We became interested in CBT after having become aware of a paper by Tognoli *et al.* that reported the inhibition of *in vitro* fusicoccin-binding by this toxin. In fact, the limited available information about the molecular features of CBT was sufficient to raise doubts about the possibility of their recognition by the fusicoccin receptor.

We have now tested several samples of CBT (obtained through the courtesy of Proff. Nasini and Assante) and have never observed a decrease of fusicoccin binding to microsomal preparations of maize or spinach tissues. Furthermore, we have been unable to duplicate the inhibition of H^+-excretion observed by Macrì *et al.*, which instead was slightly stimulated by CBT ($10^{-4} - 10^{-3}$ M). The same samples of CBT, as expected, were highly effective in inhibiting H^+-transport across microsomal vesicles of maize roots; also their phosphohydrolytic activity was decreased by the toxin. We suspect that the older sample of CBT used by Macrì *et al.* and by Tognoli *et al.* contained some contaminant compound capable of binding to the fusicoccin receptor and of inhibiting H^+-excretion from plant tissues which was eliminated during the further purification of the toxin.

This work was supported by the Italian Ministry of Agriculture and Forestry, by the Italian Ministry of University and Scientific and Technological Research, and by the Italian Research Council (CNR) - Progetto Finalizzato «Chimica Fine II».

EFFECTS OF FUSARIC ACID IN *ELODEA DENSA* LEAVES

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The mechanism of action of fusaric acid (FA) in *Elodea densa* leaves has been investigated by measuring the effects of FA on the transmembrane electrical potential difference (Em), on H^+ extrusion, on electrolyte leakage and on cell morphology.

The effects of pH of the medium on FA uptake by the leaves has also been determined. The results show that at concentration between 10^{-4} and 3×10^{-4} M FA induces a rapid, initial hyperpolarization, followed by a depolarization of Em, which increases with the increase of FA concentration, and an alkalinization of the medium associated with an increase of electrolyte leakage.

FA uptake by the leaves was markedly decreased by increasing the pH of the medium from 5 to 7, indicating that only the uncharged form permeates the cells and accumulates in the cytoplasm, in agreement with the general behaviour of weak acids (pK of FA = 3.59).

At concentrations 1 mM and higher FA induced marked morphological changes, including the disconnection of the plasmalemma from the cell wall.

These results indicate two different components in FA action: a first one depends on its nature of weak acid, and is responsible of the initial hyperpolarization of Em and of intracellular acidification, and a second one, more toxic, due to the interaction with specific cell systems, and leading to Em depolarization and to serious damage of both metabolism and membrane functions.