Editor’s Perspective

New Directions for Dementia

Cognitive impairment and dementia affect a significant portion of the global population. As a result of an aging population, dementia is increasing in prevalence worldwide. Based on age, the onset of dementia becomes a significant risk factor. For example, the prevalence of dementia is almost 25% among those over age 85, but, is approximately less than 5% for those under 75 years of age. There may also be a gender difference in the risk factors for the occurrence of the type of dementia. Men appear to have a greater risk factor for vascular dementia with aging, but women with advanced age may have a greater risk for Alzheimer’s disease (AD). Overall, it is expected that the number of individuals suffering from dementia will continue to grow to over 80 million by the year 2040.

Interestingly, AD occurs mostly in the developed countries and can involve over 50% of cases. Other types of dementia, such as vascular dementia, can represent a smaller number of cases of approximately 20%. It must be recognized that dementia is not a “clean entity” and individuals may suffer predominantly from AD, vascular dementia, or a combination of pathways that lead to AD and vascular cognitive loss. It is also expected that by 2040, almost three fourths of individuals with dementia will reside in the developed countries. These observations should not lead to conclusions that developing countries will not experience such a burden with disorders that lead to cognitive impairment. On the contrary, countries such as India, Latin America, and China have increasing prevalence of dementia in their populations and see this as a threat to the sustainability of their health systems. Yet, not all countries have similar experience with dementia and cognitive loss in their populations. For example, in the developed countries, Japan has the lowest prevalence of dementia in its population.

Given the public impact of dementia on the global population that can lead to years of disability and loss of independence, new directions for the treatment of dementia and cognitive loss are highly warranted. In this issue of Current Neurovascular Research, a number of new studies focus on novel pathways that can elucidate new strategies to treat dementia. In the paper by Wang et al. the authors examined the role of blood brain barrier disruption during postoperative cognitive impairment in a rat model and observed that alterations in the levels of tight junction proteins in the blood brain barrier following an operative procedure may contribute to an increase in the permeability of the blood brain barrier and postoperative cognitive dysfunction. The group with Li et al. evaluated constraint-induced movement therapy following experimental ischemic stroke and found that not only was angiogenesis fostered, but also neurobehavioral outcome significantly improved. Yao et al. investigated novel pathways for the onset of cognitive deficits and found that microRNA 132 (miR-132) is down-regulated during cerebral hypoperfusion and led to the loss of methyl cytidine-phosphate-guanosine (CpG) binding protein 2 (MeCP2). Restoration of MeCP2 could improve memory and learning, suggesting that miR-132 was an important factor in maintaining cognition. At a more clinical level, Menegatti et al. evaluated the prevalence of valve disorders in neurovascular disorders linked to chronic cerebrospinal venous insufficiency when compared to normal controls and have linked valve disorders to a number of neurological disorders including cognitive disease. Huang et al. also found in their clinical studies that prolonged time to maximum of the residue function (TMax) during ischemic stroke in the middle cerebral artery territory can lead to lateralized impairment in cognition functions in patients. In their review paper, Zhang and Sun provide additional information on the etiology of cognitive loss and discuss how cystatin C can become a significant component for the progression of vascular dementia. In the paper by Maiese, new avenues of investigation for dementia and cognitive loss are discussed that focus upon mammalian forhead transcription factors of the O class (FoxOs) and their involvement with erythropoietin, neurotrophins, silent mating type information regulation 2 homolog 1 (Saccharomyces cerevisiae), Wnt1 inducible signaling pathway protein 1, Wnt signaling, cancer-related pathways, apoptosis, and autophagy that could yield exciting prospects for the treatment of dementia. Additional papers in this issue of Current Neurovascular Research examine a number of other pathways that can affect vascular disease, neuronal survival, and of course how these processes can impact cognitive function. These processes can involve neurotrophic factors, steroids, genetic disease, potassium channels, and additional microRNA pathways to further highlight the multifactorial pathways that can impact cognition. Ultimately, this issue of Current Neurovascular Research offers an interesting roadmap for many new directions that can affect the treatment of dementia.

Kenneth Maiese
(Editor-in-Chief)
Laboratory of Cellular and Molecular Signaling
Cancer Center, F 1220
New Jersey Health Sciences University
205 South Orange Avenue, Newark, NJ 07101, USA
E-mail: wntin75@yahoo.com

2017 Bentham Science Publishers