deep tectal, positive response. Contrary to the usual interpretation, this wave may be due to activation of slow conducting fibers. The deep response was depressed by both threshold and supramaximum stimulation.

The contralateral visual pathway used as control excludes a systemic effect of colchicine. One hour after direct colchicine application to the tectum the postsynaptic response to optic nerve stimulation was unaffected. It thus seems unlikely that the depression of synaptic transmission could result from a direct effect of colchicine on the nerve terminals, as might be anticipated from its prompt and drastic inhibitory effect on the release of catecholamine in the adrenal medulla (*13*). Intraocular injection of Ringer solution (20 μl) did not affect the tectal potentials recorded 7 days thereafter.

There is a striking convergence among the effects of intraocular injection of colchicine, that is, reduction of axonal transport (*6, 7*), alteration of organelles in the nerve terminals (*8*), and depression of synaptic transmission. The time course of these alterations is similar; the effect on protein transport appeared earlier than the other two, and all three were reversible within the same period. From the early appearance of these effects, we are tempted to conclude that proteins or other components migrating with the fast flow are more likely to be implicated in synaptic transmission than the material migrating slowly. Colchicine interference with slowly migrating material may contribute, however, to the relatively slow recovery of the phenomena. It should be noted that length of the nerve segment distal to the site of sectioning determines the time of onset of synaptic failure; the longer the axon, the later the alteration of the postsynaptic response (*14*). These observations point also to the dependence of the synaptic transmission on material supplied by the axon. In conclusion, although other effects of colchicine cannot be excluded, our results would be consistent with the notion that material, which is normally provided by the ganglion cell body to nerve terminals and which may be arrested by this drug, plays an essential role in the maintenance of synaptic functions.

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Release of Mercury from Contaminated Freshwater Sediments by the Runoff of Road Deicing Salt

Mercury is strongly held by the bottom sediments of natural water courses, and, as binding mechanisms, Krauskopf (*1*) has suggested (i) correlation or sorption on hydrated ferric oxide, (ii) surface sorption or ion exchange, or both, with naturally occurring mineral ion exchangers, such as montmorillonite, and (iii) sorption or chemical combination, or both, with organic material, such as peat and especially sulfur-containing matter. In view of these differ-

Table 1. Mercury partitioning at 25°C; S, sandy sediment; O, highly organic sediment. Both sandy and highly organic sediments are from Nagog Pond, Acton, Massachusetts. The moisture contents of the sandy and highly organic sediments were, respectively, 27 and 79 percent; the losses on ignition of the sandy and highly organic sediments were, respectively, 0.804 and 44.3 percent. Both types of sediments released some H_2S upon acidification; the highly organic sediments released more H_2S than the sandy sediments.

Sediment type	Salt added		Hg^{2+} content (ppm)		$(Hg^{2+})_{H_2O}/(Hg^{2+})_{sed}$	pH
	Formula	Amount (g/liter)	Dry sediment	Water		
S	None		41.2	0.024	5.8×10^{-4}	6.7
O	None		1430	< 0.00002	$< 1.4 \times 10^{-8}$	5.2
O	None		2670	0.0044	1.65×10^{-6}	5.1
S	NaCl	35	45.4	1.70	3.75×10^{-2}	6.6
O	NaCl	35	800	0.004	5.0×10^{-6}	4.8
O	NaCl	35	2670	25.0	9.4×10^{-3}	4.6
O	$CaCl_2$	165	800	1.58	1.9×10^{-3}	3.7
O	$CaCl_2$	165	1535	215.0	0.14	3.6

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 well-shaken laboratory samples to range from 5.8×10^{-4} part per million (ppm) of mercury in water per part per million of mercury in dry sediment for sandy sediments to less than 1.4×10^{-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^{2+} for exchange sites, a recent report of the contamination of freshwater by the runoff of $CaCl_2$ 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 $CaCl_2$ 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 *p*H changes 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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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 ($F_{\text{days}} = 2.69$, 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-

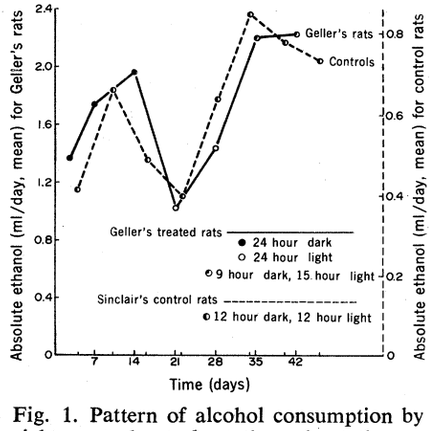

Fig. 1. Pattern of alcohol consumption by eight control rats kept throughout the experiment on a 12-hour dark, 12-hour light schedule (*2*), superimposed on the mean alcohol consumption by Geller's four rats, which were sequentially kept in three different lighting conditions (*1*). The general level 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.