Preface

This triple issue of *Molecular Neurobiology* contains the edited versions of papers presented at a Satellite Meeting of the 13th Biennial Meeting of the International Society for Neurochemistry. The Satellite Meeting, titled Molecular Mechanisms of Synaptic Plasticity, was held in Port Douglas, North Queensland, Australia, from July 21 to 25, 1991. The intimate atmosphere of the hotel, the beautiful surroundings, and the relatively small number of participants ensured a meeting that was scientifically exciting and socially relaxing. The theme of the meeting was a burgeoning one that until recently was largely confined to studies of regeneration in the peripheral nervous system—the synapse as the site of interneuronal communication and as the basic structural and functional unit of neural tissues. One of the most fundamental questions in neurobiology is how the molecular structure of the nervous system changes in response to its experience. Synaptic plasticity is the term used to describe these changes, which are responsible for the brain's ability to learn, remember, and adapt.

W. H. Gispen, from the University of Utrecht in the Netherlands, gave the plenary lecture entitled "The Role of the Growth-Associated Protein B-50/GAP-43 in Neuronal Plasticity," which set the direction for the entire meeting. Other participants included a core of invited speakers and a number of scientists who contributed of their own accord and to whom we are particularly grateful. The contributions of these volunteers well complemented the invited papers and provided a balanced program. The papers in these special issues of *Molecular Neurobiology* are divided into two groups, comprising either basic molecular aspects or applied aspects of synaptic plasticity. Protein phosphorylation studies feature prominently in both groups. This is understandable, since there now exists ample evidence to suggest that the two main calcium ion-dependent protein kinases present in synaptic structures, protein kinase C (PKC) and calcium/calmodulin kinase II (CaM-PK II), are regulators of plasticity.

Basic aspects are covered in the first eleven papers. Regulation of plasticity by PKC is achieved through substrates that function as growth associated proteins (GAPs) and modulators of synaptic function. Papers by Gispen et al., by Robinson, and by Neve et al. deal with PKC, focusing particularly on its synaptic substrate B-50/GAP-43 and on neurotransmitter release. Pfenninger contributes a short general commentary on GAPs and plasticity. CaM-PK II plays a role in plasticity through its capacity to change its sensitivity to calcium by autophosphorylation and by major changes in its abundance in the synapse. Papers by Kelly, Dunkley, and Rostas deal extensively with molecular aspects of CaM-PK II. The other equally important aspect of synaptic protein phosphorylation is its reversal by protein phosphatases, which are surveyed in a paper by Sim. The remaining papers in this section are more anatomical in nature and deal with the composition of postsynaptic densities during synaptic maturation (Rostas et al.), char-

acterization of perforated synapses (Jones et al.), and binding sites for calcitonin in the brain (Sexton).

The second group of papers is concerned with applied aspects of the subject. Research in the area has led to a number of experimental models for the study of various aspects of synaptic plasticity. An important example of these models, the intriguing phenomenon of long-term potentiation (a candidate mechanism for the storage of information in the brain) is discussed in papers by Muller et al. and by Malenka and Abrahams et al. Possible molecular factors involved in learning in the sea snail *Hermissenda* are described by Nelson and Alkon and in neonate chicks by Ng et al. Two papers by Darlington et al. and Smith et al. deal with neurochemical mechanisms involved in a specific example of plasticity in the brain stem—the vestibular compensation model. The four remaining papers are all concerned with plastic changes in CNS disease in humans, where such changes are thought to be a compensatory response of the brain to injury, disease, or aging. Alzheimer's disease is surveyed by Martins et al., Iqbal et al., and Adams; Parkinson's disease is surveyed by Donnan et al. Finally, three short communications are included (by Rees et al., by Schmid, and by Sedman et al.) based on posters presented at the meeting.

The growing evidence that the brain can exhibit plasticity in response to external stimuli, despite its minimal regenerative capacity, has profound implications for our knowledge of both normal and abnormal brain function. We are confident that the present volume represents a significant contribution to this important research area.

The meeting was made possible by grants from the International Society for Neurochemistry, by a loan from the Australian Government International Conference Support Scheme, and by support from St. Vincent's Institute of Medical Research, Melbourne. We express our gratitude to these organizations. All papers included in these issues were reviewed by two referees, and we are grateful to them for their generous help and to Humana Press for their efficient processing of the manuscripts. Finally, we wish to thank the many individuals who helped in the organization of the meeting, especially Patrick Sexton and Paul Kelly, as well as the others who helped prepare these volumes.

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