RESUME

Memory logs

H. KUMAGAI and Y. Fukao have turned petrified trees to good use (*Geophys. Res. Lett.* **19**, 1859–1862; 1992) in reconstructing events at a now extinct volcano in central Honshu. Twenty million years ago the slopes of the volcano were violently deforested in a sequence of hot avalanches and explosions. The tree trunks, caught up in the deposits and then fossilized by silica mineralization, are so well preserved that the volcanologists were able to count and correlate tree-ring sequences from many logs and deduce a year-by-year growth pattern for the population. They could recognize at least five eruptions when some trees died while others survived, in a sequence lasting 17 years — an unprecedented time resolution for ancient volcanic activity.

Dominant domain

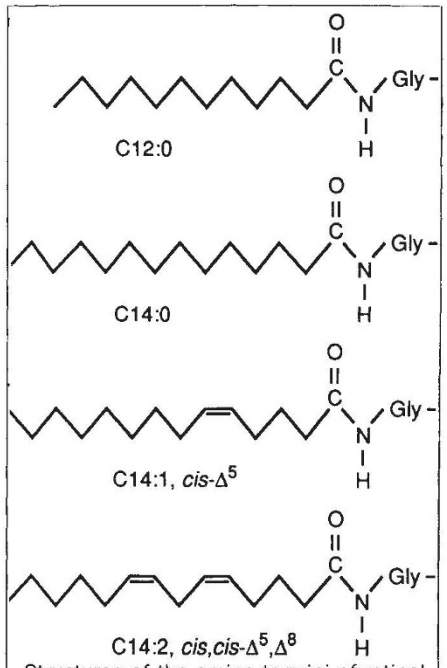
ANKYRIN is a protein that binds to a variety of other proteins — the red cell membrane anion channel and spectrin, thus attaching the membrane skeletal network to the membrane, tubulin, vimentin and the Na^+/K^+-ATPase of epithelial cells. Ankyrin contains three recognizable domains, one of which regulates the others. In red cells the protein occurs as a set of isotypes, some of which at least are splicing variants; in one the regulatory domain is deleted, and binding to other proteins is thereby enhanced. L. Davis *et al.* (*J. biol. Chem.* **267**, 18966–18972; 1992) have now shown that the isolated regulatory domain not only attaches to the truncated variant of ankyrin, but also non-competitively moderates its binding to the anion transporter. Why such fine-tuning is needed for a protein that had been thought of as no more than a kind of clothes-peg is now open to conjecture.

New for old

TEMPERATURE fluctuations in the microwave background, whose detection by the COBE satellite was so widely reported earlier this year, imply such small primordial density fluctuations that it is already hard for theoretical cosmologists to manufacture galaxies and clusters in the time available. C. Hogan is now trying to make the job even harder (*Astrophys. J.* **398**, L77–L80; 1992). According to Hogan, it is possible that much of the COBE signal is actually of rather recent vintage. Hot ionized gas associated with galaxy superclusters is not quite transparent to the passage of microwave photons, so can induce extra temperature irregularities. But then the primordial fluctuations must be smaller still, which makes galaxy formation harder yet. . . which leaves us wondering where the superclusters and their hot gas could have come from.

a crucial component of G-protein action, because subunit dissociation upon activation of the G protein by GTP binding is a requirement for efficient signal transmission[6]. A unique property of $G_{t\alpha}$ is that, unlike other α-subunits, it does not require detergent for its release from membranes. So a major role of acylation of $G_{t\alpha}$ may be to control subunit interactions rather than membrane association.

Kokame *et al.* synthesized acylated peptides corresponding to the amino terminus of $G_{t\alpha}$ and, in *in vitro* measurements of α–$\beta\gamma$ interactions, found that the C12:0-modified peptide was a much less potent competitor of this interaction than a myristoylated peptide. This observation suggests that the $G_{t\alpha}$ modified by C12:0 interacts more weakly with $\beta\gamma$ than one which is modified by C14:0. This weaker interaction could lead to a more rapid dissociation of the G_t oligomer upon light stimulation than would be possible if the α-subunit was modified by C14:0, and the authors speculate that this property could contribute to the rapidity of the visual signalling response compared with that of other systems controlled by G proteins.



Structures of the amino-termini of retinal rod $G_{t\alpha}$ species. The four distinct acyl groups are attached in an amide linkage to the amino-terminal glycine residue of the $G_{t\alpha}$ polypeptide.

If modification of $G_{t\alpha}$ in retinal rods by acyl groups less hydrophobic than myristate determines the rapidity of visual signalling, the question of how this modification arises becomes an important one. Notably, recoverin, another retinal protein involved in visual signalling, has also recently been reported to contain the same acyl groups at its amino terminus as $G_{t\alpha}$ (ref. 10). This finding supports the view that heterogeneous acylation is retinal-specific.

One possibility is that this heterogeneous protein acylation in the retina reflects the relative abundance of acyl-CoA substrates available to the myristoyl-CoA:protein *N*-myristoyltransferase (NMT), which is the enzyme thought to be responsible for this modification. Another is that there is a retinal-specific NMT with unique selectivity for acyl CoAs. The first possibility seems most likely, because the characterization of NMT has revealed that it can accept a variety of acyl-CoA species as substrates, including those with C12:0 and unsaturated acyl groups[11], even though the myristoyl group is the one that (until now) was always found on modified proteins.

So it seems likely that the heterogeneous acylation of $G_{t\alpha}$ will reflect retinal-specific production of acyl-CoA species. Analysis of other retinal counterparts of known myristoylated proteins, particularly those not involved in signalling in the rod outer segment, should settle the matter, as they should also contain the same mixture of acyl groups if retinal acyl-CoA levels are responsible. Another approach is to determine if expression of $G_{t\alpha}$ or recoverin in a heterologous cell type produces a protein with only C14:0 on its amino terminus — in the previous study with $G_{t\alpha}$ only the fate of the labelled myristate was followed, and other acyl groups attached to the protein might not have been detected.

A host of issues now present themselves. How, for example, are acyl-CoA levels in cells determined and what is the effect of those levels on protein acylation? And what is the significance of producing several forms of an acylated protein from a single translation product? □

Patrick Casey is in the Section of Cell Growth, Regulation and Oncogenesis and the Department of Biochemistry, Duke University Medical Center, Box 3686, Durham, North Carolina 27710, USA.

1. Kokame, K., Fukada, Y., Yoshizawa, T., Takao, T. & Shimonishi, Y. *Nature* **359**, 749–752 (1992).
2. Neubert, T. A., Johnson, R. S., Hurley, J. B. & Walsh, K. A. *J. biol. Chem.* **267**, 18274–18277 (1992).
3. Clarke, S. *A. Rev. Biochem.* **61**, 355–386 (1992).
4. Inglese, J., Koch, W. J., Caron, M. & Lefkowitz, R. J. *Nature* **359**, 147–150 (1992).
5. Gordon, J. I. *et al. J. biol. Chem.* **266**, 8647–8650 (1991).
6. Bourne, H. R. & Stryer, L. *Nature* **358**, 541–543 (1992).
7. Buss, J. E. *et al. Proc. natn. Acad. Sci. U.S.A.* **84**, 7493–7497 (1987).
8. Mumby, S. M., Huekeroth, R. O., Gordon, J. I. & Gilman, A. G. *Proc. natn. Acad. Sci. U.S.A.* **87**, 728–732 (1990).
9. Linder, M. E. *et al. J. biol. Chem.* **266**, 4654–4659 (1991).
10. Dizhoor, A. M. *et al. J. biol. Chem.* **267**, 16033–16036 (1992).
11. Kishore, N. S. *et al. J. biol. Chem.* **266**, 8835–8855 (1991).