and stability of the western strain virus by testing the immunity of vaccinated animals to virus derived from as widely different sources as possible.

The guinea pigs for these experiments have been vaccinated with two 0.5 cc doses of chick embryo vaccine injected subcutaneously at an interval of a week. Two weeks after the second injection their immunity has been tested by the intracerebral inoculation of 100 to 1,000 lethal doses of virus. Previous experiment[1] has shown that such animals are completely protected against massive intracerebral inoculation with the virus from which the vaccine was made. In our tests two vaccines have been employed. The viruses for both were derived from the brains of horses dying during the epidemic of 1933.[3] One has been passed uninterruptedly through guinea pigs, the other has been through both guinea pigs and horses. No differences in behavior could be seen between the two vaccines. Guinea pigs vaccinated with them have been inoculated with the following strains of virus: (1) Guinea pig passage virus from 1933 brains; (2) the same virus after 60 egg embryo passages; (3) virus[4] from a 1933 brain after five years of uninterrupted mouse passage, and (4) 1937 field virus after several passages through guinea pigs. One of these 1937 viruses[5] was from Iowa, the other from Texas. The vaccinated animals have been completely protected against each of these viruses. It is thus apparent that the virus of western encephalomyelitis has not been greatly altered in its antigenic structure by transfer to guinea pigs, mice and chicken embryos. The fact that a vaccine gave complete protection against viruses from localities as widely separated as Iowa and Texas proves that this disease is substantially the same in different parts of the country in which it occurs; since vaccine made with 1933 virus protects against 1937 viruses, we may be confident that vaccines made with previous years' viruses will be effective against the disease that is now epidemic in several mid-western States and Canada.

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[3] We are indebted to Dr. B. M. Lyon for these viruses.

[4] We are indebted to Dr. P. K. Olitzky, of the Rockefeller Institute, for this strain of virus.

[5] These virus brains have been obtained from the Bureau of Animal Industry, U. S. Department of Agriculture, through the kindness of Dr. L. T. Giltner and Dr. M. S. Shahan.

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