THE RÔLE OF CANNIBALISM IN THE SPREAD OF FUNGOUS DISEASES OF GRASSHOPPERS

FUNGI of the genera Entomophthora and Empusa have long been known fatally to infest Melanoplus and other genera of grasshoppers. The body of the host remains clinging to the stalk where it died and transmits the disease by allowing the spores of the parasite to come in contact with healthy individuals, and, under favorable conditions, to germinate in their bodies.

Uninfected grasshoppers may themselves aid in the spread of the disease by virtue of a cannibalistic tendency present in certain species. As a demonstration of this tendency, freshly killed individuals of *Melanoplus bivittatus* "planted" along an open path served as bait to attract other individuals of the same species. The cannibals, after making an incision through the first abdominal tergites, would greedily devour the softer substances of the interior of the thorax, leaving the abdominal organs, however, untouched.

Observations made on individuals of this species that had died as a result of parasitism by Entomophthora and Empusa reveal that, although most of them had been untouched by their cospecific cannibals —probably because of the slower rate of dying, or because the bodily odors are more suppressed than in the artificially killed individuals—yet in a few cases the thorax had been hollowed out in precisely the same manner as in the "planted" specimens; and in one instance the act of cannibalism was itself witnessed by the writer. If mere contact, or the chance settling of an Entomophthora or Empusa spore on Melanoplus can bring about infection under the proper conditions (as it unquestionably can), this intimate contact ought all the more to be able to do so; and the moisture transmitted by the dead insect's body to that of the prospective host should make germination of the spores, and the consequent infection, practically certain.

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THE IRON-DEFICIENCY HYPOTHESIS IN PELLAGRA

IT is desired to correct some misstatements that appeared in an article written by Nellie Halliday.[1] Her reference to an article by Bliss[2] accepts more for his views than he had any intention of claiming, and is, therefore, misleading. Bliss, far from "claiming to show that pellagra . . . and black tongue . . . are both due to an iron deficiency," stated that "none of the considerations here outlined prove that pellagra is an iron deficiency disease," and asserted that he "has adopted the working hypothesis that pellagra is an iron deficiency disease, and has set out to *prove* or *disprove the thesis.*"

One point which Miss Halliday appears to have tried to make is in connection with the probable value of inorganic iron salts in curing the bilaterally symmetrical lesions occurring in rats on a low-iron diet. The failure of $FeCl_3$ in this respect is entirely to be expected, and follows from the results published by Bliss and Thomason,[3] which may have escaped the attention of Miss Halliday. Bliss and Thomason stated that "the addition of ferric citrate to the drinking water or the addition of solid ferric citrate to the food has no demonstrable effect upon the symptoms over a period of months." This finding has been confirmed and reported by others.[4]

Familiarity with living conditions in the south would probably have prevented Miss Halliday from calling attention to the iron content of commercial molasses as an apparent discrepancy in the iron hypothesis. Farmers of the South do not use the concentrated molasses preparations in question, but syrup prepared upon their own farms. Analyses of products subjected to commercial plant manipulations throw no light on the question in hand. Pellagra in the South is essentially a rural problem.

The efficacy of inorganic iron preparations given by mouth has little to do with a decision for or against the iron hypothesis. Work in this laboratory indicates more clearly than before that in pellagra we are dealing with an iron deficiency, but we are refraining from publishing fragmentary announcements until the evidence from many angles can be assembled and evaluated.

For the present we are only desirous that any attempts to refute the iron hypothesis, whether they come from our own laboratory or another, be based upon sound work of a comprehensive character.

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[1] N. Halliday, "The Lack of Correlation between Anemia and the Pellagra-like Symptoms in Rats," SCIENCE, 74: 312, 1931.

[2] S. Bliss, "Considerations Leading to the View that Pellagra is an Iron-Deficiency Disease," SCIENCE, 72: 577, 1930.

[3] S. Bliss and M. L. Thomason, "Iron Deficiency in the White Rat and the White Mouse," *Proc. Soc. Exp. Biol. and Med.*, 28: 636, 1931.

[4] B. Sure, M. C. Kik, M. E. Smith, "Hematopoietic Function in Avitaminosis. VI., Vitamin G Deficiency," *Proc. Soc. Exp. Biol. and Med.*, 28: 498, 1931.