Butylphthalide

3-n-butylphthalide (3-NBP) is the same as butylphthalide. The “3-n” prefix in its name indicates the specific isomer of butylphthalide. Butylphthalide is a compound with multiple isomers, and 3-NBP is one of those isomers. It's often referred to by either name in the scientific literature, and they are used interchangeably to describe the same chemical compound. This compound has been studied for its potential therapeutic applications, particularly in the context of neurological and neuroprotective effects.

- Investigation of the Impact Factors and Efficacy of N-Butylphthalide (NBP) on Functional Outcomes Following Mechanical Thrombectomy in Stroke Patients
- Butylphthalide mitigates traumatic brain injury by activating anti-ferroptotic AHR-CYP1B1 pathway
- Therapeutic effectiveness of Donepezil hydrochloride in combination with butylphthalide for post-stroke cognitive impairment
- Dl-3-n-butylphthalide promotes microglial phagocytosis and inhibits microglial inflammation via regulating AGE-RAGE pathway in APP/PS1 mice
- Dl-3-n-butylphthalide attenuates cerebral ischemia/reperfusion injury in mice through AMPK-mediated mitochondrial fusion
- Effects of Butylphthalide Combined with Fasudil on Inflammatory Factors, Cognitive Function and Vascular Endothelial Function in Patients with Subarachnoid Hemorrhage Complicated with Cerebral Vasospasm
- Co-administration of dl-3-n-butylphthalide and neprilysin is neuroprotective in Alzheimer disease associated with mild traumatic brain injury
- Nanowired delivery of dl-3-n-butylphthalide with antibodies to alpha synuclein potentiated neuroprotection in Parkinson's disease with emotional stress

Butylphthalide (3-n-butylphthalide or NBP) is one of the chemical constituents in celery oil, along with sedanolide, which is primarily responsible for the aroma and taste of celery.

Studies in animal models suggest that butylphthalide may be useful for the treatment of hypertension and may have neuroprotective effects.

A investigation examine the role of immobilization stress influencing Parkinson's disease brain pathology in model experiments. In one previous report they found that mild traumatic brain injury exacerbate Parkinson's disease brain pathology and nanodelivery of dl-3-n-butylphthalide either alone or together with mesenchymal stem cells significantly attenuated Parkinson's disease brain pathology [1)].

Among patients with acute ischemic stroke receiving intravenous thrombolysis and/or endovascular treatment, NBP was associated with a higher proportion of patients achieving a favorable functional outcome at 90 days compared with placebo [2)]

NBP improves the barrier function of BBB against ischemic injury by upregulating the expression of TJ

proteins, possibly by reducing oxidative stress and activating the Akt/GSK-3β/β-catenin signaling pathway [3)]

In 2002, NBP was approved in China for the treatment of cerebral ischemia.

NBP undergoes extensive metabolism in humans.

The major metabolites in human plasma was 3-OH-NBP, 10-OH-NBP, 10-CO-NBP, 11-COOH-NBP. The AUC of metabolites was much larger than that of NBP.

Minor side effects were observed in preclinical and clinical studies. The minor bioactivation pathway of NBP was proved to be medicated via sulfation of 3-OH-NBP.

In a study, He et al. examined the neuroprotective effects and anti-inflammatory properties of Dl-3-n-butylphthalide (NBP) in Sprague-Dawley (SD) rats following traumatic spinal cord injury (SCI) as well as microglia activation and inflammatory response both in vivo and in vitro. Our results showed that NBP improved the locomotor recovery of SD rats after SCI an significantly diminished the lesion cavity area of the spinal cord, apoptotic activity in neurons, and the number of TUNEL-positive cells at 7 days post-injury. NBP inhibited activation of microglia, diminished the release of inflammatory mediators, and reduced the upregulation of microglial TLR4/NF-κB expression at 1 day post-injury. In a co-culture system with BV-2 cells and PC12 cells, NBP significantly reduced the cytotoxicity of BV-2 cells following lipopolysaccharide (LPS) stimulation. In addition, NBP reduced the activation of BV-2 cells, diminished the release of inflammatory mediators, and inhibited microglial TLR4/NF-κB expression in BV-2 cells. Our findings demonstrate that NBP may have neuroprotective and anti-inflammatory properties in the treatment of SCI by inhibiting the activation of microglia via TLR4/NF-κB signalling [4)].

A study was aimed to evaluate the neuroprotective effects of NBP in the mice models of TBI, as well as the possible role of Nrf2-ARE pathways in the assumptive neuroprotection. In mice,a modified Marmarou's weight-drop model was employed to induce TBI. ICR mice were randomly assigned to four experimental groups: sham, TBI, TBI+vehicle(V) and TBI+NBP. NBP (100 mg/kg) was administered via an intraperitoneal (i.p.) injection at 1 h following TBI. The administration of NBP significantly ameliorated the effects of the brain injury, including neurological deficits, brain water content, and cortical neuronal apoptosis. Furthermore, the level of malondialdehyde and the activity of superoxide dismutase (SOD) paired with glutathione peroxidase (GPx) were restored in the NBP treatment group. NBP promoted the translocation of Nrf2 protein from the cytoplasm to the nucleus markedly, increased the expressions of Nrf2-ARE pathway-related downstream factors, including hemeoxygenase-1(HO-1) and NAD(P)H: quinone oxidoreductase 1 (NQO1), and prevented the decline of antioxidant enzyme activities, including SOD and GPx. NBP enhanced the translocation of Nrf2 to the nucleus from the cytoplasm,verified by a western blot, immunofluorescence. Additionally, it upregulated the expression of the Nrf2 downstream factors such as HO-1 and NQO1 were also confirmed via a western blot and real-time quantitative polymerase chain reaction. In conclusion, NBP administration may increase the activities of antioxidant enzymes and attenuate brain injury in a TBI model, potentially via the mediation of the Nrf2-ARE pathway [5)].

1)

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