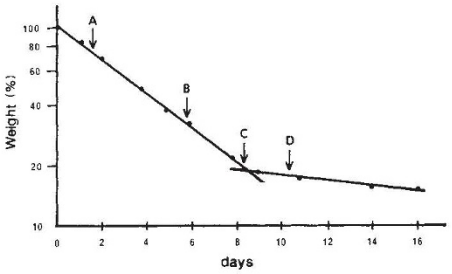

Two stages of moisture evaporation in boiled egg white kept in an ordinary refrigerator and the state of its transparency. Ordinate, weight reduction of boiled egg white. The thin edges of boiled egg white became hard and transparent (vitrified) after the first day in a refrigerator, as shown by arrow A. After 6 days (arrow B) most of the boiled egg white had become hard and transparent. At arrow C (8 days) the evaporation entered the second stage. And finally, after 10 days (arrow D), all the egg white was hard and transparent.

and neglecting the rest we estimated the average molecular mass to be 51,000. The content of the free and bound water was estimated to be 1,900 and 19,000 moles per mole of protein, respectively, in the egg white. In other words, one gram of the protein contained 0.7 gram bound water and 7 gram free water. Assuming that the average molecular mass of amino acids is 110, it can be calculated that one amino acid contains 4 and 40 moles of the bound and free water, respectively.

The vitrified egg white was a plastic pale yellow solid. The plasticity could be due to the increased entanglement of the denatured protein molecules. Vitrified raw egg white, on the other hand, is fine and forms thin fragments. X-ray diffraction of vitrified egg white reveals haloes characteristic of the amorphism seen in common glass, and the pore radius in the protein cluster was 6 angstroms. Neither the egg white nor fish eyeballs become putrified.

In the field of food sciences, it is very common to get dry protein product from its moistured material for preservation, as raw or processed products with high moisture are generally considered to putrify readily. As eggs are so common, it would be unusual and unnecessary to dry boiled eggs for preservation. Vitrification of denatured boiled egg white could therefore have been overlooked as a source of edible protein.

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Cautious searches

SIR—Skern *et al.*[1] are correct in stating that the results of computer searches for similarity between proteins must be interpreted with caution, but our paper[2] on the maize bifunctional inhibitor of α-amylase/trypsin merely suggested that both thaumatin and the tobacco pathogenesis-related protein should be re-examined for their possible effects on hydrolytic enzymes due to their strikingly extended sequence homology (52–57 per cent over 200 residues) with the inhibitor. Moreover, the sequence similarities we reported are of much greater statistical significance[3] (Z values of 41.7 and 97.8) than those found by Skern *et al.* for the limited similarity between human rhinovirus 2 and thaumatin (Z=5.8) or trypsin (Z=5.6).

The validity of our suggestion has been supported by several recent discoveries. For example, a potent inhibitor of locust gut α-amylase isolated from seeds of *Coix lachryma-jobi* is, in fact, also an endochitinase[4]. We were prompted to look for the enzyme activity only when a computer search revealed that the inhibitor had a similar sequence to endochitinases. Similarly, when a putative α-amylase/protease inhibitor isolated from barley seeds[5,6] was found to have sequence similarities with phospholipid transfer proteins[7], subsequent assays confirmed that it possessed this function[8]. The further observation that an amylase inhibitor from beans is similar to lectin (agglutinin)-like proteins[9] tends to confirm the belief that computer-aided searches will continue to provide a valuable indication of the possible, but as yet unsuspected or unproven, multifunctional nature of many defensive plant proteins.

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Killer toxins

SIR—White *et al.*[1] have reported that the yeast killer toxin encoded by the *Kluyveromyces lactis* plasmid pGKL1 is not an inhibitor of adenylyl cyclase, leaving open the question of the mechanism of irreversible growth inhibition in sensitive yeast cells. Recent experience with endochitinase genes from plants and exochitinase genes from bacteria may shed some light on the mode of action of the yeast killer toxin.

Many plants have genes that encode enzymes with chitinase activity; I am most familiar with chitinase-related genes from hybrid poplars[2]. A TFASTA (ref. 3) sequence-similarity search of the GenBank 59.0 DNA (ref. 4) and PIR 21.0 protein (ref. 5) sequence databases using a chitinase-related (Win8) protein sequence (deduced from its complementary DNA sequence) revealed the previously reported global similarity to other plant chitinases and local similarity to plant chitin-binding proteins[2].

Unexpectedly, the large subunit of the yeast killer toxin from pGKL1 (ref. 6) shares a region corresponding to the chitin-binding domain with this collection of plant proteins. When I used the toxin protein itself to search the sequence databases, an additional similarity (outside the domain shared with plant chitin-binding proteins) to the exochitinase from the bacterium *Serratia marcescens* (ref. 7) uncovered was that *Serratia* chitinase has no discernable sequence identity with plant chitinases.

Because fungal cell walls are made predominantly of chitin, and plant and bacterial chitinases are effective antifungal agents[8–10], it seems reasonable to suggest that the large subunit of the *K.lactis* killer toxin arrests the growth of target yeast cells by interfering with cell-wall biosynthesis. It may be useful to test the purified toxin both for the predicted chitin-binding and chitinase activities. If the toxin has exochitinase activity, the domain in common with the chitinase from *Serratia* could be the site of glycosidic bond cleavage.

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