Barrenscheen and Lang (*1*) found a specific ATP-ase in guinea pig and rabbit liver with pH optima of 8.2 and 9, while Satoh (*7*) reported that ATP was dephosphorylated by the combined action of phosphomonoesterase and pyrophosphatase. DuBois and Potter (*2*) report an optimum of pH 9 for rat liver ATP-ase. Studies of two specimens of pooled rat serum have failed to reveal an acid optimum for ATP-ase activity. It is possible that such species differences as have been found for prostatic phosphatase (*5*) exist with regard to "acid" ATP-ase.

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The Mode of Action of 7-Methyl Folic Acid

Gustav J. Martin, Leo Tolman, and Jack Moss

Research Laboratories, National Drug Company, Philadelphia, Pennsylvania

The primary action of sulfonamides (*1*) has been found to be directed against the incorporation of p-aminobenzoic acid into pteroylglutamic acid. 7-Methyl folic acid has been reported (*2*) as an effective folic acid displacing agent. The 7-methyl folic acid used in these experiments is N-(4-(((2-amino-4-hydroxy-7 - methyl - 6 - pteridyl) - methyl) - amino) benzoyl) - 1(+) - glutamic acid. To determine the mode of action of folic acid displacers, attempts were made to counteract the growth-inhibiting action of sulfonamides and folic acid displacers by various agents. *Staphylococcus aureus* # 209 grown in a bouillon medium was used as the test organism. Table 1 gives the results obtained.

TABLE 1

Counteraction of 7-Methyl Folic Acid and Sulfathiazole

Compound	p-Aminobenzoic acid	Pteroylglutamic acid	Pteroic acid	Glutamic acid	p-Aminobenzoyl 1(+) glutamic	Sulfathiazole
7-Methyl folic acid...	+	+	+	−	−	+
Sulfathiazole.....	+	−	+	−	−	−

+ = counteracts
− = no effect

The concentrations per 10 ml. of chemicals used were: 7-methyl folic acid, 1–10 mg.; sulfathiazole, 0.05–1.0 mg.; p-aminobenzoic acid, 1–5 mg.; pteroylglutamic acid, 1–5 mg.; pteroic acid, 1–10 mg.; glutamic acid, 1–5 mg.; and p-aminobenzoyl-1(+)-glutamic acid, 1–5 mg.

When *Staph. aureus* is the test organism, it seems that the action of the sulfonamide is to prevent the incorporation of p-aminobenzoic acid into pteroic acid. Pteroylglutamic acid does not counteract the sulfonamide, which indicates that pteroic acid and not pteroylglutamic acid is involved in *Staphylococcus* metabolism. The fact that p-aminobenzoylglutamic acid does not counteract the sulfonamide would be anticipated if pteroic acid is the vital factor in growth. Pteroic acid is more effective than p-aminobenzoic acid in counteracting sulfathiazole.

With the same test organism, methyl folic acid is counteracted by pteroylglutamic acid, pteroic acid, and p-aminobenzoic acid but not by p-aminobenzoyl-1(+)-glutamic acid, indicating the synthesis of pteroic acid as the first step in the formation of pteroylglutamic acid. Pteroic acid, p-aminobenzoic acid, and pteroylglutamic acid are equally effective on a molar basis in counteracting methyl folic acid. It seems probable that methyl folic acid interferes with the synthesis of pteroylglutamic acid at the stage of pteroic acid formation and at the stage of union with glutamic acid. It has the further capacity to displace formed pteroylglutamic acid. Thus, methyl folic acid represents another displacement compound incorporating into one molecule the capacity to inhibit the synthesis of and displace a given metabolite. As with a sulfonamide, methyl folic acid interferes with the incorporation of p-aminobenzoic acid into pteroylglutamic acid. The counteraction of methyl folic acid by sulfathiazole indicates similarity of structure resulting in mutual interference superseding that of interference with the metabolism of pteroylglutamic acid.

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Effect of Age of Infection Upon the Oxidative Metabolism of *Trypanosoma lewisi*[1]

James W. Moulder[2]

Departments of Bacteriology and Parasitology and *Biochemistry, University of Chicago*

When the nonlethal protozoan parasite, *Trypanosoma lewisi*, is inoculated into its natural host, the rat, reproduction of the trypanosomes occurs only during the first few days of the infection. Thereafter, reproduction is inhibited, and the population consists entirely of adult, nonreproducing organisms (*2*). Taliaferro and his associates (*5*) have shown that cessation of parasite reproduction is caused by an antibody which specifically inhibits the reproduction of the trypanosomes, and in 1932 Taliaferro termed this antibody ablastin.

In an investigation of the mechanism of action of ablastin upon *T. lewisi*, the oxidative metabolism of trypanosomes taken from rats at different ages of infection has been compared. The study was limited to infections 2-10 days of age in which no number crisis had been brought about by the appearance of trypanolysins (*1*). Reproduction of the trypanosomes ceased on the fourth or fifth day of infection. Table 1

[1] Supported by grants from the Dr. Wallace C. and Clara A. Abbott Memorial Fund and the John Rockefeller McCormick Memorial Fund of the University of Chicago.

[2] Mr. and Mrs. Frank G. Logan Research Fellow.