purified enzymes from other sources, *e.g.* the adenylate kinase, nucleoside diphosphatase from liver[6,7]. The six to ten times higher activity of the GMP phosphorylase in comparison with the other polyase activities is noteworthy from the point of view of specificity and heterogeneity[8–10] of the polyase enzymes and also of the tumor polynucleotides[11].

Finally, it should be mentioned that ATPase activity with characteristics of the electron-transport chain is also present. Perhaps the viral enzyme complex represents a reminiscence of those parts in the host cell which have participated in the virus synthesis[12]. The view[13] "that viruses do not act only as cell and tissue destroyers but as parts of normal animal cells" conforms with this hypothesis.

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Interference with adenine and histidine metabolism of microorganisms by aminotriazole

3-Amino-1,2,4-triazole (aminotriazole), a heterocyclic nitrogen compound used extensively in agriculture as a weed killer, bears a close structural relationship to 4-aminoimidazole. 4-Aminoimidazole is recognized as an intermediate in purine degradation in *Clostridium cylindrosporum*[1] and 4-aminoimidazole ribonucleotide is an intermediate in purine biosynthesis[2]. From these considerations, it might be

anticipated that aminotriazole could interfere with reactions of purine metabolism and indeed RABINOWITZ AND PRICER have demonstrated[1] that aminotriazole blocks the enzymic conversion of 4-aminoimidazole to formiminoglycine in extracts of *Cl. cylindrosporum*. In the present investigation, it was found that aminotriazole (10 μmoles/4 ml) was inhibitory for growth of *Escherichia coli* and a yeast, *Torula cremoris*. Adenine partially overcame the inhibition in *E. coli* (Table I). In more detailed studies with *E. coli*, not reported here, adenosine, adenylic acid, and guanine were less effective than adenine in relieving the inhibition whereas hypoxanthine and xanthine were totally without effect. The specificity of adenine in counteracting the action of aminotriazole suggested that histidine also be included in this study in view of the demonstrated involvement of adenosine triphosphate in histidine biosynthesis in microorganisms[2]. The data of Table I further illustrate that in *E. coli* the addition of histidine alone had no effect in reversing aminotriazole inhibition but when tested in the presence of adenine, the inhibition was completely reversed even at the 100 μmole level of aminotriazole. With *T. cremoris*, adenine was incapable of counteracting the effect of aminotriazole, but the single addition of histidine completely nullified the toxicity of aminotriazole. One interpretation of the data is that aminotriazole blocks the biosynthesis of histidine or possibly its utilization in both organisms but that in *E. coli* it may also block a step in the biogenesis of adenine. These findings may have a bearing on the mechanism of action of amino-

TABLE I

EFFECT OF ADENINE AND HISTIDINE ON TOXICITY OF AMINOTRIAZOLE FOR GROWTH OF MICROORGANISMS

(Additions to a final volume of 4 ml medium)

Inhibitor 3-amino-1,2,4-triazole μmoles	*Metabolite* Growth (absorbancy at 660 mμ) after 24 h				
		adenine		*histidine*	
	None	*1 μmole*	*5 μmoles*	*1.5 μmoles*	*1.5 μmoles + 1 μmole adenine*
			Escherichia coli		
0	0.69	0.73	0.75	0.74	0.75
10	0.03	0.58	0.60	0.01	0.77
20	0.01	0.43	0.39	0.00	0.75
40	0.00	0.19	0.15	0.00	0.69
80		0.00	0.00		0.71
100		0.00	0.00		0.71
			Torula cremoris		
0	1.0	1.0	1.0	1.0	
10	0.15	0.19	0.26	1.0	
20	0.08	0.09	0.12	1.0	
60	0.0	0.04	0.05	0.95	
100	0.0	0.0	0.0	0.93	

Escherichia coli, Crooks strain, was cultured in a 4-ml volume in 18 × 150 mm test tubes in the synthetic medium of DAVIS AND MINGIOLI[4]; a 24-h culture grown up in this medium was diluted 1:10 and a drop of this suspension added per assay tube as inoculum; cultures were slanted to favor aerobic conditions and incubated for 24 h at 30°. The technique for culturing *Torula cremoris* A.T.C.C. No. 2512 has been previously described[5] and assays were carried out in a final volume of 4 ml in 50-ml Erlenmeyer flasks and placed on a reciprocal shaker for 24 h at 25°. The yeast cultures were then diluted to 10 ml with water before making absorbancy measurements.

triazole as a weed killer, but also suggest that this compound may be useful in biochemical or clinical studies relative to purine metabolism.

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Formation of tryptophol in the disulfiram-treated rat

In man, the major deamination product of norepinephrine is 3-methoxy-4-hydroxymandelic acid[1]. In the white rat, however, a major metabolite of this catecholamine has been shown to be 3-methoxy-4-hydroxyphenylglycol[2]. These metabolites are presumably the oxidation and reduction products of a common intermediate, 3-methoxy-4-hydroxyphenylglycol aldehyde, which is formed *in vitro* by incubation of normetanephrine with purified monoamine oxidase[3]. The existence of this reduction product suggests that other biogenic amines also may be metabolized to alcohols. This possibility was considered for tryptamine which *in vivo* is largely metabolized to indole-3-acetic acid following oxidative deamination[4].

The postulated metabolite of tryptamine, tryptophol (indolylethyl alcohol), was synthesized by reduction of indole-3-acetic acid with $LiAlH_4$ (ref. 5) (this procedure appears useful for the preparation of alcohols from acidic metabolites of many biologically important amines). The leaflets obtained on crystallization from ether–petroleum ether melted at 59° and the picrate melted over a range of 93–96° (*cf.* ref. 6: m.p., 59°; m.p. picrate, 94–96°).

After chromatography in isopropanol–7 *N* NH_3 (4:1), and in benzene–propionic acid–water (2:2:1), the papers were sprayed with 0.5 % *p*-dimethylaminolbenzaldehyde in 50 % methanol–2 *N* HCl. A dark blue spot quickly appeared which had the R_F of 0.9 in either solvent. In contrast with tryptophol, indole-3-acetic acid yields maximal color formation with this spray in 20–40 min.

Each of 3 adult white rats was injected intraperitoneally with 10 mg tryptamine and their urines collected in dilute acid over a 24-h period. The pooled urines were adjusted to pH 1 with HCl, heated to 100° for 10 min and then extracted with 4 vol. ether. The ether solution was concentrated by evaporation and a portion of the extract transferred to Whatman No. 1 filter paper. After development in isopropanol–NH_3, the sprayed chromatogram revealed only a single blue spot which corresponded in R_F, color and rate of color development with indole-3-acetic acid.

The absence of tryptophol suggested that the presumed intermediate, indole-3-