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.

D. F. ADAMS
C. W. SULZBACH
Division of Industrial Research, Washington State University, Pullman

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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.

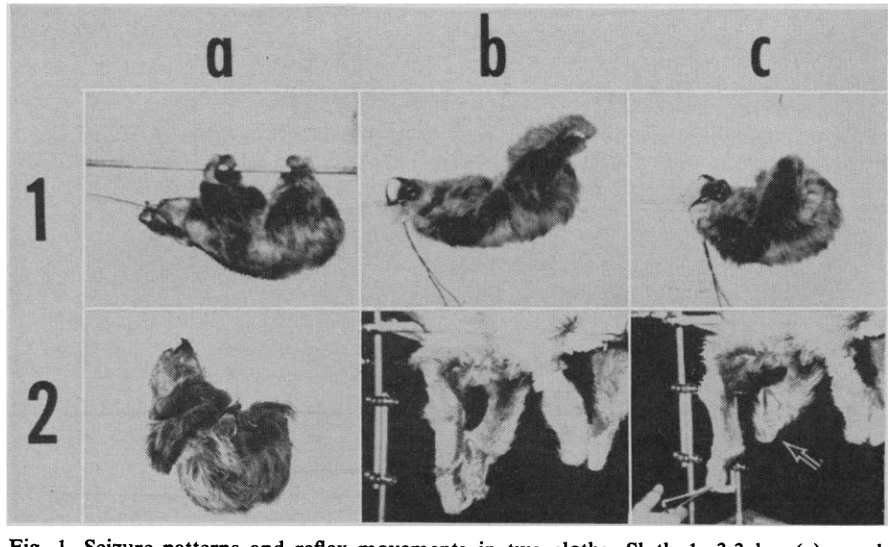

Fig. 1. Seizure patterns and reflex movements in two sloths. Sloth 1, 3.3 kg; (*a*) usual position of animal hanging on bar (restraining noose around neck); (*b*) weak extension 3 seconds after supramaximal brain stimulation; (*c*) tonic flexion 25 seconds after supramaximal brain stimulation; (*b*) 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.

The phase which precedes tonic flexion appears to be weak extension; however, there was little extensor tone, and the limbs could be manually flexed. That this phase represents a decrease in normal flexor tone is demonstrated by the fact that the animal lost its grasp if the shock was applied while it was hanging on a bar.

In seizures elicited with submaximal current, or in seizures elicited with supramaximal current during the period of postictal depression, tonic flexion was absent or greatly reduced. Thus, decrease in flexion in the sloth parallels the decrease in tonic extension seen in other animals under such circumstances (*1*).

Intracardiac injection of strychnine (1 mg/kg) rapidly produced a pure tonic flexor convulsion (Fig. 1, *2a*). This seizure is in contrast to the strychnine convulsions in most animals in which pure extension is seen. In a more fundamental sense, however, the strychnine seizure of the sloth is comparable to that seen in other animals in that the pattern of the seizure is determined solely by the most powerful muscles. Administration of pentobarbital (35 mg/kg) produced relaxation from tonic flexion and caused the appearance of flexor jerks.

Four days after the strychnine convulsion, the spinal cord was transected at the atlanto-occipital junction, and the cord was electrically stimulated by means of a needle inserted in the cord from about C_1 to C_4. The technique employed has been described previously (*3*). Spinal cord stimulation duplicated all the motor patterns seen during electroshock convulsions in the intact sloth. Single stimuli produced flexor thrusts. Stimulation at frequencies of 2 to 6 pulses per second resulted in alternating hindlimb movements which resembled the normal (upside-down) mode of locomotion of the sloth. Walking movements also are seen in the spinal cat over this frequency range (*3*). High-frequency stimulation (100 to 300 pulses per second) produced convulsions which contained all the components of the seizure produced by supramaximal brain stimulation. At a frequency of 300 pulses per second the onset of the flexor phase was at 4 seconds in sloth 2, only slightly less than the time to onset of flexion in the maximal electroshock seizure in this animal.

After spinal cord section in sloth 2, and just prior to cord stimulation, various reflexes of the hindlimbs were examined. Tapping the superficial tendons, the majority of which subserve flexor muscles, elicited brisk stretch reflexes. The most unusual reflex observed in this spinal sloth was that associated with painful stimulation of the footpad. In species previously investigated (*7*), this reflex consists of withdrawal of the limb to which the painful stimulus is applied and extension of the opposite limb. In the sloth the response to pinching of the footpad is strong flexion of all joints in the contralateral limb (Fig. 1, *2c*). Slight flexion at the knee and marked extension of the foot and claws are observed on the side stimulated. While this reflex is opposite in direction to that which has been observed in other animals, it would appear to serve the same functions during normal locomotion, namely, to avoid the painful stimulus with one limb and to support the body with the opposite limb (*8*).

DON W. ESPLIN
DIXON M. WOODBURY
Department of Pharmacology, University of Utah College of Medicine, Salt Lake City

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Haptoglobin Types in Poland

Abstract. The frequency of the Hp^1 gene in Polish subjects is 0.36. This frequency is lower than that in Western European populations and higher than that in Asiatic populations. We suggest that the increase in frequency of this gene from East to West has a regular continuous character that may be attributed to a still-unclear genetic mechanism.

Distribution in different populations of the haptoglobin types and the genes controlling their inheritance is of great anthropological interest. The frequency of the Hp^1 gene is high among African Negroes and low among Asiatic people; in European populations so far tested it ranges from 0.34 to 0.47 (the reported data are collected in the paper of Sutton *et al.*, *1*). Up to now no data have been available for any of the Slavonic populations. The present report gives the results of haptoglobin type determinations of 208 Polish subjects (unselected blood donors from Warsaw).

Table 1. Haptoglobin types in 208 Polish subjects.

Haptoglobin type	Sex (No.)		Total (No.)
	M	F	
1–1	21	2	23
2–1	75	29	104
2–2	55	26	81
Total	151	57	208

Sera containing 300 mg of hemoglobin per 100 ml were assayed by vertical starch gel electrophoresis (*2*) with subsequent staining of gels with *o*-tolidine (*3*). The results obtained are shown in Table 1. Ahaptoglobinemia was not found in any case. The Hp^1 frequency computed from these data was 0.3606.

The Polish population is characterized by a relatively low frequency of Hp^1 allele which is lower than that found in Western European populations (*1*) and close to those occurring in Norwegian (*4*) and Finnish (*5*) populations; the distribution of all three haptoglobin types in these populations is statistically indistinguishable (as proved with the chi-square test). However, haptoglobin type 1-1 occurs in 11.1 percent of Poles, 13.2 percent of Norwegians, and 14.5 percent of Finns; haptoglobin type 2-2 occurs in 38.9, 40.6, and 42.0 percent, respectively. This might correspond to a regular lowering of the Hp^1 gene frequency from the west to the east of Europe—where the only exceptions are found in some parts of Italy (*6*) and Germany (*7*)—and might be due to a historically earlier mixing of haptoglobin genes in the Polish population or to other genetic mechanisms. More detailed study of the distribution of haptoglobin types is needed to clear up this problem.

K. MURAWSKI
T. MISZCZAK
Department of Biochemistry, Institute of Hematology, Warsaw, Poland

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