Books Received

CEREBRAL REORGANIZATION OF FUNCTION AFTER BRAIN DAMAGE. 2000. Edited by Harvey S. Levin, Jordan Grafman. Published by Oxford University Press. 392 pages C$88.00 approx.

CHILD NEUROLOGY – 6TH EDITION. 2000. Edited by John H. Menkes, Harvey B. Sarnat. Published by Lippincott Williams & Wilkins. 432 pages C$102.82 approx.

ELECTRONIC COLLABORATION IN SCIENCE. 2000. Edited by Stephen H. Koslov, Michael F. Huerta. Published by Lawrence Erlbaum Associates Inc. 150 pages C$73.42 approx.

HORMONES, GENDER AND THE AGING BRAIN. THE ENDOCRINE BASIC OF GERIATRIC PSYCHIATRY. 2000. Edited by Mary F. Morrison. Published by Cambridge University Press. 359 pages C$139.65 approx.

LOCALIZATION OF BRAIN LESIONS AND DEVELOPMENTAL FUNCTIONS. MARIANI FOUNDATION PAEDIATRIC NEUROLOGY. 2000. Edited by D. Riva, A. Benton. Published by John Libbey & Company Limited. 165 pages C$99.96 approx.

MERRITT'S NEUROLOGY – 10TH EDITION. 2000. Edited by Lewis P. Rowland. Published by Lippincott Williams & Wilkins. 1002 pages C$130.83 approx.

MOVEMENT DISORDER SURGERY. PROGRESS IN NEUROLOGICAL SURGERY – VOL. 15. 2000. Edited by A. M. Lozano. Published by Karger. 404 pages C$367.13 approx.

NEUROLOGY AND MEDICINE. 1999. Edited by RAC Hughes, GD Perkin. Published by BMJ Books. 415 pages C$66.15 approx..

NEUROMUSCULAR DISEASES: FROM BASIC MECHANISMS TO CLINICAL MANAGEMENT. MONOGRAPHS IN CLINICAL NEUROSCIENCE: VOL. 18. 2000. Edited by F. Deymeer. Published by Karger. 196 pages C$241.81 approx.

PARKINSON'S DISEASE AND PARKINSONISM IN THE ELDERLY. 2000. Edited by Jolyon Meara, William C. Koller. Published by Cambridge University Press. 251 pages C$73.42 approx.

PROCEEDINGS OF THE 9TH WORLD CONGRESS OF PAIN. 2000. Edited by Marshall Devor, Michael C. Rowbotham, Zsuzsaanna Wiesenfeld-Hallin. Published by IASP Press. 1154 pages C$117.60 approx.

Book Reviews

ALZHEIMER'S DISEASE: METHODS AND PROTOCOLS. 2000. Edited by Nigel M. Hooper. Published by Humana. 408 pages C$139.30 approx.

Alzheimer's Disease: Methods and Protocols is published as part of the series of Methods in Molecular Medicine. The series spans a wide range of disease areas with an emphasis on the hands-on protocols and methods that are required in carrying out molecular biological bench research. This multi-authored text has been edited by Dr. Nigel Hooper of the School of Biochemistry and Molecular Biology at University of Leeds and has contributions from many of the molecular research leaders in Alzheimer's disease (AD).

There are two major strengths of this text. The first is the success of the authors in developing a comprehensive overview of the molecular basis of Alzheimer's disease (AD), its genetics as well as its current treatments. The second strength resides in the detailed and specific protocols that are provided for the laboratory methods for the wide range of molecular investigative techniques current to AD.

With the pace of discovery in the molecular biology of AD being as rapid as it is, clinicians, even those with particular interest in this field, have difficulty maintaining an integrated understanding of the different aspects of molecular research. The first three chapters of this book set out to rectify this need with some success. Dr. David Allsop, in the first chapter, provides a much-needed comprehensive overview of the molecular pathogenesis of AD. He provides discussion of both amyloid processing to senile neuritic plaques as well as tau protein to neurofibrillary tangles. He provides his insight into the interrelationships between amyloid processing events and tau phosphorylation, a link that has been elusive in bringing the "B-APP"tists and "Tau"ists together but which seems to be gradually reaching a level of acceptance in the field. He notes that it is the fibrillization of the 1-42 Aə form of amyloid that is seminal to the phosphorylation of tau providing a direct link between amyloid deposition and neurofibrillary tangles in AD. His discussion of tau additionally updates the current understanding of the tau gene, which has been mapped to a single gene on chromosome 17. He adds some discussion of the mutations that have been identified with familial frontotemporal dementia and Parkinsonism (FTDP 17).

Unfortunately the pace of discovery has been so rapid that the discussion of the secretases which are the critical cleavage enzymes of the amyloid precursor protein are already well out of date despite the publication date of the text being 2000. Since the preparation of the text the amyloid precursor protein (APP) cleaving beta-secretase has been isolated beyond the speculation that it existed at the time of the text publication. The gamma secretase is increasingly appearing to be presenilin though final consensus on this point has not yet been achieved. Following from the above there is unfortunately no anticipatory discussion of the emergence of secretase inhibitors as rational therapies for clinical trial investigation. Currently there are