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Effects of In Vivo Prepared Toxic Products of B. Coli Upon the Guinea Pig.*

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In a previous communication,[1] one of us (Harris) reported the production of experimental typhoid fever in the guinea pig by employing certain toxic substances of *B. typhosus*. These toxic materials were obtained by producing primarily a peritonitis by the injection of suspensions of *B. typhosus*. The exudate present in the peritoneal cavity was filtered through a Berkfeld letter N filter and the filtrate used for injections.

The object of the present work was to determine if products of the colon bacillus obtained in a similar manner as employed with *B. typhosus*, would produce the same results as manifested in the typhoid experiments. Cultures of *B. coli communior* were injected into the peritoneal cavity of guinea pigs. Fifteen pigs were employed. The exudate from the resultant peritonitis was diluted and filtered through a letter N Berkfeld filter, and the filtrate injected subcutaneously into a series of guinea pigs. A slight febrile rise was noted following the injections and the leucocytic count was slightly lowered. The animals received several injections and eventually died after 4 to 6 weeks. The post mortem examination of these animals showed but little changes macroscopically excepting in the lungs and kidneys. In the lungs, areas of marked congestion were seen and in the kidneys intense redness and swelling were noted. The spleen and other lymphoid structures of the peritoneal cavity demonstrated but little change. The lymphatic glands were in certain instances slightly enlarged. The Peyers patches did not reveal the changes as previously described for the typhoid filtrate.

The microscopic study of the various organs showed chiefly intense congestion of the blood vessels and degenerative changes of the parenchymatous structures. These findings were noted especially in the kidneys and lungs. In the latter organ hemorrhagic extravasations were often present. The lymphoid structures in addition to the congestion showed occasionally a hyperplasia of both the connective tissue and lymphoid elements. The phagocytic cells prevalent in the animals injected with typhoid toxin, were not found in the guinea pigs given the filtrate of the colon material.

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[1] Harris, W. H., PROC. SOC. EXP. BIOL. AND MED., 1928, XXV, 5, 372.

In these experiments the marked leucopenia and febrile reaction observed for the typhoid filtrate did not occur, the reaction being comparatively slight. The lesions as a whole, both gross and microscopic were those of a toxemia but unlike the representative lesions produced by the typhoid toxin.

It would appear, therefore, that the toxic moiety obtained from *B. coli* wherein a peritonitis has been excited, does not demonstrate the effects analogous to human typhoid fever previously noted when the typhoid bacillus had been employed in a similar manner.

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Afebrile Post-Scarlatinal Nephritis.*

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In many cases of scarlet fever the symptoms of the infection have passed and recovery has apparently been established, when acute nephritic symptoms without fever appear about the third week. Of these, the signs of glomerular nephritis are the most prominent. It is generally thought that the new symptoms are not those of a secondary infection or late manifestations of scarlatina. In this connection Longcope[1] and his co-workers indicate that the late nephritis following scarlet fever is the result of existing foci of streptococci in the throat and other parts of the upper respiratory tract. They could not obtain evidence that the streptococci caused the glomerular nephritis by actual invasion of the kidney, as cultures of the blood and urine were negative. These authors suggest that toxin from streptococcal foci in the throat, sinuses, etc., is eliminated by the kidney, thus causing the acute glomerular nephritis.

Our experiments with 17 healthy young dogs support Longcope's contention and furthermore prove that the late acute glomerular nephritis (at least in the dog) is not the result of retained viable streptococci in the body but is due solely to the *in vivo* prepared streptococcal lysate (endotoxin) of Duval and Hibbard.[2] In the recovered experimental dogs which had been infected with living cultures of scarlatinal streptococci, a second or a third dose of

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[1] Longcope, W. T., *et al.*, *J. Clin. Invest.*, 1927, v, 7.

[2] Duval, C. W., and Hibbard, R. J., *J. Exp. Med.*, 1927, xlvi, 379.