Fig. 2. Ultraviolet spectra of Perkin's original sample of alizarin (A) and a sample of alizarin synthesized and purified in 1969 (B). The solvent was water, pH 11.7.

had serious repercussions on the madder growing industry, resulting in bankruptcy for most of the growers. In some quarters, however, it was considered that the advent of artificial alizarin brought many benefits, including "vast tracts of land formerly used for the cultivation of madder thus made available for the production of the necessities of life" (Knecht, Rawson and Loewenthal, *Manual of Dyeing*, 2, 571; Griffin, London, 1910).

The Science Museum in London also has a sample of Perkin's artificial alizarin, and Gurr has recorded its absorption spectrum too. Although the first record of its spectral curve (in dilute aqueous sodium hydroxide) has already been published (Gurr, *J. Soc. Dyers and Colourists*, 85, 473; 1969), Fig. 2 is new; it compares the spectral curves of Perkin's original specimen in the ultraviolet region with a pure specimen of alizarin produced recently. Gurr found that colour changes corresponding to different pH values were the same for both old and new specimens.

AUXIN

Transport by Carriers

from our Plant Physiology Correspondent

A NEW and provocative hypothesis to explain the transport of auxins in stems has been put forward by D. J. Osborne and M. G. Mullins (*New Phytol.*, 68, 977; 1969). The theory is highly speculative, but it provides rather neat solutions to several of the more intriguing problems of auxin physiology—for example, the polarity of auxin movement, and the effect of ethylene gas on the transport process.

Most research on the transport of auxins, the plant hormones which regulate, among other things, stem extension, has involved cutting short segments of either stem or leaf stalk (known as the petiole), applying an agar block containing auxin to one end, and catching auxin transported through the segment in a plain agar block at the other end. More than forty years ago, F. W. Went demonstrated that the movement of auxin in this system is in one direction only, towards the morphological base of the stem or petiole. Later work showed that this movement is much too rapid to be attributable to simple diffusion, that it depends both on metabolic energy and temperature, and that it is confined chiefly to those tissues which have the capacity for extension growth. Nobody, however, has yet provided a satisfactory explanation for the underlying mechanism.

The starting point in the new theory is the observa-

tion that segments of bean petiole rapidly lose the ability to transport auxin after removal from the plant. If, however, auxin or kinetin, a hormone analogue which stimulates protein synthesis, is supplied to the apical end of the segment immediately after cutting, a normal rate of transport can be maintained for many hours. Ethylene gas, which is exciting interest as a possible natural plant growth regulator, is also known to reduce the capacity of freshly cut segments to transport auxin, but again this effect can be completely nullified by supplying auxin to the freshly cut tissues. Kinetin, however, is only partially effective in protecting the tissue against ethylene.

Osborne and Mullins have combined these diverse effects in a unified concept by drawing an analogy with animal hormone systems, where it is known that specific carrier proteins are implicated in hormone movement. They suggest that such a protein, perhaps a transpermease, is operative in hormone transport in plants. Polarity of movement could then be explained by a greater concentration of this carrier at one end of the cell than at the other. This asymmetry need be quite small; Hertel and Leopold pointed out that minute differences in the amounts of auxin moved through each end of a cell, when multiplied over a large number of cells, would be realized as a significant polarity (*Planta*, 59, 535; 1963). Osborne and Mullins propose therefore that for an auxin molecule to be transported across a cell membrane, it must be bound to the carrier at a specific site. The carrier, however, has a second active site which can bind ethylene. When one of these sites is occupied, the other site is blocked; in other words, the carrier could be an allosteric protein.

In natural conditions there would be a continuous supply of auxin to the carrier molecules from the apical regions of the plant, which would maintain the transport process, but if a segment was cut from the plant this source of auxin would be lost. Consequently the auxin sites on the carrier molecule would gradually become vacant, and ethylene molecules could then become bound to the carrier, resulting eventually in a loss of capacity to transport auxin. In this respect it is interesting that wounding is known to stimulate the production of ethylene. If, on the other hand, auxin is applied artificially to the segment immediately after cutting, the supply of auxin to the carrier continues unabated and the transport system is effectively protected. The influence of kinetin in maintaining transport seems to be quite different from that of auxin, for it confers no protection against ethylene. It seems possible that kinetin stimulates overall protein synthesis, and so enhances the production of new carrier protein.

The scheme is not without its shortcomings; there is no explanation of why such molecules as triiodobenzoic acid and naphthylphthalamic acid are such potent inhibitors of polar auxin transport, and similarly there is no real evidence either for the presence of the proposed carrier molecule, or for the asymmetrical distribution of such a molecule in the cell. And, of course, there are those who wonder how relevant experiments such as these are to processes actually occurring in plant tissues. Nevertheless, the scheme is ingenious, and should provide a basis for further investigation into a process which lends itself easily to speculation, but which equally strongly resists experimental evaluation.