TABLE 2
NEUTRALIZATION TESTS ON SERUM OF MAMMALS

	St. Louis Neg.	St. Louis Pos.	W. Equine Neg.	W. Equine Pos.
Domestic or in Captivity:				
Cat, House (*Felis domesticus*)	8	0	8	0
Cow (*Bos taurus*)	10	6	9	5
Dog (*Canis familiaris*)	8	3	6	2
Goat, Domestic (*Capra hircus*)	3	3	3	3
Horse (*Equus caballus*)	0	12	0[T]	9[T]
Pig (*Sus scrofa*)	3	1	3	1
Sheep, Domestic (*Ovis aries*)	8	2	10	1
*Miscellaneous	8	2	9	2
Total Domestic	48	29	48	23
Wild:				
Chipmunk, Great Basin (*Eutamias minimus*)	5	0	6	0
Ground Squirrel, Townsend (*Citellus townsendii*)	11	0	12	0
Mouse, Field (*Microtus montanus*)	8	0	8	1
Mouse, White-footed (*Peromyscus maniculatus*)	2	0	4	1
Pocket Gopher (*Thomomys talpoides*)	5	0	4	0
Rabbit, Cottontail (*Sylvilagus nuttalli*)	7	1	9	0
Rabbit, Jack (*Lepus californicus*)	5	1	10	0
Rat, Brown (*Rattus norvegicus*)	4	3	10	0
Rat, Black (*Rattus rattus*)	4	1	5	0
Weasel (*Mustela frenata*)	4	0	3	2
*Miscellaneous	4	0	3	0
Total Wild	59	6	74	4
Total	107	35	122	27

* Only one or two examined of each species.
[T] Not vaccinated for Western equine encephalomyelitis.

against 15.0 per cent. of 87 wild; and for the equine virus the respective percentages are 48.7 per cent. of 74 and 20.0 per cent. of 80. Of 77 domestic mammals tested, 37.7 per cent. protected against the St. Louis virus in contrast to 9.2 per cent. of 65 wild, and for the equine virus 32.4 per cent. of 71 domestic as against 5.1 per cent. of 78 wild. Caution is suggested in interpretation of these differences between the domestic and wild animal groups until both the areas of sampling and the species sampling can be more carefully analyzed. However, both the domestic and wild species were collected principally in areas where cases of encephalitis had occurred in 1939, 1940 or 1941. The only species of which an overly large sample was taken, which might exaggerate the above difference, is the Brewer blackbird.

If the apparent significance of these findings is confirmed, it will indicate a much more wide-spread potential reservoir for both viruses than has generally been suspected, especially for the St. Louis virus. It would appear that barnyards and fowl runs, found in large numbers in small towns, rural and suburban areas, are the principal foci of infection for encephalitis of either the Western equine or the St. Louis type. The distribution of human and obviously of horse cases has conformed with this pattern.[4,10,3] Final results, together with other aspects of the survey and with more adequate discussion of the potentialities of the findings, will be published following completion of the survey.

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A SYNDROME IN MACACUS RHESUS AFTER INOCULATION OF STOOL FROM CARRIERS OF POLIOMYELITIS VIRUS[1,2]

DURING a study of stool specimens from contacts and patients in an outbreak of infantile paralysis in a rural community, certain monkeys (*Macacus rhesus*) that failed to develop anterior poliomyelitis nevertheless showed signs of disease. The clinical symptoms ordinarily appeared from the tenth to the fifteenth day after inoculation. They consisted of some change in behavior, that is, lethargy or excitement, diarrhea and loss of appetite, associated with a slight febrile temperature lasting from twenty-four to forty-eight hours. After it became apparent that such mild illness followed stool inoculation and that the incubation period compared closely with that in monkeys developing poliomyelitis, all animals were killed and autopsied after the twenty-first day following inoculation.

Tissue was taken from the following regions of the

[10] *Public Health Bulletin*, No. 214, 1935.
[1] A preliminary report.
[2] Aided by a grant from the National Foundation for Infantile Paralysis, Inc.

central nervous system: the intervertebral ganglia; the sympathetic chains; the ganglia of both vagus nerves external to the skull; the Gasserian ganglia; the olfactory bulbs; the sacral, lumbar, upper and lower dorsal, and cervical regions of the spinal cord; from the medulla; the mesencephalon; the diencephalon, and from each of the major lobes of the cerebral cortex of both hemispheres of the brain, as well as from the cerebellum, including the roof nuclei. These tissues were fixed immediately in 70-per-cent. alcohol and the sections eventually stained with thionin.

It was observed that lesions resembling those ordinarily found in the intervertebral ganglia in cases of typical anterior poliomyelitis were also present in the intervertebral ganglia of most of these monkeys. Moreover, similar lesions were almost invariably present in the large-celled portion of one or both vagus ganglia and, rarely, in one or both Gasserian ganglia. The lesions varied from extremely mild, doubtful ones to florid pathologic changes. They consisted of focal interstitial ganglionitis, with focal destruction of neurons and neuronophagia. Some degree of perivascular "cuffing" was usually present. The exudative cells were predominantly mononuclear types. These lesions followed intraperitoneal inoculation of treated stool from patients with clinical anterior poliomyelitis or from contacts. If, in addition to the intraperitoneal route, stool was exhibited to monkeys intranasally, then lesions of the olfactory bulbs sometimes occurred. In the olfactory bulbs, the lesions consisted of perivascular "collaring" with foci of cell necrosis and exudative accumulations principally in the mitral-cell layer. Degeneration of mitral cells had occurred, and although neuronophagia was not always easy to identify in the manner that one does in anterior-horn cells, evidence of it appeared to be present. No changes were noted in sections that were studied from the remainder of the central nervous system of these animals.

In the seven instances in which this syndrome was observed, other monkeys were subsequently inoculated with larger doses of the original stool specimens. In five of the seven, a monkey eventually contracted classical anterior poliomyelitis. Sometimes as many as four attempts in as many monkeys, with repeated occurrence of the mild disease described, were necessary to achieve, in one monkey, the accepted endpoint, that is, fever, flaccid paralysis and anterior-horn cell necrosis with neuronophagia and perivascular "cuffing." In one case, stool inoculation, intranasally and intraperitoneally, was made in two different monkeys. Although neither developed typical poliomyelitis, both showed lesions in the vagus and spinal ganglia and in the olfactory bulbs. The patient from whom this specimen of feces was obtained had clinical poliomyelitis. In the seventh instance, no illness and no lesions were obtained after the inoculation was repeated.

It is common knowledge that a characteristic pathologic picture may be found in intervertebral ganglia and olfactory bulbs in experimental and human anterior poliomyelitis. That similar changes may be present in the ganglia of the vagus nerves is not usually recognized. It may be opportune at this point to call attention to a publication of Goodpasture in 1925,[3] in which, after studying tissue obtained at autopsy from a patient with "polio-encephalomyelitis," he makes the following statement: "A case of polioencephalomyelitis in a boy is described in which medullary lesions were found which appear to be directly related to the central distribution of the ninth and tenth cranial nerves. It is suggested that the virus of poliomyelitis in human infections may enter the brain through peripheral nerves."

Further work is in progress to determine whether or not the syndrome described is truly atypical anterior poliomyelitis, and what possible significance lesions of the vagal ganglia may have. However, these observations demonstrate the necessity of killing and examining all inoculated monkeys.

SUMMARY

A mild clinical syndrome in *Macacus rhesus,* accompanied by pathologic changes in the sensory portions of the vagus ganglia, intervertebral ganglia, and sometimes in the Gasserian ganglia is described. This syndrome occurred following intraperitoneal inoculation of fecal material obtained from contacts and from patients with infantile paralysis in an epidemic in a rural community. When intranasal as well as intraperitoneal inoculation was practiced, the olfactory bulbs were sometimes involved.

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RESYNTHESIS OF BIOTIN FROM A DEGRADATION PRODUCT[1]

IN a forthcoming paper[2] we are presenting data which indicate that biotin ($C_{10}H_{16}O_3N_2S$) is a cyclic urea derivative. The basis of this conclusion was the formation of a diaminocarboxylic acid ($C_9H_{18}O_2N_2S$) from biotin by the action of $Ba(OH)_2$ at 140°. The loss of one carbon atom and one oxygen atom, the

[3] E. W. Goodpasture, *Amer. Jour. Path.*, 1: 29–46, 1925.

[1] We wish to thank Mr. W. O. Frohring, of the SMA Corporation, for supplies of biotin concentrates used by us in the preparation of the crystalline biotin and for a research grant which has aided us in this work.

[2] K. Hofmann, D. B. Melville and V. du Vigneaud, *Jour. Biol. Chem.*, 141: 207, 1941.