adenylyl cyclase—thus providing a versatile tool for studying cellular effects of cAMP as well as opening avenues to understanding the structure and function of the GTP-bound active forms of G proteins. As described in a chapter by Ui, ADP-ribosylation by the exotoxin of *Bordetella pertussis* led to the discovery of the G_i proteins and, later, of the G_o proteins, which are ubiquitous in the brain. By blocking activation of substrate G proteins, pertussis toxin furnished a simple and specific way of determining whether a G protein participates in a transmembrane signaling pathway—thereby implicating G_o or G_i proteins as mediators of hormonal inhibition of adenylyl cyclase, opening and closing of ion channels, and (in some cells) stimulation of phospholipases C or A_2. Similarly, the botulinum-derived ADP-ribosyltransferases will help to elucidate cellular functions of actin (a substrate of botulinum C2 toxin) and of several small GTPases modified by another botulinum protein, called C3 transferase.

The exotoxins are fascinating in their own right. Each is divided into two functionally distinct portions, one of which binds to the exterior of a target cell and somehow transfers the second portion, the ADP-ribosyltransferase proper, across the lipid bilayer into the cell. Unraveling the mechanisms used by these transmembrane injection devices may provide fundamental insights into protein-lipid interactions involved in membrane fusion, protein translocation across membranes, and assembly and function of protein channels, pores, and transporters.

Diphtheria toxin and exotoxin A of *Pseudomonas aeruginosa* arrest protein synthesis by ADP-ribosylating EF 2. Their amino acid target, diphthamide, poses a real conundrum. Diphthamide, a chemical derivative of histidine, is found in EF 2 of all eukaryotes studied, from yeast to man—but nowhere else. Nobody knows why evolution has gone to the trouble of putting diphthamide on EF 2 and keeping it there. Mutant cells in culture appear to get along just fine without the several host enzymes that participate in diphthamide biosynthesis (Bodley and Veldman). There are hints (Iglewski and Fendrick) that an enzymatic activity of the host, perhaps occurring in EF 2 itself, may also ADP-ribosylate diphthamide. The evolutionary puzzle, however, remains unsolved.

What are the selective advantages for bacteria for expressing these ADP-ribosyltransferases? What roles do their host substrates play in pathogenesis of actual disease? The 30 percent of this book devoted to these enzymes does not ask such questions.

Most of the rest of the book is devoted to G proteins as transmitters of chemical signals across the plasma membrane. Several useful and interesting chapters cover receptors that act via G proteins (Caron, Lefkowitz, and colleagues), genes encoding G protein α subunits (Kaziro), regulation of ion channels and other effectors (Birnbaumer, Brown, and colleagues), phospholipid turnover (De Vivo and Gershengorn), and relations between structure and function of GTPases (Price, Barber, and J. Moss).

Overall, however, the treatment of G proteins is uneven and idiosyncratic. Although its signaling role is not well defined, the α subunit of G_o is allotted a chapter all to itself (Moss and colleagues), whereas the much more thoroughly studied α subunit of retinal transducin is not. A summary of the confusing literature on G proteins and neutrophil function (Snyderman and colleagues) is useful, but one seeks in vain a scholarly account of hormone-sensitive adenylyl cyclase—the signaling system, after all, in which the first G protein was discovered.

Three books on G proteins (not including symposium volumes) have appeared in the last year—surely enough to satisfy all but the most ravenous appetites. They overlap not only in subject matter but also in contributors; several authors have contributed papers on the same subject to all three. Compared to the present volume, *G Proteins*, edited by Birnbaumer and Iyengar (Academic Press, 1989) is more comprehensive but lacks detailed coverage of bacterial exotoxins. Both books cover G proteins in more detail than does the slender (232 pages) *G-Proteins as Mediators of Cellular Signaling Processes*, edited by Houslay and Milligan (Wiley, 1990).

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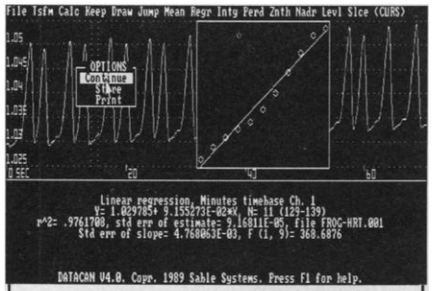

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