integration into brainstem neural networks by the 2nd week of postnatal life, the bladder is not capable of organized reflex response if these central pathways are acutely interrupted.

WILLIAM E. BRADLEY
FRANCIS S. WRIGHT
Division of Neurology, University of Minnesota Hospitals, Minneapolis

References and Notes

1. M. Kuru, *Physiol. Rev.* **45**, 425 (1965).
2. F. J. F. Barrington, *Brain* **44**, 23 (1921).
3. Crown-rump length was not measured. This may be of value in subsequent interpretation of data on the development of the micturition reflex.
4. Model 600-1200, Harvard Apparatus Co., Dover, Mass.
5. The frequently increased amplitude of the reflex detrusor contraction after transection of the spinal cord may or may not represent release from cephalad inhibition.
6. S. Skoglund, *Acta Physiol. Scand.* **49**, 299 (1960); *ibid.*, p. 318.
7. T. C. Ruch, in *Handbook of Physiology*, J. Field, Ed. (Williams & Wilkins, Baltimore, 1960), p. 1207.
8. Supported by PHS grants NB 05242 and NB 05319.

7 February 1966

Naphthaleneacetic Acid: Localization in the Abscission Zone of the Bean

Abstract. *When ^{14}C- naphthaleneacetic acid, labeled in the ring, was applied to the petiolar stub of debladed bean (*Phaseolus vulgaris *L.) plants, it accumulated in or between cell walls of tissue immediately adjacent and distal to the abscission layer. There was no localization in the abscission layer per se. Similar distribution patterns were observed in the abscission zone after naphthaleneacetic acid applications, which either delayed (10^{-3}M) or accelerated (10^{-5}M) abscission.*

It is generally accepted that of the many factors influencing abscission, auxin appears most important. Two theories have been advanced to explain the action of auxin in abscission. Addicott *et al.* (*1*, *2*) suggested that a gradient from relatively high auxin, distal to the abscission layer, to relatively low auxin, proximal to the abscission layer, is necessary to prevent abscission. A lowering of this gradient initiates or accelerates abscission. Gaur and Leopold (*3*), however, proposed that the total quantity of auxin applied was the controlling factor, and not the gradient. Using naphthaleneacetic acid (NAA), Biggs and Leopold (*4*) demonstrated a two-phase action: low concentrations accelerated, and high concentrations delayed, abscission. Significantly, they concluded that the primary action of auxin was directly on the abscission zone. Our experiments provide a test for both the auxin gradient and concentration theories and establish the localization of NAA in the abscission zone.

Bean (*Phaseolus vulgaris* L. cv 'Contender') seedlings, started in sand, were placed in nutrient solution and transferred to a controlled environment at 25°C and a light intensity of 4400 lu/m^2 (fluorescent, cool white). The seedlings were debladed, and approximately 15 mg of a 10^{-3} or $10^{-5}M$ lanolin emulsion of ring-labeled (*5*) naphthaleneacetic acid (^{14}C-NAA) was applied to the 1-cm petiolar stub.

Two plants were harvested from each concentration daily from 2 to 6 days (inclusive) after treatment, and a tissue explant containing the lower pulvinus was removed from the treated petiole. The tissues were quickly frozen on a Freon block, sectioned (58 μ) with a cryostat, and affixed with adhesive onto glass slides. After the slides were dried

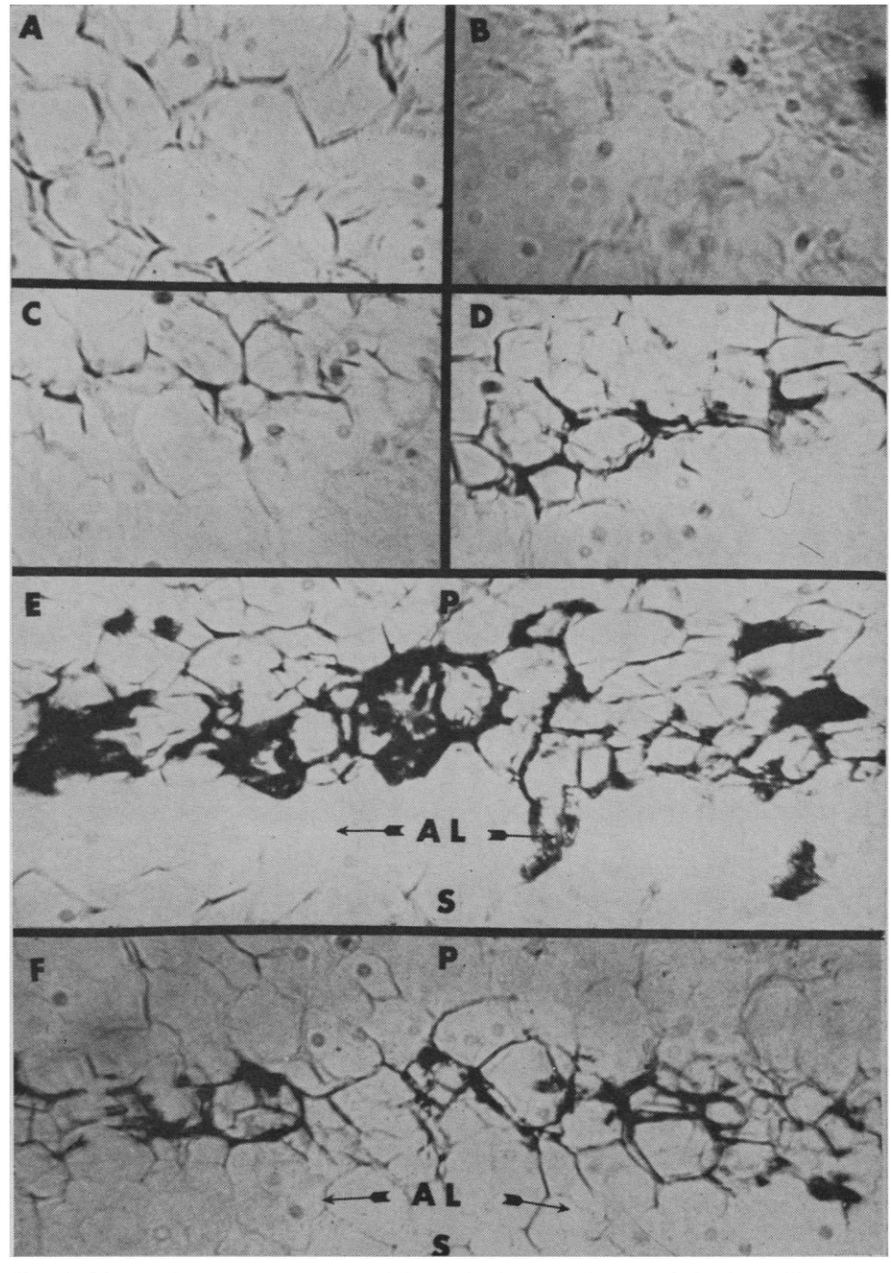

Fig. 1. Microradioautograms prepared from the lower pulvinus of the bean illustrating the localization of ^{14}C-NAA: (*A*) 2 days, (*B*) 3 days, (*C*) 4 days, (*D*) 5 days, and (*E*) 6 days after deblading at $10^{-3}M$ NAA; (*F*) application of $10^{-5}M$ NAA 6 days after deblading. *P*, petiole; *AL*, abscission layer; *S*, stem (× 200).

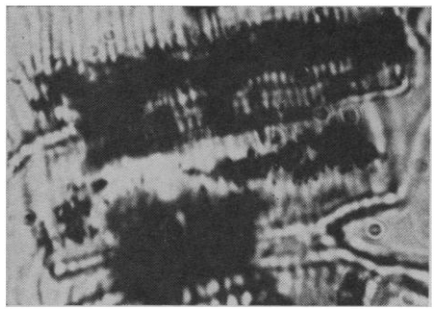

Fig. 2. Microradioautogram of ^{14}C-NAA in the xylem tissue of a petiole 2 days after the tissue was debladed and $10^{-3}M$ ^{14}C-NAA was applied to the petiolar stub ($\times$ 400).

in air, liquid emulsion (Type NTB-2, Eastman Kodak) was spread evenly over the sections, dried, and exposed in a volatile-free box containing $CaCl_2$. The microradioautographs were developed, and the sections were stained with Azure B.

The distribution of ^{14}C-NAA in the lower pulvinus during development of the abscission layer is depicted in the microradioautograms in Fig. 1, *A* through *E*. The abscission layer was first evident 3 days after application of $10^{-3}M$ ^{14}C-NAA to the petiolar stump. The first localization of labeled NAA occurred at this time and became more pronounced at 4 days (Fig. 1, *B* and *C*). By the 5th and 6th day the abscission layer was readily evident, and considerable radioactivity was localized in or between the cell walls of tissue immediately adjacent and distal to the abscission layer (Fig. 1, *D* and *E*). There was no localization of ^{14}C-NAA in the abscission layer (Fig. 1*E*).

Conductive tissue appeared to be a pathway for basipetal movement, since numerous xylem elements were heavily labeled (Fig. 2).

When ^{14}C-NAA was applied at $10^{-5}M$, the abscission layer was well-defined 2 days after treatment. Abscission of the petiole was promoted at this concentration, whereas at $10^{-3}M$ NAA abscission was delayed. No labeled NAA accumulated in the abscission layer per se, but rather in tissue adjacent and distal to the abscission layer (Fig. 1*F*).

We interpret the absence of ^{14}C-NAA localization in the abscission layer to mean that the abscission layer is not the primary locus of NAA action. More likely, NAA influences abscission indirectly through tissue distal to the abscission layer.

Another possible interpretation is that the abscission layer forms a physical barrier resulting in accumulation distal to it. We have found that basipetal movement of phosphorus and ribidium through the abscission zone increased after the 3rd day (following deblading); however, calcium decreased (*6*). Further, if ^{14}C-NAA localization was related solely to the presence of the abscission layer, then accumulation would be expected to occur earlier at $10^{-5}M$ than $10^{-3}M$, and this was not the case.

Of further significance is the similarity of ^{14}C-NAA distribution patterns (Fig. 1, *E* and *F*) observed subsequent to treatment at concentrations known (*3*, *4*) to delay ($10^{-3}M$) and accelerate ($10^{-5}M$) abscission. In these studies (data not reported) $10^{-3}M$ NAA delayed abscission by 3 days and $10^{-5}M$ accelerated abscission by 2 days as compared to a lanolin control. At $10^{-3}M$ both the auxin gradient and concentration should favor a delay in abscission. Presumably, also at $10^{-5}M$ the auxin gradient and concentration would be expected to delay abscission, but abscission was accelerated. Thus, our data do not support either the gradient or concentration concept as an explanation for auxin control of abscission, but do confirm the absence of a correlation between distally applied NAA detectable on the two sides of the abscission layer and the abscission process (*7*).

Addicott (*2*) visualizes the acceleration of abscission by low concentrations of NAA as an antagonism to auxin whereby the effective concentrations of endogenous auxin are lowered. Rubinstein and Leopold (*7*) and Rasmussen (*6*) have found an induction period during which auxin delayed abscission, but after its completion accelerated abscission. In view of these findings, another explanation may be that the auxin optima of the two processes are such that the effect of low auxin is mainly on the postinductive process, that is, the induction period is not delayed but the postinduction period is accelerated, as suggested by Rubinstein and Leopold.

H. P. RASMUSSEN*
M. J. BUKOVAC
Department of Horticulture, Michigan State University, East Lansing 48823

References and Notes

1. F. T. Addicott, R. S. Lynch, H. R. Carns, *Science* **121**, 644 (1955).
2. F. T. Addicott, *Encycl. Plant Physiol.* **15**(2), 1094 (1965).
3. B. K. Gaur and A. C. Leopold, *Plant Physiol.* **30**, 487 (1955).
4. R. H. Biggs and A. C. Leopold, *Amer. J. Bot.* **45**, 547 (1958).
5. Specific activity, 1.25 mc/mmole.
6. H. P. Rasmussen, thesis, Michigan State University, East Lansing (1965).
7. B. Rubinstein and A. C. Leopold, *Plant Physiol.* **38**, 262 (1963).
8. Journal article No. 3744 from the Michigan Agricultural Experiment Station, East Lansing.

* Present address: Department of Soils and Climatology, Connecticut Agricultural Experiment Station, New Haven.

5 November 1965

Scolytid Beetles Associated with Douglas Fir: Response to Terpenes

Abstract. *Douglas-fir oleoresin and the terpene hydrocarbons (α-pinene, β-pinene, limonene, camphene, geraniol, and α-terpineol) attracted various bark and timber beetles associated with Douglas-fir forests during their flight. In responding to these volatile terpenes the bettles are directed to favorable breeding material.*

Several species of bark and timber beetles (family Scolytidae) select and invade physiologically weakened or damaged or felled trees shortly after flight begins in the spring. Bark beetles that prefer freshly cut trees invade them virtually within minutes of cutting (*1*). How do the first invading or pioneer beetles find their host and what mechanism guides them to it? Because there is often no discernible difference in shape or color between vigorous and weakened trees or between freshly cut and old logs, it may be assumed that what attracts the pioneer beetles is olfactory in nature and is effected by some substance or substances of the host tree. From mechanically damaged or freshly cut Douglas fir, exuding oleoresin contacts the air at once. In fact, response of *Dendroctonus valens* Lec. to oleoresin of ponderosa pine has been observed in the field (*2*), and in the laboratory various bark beetles, associated with pine and spruce forests, were attracted by very low concentrations of α-pinene, β-pinene, limonene, and α-terpineol (*3*).

Various solutions of Douglas-fir oleoresin and of its various fractions were tested amid stands of Douglas fir, *Pseudotsuga menziesii* (Mirb.)