trapping mechanism is unusual, nevertheless the anomaly suggests the possibility that a meteroid impact or major volcanic event has been a contributing cause to the world-wide extinction[18]. There are still problems to be settled, however, particularly in regard to the synchroneity of the event in terms of conodont zonation.

(3) The Hangenberg event, towards the close of the Famennian, was another euxinic black shale event in Europe that caused a relatively extended decline and extinctions which included the end of the peculiar Devonian ammonoid group, the clymeniids. The coral and brachiopod faunas of the late Famennian are nevertheless more closely related to succeeding Carboniferous forms than to any previously existing in the Frasnian.

While House[9] suggests that the events he describes may have a eustatic or euxinic immediate cause, he is prepared to speculate on the possibility of various ultimate causes. Recently he has published on Devonian eustatic events, and has attempted to recognize rhythms and cycles in the New York Devonian and their correlates across the Atlantic, based on sedimentological and biostratigraphical data[16]. In the current paper, he finds that facies microrhythms may, in some instances, be correlated with Milankovitch cycles of the order of 100 Kyr. Other possibilities, including climatic change and carbon variations, would seem to make a simple rhythmic interpretation for all the observed events unlikely. Nor is it easy to decide if larger-scale periods are involved. At least some of the events described by House do not seem to be regularly spaced and there is variation in the magnitude between them. He notes recent ideas linking periodicity in extinctions with rhythmic episodes of enhanced meteoroid or cometary impact, but he concludes that, at least in the Devonian and Carboniferous, rhythms and events did not exhibit periodicity. His evidence suggests a more complex scenario and he concludes that thorough investigation of the type of events he describes might lead to an accurate chronology and ultimately to an objective global time scale. □

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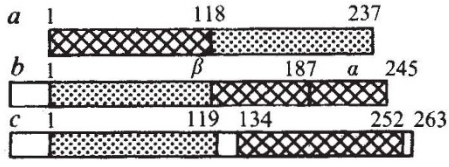
Plant biochemistry

Seed protein construction

from John A. Gatehouse and Donald Boulter

PRODUCTION of seed proteins by the enzymatic cleavage of precursor molecules into their component parts is common place. But on page 64 of this issue, D.M. Carrington, A. Auffret and D.E. Hanke make the provocative suggestion that concanavalin A (con A), a seed protein of jackbean (*Canavalia ensiformis*), is produced by the rejoining, in transposed order, of the two main cleavage products of its precursor[1].

Con A belongs to the class of carbohydrate-binding proteins known as lectins. These are specifically accumulated in the seeds of many plants, although usually to a lesser extent than the storage proteins, and may function in the defence of seeds against insects and other predators. Lectins have been purified from a range of legume seeds and a considerable body of data has shown that the lectins of pea, broad bean, lentil and related species have highly related amino acid sequences. The sequence of con A is also related if a circular permutation of sections of its sequence is taken into account[2,3]. Specifically, pea lectin is synthesized as a precursor containing a leader sequence, followed by its 187-amino acid β subunit and then its 58-amino acid α subunit[4], whilst con A is a tetramer of a 237-amino acid polypeptide, of which amino acids 1–118 are homologous to 105–240 of pea lectin, and amino acids 119–237 are homologous to 1–105 of pea lectin (see figure).



Schematic structure of con A (a), pea lectin (b) and the suggested con A precursor (c). Corresponding regions are in the same shade. Numbers refer to amino acids.

Enzymatic cleavage of con A between amino acids 118 and 119 (ref.5), and possibly at other positions too[6], has seemed to be a slow, but natural, modification, accounting for the frequent isolation of the two fragments of the lectin. But Carrington *et al.* now present evidence that the cleaved fragments are, in fact, precursors to a 237-amino acid protein that is formed by polypeptide ligation. (The isolation of fragments is thus ascribed to incomplete ligation). This interpretation is based on the isolation of a jack bean seed cDNA (produced from mRNA) that seems to contain the complete con A coding sequence but rearranged in a way that makes it similar to the sequence of pea lectin (see figure). Specifically, the cDNA encodes a 29-amino acid leader sequence, followed by amino acids 119–237 of con A, a 15-amino acid 'linker' peptide, amino acids 1–118 of con A, and a 9-amino acid carboxy-terminal peptide. To produce concanavalin A from this precursor would require at least four proteolytic cleavages and the ligation of the 1–118 fragment to the 119–237 fragment. At least one further proteolytic step must be involved, since a form of con A isolated from immature seeds contains an extra four amino acids corresponding to the last few of the linker fragment at its amino-terminus. In addition, the linker fragment carries a glycosylation site, so that the precursor is probably glycosylated whereas con A itself is not.

Most of this sequence of events is not unreasonable when compared to the synthesis of other seed proteins, particularly since, as with other legume seed proteins[7], all the proteolytic cleavages (except that of the leader sequence) occur on the carboxy-terminal side of asparagine. But the postulated final ligation step is unprecedented and should be treated with caution. Is it possible that Carrington *et al.* have sequenced a cDNA corresponding not to con A but to a related protein or to a precursor that gives rise to the 'fragmented' subunits seen *in vivo*, whereas another cDNA encodes the 'intact' subunits? The authors think not, since they claim that only one con A gene per genome can be detected with their cDNA, although complete evidence is not presented. They also claim that the precursor form of con A predicted by the cDNA sequence is detected *in vivo* as a polypeptide antigenically-related to con A, and can be shown to give rise to all the con A polypeptides seen *in vivo*. Again, however, they provide only incomplete evidence (pulse-chase data) that this is the case. Furthermore, the possible presence of other sites of proteolytic cleavage is not considered.

Finally, one must ask why such a perverse synthetic route should be used. And if it is used, what are the characteristics of the ligation enzyme? One suspects this system still has a few tricks up its sleeve. □

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