ent mechanisms, it is not surprising to find that the mercury content in stream sediments varies widely with the type of sediment (2).

We have observed the equilibrium partitioning of mercury in wellshaken laboratory samples to range from  $5.8 \times 10^{-4}$  part per million (ppm) of mercury in water per part per million of mercury in dry sediment for sandy sediments to less than 1.4  $\times$  $10^{-8}$  ppm of mercury in water per part per million of mercury in dry sediment for sediments that are rich in organic material (see Table 1). Cation adsorption also depends on particle size (3) as well as on the chemical nature of the sedimentary material.

Inasmuch as chloride ion complexes strongly with mercury, and sodium and calcium ions can compete with Hg<sup>2+</sup> for exchange sites, a recent report of the contamination of freshwater by the runoff of CaCl<sub>2</sub> and NaCl used for deicing roads raised the possibility that road salt could release mercury from bottom sediments (4). The results tabulated in Table 1 show such to be the case, with the addition of NaCl or CaCl<sub>2</sub> increasing the relative amount of mercury in the water in equilibrium with the sediments by two to five or more orders of magnitude. The effect tends to increase as the mercury burden of the sediments increases. The pHchanges consequent upon salt addition probably also contribute to the release of mercury.

In addition to being a serious contaminant itself, road salt in natural waters can acerbate contamination by mercury and undoubtedly by other toxic heavy metals. The results presented here are also of interest in connection with the chemistry of heavy metals in the estuarine environment where sediment-laden freshwater and saltwater are mixed.

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- 10 MARCH 1972

## Ethanol Consumption by Rats under **Different Lighting Conditions**

Geller (1) has reported that male albino rats drink more alcohol as a result of having been held for a prolonged period in constant darkness. Some work of mine supports this finding, but limits its applicability to young animals, as is shown below. Comparison of his data with mine also helps to eliminate an apparent contradiction in Geller's work.

Rats have been found to show significant changes in alcohol consumption during the first 6 weeks of access to it (2, 3). Because Geller failed to include control groups that did not have the lighting conditions changed, it is not possible to determine from Geller's data alone whether the changes in alcohol consumption he reported were caused by the illumination shifts, or by the natural pattern of variation which would have occurred regardless of the lighting.

Figure 1 shows the data from such a control group [from (2)] superimposed on Geller's data. The pattern of change in alcohol drinking is similar in both groups, indicating that the changes Geller attributed to lighting differences may be artifacts. If, for instance, the periodic (9 hours dark, 15 hours light) lighting had been imposed in the middle of the 6 weeks, and the constant illumination last, the rats might have consumed more alcohol on a 24-hour light cycle than in either of the other conditions.

The very high alcohol intake during the periodic lighting condition (Fig. 1) was contradictory (i) to findings in Geller's second study in which the rats drank much more alcohol during constant dark than in the periodic situation, (ii) to Geller's hypothesis that darkness induces increased alcohol drinking, and (iii) to my finding that young albino male Sprague-Dawley rats consume almost exactly the same amount of alcohol regardless of whether they are in constant light or on a periodic 12-hour dark, 12-hour light schedule (F = 0.012, d.f. = 1;30) (2). If, however, the high consumption by Geller's rats in the final 2 weeks (Fig. 1) is artifactual, and not caused by periodic lighting, these contradictions are eliminated.

The 24 albino male Sprague-Dawley rats that were kept in constant light also showed a pattern of changes in alcohol consumption similar to that shown in Fig. 1. A repeated-measures analysis of variance showed that these changes were highly significant ( $F_{days} = 2.69$ , d.f. = 23;529, P < .01). Comparison of the pattern for these constant-light rats with that for the animals in periodic light produced F for the product days times lighting = 0.121 (d.f. = 23;690, P >.05). A similar pattern has also been found with two male and two female young albino Long-Evans rats housed in constant light, but was not seen in 12 hooded and black rats from the same two litters.

The evidence that darkness increases alcohol consumption under certain circumstances is very strong. In Fig. 1, although the patterns over days are similar, the absolute amount of alcohol consumption is much higher in Geller's rats (different ordinates were used for the two groups). Similarly, the alcohol intake by six rats in constant dark in Geller's second study is much higher than what I have observed for Sprague-Dawley albinos (housed in either continual or periodic light), even though the initial alcohol consumption by his rats, before being placed in the dark, is very close to that for my animals. The most plausible explanation for these differences seems to be that the complete darkness to which Geller's rats were subjected, produced an increase in their alcohol intake. Further-



Fig. 1. Pattern of alcohol consumption by eight control rats kept throughout the experiment on a 12-hour dark, 12-hour light schedule (2), superimposed on the mean alcohol consumption by Geller's four rats, which were sequentially kept in three different lighting conditions (1). The general level of intake was much higher for Geller's rats (as shown by the use of different ordinates for the two groups), perhaps because of the initial darkness; but the temporal patterns were similar, suggesting that changes in consumption by Geller's rats may not have been caused by changes in lighting.

more, once raised to this higher rate of intake, the rats (in Geller's first study) continued to drink at an elevated rate, although they still showed the fluctuations over time normally seen with these rats.

The increase in alcohol consumption by Geller's albino rats in 24-hour darkness is similar to that usually found with hooded Long-Evans rats kept in continuous light (2). The pattern, however, for all albinos tested so far in either continuous or periodic light is that shown in Fig. 1 and results in a much lower total consumption of alcohol. This result suggests the possibility that in normal lighting the quantity of light stimulating or passing through the unpigmented eyes of albinos is so great that it somehow interferes with the process that would otherwise increase alcohol drinking during the first few weeks of access. This light thus results in the decline of alcohol drinking after the second week. With albinos placed in nearly total darkness, or with hooded rats, whose eyes, and brain areas behind the eyes, are protected by pigmentation, this process continues unhindered; these rats, therefore, eventually drink much greater quantities of alcohol than the albinos do in stronger lighting.

I have also observed increased alcohol drinking with young male albino Wistar rats that have been kept in nearly total darkness for 2 weeks before and during the period that they have access to alcohol (Table 1). Older (220 days) male albino Wistar rats, however, drank significantly more alcohol in the light than in the dark (2). Similarly, slight increases in illumination, arising from cage position on the rack, have been found to be highly positively correlated (r = .742, P < .01) with increased alcohol consumption in 18 older (180 days) male hooded Long-Evans rats (4).

The mechanism by which light is able to produce such strong, long-lasting, but opposite, influences on alcohol consumption of young and older rats is still unknown. Perhaps, as Geller suggests, melatonin may be involved, but his evidence for this (from two rats injected daily with melatonin, without controls) is not convincing. Although daily hypodermic injections do not increase alcohol intake

Table 1. Mean alcohol consumption on the first 2 days of access for 24 young (90 days) and 24 older (220 days) male albino Wistar rats, under conditions of continuous light or continuous dark for the 2 weeks before and during access. F for the interaction between age and lighting conditions is 7.40; d.f. = 1.32; P < .02 (2).

Condition	Consumption (ml/day)		
	Young	Older	Mean
Light	0.82	1.78	1.30
Dark	1.16	0.61	0.88
Mean	0.99	1.20	1.09
	E/T (perce	entage)*	
Light	33.0	71.3	52.2
Dark	46.3	24.3	35.3
Mean	39.7	47.8	43.8

\*The alcohol solution used was 5 percent, by volume. E/T is the ratio of alcohol consumed to total consumption per rat per day.

in C57BL mice (5), they might increase drinking in rats; even handling rats for 1 minute per day significantly increases their alcohol consumption (2). Nevertheless, the interaction between alcohol. melatonin (and dopamine), and the addictive alkaloids in the brain is intriguing and warrants additional investigation.

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- These animals had been on a program of periodic free access and intubation of alcohol for 116 days prior to the measurement of this correlation. The program has resulted in extremely high voluntary consumption of alcohol: for day 116, they drank 4.78 ml of absolute ethanol per rat, 10.17 ml of absolute ethanol per kilogram of body weight. [These figures are slightly higher than the normal daily intake because of a switch from a 7 percent to a 10 percent (by volume) of alcohol solution on day 115.]
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Sinclair presents data from a control group in his laboratory and alleges that it shows a pattern of variation comparable to that produced by experimental manipulation in my studies (1). This compari-

son is relatively meaningless given (i) the tremendous difference in the ordinates of the two curves shown in his figure 1, (ii) the likely differences in conditions between the two laboratories, and (iii) the lack of some measure of variation for the means of his control group. Later, Sinclair uses the same data to argue that "the evidence that darkness increases ethanol consumption is very strong."

Sinclair also states that the increased alcohol drinking during the normal light-dark cycle of the first study was contradicted by my second group of rats which showed, if anything, a greater preference for water than alcohol under the same conditions of lighting. In order to make a valid comparison between these two groups of rats, one must take into account the differences in their drinking histories. The first group had a history of 21/2 weeks in total darkness during which alcohol intake was markedly increased. This historical condition is quite important, since both Sinclair and I have observed that either light or initial periodic darkness does not necessarily result in increased alcohol intake.

Another criticism made by Sinclair is that the melatonin data that I presented were not convincing. While the data available thus far are not compelling, they are highly suggestive, and that is precisely what I said. In fairness, Sinclair did allow that the melatonin data were sufficiently intriguing to warrant further study. I agree.

Although Sinclair has not substantiated his criticisms of my work, he has extended my observations by demonstrating that the effects of darkness may be modulated by an age-related variable. I find that to be of extreme interest and assure him that I will remain cognizant of it in my future research.

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