Neural Plasticity

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Special Issue on

# DNA Damage, Neurodegeneration, and Synaptic Plasticity

CALL FOR PAPERS

Unrepaired DNA lesions and deficiencies in DNA repair systems are implicated in the progressive neuronal loss observed in many neurodegenerative pathologies including Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and amyotrophic lateral sclerosis (ALS). Whether the increased level of DNA damage in neurodegenerative disorders is a consequence rather than the cause of previous neurodegenerative events remains to be established.

Neuronal DNA damage and repair may also play a role in cognitive function and dysfunction.

The formation of double strand breaks (DSBs), the most lethal form of DNA damage, may be a physiological process that modifies chromatin organization and gene expression involved in information processing, learning, and memory and may be caused by normal brain activity. Conversely, pathologically elevated β-amyloid peptide (Aβ), a major culprit for the pathogenesis of AD, worsens DNA damage by eliciting aberrant synaptic activity.

Interestingly, the involvement of ataxia-telangiectasia mutated protein (ATM, a PI 3-kinase DSB repair protein) in synaptic plasticity provides a new attractive explanation for the severe neurological symptoms observed in ataxia-telangiectasia patients and also suggests a possible function of ATM not associated with DNA repair.

We invite authors to submit original research articles as well as review articles that will contribute to and stimulate our knowledge of the mechanisms underlying DNA damage in neurodegenerative disorders, the understanding of the molecular and cellular basis of neurodegenerative diseases, and the mechanisms involved in cognitive impairment associated with neurodegeneration.

Potential topics include, but are not limited to:

- Role of DNA damage in neurodegenerative diseases
- DNA damage in the "normal" aging brain
- Neurological consequences of unrepaired DNA damage
- Consequences of the failure of DNA repair
- Natural and transgenic animal models for studying neurodegenerative disorders
- Defects in synaptic plasticity associated with neurodegeneration
- DNA damage/repair and its links to cognitive functions
- Innovative therapeutic strategies for treatment of neurodegenerative diseases

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/np/dnad/.

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