

# Neurological Disorders in N-Acetylcysteine

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## Editorial

Due to its vital medical applications, N-acetylcysteine (NAC), an acetylated cysteine molecule, has piqued scientific curiosity for decades. It also functions as a dietary supplement for humans. NAC is a glutathione precursor with anti-inflammatory and antioxidant properties. NAC may be used in therapies to combat neurological and mental health illnesses, in addition to the uses mentioned in the literature. Furthermore, the neuroprotective potential of this chemical in the prevention of cognitive ageing dementia has been investigated. NAC is a low-cost, commercially available supplement with no discernible negative effects after use.

The goal of this study is to provide an overview of NAC's effects and applications in Parkinson's disease, Alzheimer's disease, neuropathic pain, and stroke. N-acetylcysteine (NAC) is a precursor to L-cysteine and is commonly used in mucolytic therapy and to treat paracetamol overdose. The World Health Organization (WHO) recognises NAC as a necessary drug in a basic health system. NAC can be taken orally, inhaled, or injected intravenously, and it is considered an important drug [1]. NAC is also regarded as a medicine with pro-neurogenic and neuroprotective characteristics. It's also employed in anti-neurodegenerative and anti-psychiatric treatments [2,3].

According to the literature, taking glutathione (GSH) as an oral drug alone is insufficient to restore GSH levels. In reality, GSH is rapidly hydrolyzed in body regions such as the liver and intestines [4], and its ability to penetrate the Blood-brain Barrier (BBB) is limited. Similarly, due to its metabolic activity, oral ingestion of L-cysteine has been demonstrated to have a negative impact on the recovery of GSH in the brain [5]. Oral NAC administration suggests a whole other problem. Its use causes a rise in plasma cysteine levels, which then leads to an increase in plasma GSH. According to studies on animal models published in scientific literature, NAC has the ability to permeate the BBB and increase GSH levels in the brain.

This uniqueness of NAC could be a key role in the treatment of neurological illnesses, where alterations in GSH levels and redox pathways have been reported. Similarly, excitotoxic damage is linked to the activation and control of N-methyl-D-aspartate (NMDA) glutamate receptors, which causes a variety of neuronal damage and degeneration. NAC has been demonstrated to have

antioxidant and anti-inflammatory properties, among other things. NAC acts as an antioxidant, neutralising free radicals before they cause cell damage. NAC boosts cysteine/GSH levels in cells while also acting as an oxidant scavenger. NAC is an acetylated cysteine molecule with an acetyl group linked to the nitrogen atom that can be oxidised by a number of radicals. NAC works by restoring the antioxidant potential of cells by replenishing GSH that has been depleted by free radicals and scavenging Reactive Oxygen Species (ROS).

NAC works as an anti-inflammatory, limiting the release of cytokines during the early stages of immunological proliferation. NAC is a chemical that has been shown to have antioxidant and anti-inflammatory properties. In this study, we discussed how NAC can be utilised as an adjuvant therapy in neurodegenerative disorders like Parkinson's disease and Alzheimer's disease, as well as neuropathic pain and stroke, due to its activities. Clinical trials in which NAC has been assessed in combination therapy support this hypothesis of linkage with other medicines.

## Conflict of Interest

None.

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