genic.[12] Hence, it must be concluded that the influence of goitrogens on "the general adaptation syndrome" is an open issue.

Penicillin has lately been reported to afford protection against the toxic action of the gonococcal endotoxin.[13] It will be of interest to determine whether this protection is based on goitrogenesis.

Another question arising from the data presented in this paper concerns the role and value of fever in resistance within the body to the toxemia of infections caused by Gram-negative bacteria. From our data it would seem that the heightened metabolism of the febrile state would enhance the toxicity of any antigen released during the course of an infection; and that low temperature or lowered metabolism would have an opposite and helpful effect. On the other hand, it is supposed that fever promotes within limits both antibody formation and phagocytic activity.[14] Whether in therapy one of these actions should be supported at the expense of the other, cannot be said in the absence of adequate quantitative information.

Summary. (1) An extensive list of pharmacological agents is presented, whose protective effects against the *Salmonella* endotoxin is reported as negative. The endotoxin of *S. typhimurium* was chosen for such tests because it is considered to be typical of the endotoxins generally found in Gram-negative bacteria. (2) Thiouracil and *p*-aminobenzoic acid have a limited protective effect against the lethal action of the endotoxin. Thiouracil protects against endotoxin-induced tumor hemorrhage. It is concluded that these effects, as well as the similar effects of the sulfonamides, is via a common mechanism, *viz.*, interference with thyroid activity. (3) Thiouracil has some protective effect against 1,2-dichloroethane, but is ineffective against colchicine. *p*-Aminobenzoic acid is likewise ineffective against colchicine. (4) Mice maintained at high environmental temperatures are more vulnerable to the endotoxin than those kept at lower temperatures. (5) The application of Selye's concept of "the general adaptation syndrome" to the action of certain poisons is briefly discussed.

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15670 P

Growth Inhibition of Tubercle Bacilli by Fusarium, Sp.

CHARLES H. BOISSEVAIN.*

From the Laboratory, The Colorado Foundation for Research in Tuberculosis, Colorado College, Colorado Springs, Col.

Growth of tubercle bacilli on media-containing egg or similar complex substances is inhibited by contamination of the medium with either fungi or bacteria. This inhibition is apparently not specific but results from the production of protein split product, deprivation of oxygen, etc.

On simpler mediums (*e.g.* Long's) the picture is very different. While some fungi are unable to grow on those mediums, others grow freely. As a rule they do not interfere with the growth of the tubercle bacillus and may even favor it.

A search was made for fungi capable of producing antibiotic agents. The following technic was used.

An alundun capsule (20 × 2 cm, dense) was introduced into a 250 cc Erlenmeyer flask containing 100 cc of Long's medium.

* Died October, 1946.

The fluid inside the capsule (±5 cc) was inoculated with the fungus under investigation, while the outside fluid was inoculated with a recently isolated strain of virulent tubercle bacilli. While a rapid and easy exchange of metabolic product occurred through the alundun wall, the coarse fungus filaments did not as a rule penetrate it and invade the outer fluid. In the few cases in which this happened, the filtrate from the fungus growth was tested for the presence of growth inhibiting substances.

Of a large number of fungi, recently isolated from a variety of sources, only one, a Fusarium, sp., inhibited growth of the tubercle bacillus.

The inhibiting substance passes through fritted glass filters and Mandler filters but is partially inactivated by passage through Seitz filters. The crude filtrate is active up to a dilution of 1/20 and withstands heating to 100° for 15 minutes without loss of activity.

Experiments are now in progress to isolate the active principle and determine its activity *in vivo*.

C. D. Sherbakoff, Head Dept. of Plant Pathology, The University of Tennessee, Agricultural Experiment Station, has kindly identified the Fusarium as *Fusarium scirpi* Lamb. et Fautr. v. accuminatum (Ell. et Ev.) Wr. Wollenweber in his monograph on Die Fusarien, Z. f. Parasitenkunde, Berlin, 1931, places this Fusarium as No. 930 in a list of 1100.

15671 P

Urinary Excretion of Orally Administered Pteroylglutamic Acid.*

RUTH STEINKAMP, CARROLL F. SHUKERS, JOHN R. TOTTER, AND PAUL L. DAY.

From the Department of Physiological Chemistry, School of Medicine, University of Arkansas, Little Rock.

Introduction. Few studies on the urinary excretion of pteroylglutamic acid have been reported.[1-4] No published information concerning the excretion of this vitamin after single large doses is available. Consequently, we are reporting results of such studies on 9 normal subjects and 9 hospitalized patients representing assays on 54 24-hour urine samples.

Methods. Since the primary purpose of the study was to determine the recovery of the free pteroylglutamic acid, no resort was made to the use of enzymes to liberate any conjugated vitamin.

One or more preliminary 24-hour urine samples were obtained in all but 2 instances. The test dose of synthetic pteroylglutamic acid was given orally (5.0, 5.1, 10 or 16 mg) and samples of urine were collected at varying intervals for the 24-hour period immediately following. The samples were preserved under benzene and aliquots were suitably diluted for determining the PGA microbiologically[5] with *Streptococcus faecalis*

* Research paper No. 830, Journal Series, University of Arkansas. This work was supported in part by a grant-in-aid from the Nutrition Foundation, Inc., New York. We are indebted to the Lederle Laboratories, Inc., for supplies of synthetic pteroylglutamic acid ("Folvite").

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