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# Various Staining Techniques used in Neurodegeneration

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## Introduction

Neurodegeneration is a process that results in irreversible neuronal damage and death, and it is a common final pathway in ageing and neurodegenerative diseases. Many of your body's activities are affected by degenerative nerve diseases, including balance, movement, talking, breathing, and heart function. Many of these diseases are passed down through families. A medical condition, such as alcoholism, a tumour, or a stroke, can sometimes be the cause. Toxins, chemicals, and viruses are some of the other possible causes [1]. Alcoholic neurodegeneration is widespread, with the frontal cortex suffering the most severe loss. Binge drinking activates neuroimmune genes, which causes neurodegeneration by increasing oxidative stress, specifically NADPH oxidase-induced oxidative stress. Furthermore, HMGB1-TLR4 and innate immune NF-B target genes are upregulated, resulting in persistent and sensitised neuroimmune responses to ethanol and other agents that release HMGB1 or directly stimulate TLR and/or NMDA receptors. Alcoholic neurodegeneration is linked to neuroimmune signalling and glutamate excitotoxicity. Models of adolescent alcohol abuse show significant frontal cortical degeneration as well as the most severe loss of hippocampal neurogenesis. Adolescence is a critical period for ethanol-induced neurodegeneration, changes in brain structure, gene expression, and adult phenotypic maturation [2].

## Description

Neurodegeneration produced by Meth has been linked directly to COX-mediated inflammation. Despite the fact that COX has been linked to Meth-induced neurodegeneration, there are some caveats [3]. COX-2 knockout protects against Meth-induced dopamine depletion, but COX inhibitors do not protect against striatal dopamine depletion. It is unclear how COX-2, in and of itself, contributes to Meth-induced neurodegeneration is not neurotoxic at doses comparable to those that cause neurotoxicity in Meth. As a result, activation of COX-2 alone may not be enough to cause neurotoxicity to amphetamines.

## Conclusion

Neurodegeneration is a multifactorial process that causes neuronal death in the brain and spinal cord. Oxidative stress, axonal transport deficits, protein oligomerization, aggregation, calcium deregulation, mitochondrial dysfunction, abnormal neuron–glial interactions, neuroinflammation, DNA damage, and abnormal RNA processing are all associated with neurodegeneration. Neurodegeneration can occur as a result of neurotraumatic, neurodegenerative, or neuropsychiatric disorders [4]. Paralysis, muscle weakness, poor coordination, seizures, confusion, and pain are caused by structural, neurochemical, and electrophysiological abnormalities in the brain, spinal cord, and nerves. Strokes, TBI, SCI, and CTE are all common neurotraumatic disorders. Neurons degenerate rapidly in ischemic and traumatic brain and spinal cord injuries due to a sudden lack of oxygen, a rapid drop in ATP, and an alteration in ion homeostasis. Alzheimer's disease, Parkinson's disease, Huntington's disease, and ALS are examples of common neurodegenerative diseases. Misfolded proteins accumulate in neurodegenerative diseases, causing neurodegeneration in a specific population of neurons. Some oxygen, nutrients, and ATP are available to neurons in neurodegenerative diseases, ion homeostasis is maintained to a limited extent, and neurodegeneration takes a longer time period (years) to die. Depression, schizophrenia, some forms of bipolar affective disorders, autism, mood disorders, attention deficit disorder, dementia, tardive dyskinesia, and chronic fatigue syndrome are examples of neuropsychiatric disorders [5]. Anomalies in signal transduction processes in the cerebral cortex and limbic system are at the root of neuropsychiatric diseases (thalamus, hypothalamus, hippocampus, and amygdala).

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