infrared spectrum indicates a carboxyl group and possible amide carbonyl.

The antibiotic gives negative Tollens, 2,4-dinitrophenylhydrazine, and ninhydrin tests. In glacial acetic acid or carbon tetrachloride, bromine is rapidly absorbed with evolution of hydrogen bromide. The color of permanganate is quickly discharged by the antibiotic in aqueous solution. On heating a strongly alkaline solution in the presence of lead acetate a slight darkening results and ammonia is evolved. A white precipitate results with mercuric chloride, but no reaction with mercuric oxide.

Aqueous solutions of the antibiotic are quite stable over a wide *p*H range at room temperature. The crystalline antibiotic can be stored for long periods of time with no loss in potency.

This antibiotic exhibits a very low order of toxicity, but preliminary animal protection studies indicate that the new antibiotic is not active *in vivo*.

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RECEIVED MAY 16, 1952

ACIDIC BEHAVIOR EXHIBITED BY METHYL BORATE TOWARD AMINES[1]

Sir:

The question of the ability of alkyl borates to form addition compounds with amines under ordinary conditions has not as yet been settled. Such compounds have not been reported, and it has been suggested[2] that they do not form.

We have found, however, that the lowest boric ester (and presumably the least hindered), methyl borate, forms white solid addition compounds when treated with a number of amines, including dimethylamine, diethylamine, di-*n*-propylamine, di-*n*-butylamine, di-*n*-amylamine, triethylamine, tributylamine, ethylenediamine, piperidine, methylamine, and *t*-butylamine. In the case of the last four amines, the compounds are stable enough to be purified by sublimation *in vacuo*, weighed, and analyzed. No evidence of interaction has been obtained in the case of the weaker bases pyridine and quinoline.

These addition reactions are strongly catalyzed by the lower aliphatic alcohols, the degree of catalysis increasing with the acidity of the alcohol.

The addition compounds thus far characterized are listed in the accompanying table.

Compound	M.p., °C.	Analyses, % Calcd.	Found
$(CH_3O)_3B{:}NH_2CH_2CH_2NH_2$	81–82	B, 6.71	6.72
$(CH_3O)_3B{:}H{-}N$⟨⟩	75	B, 5.82	5.77
$(CH_3O)_3B{:}NH_2CH_3$	67	N, 10.37	10.44
$(CH_3O)_3B{:}NH_2C(CH_3)_3$	67–70	B, 6.01	6.22

Solid compounds do not seem to separate when the amines and ethyl borate are mixed under the same conditions. However, there is considerable heat evolved during mixing, indicating some chemical interaction between components. When amines are added to the higher esters, *n*-butyl- and *n*-amyl borates, there is no appreciable heat effect. It seems likely, therefore, that the stabilities of such amine-borate complexes are governed largely by steric factors.

We are at present unable to propose a reasonable mechanism explaining the catalysis of these addition reactions by alcohols. Studies on the heats of formation of these complexes are being carried out.

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SOME ANTIMETABOLITES OF SEROTONIN AND THEIR POSSIBLE APPLICATION TO THE TREATMENT OF HYPERTENSION

Sir:

The recent elucidation of the structure of serotonin, the vasoconstrictor of serum,[1-3] has provided an opportunity for the testing of a basic postulate in the chemotherapy of non-infectious diseases. This postulate is that if such diseases arise from excess of specific hormones or other metabolities, they may be susceptible to treatment by antimetabolities, which would thus nullify these extra amounts.[4] Experimental models to test this idea have been described for thyroxine, and for other metabolities.[5,6] The use of some of the antihistamines in medicine is an unconscious application of the same principle.[6] If serotonin, which is the naturally occurring vasoconstrictor in mammals were to be increased in an animal, either by excessive synthesis or decreased destruction, it would not be difficult to envision it as the cause of certain clinical hypertensions. We have therefore attempted to produce antimetabolities of serotonin, in the hope that they may be useful pharmacological agents.

Several new 5-aminoindoles with alkyl groups in positions 2 and 3 have been made by reduction of the corresponding 5-nitroindoles prepared by the Fischer synthesis.[7,8] The structural resemblance to serotonin, 3-aminoethyl-5-hydroxyindole, is clear. These were tested on ring-shaped segments of sheep carotid artery for ability to prevent the constriction which serotonin causes. A roughly quantitative test was developed to allow comparison of various analogs, and to permit study of the competitive nature of the antagonism.

The most active antimetabolite examined was 2-methyl-3-ethyl-5-aminoindole, m.p. 148–149° (calcd. C, 75.84; H, 8.10; N, 16.08; found, C, 75.77; H, 7.83; N, 16.33). A maximal contraction

(1) Based on research carried out under Signal Corps Contract DA 36-039 Sc-5492 between the Squier Signal Laboratory and the Polytechnic Institute of Brooklyn.

(2) N. V. Sidgwick, "Chemical Elements and Their Compounds," Oxford University Press, London, 1950, p. 403.

(1) M. Rapport, *J. Biol. Chem.*, **180**, 961 (1949).

(2) K. E. Hamlin and F. E. Fischer, THIS JOURNAL, **73**, 5007 (1951).

(3) M. E. Speeter, R. V. Heinzelman and D. I. Weisblat, *ibid.*, **73**, 5515 (1951).

(4) D. W. Woolley, *Science*, **100**, 579 (1944).

(5) D. W. Woolley, *J. Biol. Chem.*, **164**, 11 (1946).

(6) D. W. Woolley, "A Study of Antimetabolities," John Wiley & Sons, New York, N. Y., 1952.

(7) H. Bauer and E. Strauss, *Ber.*, **65**, 308 (1932).

(8) K. Schofield and R. S. Theobald, *J. Chem. Soc.*, 1505 (1950).