BIOCHEMICAL JOURNAL LETTERS

On the action of ribosome-inactivating proteins: are plant ribosomes species-specific?

Several plant proteins are known that inactivate eukaryotic ribosomes in a catalytic (enzymic) manner, rendering the 60S ribosomal subunit incapable of binding the elongation factor 2, and thus arresting protein synthesis. Some of these proteins consist of an A (active) chain joined to a B (binding) chain to constitute toxins [ricin, abrin (Olsnes & Pihl, 1977), modeccin (Olsnes *et al.*, 1978; Gasperi-Campani *et al.*, 1978) and presumably *Viscum album* (mistletoe) toxin (Stirpe *et al.*, 1980b)] whilst other, and more numerous, are single, A-chain-like proteins [the pokeweed antiviral protein ('PAP') from the leaves of *Phytolacca americana* (Obrig *et al.*, 1973), tritin from wheat germ (Roberts & Stewart, 1979), an inhibitor from *Momordica charantia* seeds (Barbieri *et al.*, 1980), gelonin from the seeds of *Gelonium multiflorum* (Stirpe *et al.*, 1980a), dianthins from *Dianthus caryophyllus* (carnation) leaves (Stirpe *et al.*, 1981)]. There is evidence that similar proteins may be present in many other plants (Gasperi-Campani *et al.*, 1977, 1980), and indeed they were identified, in variable concentrations, in almost all plant materials examined with a more refined procedure (L. Barbieri, M. G. Battelli, A. Gasperi-Campani & F. Stirpe, unpublished work).

The pokeweed antiviral protein, the first to be known, actually was identified and purified as a factor preventing infection of plants by tobacco-mosaic virus (Wyatt & Shepherd, 1969), and when its effect on ribosomes was demonstrated (Obrig *et al.*, 1973) it was shown that this is the mechanism whereby it inhibits viral replication (Owens *et al.*, 1973).

It has been shown recently that all the ribosome-inactivating proteins tested, either complete toxins or single-chain ones, prevent local lesions by tobacco-mosaic virus (Stevens *et al.*, 1981). Thus they seem identical with at least some of the factors responsible for the antiviral activity of many plant extracts (Bawden, 1954); this was demonstrated for carnation leaves with the identification of dianthins, which have both the ribosome-inactivating and the antiviral activity (Stirpe *et al.*, 1981).

It has been known for many years (Duggar & Armstrong, 1925) that several plant extracts have antiviral properties, but prevent viral infection only in heterologous plant species, with little effect on infection of the plants they come from. This led to the conclusion that the active principles do not act on viruses, but rather on the host plant itself (Gendron & Kassanis, 1954; Bawden, 1954). If the antiviral activity of these factors is due to their effect on ribosomes, this implies that they must act only on ribosomes from other plant species; indeed, the pokeweed antiviral protein inactivates ribosomes from wheat germ and from cowpea, but not those from pokeweed (Owens *et al.*, 1973), whilst tritin does not affect wheat germ ribosomes (Coleman & Roberts, 1981). Further, the fact that ricin is 100–1000-fold more active on mammalian than on wheat-germ ribosomes (Cawley *et al.*, 1977, 1979) is presumably a reflection of the evolutionary distances which separate these ribosomes from those of the castor bean. This, and the widespread distribution of ribosome-inactivating proteins in plants, strongly suggests (i) that ribosomes must differ between plants, possibly with species specificity, as postulated already for other reasons (reviewed by Ciferri & Parisi, 1970), and (ii) that many, and probably all, plants possess proteins capable of recognizing ribosomes different from their own and of inactivating them through a still unknown mechanism and for as yet unknown reasons.

Fiorenzo STIRPE
*Istituto di Patologia generale
dell'Università di Bologna,
I-40126 Bologna, Italy*

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