poral interplay between a light-sensitive system and a cooperating "dark" system.

The chief evidence for this interpretation lies in the cited irrelevance for inversion of the light pattern evoking it. A depletion effect is also suggested by the fact that reversed bending does not start until after 2 to 3 minutes of unusually rapid elongation; in fact, the maximum rate of reversed bending just about coincides in time with the maximum rate of elongation. Further, in studying the distribution of elemental curvature along the growth zone during bending and inversion, I found that the maximal initial rate of reversed bending occurs at or very near the locus of maximal curvature; this would be that level in the growth zone where, across the cell, the greatest accumulated difference in growth capacity exists, and where a burst of light ought to initiate the fastest reversed bending.

The spatial distribution of bending velocity changes with time in a way suggesting that capacity deficit first develops high in the growth zone because supply is basically from below (*4*). I attribute the strict polarization of reversed bending in the plane of prior curvature to the pattern of altered growth capacity established by the different growth rates during prior bending. This pattern may well involve different states of extension of the cell wall's microfibrils which are engaged in multinet growth (*5*).

Growth capacity evidently works with adaptation but overrides it under conditions where a burst of light unmasks latent capacity differences across the cell's diameter. One might choose to consider growth capacity as a normally concealed supply problem supplementary to adaptation. But operationally, adaptation is known only by what a system does: whether, how much, and in what direction it responds. Hence the scheme of Delbrück and Reichardt for the light responses of *Phycomyces* (*6*) must be judged imperfect. Its deficiency confounds Reichhardt and Varjú's analysis of inversion.

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9 January 1961

Nitrogen Deficiency and Fluoride Susceptibility of Bean Seedlings

Abstract. Groups of bean seedlings (*Phaseolus vulgaris*) were grown concurrently in cleaned and fluoride-containing (4 to 7 μg of F^- per cubic meter) air in plant-growth chambers for 10 and 20 days with six water-culture treatments: Hoaglund's formulation and Hoaglund's minus potassium, calcium, nitrogen, phosphorus, and iron. An inadvertent 15-hour exposure on day 15 (at 42 $\mu g/m^3$) produced foliar fluorosis symptoms only on the nitrogen-deficient plants fumigated 20 days, although higher fluoride concentrations were present in the foliage of similar plants grown in the other nutrient-deficient solutions.

The limited published data relating nutrition levels to susceptibility or resistance of plants to atmospheric fluoride exposure appear to be somewhat paradoxical. Brennan *et al.* (*1*) studied visible fluoride-induced, foliar toxicity in tomatoes as modified by alterations in the nitrogen, calcium, and phosphorous nutrition. They concluded that medium levels of nitrogen and calcium nutrition favored sorption of toxic quantities of fluorine, whereas low or deficient nutrition levels prevented fluorine injury. Fumigation concentrations of 48 and 470 parts of hydrogen fluoride per billion were used in these experiments.

Applegate and Adams (*2*) recently reported the effect of nutritional and water deficiencies on respiration and relative fluoride uptake of bean seedlings (*Phaseolus vulgaris*) at low fumigation levels of fluoride. Ten- and twenty-day fumigations were conducted at approximately 2 μg of F^- per cubic meter (1.6 parts per billion) of hydrogen fluoride, which is well below exposure levels required to produce visible, fluoride-induced foliar necrosis. The data showed that deficiencies in potassium, phosphorus, and iron accelerated the uptake of atmospheric fluoride. Direct comparison of data was impossible because of basic differences in the experimental techniques that were used in these separate investigations (*1*, *2*).

Studies at low fumigation concentrations are continuing in this laboratory. All plants are grown in an air pollution phytotron (*3*), and the experimental techniques have been described previously (*2*). Recently, one set of beans was inadvertently exposed to a high concentration of atmospheric hydrogen fluoride. For a period of 15 hours on day 15 of a 20-day exposure, the average concentration was 42 $\mu g/m^3$ (34 parts per billion). This acute exposure produced foliar necrosis in all plants subjected to one of the six nutrient treatments being studied. This report presents some observations on the influence of nutrition upon the susceptibility of beans to visible foliar injury from atmospheric fluorides (*4*).

One-half of the 20-day fumigated and control bean plants are shown in Fig. 1. The duplicate plants (not photographed) showed similar characteristic growth patterns and foliar markings. No fluorine-induced necrosis was observed in the group of 48 plants fumigated for 10 days and removed from the phytotron prior to the accidental 15-hour exposure at 42 $\mu g/m^3$. Foliar fluorosis was observed on all eight of the plants grown in nitrogen-deficient nutrient and fumigated 20 days. No fluoride-induced foliar symptoms were

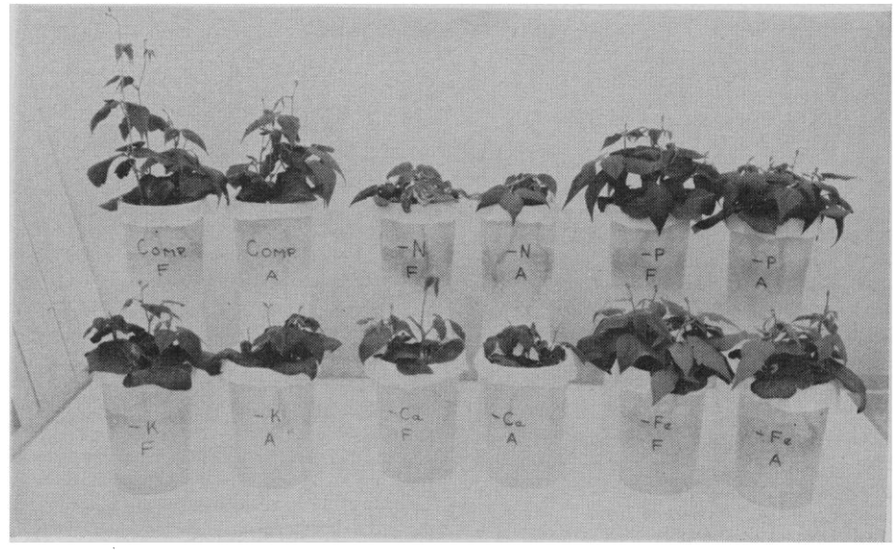

Fig. 1. Twenty-day fluoride-fumigated (F) and control (A) bean plants (*Phaseolus vulgaris*) grown in complete (Hoaglund's) nutrient solution "COMP" and nutrient solutions from which nitrogen (— N), phosphorus (— P), potassium (— K), calcium (—Ca), and iron (—Fe) have been withheld.

present on any of the other 40 plants in the 20-day set.

The fluoride-induced foliar symptoms expressed by the plants grown in nitrogen-deficient nutrient, although typically marginal, were also interveinal in nature. Interveinal effects are frequently produced by acute fluoride fumigations (*5*). The fluoride concentration in the leaves of the plants grown in nitrogen-deficient nutrient at the onset of acute episode on the 15th day was, of course, not specifically known. However, the foliar concentrations were known to be somewhere between 211 (the 10-day concentration) and 360 parts per million (the 20-day concentration) on a dry-weight basis. The plants grown in a complete nutrient showed, by way of comparison, 183 parts of F^- per million after 10 days of fumigation and 348 parts per million after 20 days. The 10-day average fumigation concentration was 4.7 $\mu g/m^3$, and the 20-day exposure level was 6.9 $\mu g/m^3$.

It is quite significant that plants treated with formulations deficient in other nutrients, although having higher foliar fluoride concentrations (10-day foliar fluoride range, 213 to 360 parts per million; and 20-day range, 394 to 463 parts per million) than the plants grown in either the complete nutrient or in nitrogen-deficient nutrient, did not show any visible expression of foliar damage. Thus additional evidence is provided, within a single variety, that production of necrosis is not solely related to the fluoride concentration in the tissue. Fluoride susceptibility must, therefore, be biochemically associated with other metabolic processes.

Typical growth patterns and differences in fluoride response related to the absence of each of the five nutrients were readily observed. One additional characteristic growth phenomenon is obvious from Fig. 1. All plants fumigated with fluoride for 10 or 20 days exhibited a longer initial internodal growth than the control plants. This phenomenon (*6*) has been observed repeatedly in a variety of experimental exposures and conditions during the last 2 years.

Studies are being conducted to elucidate these and previously reported (*2*) nutrient-related phenomena.

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19 December 1960

Spinal Reflexes and Seizure Patterns in the Two-Toed Sloth

Abstract. In striking contrast to the seizure patterns of other animals, the electroshock seizure of the sloth consists of weak extension followed by tonic flexion and terminal clonus. A similar pattern is seen with direct spinal cord stimulation. Strychnine produces a pure flexor convulsion. In the spinal sloth, painful stimulation of one foot causes extension at some joints of that limb and strong flexion at all joints of the contralateral limb.

The maximal seizure pattern in man and most laboratory animals consists of the sequence of tonic flexion, tonic extension, and clonus (*1*). Previous investigations in our laboratory (*2*, *3*) have shown that the final determinants of the motor patterns of seizures are the reflex systems of the spinal cord. From this finding stems the prediction that the tonic components of the seizure pattern of the sloth should be in reverse sequence to those of most laboratory animals, since in this species the most powerful limb muscles are the flexors, which serve an antigravity function. Richter and Bartemeier (*4*) have shown that the decerebrated sloth assumes a posture of flexor rigidity.

Various types of seizures were investigated in the two-toed sloth (*Choloepus hofmanni*). Of four animals obtained (*5*), only two (*1* and *2* of Fig. 1) were in satisfactory condition for investigation of seizure patterns. These sloths were each studied for a period of about 2 weeks; they ate well (citrus fruits, bananas, and green vegetables) and remained in good condition during that time.

Fifteen electroshock seizures, elicited by stimuli of varying parameters from an a-c electroshock apparatus (*6*), were observed. Current was delivered through clips attached to the eyelids. In both sloths the motor patterns following supramaximal brain stimulation (0.5 amp, 1.0 sec) consisted of weak extension (Fig. 1, *1b*), followed by rigid tonic flexion of limbs, trunk, and neck (Fig. 1, *1c*). Terminal clonus, principally of the claws and of the jaw, persisted for about 1 minute after relaxation from flexion. The seizure was followed by a period of profound postictal depression. Although the seizures produced by supramaximal brain stimulation were of identical pattern in the two sloths, the times to onset and termination of the flexor phase differed: average times from the end of brain stimulation to the beginning and to the end of flexion, were, respectively, 11 and 35 seconds for sloth 1, and 5 and 27 seconds for sloth 2.

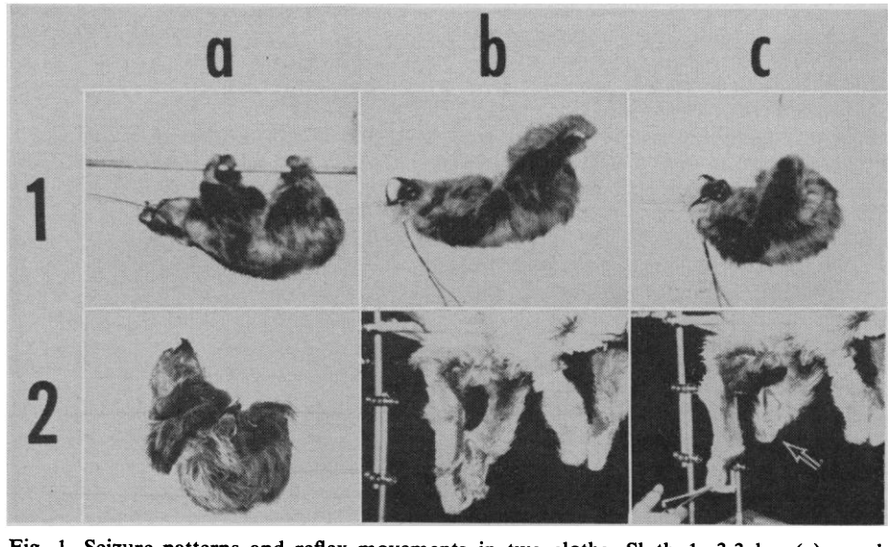

Fig. 1. Seizure patterns and reflex movements in two sloths. Sloth 1, 3.3 kg; (*a*) usual position of animal hanging on bar (restraining noose around neck); (*b*) weak extension 3 seconds after supramaximal brain stimulation; (*c*) tonic flexion 25 seconds after supramaximal brain stimulation; (*b*) and (*c*) are from a motion picture record of one convulsion. Sloth 2, 4.5 kg; (*a*) tonic flexion produced by intracardiac administration of 1 mg/kg of strychnine; (*b*) and (*c*) sloth suspended in a harness following spinal transection; (*b*) normal position hanging in harness; (*c*) reflex position assumed during pinching of right footpad. Note strong flexion of limb and claws on the left side, identified by the arrow. Extension of the foot and of the claws can be seen on the right side.