that the saliva functions to supply a carbon source of energy-rich lipids (*12*) and that the proctodeal fluids constitute, at least for the beetles, a major source of nitrogen via the protoplasm of ingested symbiotes (*13*). Since there is no evidence of the beetles' proffering any of their digestive fluids to the termites, their trophic relationship with the termites seems to be undirectional.

Much remains unknown regarding the trophic interactions between these beetles and their hosts. A quantitative measurement of the relative frequency of each type of feeding behavior as well as a biochemical characterization of the stomodeal, proctodeal, and surface lipid reservoirs will be necessary before a detailed understanding of the trophic interactions can be obtained.

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2. A record of the convoluted history of various classification schemes for the relationships between termitophiles and their hosts has been summarized (*10*). I find the classification scheme developed by Kistner [D. H. Kistner, in *Biology of Social Insects*, H. Herman, Ed. (Academic Press, New York, in press), vol. 1, chap. 8] to be most useful. Thus, "an integrated termitophile is one which from its behavior and its host's behavior can be clearly seen to be incorporated into the social life of the colony. In contrast, a nonintegrated termitophile is one which is not incorporated into the social life of the colony but rather which has adapted to the nest as an ecological niche. If it is not known whether a species is integrated or not, judgment is suspended until the requisite information is available."
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7. The taxonomic assistance of Dr. J. K. Mauldin, U.S. Department of Agriculture, Forest Service, Gulfport, Miss., and Dr. B. M. Honigberg, Department of Zoology, University of Massachusetts, in identification of these protozoa is acknowledged.
8. This fluid was consistently perceived as clear under my experimental conditions, suggesting that it was probably saliva rather than regurgitated crop contents. This conclusion would support the observations of McMahan, working with *Cryptotermes brevis* (5). She noted that regurgitated food is not fed to the dependent castes, and may, in fact, be exchanged rather infrequently between the other members of the colony.
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Disulfiram Enhances Pharmacological Activity of Barbital and Impairs Its Urinary Elimination

Abstract. *Disulfiram or diethyldithiocarbamate significantly enhanced the sleeping time induced by barbital in rats. At identical time intervals after rats were injected with barbital the concentration of barbital in the blood or brain of animals that had previously received disulfiram was significantly higher than the concentrations in the corresponding tissues of control animals. Urinary excretion of barbital was significantly reduced in disulfiram-treated animals.*

Disulfiram has been in use in avoidance therapy in certain cases of alcoholism for many years. Although disulfiram by itself is generally regarded as a relatively safe drug, several cases of exaggerated responses to some drugs, and sometimes toxic manifestations, were reported in patients receiving disulfiram. For example, enhanced reduction of prothrombin level and evidence of bleeding were reported when an anticoagulant, warfarin, was administered to patients taking disulfiram (*1*). Typical symptoms of phenytoin overdosage were observed in patients receiving this drug and disulfiram (*2*). Psychosis and confusion were reported in patients taking both disulfiram and metronidazole (*3*). It has been suggested that disulfiram impairs the biotransformation of these drugs and thus enhances their pharmacological activity (*4*). Rats treated with disulfiram (200 mg/kg, intraperitoneally) 2 hours before the administration of hexobarbital slept three times as long as control rats (*5*). This effect was presumed to be due to a potentiation of the hexobarbital action in the central nervous system, because the amount of hexobarbital metabolized by the whole body in 30 minutes was not significantly altered by prior administration of disulfiram.

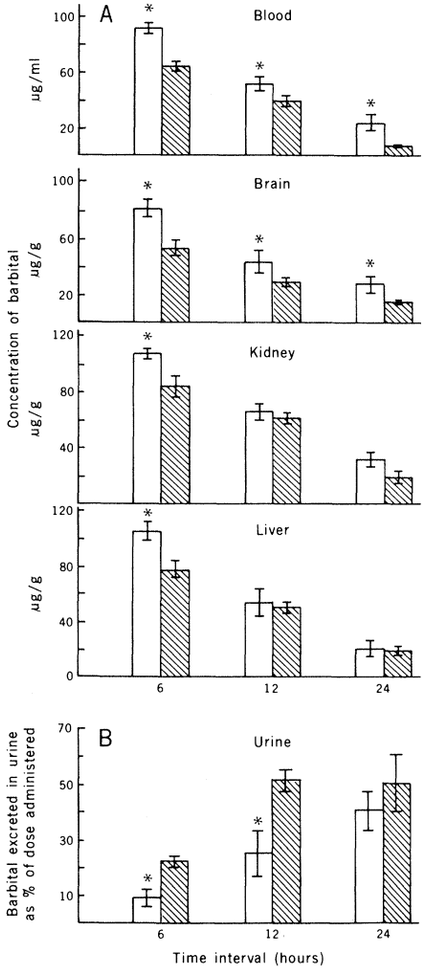

Fig. 1. The concentrations of barbital in blood, brain, kidneys, and liver (A), and the amounts of barbital excreted in urine (B) at 6, 12, and 24 hours after an intraperitoneal injection of barbital (100 mg/kg). The data in (B) are expressed as percentages of the dose administered. Unshaded bars, disulfiram-treated rats; shaded bars, CMC-treated rats. Each value represents the mean ± standard error of the mean obtained from a different group of six rats each. Asterisks indicate results statistically different from control ($P < .05$) by two-tailed Student's t-test.

We have observed that disulfiram (400 mg/kg, injected intraperitoneally) enhances the pharmacological activity and toxicity of barbital in the rat (*6*), although barbital undergoes virtually no metabolic transformation in this species and is eliminated almost entirely in the urine (*7*). This suggests that enhancement by disulfiram of the pharmacological activity of certain drugs could involve some aspects other than the impairment of enzymes involved in the biotransformation of drugs.

Here we report that treatment of rats with disulfiram is associated with changes in the distribution of barbital in blood, brain, liver, and kidneys, and that urinary excretion of barbital is significantly reduced in disulfiram-treated rats.

Male Sprague-Dawley rats (180 to 200 g) were used in all experiments. They were housed in temperature-regulated quarters (23° to 25°C) on a 12-hour light-dark cycle (lights on from 0700 to 1900) and given free access to food and water. The animals were divided into groups of six rats each. The rats in some of the groups received an intraperitoneal injection of 1 percent carboxymethyl-

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Table 1. The influence of disulfiram (200 mg/kg) and DDC (200 mg/kg) on sleeping time induced by barbital, and the concentration of barbital in the brain and blood upon return of the righting reflex in rats. Barbital (200 mg/kg), was injected 1 hour after CMC, disulfiram, or DDC. Upon return of the righting reflex the rats were decapitated and the concentration of barbital in blood and brain was measured as described in the text. Values represent the mean ± standard error of the mean from a different group of six rats each.

Treatment	Sleeping time (minutes)	Barbital concentration on awakening*	
		Brain (μg/g)	Blood (μg/ml)
CMC	153 ± 23	152 ± 3	195 ± 5
Disulfiram	367 ± 48†	159 ± 6	202 ± 8
DDC	307 ± 42†	158 ± 11	208 ± 10

*Return of righting reflex. †Statistically different from control ($P < .05$) by two-tailed Student's t-test.

cellulose (CMC) (2 ml/kg). The rats in the other groups received an intraperitoneal injection of disulfiram (200 mg/kg) suspended in a 1 percent solution of CMC. One hour later the rats in all groups were given an intraperitoneal injection of barbital sodium (100 mg/kg). They were then placed individually in metabolic cages (23 by 25 by 18 cm) to allow for urine collection. At different time intervals (6, 12, and 24 hours) after barbital administration, the animals were decapitated and blood was collected in 50-ml beakers containing about 500 units of heparin in 0.1 ml of 0.9 percent NaCl solution. The blood was then immediately analyzed for barbital content. The brain, liver, and kidneys were immediately removed after decapitation. The barbital content of blood, brain, liver, kidneys, and urine collected during the 6-, 12-, and 24-hour periods was measured spectrophotometrically as described previously (*8*). This method involves extraction of barbital into diethylether from blood diluted with phosphate buffer (*p*H 6), or from tissue samples homogenized in the same buffer. A portion of the ether layer is then aspirated and the barbital it contains is extracted with phosphate buffer (*p*H 11) and its optical density in the buffer is measured. The concentration of barbital is found from a standard curve relating optical density to concentration. Percentage recoveries varied between 85 and 89 percent. This method proved specific for barbital since the use of liquid scintillation spectrometry to measure ^{14}C-labeled barbital in some biological fluids yielded similar results.

As shown in Fig. 1A, disulfiram-treated animals had a significantly higher ($P < .05$) concentration of barbital in blood and brain at the three time intervals examined. The concentration of barbital in kidneys and liver was significantly higher ($P < .05$) at the 6-hour interval. Figure 1B indicates that the amounts of barbital excreted in urine during 6- or 12-hour periods by disulfiram-treated rats are significantly less ($P < .05$) than the amounts excreted by CMC-treated rats.

In addition, we found that the same dose of disulfiram (200 mg/kg) administered 1 hour before barbital (200 mg/kg) significantly increased ($P < .05$) the sleeping time induced by barbital. The concentrations of barbital in the brain or blood in disulfiram- or CMC-treated groups were similar when rats gained their righting reflex. With diethyldithiocarbamate (DDC), believed to be an active metabolite of disulfiram, similar results were observed (Table 1). Thus, it seems that the sensitivity of the brain to barbital is not affected by disulfiram or DDC.

Our results indicate that the urinary elimination of barbital in disulfiram-treated animals is impaired. This impairment could account for the prolongation of sleeping time induced by barbital, particularly since it is associated with an increased concentration of barbital in blood and brain. We previously reported (*9*) that disulfiram significantly enhances morphine-induced analgesia and catalepsy in the rat. When we examined the elimination of morphine and metabolites in disulfiram- and CMC-treated rats, we found that both urinary and biliary excretion of morphine and metabolites were significantly impaired in the disulfiram-treated group (*9*).

Previous studies of the enhanced pharmacological activity of certain drugs by disulfiram have usually been limited to measuring the concentration and half-life of the drugs in the plasma. It has often been concluded that the biotransformation of the drug in question is impaired (*4*, *10*). Indeed, it has been shown that disulfiram impairs the activity of several enzymes implicated in drug biotransformation (*11*). However, in view of the results obtained with barbital and morphine, the influence of disulfiram on drug excretion should be considered when attempts are made to explore the mechanisms underlying the enhancement of the pharmacological activity of certain drugs by disulfiram. In man, retardation of drug elimination might have therapeutic implications.

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Iconic Storage: The Role of Rods

Abstract. *The hypothesis that rods mediate iconic storage was tested by presenting letters of one color against a field of another. The colors were chosen to be discriminable only by the cones, only by the rods, or both. Under dark adaptation, the rods had little if any effect on partial-report advantage; however, they were important in determining the phenomenal persistence of the stimulus. Under light adaptation, the rods played no apparent role in either type of persistence.*

The information in a briefly displayed visual stimulus is not lost as soon as the display ceases. Rather, it persists for a fraction of a second, almost as if the physical stimulus were still present.

This can be shown by the partial-report technique introduced by Sperling (*1*). An array of letters (say, three rows of four letters each) is briefly displayed and followed by a cue to read the letters in one row. If the cue is delayed by more than a few hundred milliseconds, per-

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