

persons had cancer, and indeed some of the lung extracts which were not toxic were obtained from lungs containing primary carcinomas or tumor metastases.

The possibility that the toxic factor was absorbed from or through the denuded surfaces (ulcers) in the bowel was entertained. This idea was supported by the absence of toxicity in the livers from one case of quiescent non-specific ulcerative colitis, several cases of ulcerated carcinoma of the colon, and one case of superficial ulcerative colitis believed to have been the result of a gold-sodium-thiosulphate reaction, because in none of these colons were the ulcerations extensive. By this explana-

tion, however, the toxicity found in the infant lungs remains unexplained, if it be assumed that the toxic substances are identical. The same objection applies to regarding this toxic factor as the cause of the ulcers during its excretion through the mucosa.

Summary. A toxic factor was demonstrated in extracts of the liver and lungs in cases of nonspecific ulcerative colitis. A factor with similar toxic properties was present in lesser amounts or in milder form in an occasional control extract of liver, and in lungs of stillborn infants, but not in adult control lungs or infant livers.

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The Flea *Malareus telchinum* a Vector of *P. pestis*.

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In the course of an extensive survey instituted in an important section of the San Francisco Bay region in order to determine the source of the *P. pestis* demonstrated in pools of fleas, 1,356 rodents (Meadow mice—*Microtus californicus* 952, Deer mouse—*Peromyscus maniculatus* 347, Brown rat—*Rattus norvegicus* 40, and Harvest mice—*Reithrodontomys megalotis* 17) were trapped. Their ectoparasites were collected and adequate samples of fleas, both males and females, were cleared and identified. The species determinations and host data are listed in Table I.

It was found that *Malareus telchinum* was by far the most numerous of the fleas found on *Microtus californicus*, and that this flea outnumbered *Nosopsyllus fasciatus* about 2 to 1 on *Rattus norvegicus*. *Opisodasys nesiotus* was slightly more numerous than *Malareus telchinum* on *Peromyscus maniculatus*.

Since *Malareus telchinum* was the only species of flea found to parasitize all 3 species of rodents known to be spontaneous hosts for *P. pestis*, it was deemed imperative to undertake transmission experiments. Such experi-

ments were prompted by the observations of Eskey and Haas¹ who noted that 74 *Malareus telchinum* fed on a plague-infected guinea pig were apparently unable to transmit *P. pestis* to guinea pigs despite the fact the flea developed blockage.

Wheeler and Douglas² using white mice as sources for the infection reported the failure of *Malareus telchinum* to transmit *P. pestis* in mass feeding experiments on white mice. In the experiments herewith reported, *Peromyscus maniculatus*, *Microtus californicus*, and the white mouse were used as a source for the infection. The pools of the fleas so infected were permitted to feed on *Microtus californicus*.

Ten experiments have thus far been completed, and 4 positive transmissions have been obtained as follows:

(1) Seventy-five fleas were fed on a moribund plague-infected *Peromyscus maniculatus*. Six of the fleas were triturated in a mortar

¹ Eskey and Haas, *Pub. Health Bull.*, No. 254, 1940, 42.

² Wheeler and Douglas, *Proc. Soc. Exp. Biol. and Med.*, 1941, 47, 65.

TABLE I.

Host	Species of flea
<i>M. californicus</i>	<i>Catallagia vonblockeri</i> (Augustson, 1941) <i>Atyphloceras multidentatus</i> (C. Fox, 1909) <i>Hystrihopsylla gigas dippiei</i> (Rothschild, 1902) <i>Malareus telchinum</i> (Rothschild, 1905) <i>Peromyscopsylla brighti</i> (C. Fox, 1926)
<i>P. maniculatus</i>	<i>Atyphloceras multidentatus</i> <i>Hystrihopsylla gigas dippiei</i> <i>Opisodasys nesiotus</i> (Augustson, 1941) <i>Malareus telchinum</i> <i>Catallagia vonblockeri</i>
<i>R. norvegicus</i>	<i>Malareus telchinum</i> <i>Nosopsyllus fasciatus</i> (Bosc, 1801) <i>Orchopeas sexdentatus</i> (Fox, 1909)
<i>R. megalotis</i>	<i>Catallagia wymani</i> (Fox, 1909)

with salt solution, and inoculated into a guinea pig which died on the 8th day with lesions of plague. A *Microtus* was placed in the crock with the remaining 69 fleas. The animal died in 13 days with the anatomical findings and the cultural isolation of *P. pestis*.

(2) One hundred *Malareus telchinum* were placed on a *Microtus* with a terminal plague septicemia; the rodent died within a few hours. Three days later a *Microtus* placed in the crock with the fleas contracted plague and died on the 15th day after exposure.

(3) Seventy-five fleas were placed on a white laboratory mouse with many plague bacilli in the blood. Twenty-four hours after

the death of the mouse, a *Microtus* was exposed in the crock. Six days later it was found dead from plague.

(4) An identical experiment was conducted with 25 *Malareus*. The *Microtus* contracted plague and died on the 6th day after exposure. Experiments to explain the earlier failures and to evaluate the vector efficiency are in progress.

The valuable assistance of Mr. Robert Holden-ried in collecting the specimens and in making the specific determinations of the mammals, and of Dr. M. A. Stewart with the flea identifications is gratefully acknowledged.

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On the Specificity of Bacterium-Decarboxylase.

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In a recent study¹ on the histamine-formation in fish muscle it was found that a minor fraction of histamine-units produced post mortem by bacterial action remains within a protein complex and can be liberated only by acid hydrolysis or by fermentative proteolysis. It was assumed, therefore, that histidine groups need not be present in a free form in

order to be converted into histamine groups. This implies that some bacterial ferments are able to split such peptide-linkages which involve the carboxyl group of histidine groups without attacking simultaneously those linkages by which the histamine group is attached to the protein molecule. The possible formation of such compounds was discussed by Guggenheim² who assumed on a theoretical

¹ Geiger, E., Courtney, G., and Schnakenberg, *Arch. Biochem.*, in press.

² Guggenheim, M., *Biochem. Z.*, 1913, **51**, 369.