In a study, Liu et al. investigated the effect of TC on kainic acid-induced seizure activity caused by oxidative stress and pro-inflammation. They found that TC pretreatment significantly decreased seizure activity score compared to kainic acid treated group. Importantly, TC pretreatment leads to lowering the mortality in kainic acid treated mice. In addition, TC was found to significantly inhibit KA-induced generation of malondialdehyde. TC pretreatment also preserved the activity of GPx, SOD, and CAT. Notably, our data shows that an important property of TC is its capacity to exert cerebral anti-inflammatory effects by mitigating the expression of proinflammatory cytokines, such as TNF-α and IL-1β. These data suggest that TC has a potential protective effect on chemical induced seizure and brain damage. [1)].

BCP pre-treatment prior to the initiation of OGD/R significantly (i) decreased BBB permeability and neuronal apoptosis, (ii) mitigated oxidative stress damage and the release of inflammatory cytokines, (iii) down-regulated Bax expression, metalloproteinase-9 activity and expression, and (iv) up-regulated claudin-5, occludin, ZO-1, growth-associated protein-43 and Bcl-2 expression. Thus, BCP pre-treatment exerted multiple protective effects on NVU in the context of OGD/R-induced injury. These protective effects potentially occur via reductions in oxidative stress damage and inflammatory cytokines that induce BBB breakdown, subsequently resulting in reduced neuronal apoptosis [2)].

Cerebral blood flow (CBF) monitoring results indicated that HPβCD/BCP can promote the recovery of CBF. Moreover, molecular biology experiments showed that HPβCD/BCP can increase the expression levels of CB2 in brain tissues, particularly the hippocampus and white matter tissues, as well as the expression levels of PI3K and Akt. Overall, the findings demonstrated the protective effects of HPβCD/BCP against cognitive deficits induced by chronic cerebral ischemia and suggested the potential of HPβCD/BCP in the therapy of vascular dementia in the future [3)].

Amyloid betaβ (Aβ) is the major component of senile plaques (SP) in the brains of Alzheimer's disease (AD) patients, and serves as an inflammatory stimulus for microglia. Trans-caryophyllene (TC), a major component in the essential oils derived from various species of medicinal plants, has displayed neuroprotection in previous studies. However, whether TC has a protective role in AD remains unknown.

In a study, the effects of TC on Aβ1-42-induced neuroinflammation were investigated. They found that TC reduced the release of LDH in BV-2 microglial cells treated with Aβ1-42. In addition, pretreatment of BV2 microglia with TC at concentrations of 10, 25, and 50μM prior to Aβ stimulation led to significant inhibition of nitric oxide (NO) and prostaglandin E2 (PGE2) production, expression of inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2), and secretion of pro-inflammatory cytokines. Notably, our results indicate that TC remarkably attenuated Aβ1-42-activated overexpression of toll-like receptor 4 (TLR4). We further demonstrated that TC markedly reversed Aβ1-42-induced phosphorylation and degradation of IκBα, nuclear translocation of p65, and NF-κB transcriptional activity. These findings suggest that TC may have therapeutic potential for the treatment of AD [4)].

1)
Liu H, Song Z, Liao D, Zhang T, Liu F, Zhuang K, Luo K, Yang L. Neuroprotective effects of trans-caryophyllene against kainic acid induced seizure activity and oxidative stress in mice. Neurochem

Res. 2015 Jan;40(1):118-23. doi: 10.1007/s11064-014-1474-0. Epub 2014 Nov 23. PubMed PMID: 25417010.
2)

Tian X, Peng J, Zhong J, Yang M, Pang J, Lou J, Li M, An R, Zhang Q, Xu L, Dong Z. β-Caryophyllene protects in vitro neurovascular unit against oxygen-glucose deprivation and re-oxygenation-induced injury. J Neurochem. 2016 Dec;139(5):757-768. doi: 10.1111/jnc.13833. Epub 2016 Sep 19. PubMed PMID: 27565895.
3)

Lou J, Teng Z, Zhang L, Yang J, Ma L, Wang F, Tian X, An R, Yang M, Zhang Q, Xu L, Dong Z. β-Caryophyllene/Hydroxypropyl-β-Cyclodextrin Inclusion Complex Improves Cognitive Deficits in Rats with Vascular Dementia through the Cannabinoid Receptor Type 2 -Mediated Pathway. Front Pharmacol. 2017 Jan 19;8:2. doi: 10.3389/fphar.2017.00002. eCollection 2017. PubMed PMID: 28154534; PubMed Central PMCID: PMC5243824.
4)

Hu Y, Zeng Z, Wang B, Guo S. Trans-caryophyllene inhibits amyloid β (Aβ) oligomer-induced neuroinflammation in BV-2 microglial cells. Int Immunopharmacol. 2017 Aug 15;51:91-98. doi: 10.1016/j.intimp.2017.07.009. [Epub ahead of print] PubMed PMID: 28821008.