and second, there is a twofold difference in the productivity of the vegetation on the control plots. Odum *et al.* (*2*) observed comparable differences in productivity during old field succession, but these were attributable to previous fertilization and changes in the growth forms of the dominants, factors not adequately discussed by Hurd *et al.* We have also observed significant differences in the primary production of herbaceous communities among fields of different quality, but not among fields differing principally in age (*3*). These data suggest that site quality rather than age may have been the more important of the confounded variables in the experiment of Hurd *et al.*

BUFORD HOLT
Biology Department,
Brookhaven National Laboratory,
Upton, New York 11973

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We do not feel that we have confounded age with location in our studies. As we said, "we studied two adjacent abandoned hayfields," and "macroclimate and soil series are identical." Both fields were planted in timothy (*Phleum pratense*) and are slowly decaying into "natural" ecosystems characteristic of upstate New York. Since timothy is hardly a species characteristic of impoverished sites (*1*), we assume that whatever impoverishment leads to invasion of *Poa compresa* and *Hieracium pratense* arises out of the successional aging process. We have extensive soil data on the two fields as well as a description of their previous land usage from the land's owner. These data could not be included in a report in *Science*.

L. E. HURD
M. V. MELLINGER
L. L. WOLF
S. J. MCNAUGHTON
Biology Department,
Syracuse University,
Syracuse, New York 13210

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Tree Seedling Growth: Effects of Shaking

Neel and Harris (*1*) reported an interesting effect of shaking on growth of *Liquidambar* seedlings and proposed that a hormonal mechanism was involved. They also discussed the effects of wind on leaf size.

First, they quoted experiments showing reduced growth of vine leaves subjected to breeze (*2*). Such a reduction could, however, be caused by partial drying of the leaves through two known mechanisms. Lowered turgor could prevent cell expansion directly. Stomatal closure could also occur, with a reduction in photosynthesis. These kinds of interpretation have been applied to the results of similar experiments (*3*).

Second, Neel and Harris stated that the larger leaves found on seedlings grown indoors (compared with those grown outside) are somehow "in line with" their observations. But the increase of leaf size with decreasing light is also well known (*4*) and could be involved in the present case. Moreover, differences in water stress could again occur between plants in the two environments with resulting differential growth similar to that outlined above. In addition to the fact that those outside might be less well watered or exposed to drier air, different ventilation conditions need to be considered. The effect of wind alone on transpiration rate is complex and, depending upon water availability and net radiation, can lead to either an increase or a reduction in water loss.

Thus, neither of these points necessarily supports the argument that a hormone is involved. The introduction of a comparison between staked and unstaked plants is more relevant. However, even if conditions for exchange of water, carbon dioxide, and radiation were not influenced by shaking, it is still possible that the movement of water in the stem can be affected. The incidence of blocked vessels due to cavitation (*5*) would probably be higher in the shaken trees and the water supply to the shoots would therefore be lower. This latter hypothesis could be tested with a pressure chamber (*6*). Growth in height and setting of terminal buds could both be moderated by water status.

A shaking effect certainly seems to exist and cavitation is not necessarily involved in it. Although hormonal control would be a plausible evolutionary adaptation, alternative explanations are available for all of the other effects quoted by Neel and Harris in support of their claim and they have not given sufficient evidence that hormonal effects are actually involved.

D. F. PARKHURST
G. I. PEARMAN
Division of Atmospheric Physics,
Commonwealth Scientific and Industrial
Research Organization, Aspendale,
Victoria 3195, Australia

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The purpose of our report (*1*) was to describe the marked influence on tree growth of daily short periods (30 sec) of trunk movement.

Parkhurst and Pearman's comments that shoot growth and reduction of leaf size could be the result of wind (and trunk movement) affecting moisture relationships, including cavitation, are well taken. However, three observations indicate that cavitation was not a factor in reducing shoot growth.

This dramatic influence on shoot growth was first noted in a light-quality experiment in which the terminal 30 cm (12 inches) of some of the trees were

Fig. 1. Corn plant shaken 30 seconds daily for 25 days (left); not shaken (right). Plants were grown in greenhouse. Arrow, height of plants at beginning.

covered with a cloth bag for 20 minutes each evening. A black bag was carefully placed over the topmost lateral branches of each tree. A wire supported the bag above the growing point. Any movement of the trunk was gentle. Without exception, every terminal section that was bagged made less growth than those not bagged. Putting the bag on and immediately removing it gave similar results.

Shoot growth was slowed regardless of whether the plants were shaken at 8:30 a.m. or at 8:30 p.m. P.D.T. (*1*).

More recently, daily shaking of corn, *Zea mays*, plants for 30 seconds resulted in reductions of 50 percent in height growth, 30 percent in leaf number, and 15 percent in leaf length (Fig. 1). However, when the shaken plants were no longer shaken each day, height-growth rate equal to that of the unshaken plants was evident within 3 days. It seems doubtful that cavitation would have been the main cause in growth reduction in these trials.

Leaf-size reduction has been noted in handling leaves during their measurement (*2*, *3*). Gentle stroking of leaves of *Bryonia dioica* resulted in smaller leaves and shorter internodes (*4*). Auxin concentrations in the touched plants were less than in the untouched. Growth rate was restored to normal by adding auxin. In addition, shaking for 30 seconds daily resulted in shorter corn leaves (note above) and *Cucurbita melopepo* petioles (*3*).

Such marked responses from short periods of handling or movement suggest that another factor in addition to those usually ascribed to slowing growth may be involved. A growth-influencing mechanism of a hormonal nature does seem to be indicated. Ethylene has been shown to reduce longitudinal growth accompanied by increased radial expansion in pea epicotyls (*5*). An ethylene-enriched atmosphere (5 to 20 parts per million) around the base of young Monterey pine and *Liquidambar* resulted in increased radial growth of xylem and phloem (*6*).

P. L. Neel
Agricultural Research Center,
Fort Lauderdale, Florida 33314
R. W. Harris
Environmental Horticulture,
University of California, Davis 95616

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6-Hydroxydopamine, Noradrenergic Reward, and Schizophrenia

Stein and Wise (*1*) propose "a novel physiological and chemical etiology for schizophrenia" which involves the endogenous buildup of 6-hydroxydopamine or its metabolites and a resultant deterioration of noradrenergic pathways that mediate reward. Data-related evidence rests principally on their finding that 6-hydroxydopamine hydrobromide impairs lever pressing for rewarding electrical stimulation (self-stimulation) of the brain. Our results do not fully support this finding. Rats were prepared with hypothalamic electrodes and indwelling lateral ventricular cannulas (*2*), were tested for self-stimulation, and were then given two, 200-μg intraventricular injections (72 hours apart) of 6-hydroxydopamine hydrobromide in a vehicle of 0.9 percent saline and 0.1 percent ascorbic acid. Although the temporary decrement in self-stimulation rates observed by Stein and Wise is replicable, priming (experimenter-delivered stimulation not contingent on lever pressing) during sessions on the first day after treatment with 6-hydroxydopamine can bring animals to the rates of self-stimulation prior to treatment. Priming becomes less necessary, then completely unnecessary within less than a week. Our data suggest that permanent impairment of most norepinephrine-containing terminals does not adversely affect significant aspects of the self-stimulation phenomenon, and that temporary behavioral changes are most likely related to depression of an adrenergic arousal mechanism, or to a complex of short-term changes or toxic reactions, or to both, rather than to selective interference with a positive reinforcement system.

To verify that our injections did indeed deplete brain norepinephrine (NE), we assayed for telencephalic NE in two animals (*3*). Telencephalic NE in both animals was found to be depleted 90 percent relative to control values, a greater depletion than Stein and Wise have reported.

It is our contention that Stein and Wise would not have obtained even a temporary decrement in the rate of self-stimulation after 6-hydroxydopamine had they given their animals priming stimulation, and that they are monitoring changes that do not involve any basic shift in responsiveness to reinforcement parameters. Roll (*4*) advanced a similar argument earlier against Wise and Stein (*5*) in experiments in which each utilized disulfiram for NE depletion. Roll found that animals treated with disulfiram became soporific, yet when aroused by the experimenter, their rates of self-stimulation were as high as those prior to the treatment.

In addition, there is no evidence that durable changes occur in an animal's response to natural rewards after 6-hydroxydopamine. Stein and Wise cite a paper by Schoenfeld and Zigmond (*6*) as evidence that deficits occur, yet the behavior of animals in that study returned to normal levels within 3 days, and Schoenfeld and Zigmond make the telling point that a later injection of 6-hydroxydopamine had no further effect on NE amounts, but disrupted behavior to the same extent as earlier doses. We find food and water intake reduced as much as 75 percent after 6-hydroxyopamine, but complete recovery occurs in 2 to 3 days. The copulatory behavior of male rats is unaffected by our 6-hydroxydopamine regime, perhaps because priming is continually present in the form of a moving partner giving multisensory input.

Only one mechanism (receptor supersensitivity due to NE depletion) might enable the NE mediation of reward hypothesis to encompass such data. However, available evidence (*7*) suggests that changes in receptor sensitivity to NE (unlike the rapid sensitization to exogenous NE that follows denervation and is due to interference with NE reuptake by presynaptic terminals) do not occur quickly enough to explain these data. Also, we have given intraventricular injections of 50 and 100 μg of phentolamine, an alpha-adrenergic receptor blocking agent, to animals treated with 6-hydroxydopamine (their self-stimulation rate had