Neurodegeneration is a complex process and involves myriad of physiological changes leading to chronic pathological states. Current therapies for neurodegenerative disorders, such as Alzheimer’s (AD) and Parkinson's diseases (PD), are only symptomatic. Hence, there is an urgent need to design and develop therapies that are disease modifying.

A group of compounds termed Cu\textsuperscript{II}bis(thiosemicarbazones) or Cu\textsuperscript{II}(btsc) have been recently identified as having biological activities that could be therapeutic in AD and PD. These group of compounds, such as Cu\textsuperscript{II}(atsm) and Cu\textsuperscript{II}(gtsm), are able to inhibit nitro-oxidative stress, alter nitric oxide signaling as well as promote neuronal survival via the AKT/PI3K pathway. These compounds are also bioavailable and readily cross the BBB making them ideal CNS drugs. Cu\textsuperscript{II}(btsc) compounds were promptly investigated for their ability as therapeutic agents in animal models of AD and PD.

Cu\textsuperscript{II}(gtsm) administration to AD transgenic (APP/PS1) mice resulted in an increase in intracellular copper bioavailability. This led to an inhibition of GSK3\(\beta\), reduction in Ab trimers and phosphorylated tau, and restoration of cognition. On the other hand, Cu\textsuperscript{II}(atsm) inhibited nitro-oxidative stress in multiple PD mouse models (MPTP and 6-OHDA lesioned, and \(\alpha\)-synuclein transgenic mice) resulting in reduced alpha-synuclein nitration and oligomerisation in the substantia nigra (SN). Increased survival of dopaminergic neurons and improved motor function and coordination were also observed and were accompanied with increased TH and VMAT2 levels.

These findings establish the therapeutic effects of Cu\textsuperscript{II}(btsc) compounds in animal models and suggest that these compounds could be effective disease modifying agents for neurodegenerative diseases.