SPECIAL ARTICLES

VENEZUELAN EQUINE ENCEPHALOMYELITIS

As is well known, severe epidemics of equine encephalomyelitis have occurred this year in Canada and the United States. A similar disease has now broken out in northern South America. Through the courtesy of the Ministry of Agriculture of the Venezuelan government and of Dr. Gunnar Tryde, of Caracas, we have received portions of brains of animals dying of this disease.

Virus has been recovered from these brains and preliminary observations have been made on the disease it produces in guinea pigs, mice and chicken embryos. The symptoms resemble those of Eastern encephalomyelitis. Nevertheless, it is clear that this virus is significantly different from either of the known American strains. Guinea pigs receiving it intracerebrally show first signs of paralysis after about 48 hours; they died in from three to five days. In young mice the disease follows a similar course. Guinea pigs can be infected by subcutaneous inoculation. Virus dropped onto the chorio-allantoic membrane of a developing chick embryo rapidly infects the entire embryo, which dies within a day. Brains of guinea pigs moribund with the Venezuela disease are especially rich in virus. Four that we have titrated have all been infectious for mice in a dilution of 10^{-7}. This indicates a virus concentration 10 to 100 times greater than that usually found in Eastern diseased brains. Several embryo-cultured virus preparations have had dilution end-points of 10^{-8}. They thus have contained about the same amount of virus as Western strain diseased embryos; Eastern strain embryos are ordinarily more infectious.

It has been shown[1] that formalinized virus vaccines will give solid immunity against corresponding forms of encephalomyelitis. We have made similar vaccines with Venezuela virus. All guinea pigs thus far vaccinated with them have successfully withstood the intracerebral injection of massive doses of Venezuelan virus.

Cross-protection tests have been made involving Venezuelan disease on the one hand and Western and Eastern encephalomyelitis on the other. Western strain vaccinated guinea pigs injected with Venezuelan virus have all succumbed with no evidence of protection. Eastern immune pigs diseased with Venezuelan virus have also all died, though in many instances a day or so later than unvaccinated controls. In a third experiment two groups of animals protected with the Venezuelan vaccine were given 10 to 100 m.l.d. of Eastern strain virus. They all died of encephalomyelitis, but those injected with the smaller dose outlived their controls.

These results demonstrate that our Venezuelan strain of encephalomyelitis is immunologically different from the familiar Eastern and Western forms of this disease. Though they are as yet insufficient to settle the question, they suggest that a remote relationship may exist between the Eastern and Venezuelan strains. No comparisons have been possible with the viruses of Borna's disease or of Russian encephalomyelitis. Work with the Venezuelan disease is being continued.

C. E. BECK
RALPH W. G. WYCKOFF

LEDERLE LABORATORIES,
PEARL RIVER, N. Y.

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THE PROTECTIVE EFFECT OF SULFANILAMIDE IN MICE AGAINST GONOCOCCAL "TOXIN"

SINCE sulfanilamide was first introduced for the treatment of gonococcal infections, we have been interested in the possibility that the favorable action of the drug might be concerned with the "toxin" of the gonococcus. Although the bactericidal effect of sulfanilamide *in vitro* has previously been demonstrated,[1,2] we have recently obtained an *in vitro* inactivation of gonococcal "toxin" with the compound.[3] Clinical data indicate that factors other than a direct effect of the drug on the gonococcus may contribute to its therapeutic efficacy. Thus, it is well known that certain patients recover with a minimal concentration of the drug in the tissues, while others fail to be cured when a high concentration is present. On the other hand, it has been frequently observed that the gonococcus is still present in infected areas after the symptoms have subsided subsequent to treatment with sulfanilamide. These facts point to an interaction of sulfanilamide with a gonococcal "toxin." Although some doubt has prevailed as to the existence of a substance which can rightly be termed a "toxin," our studies substantiate the findings of earlier investigators,[4,5] whose work indicates that a "toxin" is formed by the gonococcus.

By employing a technique similar to that described by Clark, Ferry and Steele[4] for the production of gonococcal "toxin," whole cultures, filtrates and supernates were prepared from several strains of the

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[4] J. DeChristmas, *Ann. de l'Inst., Pasteur*, II: 609, 1897.

[5] L. T. Clark, N. S. Ferry and M. H. Steele, *Jour. Immunology*, 21: 233, 1931.