Friday afternoon was devoted to division meetings, at which about eighty papers were read and discussed. Also the new Psychological Laboratory of DePauw University was dedicated with an address by Archie G. Bills, of the University of Cincinnati.

The annual dinner was held on Friday evening with one hundred fifty in attendance after which the president's address was given by Paul Weatherwax, Indiana University, on "The Indian as a Corn Breeder." The following officers were then chosen for the coming year: *President,* M. G. Mellon, Purdue University; *Vice-president,* Theodore Just, Notre Dame University; *Secretary,* Winona H. Welch, DePauw University; *Treasurer,* William P. Morgan, Indiana Central College; *Editor of the Proceedings,* P. D. Edwards, Ball State Teachers College; *Press Secretary,* C. M. Palmer, Butler University; *Member of Research Grant Committee,* A. L. Foley, Indiana University; *Division Chairmen:* Archeology, C. F. Voegelin, Indiana University; Bacteriology, L. S. McClung, Indiana University; Botany, Ray C. Friesner, Butler University; Chemistry, K. N. Campbell, Notre Dame University; Geology and Geography, G. T. Wickwire, Hanover College; Mathematics, Will E. Edington, DePauw University; Physics, O. H. Smith, DePauw University; Zoology, T. M. Sonneborn, Indiana University. J. L. Riebsomer, DePauw University, was elected a fellow of the academy.

Saturday was devoted to meetings of the Indiana Society of Taxonomists under the chairmanship of M. S. Markle, Earlham College, and the Indiana Society of Entomologists, with George E. Gould, Purdue University, as chairman.

The Junior Academy of Science, composed of forty high-school science clubs, also held its meetings and exhibits on Saturday under the sponsorship of Howard E. Enders, Purdue University. About three hundred high-school students and teachers were in attendance and a number of papers were presented on their program. A special feature was a lecture and demonstration of liquid air by F. J. Allen, Purdue University. The following officers were chosen for 1942: *President,* James Sarasien, Elmhurst High School, Fort Wayne; *Vice-president,* Jack Moseley, Greencastle High School; *Secretary,* Selma Heaton, Mishawaka High School. Honorary memberships in the American Association for the Advancement of Science were awarded to Don Courtney, Sullivan High School, and Jean Ross, Hammond High School.

The next annual meeting of the academy and the junior academy will be held at Notre Dame University as a part of the Centennial Celebration of that university.

WILL E. EDINGTON,
Press Secretary

DEPAUW UNIVERSITY

SPECIAL ARTICLES

EXPERIMENTAL AIR-BORNE INFECTION WITH POLIOMYELITIS VIRUS[1]

THE work of W. F. Wells and his associates[2] has shown the importance of transmission of both bacterial and virus infections by dried droplet nuclei, such as are formed by evaporation in the air of moist droplets expelled from the nose and mouth. These minute nuclei remain suspended in air for considerable periods of time, during which they constitute a potential source of infection by inhalation. As far as we are aware, the possibility of poliomyelitis being transmitted in this manner has never been put to experimental test, probably because the apparatus and technique developed by Wells have not been widely available. During the past year we have constructed an apparatus based on that of Wells[3] but modified in some respects, with which we have made a number of experiments and obtained some positive results. These, we believe, justify a preliminary report.

Apparatus. The infecting chamber consists of a Monel metal box 18″ × 18″ × 24″ with four port holes with rubber diaphragms through which the heads of the monkeys are inserted. The atomizing apparatus used in the first two series (32 monkeys) was found to give a poor delivery of material into the infecting chamber and was replaced by a better one designed for us by Mr. F. H. Osborn of this city. Compressed air mixed with 5–10 per cent. CO_2 (to increase the depth of respiration) was passed through a flow meter into the atomizer at an average rate of about 6.6 liters per minute, run continuously throughout each experiment. Test runs with suspensions of Chr. prodigiosum showed an even distribution of colonies on plates placed in the position later occupied by the animals' heads—evidence of satisfactory formation and dispersion of droplet nuclei and absence of large, moist droplets.

Animals used. A total of 48 rhesus monkeys (*M. mulatta*) and of 13 cynomolgus monkeys (*M. irus*) have been exposed. Of these, 31 rhesus and 6 cyno-

[1] From the Department of Pediatrics, Stanford University School of Medicine, San Francisco, California. Sponsored by the National Foundation for Infantile Paralysis, Inc.

[2] (a) W. F. Wells, *Am. Jour. Hyg.,* 20: 611, 1934. (b) W. F. Wells and H. W. Brown, *Am. Jour. Hyg.,* 24: 407, 1936. (c) W. F. Wells and M. W. Wells, *Jour. Am. Med. Assn.,* 107: 1698 and 1805, 1936.

[3] W. F. Wells, SCIENCE, 91: 172, 1940.

molgus were subjected to preliminary blockade of the olfactory mucosa with 1 per cent. zinc sulfate solution; and 16 rhesus and 7 cynomolgus were not so treated.

Virus used. The first tests were made with the old MV strain (mixed with the Armstrong strain), kindly supplied by Dr. E. W. Schultz. All other tests in rhesus were made with the O–H strain in the second and third passages (kindly supplied by Dr. James D. Trask). The tests with cynomolgus were made with a recently isolated strain (designated Sab) in the sixth and eighth passages, kindly supplied by Dr. Albert F. Sabin. Supernatant fluid from 20 per cent. suspension of cord was used in amounts varying from 25 to 85 cc for each series of monkeys; the maximum amount available to each monkey based on an assumed even dispersion of nuclei, ranged from 1.6 to 12.1 cc. The actual amount inhaled can not at present be estimated, but was undoubtedly much less than this, since much of the virus in the air must have been evacuated through the exhaust outlet.

The animals, under nembutal anesthesia, were exposed in groups, usually of four, for individual periods of 1–2 hours; repeated in some instances on two or three successive days.

The experiments and the results obtained are summarized in Table 1.

TABLE 1

Series	Virus	Total number	Olfactory blockade				No olfactory blockade				Exposure			
			Number	Polio.	No Polio.	Fever	Number	Polio.	No Polio.	Fever	Number	Total hours	Total virus cc	Virus per monkey cc
Rh I	MVA	16	8	0	8	0	8	0	7	1	1	1	25	1.6
Rh II	O–H[2]	16	8	0	8	0	8	0	5	3	1	1	37	2.3
Rh III	O–H[3]	8	8	1	4	3	..	..	..	..	3	3	75	9.4
Rh IV	O–H[3]	8*	8	1	5	1	..	..	..	..	2	3	75	9.4
Total rhesus		48	32	2	25	4	16	0	12	4				
Cyn I	Sab[3]	7	..	..	..	..	7	6	1	0	2	2	85	12.1
Cyn II	Sab[3]	6	6	2	4	0	..	..	..	..	1	2	40	6.7
Total cynomolgus ..		13	6	2	4	0	7	6	1	0				

* One monkey killed immediately after exposure.

In all, up to the present time, 10 monkeys of the 60 tested have developed typical poliomyelitis, proved by the presence of typical lesions, by subinoculation, or by both. Of these, four had had olfactory blockade and six had not. Of the four with olfactory blockade, two were rhesus and two were cynomolgus. In the positive rhesus experiments, weakness first appeared in the legs, on the seventeenth and eighth days after exposure, respectively, and no bulbar disturbances were observed. The incubation periods in both cynomolgus monkeys were 11 and 14 days to the appearance of paralytic signs. One of these had right facial paralysis and some respiratory difficulty. The other had severe respiratory difficulty, probably of the central type, but no other paralyses. Of seven cynomolgus monkeys in which the olfactory mucosa was not treated with zinc sulphate, six developed poliomyelitis proved by typical histological lesions. The seventh, which escaped, had a severe diarrhea for four weeks after exposure. A fairly uniform symptomatic pattern occurred in all the positive cases of the last series: fever at 4 to 7 days after exposure, followed by tremors and varying amounts of weakness or paralysis, the latter in all cases being in the extremities without apparent involvement of the cranial nerves. Examination of serial sections of the olfactory bulbs showed lesions in all; but in at least two of the cases, these were superficial and slight and no destruction of the mitral cells was found. Such lesions resemble those described by Howe and Bodian[4] in some of their human cases, in which the writers felt that the lesions were secondary rather than primary and probably not indicative of entry through the olfactory portal.

In 4 rhesus monkeys with, and in 4 rhesus without olfactory blockade, transient rises of temperature occurred from 6 to 17 days after exposure, without other evidences of infection except in a few of mild malaise, tremors or ruffling of the fur. One of these was killed; subinoculations from various parts of the central nervous system were all negative; histological examination showed no cellular infiltrations or neuronophagia; in the anterior horns at various levels of the cord, a rather marked satellitosis was noted and a number of ganglion cells showed vacuolization of the cytoplasm. The significance of these changes is not clear. The fever and occasional slight symptoms, however, suggest an abortive type of the disease which can not at present be considered as proved. More complete histological examinations are in progress.

We feel that the successful infection of two rhesus monkeys by inhalation has a special significance in relation to the portal of entry. The olfactory route in both was excluded by the previous blockade of the olfactory mucosa with zinc sulfate and by the

[4] H. A. Howe and D. Bodian, *Bull. Johns Hopkins Hosp.*, 69: 183, 1941.

absence of lesions in the olfactory bulbs as determined by serial sections. The oropharyngeal and gastrointestinal routes can also, in all probability, be considered as excluded on the basis of general experience with the rhesus monkey extending over more than thirty years during which it has been the standard test animal in poliomyelitis research, showing that animals of that species can only be infected non-traumatically by the olfactory route. Several extensive studies, of which those of Clark, Roberts and Preston[5] and of Flexner[6] may be cited, testify to the uniformly negative results of feeding virus to rhesus. It therefore seems highly probable that in the positive inhalation experiments with rhesus here reported, infection entered through the lower respiratory mucosa, at or below the epiglottis. The character of the symptoms in the two positive cynomolgus experiments with olfactory blockade, on the other hand, suggest entry through the oropharynx.

Further work along the same lines is in progress.

SUMMARY

Infection has been obtained in both rhesus and cynomolgus monkeys by inhalation of poliomyelitis virus in the form of droplet nuclei. The olfactory route was excluded in part of the animals successfully infected. The gastrointestinal route is believed to have been excluded in the rhesus monkeys. It seems most probable that the portals of entry were the lower respiratory mucosa in the case of the rhesus monkeys and the oropharyngeal mucosa in the case of the cynomolgus monkeys. Fever and occasional mild symptoms in 8 other rhesus monkeys suggest that an abortive form of poliomyelitis may have resulted from inhalation, but this can not at present be considered as proved. The experiments open up the possibility that human poliomyelitis may, at least sometimes, be an air-borne infection and that the lungs may be a portal of entry. Neither of these aspects of the disease has hitherto, so far as we are aware, been studied experimentally. The presence of virus in the human nasopharynx, which has been repeatedly demonstrated,[7, 8] provides an obvious source of air contamination by patients and carriers; and direct contact has been traced during epidemics in a considerable fraction of cases,[9] amounting to about one third in the report of Top and Vaughan.[10] The relative importance of transmission by air and by ingested material remains to be determined. It would seem probable, however, that both modes of infection must be taken into account.

HAROLD K. FABER
ROSALIE J. SILVERBERG

INHIBITORY EFFECTS OF SULFONAMIDES ON CULTURES OF ACTINOMYCES HOMINIS[1]

PRACTICALLY no information is available on the possible effects of *in vitro* sulfonamides on fungi, although there have been several reports of clinical benefits from the use of sulfonamide drugs in fungous infections.[2] This has resulted in an uncertainty as to whether the effect of the sulfonamides is on the fungi themselves or on secondary bacterial invaders. This paper reports the demonstration of direct inhibitory effects of sulfonamide drugs on cultures of *Actinomyces hominis*.

A stock strain (S) of *Actinomyces hominis* was cultured on Krainsky's glucose agar, and thioglycollate medium.[3] Another strain (M) was isolated on the thioglycollate medium from a draining sinus from the jaw of a patient and thereafter grown on veal agar medium. It differed from the former strain in that the organisms were shorter, seldom branched and often clubbed. The following drugs and concentrations were used: sulfanilamide, sulfathiazole and sulfadiazine,[3] in concentrations of 10, 50 and 100 mg per cent. incorporated in the media before autoclaving. The results obtained may be summarized according to aerobic and anaerobic conditions.

AEROBIC CONDITIONS

The S strain grown aerobically on Krainsky's medium was delayed in growth by concentrations of sulfanilamide of 10 and 50 mg per cent. but at the end of one month the growth in both concentrations equalled that of the controls (10 cultures). A concentration of 100 mg per cent., however, completely inhibited growth in some instances and allowed only slight growth in others by the end of one month (7 cultures). Sulfathiazole allowed moderate growth in 10 mg per cent. concentration, slight growth in 50 mg per cent. concentration, and no growth in 100 mg per cent. concentration (9 cultures). The observations in each case extended over one month. The results with

[5] P. F. Clark, D. J. Roberts and W. S. Preston, *Jour. Prev. Med.*, 6: 47, 1932.

[6] S. Flexner, *Jour. Exp. Med.*, 63: 157, 1936.

[7] S. D. Kramer, B. Hoskwith and L. H. Grossman, *Jour. Exp. Med.*, 69: 49, 1939.

[8] A. B. Sabin and R. Ward, *Jour. Exp. Med.*, 73: 771, 1941.

[9] Survey by International Committee for the Study of Infantile Paralysis Organized by Jeremiah Milbank. Williams and Wilkins Company, 1932, pp. 370 *et seq.*

[10] F. H. Top and H. F. Vaughan, Special Report of the Detroit Dept. of Health on Epidemiology of Poliomyelitis in Detroit in 1939.

[1] Aided, in part, by the Rockefeller Fluid Research Fund of the Stanford University School of Medicine.

[2] L. G. Dobson, E. F. Holman and W. C. Cutting, *Jour. Am. Med. Assoc.*, 116: 272, 1941.

[3] We wish to thank the Baltimore Biological Laboratory for supplies of thioglycollate medium, and the Lederle Laboratories for sulfadiazine.