Editorial

New Therapeutic Targets for Neuroprotection Against Acute Brain Injuries

Acute CNS injuries caused by ischemic and traumatic insults are the major promoters of long-term disabilities in humans. Many synergistic pathophysiologic mechanisms including inflammation, ionic imbalance, apoptosis, edema, transcriptional failure, oxidative stress and endoplasmic reticulum stress are currently considered as the proponents of the post-injury neuronal death leading to neurological dysfunction. In the past 20 years, laboratories all over the world tested hundreds of compounds for their neuroprotective potential in animal models of CNS injury. However, not a single therapeutic drug that can prevent neuronal death in humans after an injury is currently available. With the emerging of molecular technologies, laboratories all over the world are continuing the efforts to identify newer target genes and proteins which might play a role in post-injury neuronal death and CNS dysfunction. Furthermore, compounds that can modulate these targets are being tested in animal models of CNS ischemia and trauma. This special issue is a collection of some cutting-edge reports on the new lines of research being conducted in the laboratories at the fore-front of CNS injury research.

As superoxide is neurotoxic, Kim *et al*. discussed the significance of NADPH oxidase which is a major source of superoxide and the therapeutic potential of its inhibitor apocyanin in inducing neuroprotection in animal models of CNS injury. Promoting new blood vessel formation after an injury can help restoration of blood flow to the damaged area. Hence, Chen and Chopp reviewed the exciting possibility of using niacin (vitamin B3) as a drug to induce angiogenesis and further neurological function recovery in rodents subjected to experimental stroke. Using endogenous compounds like niacin as neuroprotective agents is very appealing due to fewer side effects. In this context, the review by Boison and Shen highlights the therapeutic potential of adenosine after cerebral ischemia. In similar lines, Samantaray *et al*. reviewed the benefits of estrogen and melatonin to induce neuroprotection after spinal cord injury. Bales *et al*. highlighted controlling dopaminergic system as a dual-acting therapeutic paradigm to protect neurons and to induce functional recovery following traumatic brain injury. Crain *et al*. discussed the significance of cross-talk between estrogen and purine receptors in controlling post-injury inflammation and strategies to target these receptors for developing efficient neuroprotective drugs. As breakdown of extracellular matrix (ECM) after an injury is a proponent of neuronal death, Clark and Bix discussed the role of ECM fragments in modulating angiogenesis and self-repair after stroke. Identifying easy to use neuroprotective paradigms is important for preventing secondary brain damage. Hence, Zhao reviewed the molecular mechanisms of mechanical and pharmacological post-conditioning which is a novel strategy for inducing neuroprotection. Hypoxic ischemia of brain is a considerable problem in neonates that promotes significant brain damage. Kleman *et al*. reviewed the recent preclinical studies and development of drugs for therapeutic approaches to prevent excitotoxicity and brain damage following hypoxic ischemia. Overall, this special issue shows the exciting neuroprotective therapies being tested currently. In addition, the special issue also highlights the need for identifying newer targets and developing novel ways to design drugs for preventing secondary brain damage following ischemic and traumatic injuries to CNS. Financial support was provided by a NIH grants NS049448, NS061071, NS067274.

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1877-3818/10 2010 Bentham Open