Introduction

The VIIIth European Conference on Glial Cells in Health and Disease was held at Imperial College, London early in September 2007. The meeting was attended by more than 750 people and offered over 20 Symposia, 9 Plenary Lectures and some 450 Poster Presentations. The fact that this was the largest meeting held to date on glial cell biology is likely to reflect the rapidly increasing awareness of the essential role of glial cells in development, function and pathology of the nervous system.

This issue of Neuron Glia Biology explores some of the numerous topics that were discussed at the meeting.

Recent work on astrocytes and their ability to respond to, and release, signalling molecules, some of which also serve as neurotransmitters, has given rise to the exciting idea that astrocytes can exert significant control over synaptic transmission. This view of astrocytes as active participants in synaptic function was advocated in several talks at the meeting, and appears on the way to becoming mainstream thinking. The essay by Kimmelberg (see p. 181) is therefore timely, since it takes a critical view of the evidence, and warns against premature shift from the classical view of astrocytes as cells that primarily carry out general support functions for neurones. Perhaps less controversial is the role of astrocytes in regulating the interface between the blood and cells of the central nervous system, the blood brain barrier. The latest developments in this field are discussed by Savidge (see p. 191), who argues for a more broadly based view of the mechanisms involved, drawing attention to the fact that the barrier function of the microvasculature in the brain functionally resembles barrier functions carried out by epithelia in a number of other tissues. There is little doubt that astrocyte function, be it synaptic control, neuronal support or barrier maintenance, depends on the ability of astrocytes to communicate with each other through gap junctions. Scemes et al. (see p. 199) write about the molecular basis of this critical ability, particularly the role of connexins. They also draw attention to the more recently identified pannexins and the idea that these molecules may form channels that allow astrocytes to release substances that act in a paracrine manner on their neighbours.

An attractive way to repair the damage caused by oligodendrocyte death and demyelination is to harness and amplify the endogenous regenerative response to injury. Aguirre and Gallo (see p. 209) address this issue, with emphasis on the role of EGF receptor signalling and the intriguing NG2 expressing progenitor cells in myelin repair responses. The molecular organisation of the transition from proliferating cells to differentiating cells that make myelin is a fundamental issue equally relevant to repair responses and oligodendrocyte development. He et al. (see p. 221) write about the interplay between epigenetic factors, in particular histone deacetylases, and transcription factors in this process.

While the control of Schwann cell proliferation and differentiation are clearly altered in peripheral nerve tumours such as neurofibromatosis type 1, Monk et al. (see p. 233) argue that another cell type, the mast cell, and interactions with Schwann cells has in fact a vital role to play in these changes and in generating the nerve pathology seen in neurofibromatosis type 1.

Microglia are central to the response of the brain and spinal cord to injury and disease. Two new developments in this field are discussed in this volume. Miller and Streit (see p. 245) present the current picture of how aging and disease affects microglia and argue that these cells are subject to cellular senescence, an issue of considerable significance since inflammation and microgliosis are thought to be involved in age-related diseases such as Alzheimer’s disease. Exciting data from a number of laboratories suggest that glial cells are involved in the control of pain. The article by Suter et al. (see p. 255) review the current evidence with respect to microglia and conclude that these cells have a major role in pain control at the level of the spinal cord.

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REFERENCES


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