in SDS PAGE. Eluted monomeric peptides are inactive but reaggregate to M_r 56,000, regaining biological activity[5]. The protein expressed from the IL-16 cDNA demonstrates all the functions and chemical features of the native protein, including an identical pI, and autoaggregation into functional tetramers. Antibodies against recombinant IL-16 identify native protein of similar size in western blots and block its biological activity. This unique constellation of activities and chemical characteristics is not likely to be fortuitously shared by unrelated proteins.

Further, deletional analysis of recombinant IL-16 demonstrates that all its biological activity resides in the C-terminal 114 residues which are completely within the 130-residue published sequence[2]. Based on sequencing of a murine IL-16 clone, we do agree that IL-16 is synthesized as a precursor molecule, but we cannot confirm the M_r 42,000 size predicted by Bazan and Schall[1] by western blot analysis.

Bazan and Schall argued also that the presence of GLGF sequences in IL-16 is incompatible with its role as a secreted protein, so is evidence against a specific interaction between IL-16 and its receptor CD4. The function of the GLGF motifs in IL-16 is unclear: however their presence does not provide evidence against secretion of the C-terminal peptide as this motif is found in other secreted proteins[6]. Further, the experimental evidence for a ligand receptor-like relationship between recombinant IL-16 and CD4 is substantial, even if the GLGF sequence is involved in binding.

The structure of IL-16 and its relationship to CD4 raises interesting questions about its role as an interleukin. Its gene structure and chromosomal location are distinct from other interleukins, suggesting that it may indeed have roles other than in the immune system. The binding of IL-16 to CD4 may indicate a role for IL-16 in suppression of HIV-1 replication. Nevertheless, we believe that it is unlikely that IL-16 inhibits HIV-1 replication by competing for binding to CD4, but it may induce CD4-dependent signal-transduction events mediating repression of HIV-1 promoter activity.

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Tobacco-plant desiccation tolerance

SIR — Holmström *et al.*[1] reported the transformation of tobacco plants with a gene, *TPS1*, which enables the transgenic plants to synthesize trehalose, a disaccharide whose biophysical properties and presence in anhydrobiotic organisms suggests that trehalose produces desiccation tolerance in organisms[2].

Holmström *et al.* clearly demonstrated an increase in drought tolerance[3] in the *TPS1*-transgenic plants in that the development of drought stress in the protoplast was retarded in the transgenic lines. But their conclusion that, because trehalose concentrations in the cytosol seem too small for osmotic adjustment, stabilization of cellular enhanced structures and macromolecules by trehalose may underlie both the improved water retention and desiccation tolerance, do not seem to me to be completely justified by their data.

There is an explanation other than osmoregulation for the improved retention of water. In their figure (*a*), Holmström *et al.* showed that drying tobacco leaves display the typical water-loss pattern of detached well-hydrated leaves, that is an initial rapid time-rate of water loss (when stomata are wide open) and a late slow rate of water loss (as drought stress causes stomata to close, thereby restricting water diffusion from the leaf). Viewed in this way, their figure indicates that, relative to nontransgenic plants, transgenic plants passed into the slow water-loss phase at higher fresh weights, and thus that transgenic stomata commenced closing at milder drought stress; as a result water was retained for a longer time. During plant cultivation then, carbon dioxide supply for photosynthesis would be restricted more frequently by stomatal closure, causing slower growth in the transgenic plants, as noted by Holmström *et al.*

Further, Holmström *et al.* did not report the determination of protoplasmic drought tolerance of the tobacco lines, therefore no conclusion can yet be reached on their desiccation tolerance. The line 8 transgenic seedlings recovered from 7 hours of drying in air of 50% relative humidity (RH) (followed by 48 hours' rehydration), whereas the control plants failed to recover (figure *b* of ref. 1). However, Holmström *et al.* gave no measure of the water status of the seedlings at the end of the drying period. The best estimates for seedling water status that can be derived from their data are those of detached leaves at 6 hours' drying (25% RH) (figure *a* of ref. 1) — line 8 transgenic leaf fresh weight = about 45% of initial fresh weight and control leaf fresh weight = about 32% of initial fresh weight The protoplasmic drought tolerance of transgenic seedlings then would correspond to some leaf fresh weight less than 45% initial fresh weight and that of nontransgenic seedlings to a leaf fresh weight greater than 32% initial fresh weight. The overlap of these value-ranges permits no conclusion on the relative protoplasmic drought tolerance of the lines. Furthermore, comparison of these drought tolerances in terms of leaf water potential would be necessary, as this parameter is basic to an understanding of plants' water status in relation to air-dryness. A value below –100 MPa is needed for a plant to qualify as desiccation tolerant; the water contents reported above (30–45% fresh weight) would correspond to water potentials of the order of only –10 MPa.

Holmström *et al.*, in producing stable transgenic tobacco plants capable of synthesizing trehalose, report an exciting result. It is to be hoped that with further experimentation on the transgenic tobacco lines they will establish if or how much trehalose contributes to plant desiccation tolerance.

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Erratum

IN the Scientific Correspondence "Clutch size and malaria resistance " by A. Oppliger, P Christe & H. Richner (*Nature* **381,** 565; 1996), the figure was incorrect. The correct version is shown below.