

While the findings thus enumerated correspond to those reported in the rabbit with generalized tuberculosis, the more chronic type of the disease in the human, and the greater tendency to differences in localization and to the complication by other disease factors, makes the findings in the human more difficult to analysis, and hence requires a greater number of studies before conclusions may be drawn.

¹ Cunningham, R. S., Sabin, F. R., Sugiyama, S., and Kindwall, J. A., *Johns Hopkins Hosp. Bull.*, 1925, xxvii, 231-280.

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Oral Immunization of Rats Against Pneumococcus Types 2 and 3.

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We have previously reported on the successful immunization of white rats against pneumococcus Type 1 by oral administration of the living¹ or acid killed germ.² We wished to see whether our results with Type 1 could be duplicated with Types 2 and 3. The organisms were grown on glucose meat extract media but no HCl acid was added to kill the growth. After centrifuging, the germs were mixed with cracker meal and water, and growth from 50 ccm. was fed to each rat daily for 18 to 21 days. Tests were made by injecting intraperitoneally in volume of 0.20 ccm.

When the first feeding experiment with Type 2 was completed it was found that the treated animals tolerated large doses, but that the controls did the same. On reexamining virulence tests which had been made earlier, and, on extending these tests, it was learned that small untreated rats (up to ca. 85 to 95 grammes) were relatively as susceptible (with exceptions) to Type 2 as to Type 1, 10^{-7} and 10^{-6} cc. being fatal doses. However, rats, from this range of weight and above, showed a distinct tendency to rapidly increasing resistance. The age or weight at which this relative immunity begins to develop cannot be given more definitely, since individuals in the same group as well as whole groups of animals vary in this respect. The increased resistance is entirely out of proportion to the increased weight, and at its maximum is of the magnitude obtained to Type 1 when this germ is fed. It may be that the tolerance of the larger animals to Type 2 follows, with changing diet, the ap-

pearance among the intestinal flora of an organism closely related immunologically to this germ.

The results of the first feeding experiment, before it was known that the *larger* animals develop a natural immunity, show that feeding of Type 2 to such rats does not produce an additive effect. The Type 2 experiment was repeated using small rats in the hope that they could be treated and tested with controls before either group developed the normal resistance exhibited by larger animals. Table 1 gives the data. On January 7 no immunity had yet been established. On the 19th both controls and treated rats survived 10^{-7} and 10^{-6} cc., illustrating an increased resistance of untreated rats referred to above. On the 21st 10^{-6} cc. and 10^{-5} cc. killed controls, but not experimental rats. On the 22nd 2×10^{-3} , 10^{-4} and 10^{-5} cc. failed to kill treated rats whereas the controls succumbed. On the 25th the results are somewhat irregular, the natural immunity of the growing rat is again evident. On the 27th, 29th and 31st the increased resistance of the treated animal is demonstrated. The protective effect of feeding is apparent, although more difficult to show than for Type 1.

In Table 2 are given the results obtained with feeding of Type 3. Untreated large rats are not less susceptible to Type 3 than young rats, hence the demonstration of the immunity produced by feeding is not encumbered by the difficulty experienced with Type 2. The data show clearly the protection afforded by feeding Type 3.

We have not yet attempted feeding Group 4. However, preliminary virulence tests with a member of this group indicate that, just as for Type 2, the larger animals normally exhibit an immunity not possessed by young ones.

¹ Ross, Victor, *J. Immunol.*, 1926, xii, 237-249.

² Ross, Victor, *J. Lab. and Clin. Med.*, 1927, xii, 566-571.