istered subcutaneously 30 minutes before training (*5*), we sought to determine whether such injections of amphetamine also antagonize the effects of cycloheximide on activity. Mice were first injected with cycloheximide; 3 hours and 45 minutes later, they were injected subcutaneously with amphetamine (1 mg/kg) or saline. This interval between drug injections is the same one employed in memory studies, where amphetamine, but not saline, was found to antagonize the effect of cycloheximide. Activity was measured continuously during the 1st hour after amphetamine or saline injection. Mice given cycloheximide and saline ($N = 8$) exhibited marked inhibition of activity during this period; their activity was significantly less than that of mice given only saline (Fig. 1) before activity ($P < .01$). Amphetamine ($N = 8$) did not antagonize the depression of activity. Thus, the effect of amphetamine on memory in cycloheximide-treated mice (*5*) is not correlated with a measurable increase in activity.

We conclude from these studies (i) that cycloheximide affects activity by acting on the brain, (ii) that this action is unrelated to its inhibition of protein synthesis, and (iii) that these effects of cycloheximide on activity do not appear to be responsible for its amnesic action. It is possible, of course, that cycloheximide has some other property, unrelated to inhibition of cerebral protein synthesis, that is responsible for its amnesic effect.

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6. Activity was measured in a soundproof chamber 45.72 cm by 45.72 cm and 27.94 cm high. The floor of the chamber was divided into quadrants, and an index of gross activity was obtained from the number of times each animal crossed from one quadrant to another. In addition, an index of rearing was obtained from touch plates that were placed along the walls of the chamber at 5.08 cm above the floor. All data were automatically recorded.
7. A 12-mg sample of isocycloheximide was provided by Francis Johnson, Dow Chemical Company, Wayland, Mass. The small amount of the drug limited our study to effects of intracerebral injections. In accordance with previous observations (*1*), we determined that this sample had a negligible inhibitory effect (12 percent) on cerebral protein synthesis.
8. For training, a mouse was placed in the stem of the T maze; after 5 seconds, shock (0.4 ma) was applied through the grid floor. The shock was terminated when the mouse entered the dark limb of the maze. The mouse was removed 5 seconds later. Training was continued with an intertrial interval of 30 seconds and a criterion of five out of six correct responses. Savings was calculated as described by S. H. Barondes and H. D. Cohen [*Science* **151**, 594 (1966)]. Mice that required more trials to reach criterion on retest than on original learning were scored as having zero savings. The savings of mice given cycloheximide were significantly less than the savings of mice given saline ($P < .02$) or isocycloheximide ($P < .02$).
9. Supported by PHS grant MH-18282.

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Models for the Transport of DDT: Verification Analysis

In their article Harrison *et al.* (*1*) presented interesting models for the transport of DDT (*2*) through several trophic levels. Their models essentially represent a series of mass transfer equations in a homogeneous spatial medium. Harrison *et al.* claim that the models they propose are "sufficiently descriptive to yield the general nature of population response, and to make possible the prediction that a significant variation of a predator population would cause upsets throughout the entire system, some of which might be of sufficient magnitude to create 'out-of-control' conditions." However, nowhere in the article does there appear a verification analysis of the models proposed which would lend even minimum support to such a contention.

A verification analysis should compare the analytical model to observed data from the real world. Such an analysis is basic to a systems analysis of any phenomenon. The presentation of a model without some indication of how well the model does in "independent" test predictions represents only part of the systems analysis. One must always be prepared to demonstrate the utility of the model for decision-making. For this reason it is necessary to go substantially beyond mere structuring of the analytical model. Numerous changes in the model may be necessitated by such a verification analysis. Certainly this step must be carried out before any claim is made about the ability of the model for prediction purposes.

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Activity Change as the Cause of Apparent Aversiveness during Prolonged Hypothalamic Stimulation

Mendelson (*1*) reported that rats in the presence of relevant goal objects (for example, food for a stimulus-bound eater) while receiving rewarding hypothalamic stimulation turn off the stimulation less frequently than rats do when there are no relevant goal objects. The rats could switch the stimulation ON by moving into one half of a shuttle box and switch it OFF by running into the other half. Mendelson states that the phenomenon is due to a suppression of the normally aversive effects of long lasting stimulation by consummatory behavior. He then proposed that the aversive effects of the brain stimulation (*2, 3*) are due to the excessive arousal of drive, and that the opportunity to engage in consummatory behavior reduces the drive and makes the stimulation less aversive. However, a much simpler hypothesis can account for his result. During hypothalamic stimulation, the rat shows a high level of activity such as sniffing, searching, and moving, but if the relevant goal object is supplied during stimulation, such activity is reduced because the rat becomes engaged in eating, drinking, or gnawing (*4*). This simple "activity" hypothesis can account for Mendelson's finding that the rat would prefer a longer duration of brain stimulation with a goal object than without one. Such a sim-