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Possible Relation of the Inclusion Body of Trachoma to Pathogenesis.*

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Among other reasons for regarding the causative agent of trachoma as a virus is the cytoplasmic inclusion of the epithelial cells of the conjunctiva, and occasionally even of the cornea. Marshalling together evidence that is essentially morphological, a number of investigators beginning with Halberstaedter and Prowazek¹ have attempted to prove that the inclusion represents a mass or "colony" of infectious units. While conducting a broader study† on the inclusion of trachoma and clinically allied diseases in this laboratory, observations have been made by indirect approach which are sufficiently interesting on this phase of the subject to merit their publication.²

In testing out the infective capacity of conjunctival tissues from different patients with trachoma, monkeys in one group were inoculated with individual tissues, all of which were found to contain inclusions. In a second group, monkeys were inoculated in a similar manner with tissues lacking inclusions. In a third group, monkeys were inoculated with pooled tissues, some of which were inclusion-bearing and others inclusion-deficient. A study of the data pertinent to these experiments reveals several interesting and suggestive results. Thus, 70 monkeys (*Macacus rhesus*) were inoculated with individual tissues from 37 patients, all containing inclusions. Typical experimental trachoma occurred in 35, or 50%, of the animals tested. On the other hand, 158 monkeys were inoculated with separate tissues from 112 patients in whom inclusions were not demonstrated. Of these animals 35, or 22%, were specifically infected. The pooled tissues which were derived from 89 patients, 44 with and 45 without inclusions, were inoculated

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† A complete report will be published in the *American Journal of Ophthalmology*.

¹ Halberstaedter, L., and Prowazek, S., *Deut. med. Woch.*, 1907, **33**, 1285.

² For a review of the subject of inclusions in trachoma, see Julianelle, L. A., *The Etiology of Trachoma*, Chap. VI, The Commonwealth Fund Division of Publications, New York, 1938.

into 106 monkeys and of these, 41, or 38%, were infected. If the figures for monkeys infected in each group are plotted in the form of a curve, the result is practically a straight line, with inclusion-bearing tissues inducing most infections, the inclusion-lacking tissues inducing least or approximately half as many infections, and the tissues pooled as indicated occupying an almost mathematically half-way position between the two extremes.

Because the above result seemed to have a certain significance, the data were reassembled to determine whether the same conclusions were approachable on some other basis. Thus, of 18 separate experiments performed with tissues containing inclusions, 13, or 72%, were considered positive (*i. e.*, animals were specifically infected). Of 42 experiments conducted with material lacking inclusions, 14, or 33%, were positive, while of 24 experiments with mixed tissues, 14, or 58%, were positive. A curve representing the data runs a remarkably parallel course with that of the preceding observations.

This is not the place to discuss the possible significance of these results. However, the implications are obvious that the presence of inclusions accompanies greatest infective capacity of trachomatous tissues, and the absence of inclusions the least, while both tissues pooled approximately on a 1:1 basis approach a value half-way between both—a result certainly to be anticipated if the pooling were to represent a simple process of dilution. While it cannot be said that the evidence is conclusive, the data are of sufficient magnitude to suggest not an accidental outcome, but an actual relation of inclusion to infectivity, even to the extent that the inclusion represents an agglomeration of virus particles. This, however, is an opinion requiring further elaboration before it can be finally accepted.