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Hericium erinaceus: A possible future therapeutic treatment for the prevention and delayed progression of Alzheimer's disease? – A Systematic Review

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Alzheimer's disease (AD) is the most common progressive central nervous system neurodegenerative disease globally[(1)]. At present, the treatment of AD involves only symptomatic medications which have continually demonstrated little efficacy[(2)]. Hericium erinaceus (HE), commonly known as lion's mane mushroom, has not yet been fully utilised among western pharmacology for its medicinal purposes, demonstrating a possible omittance of a highly beneficial neuro-altering substance. To date, studies that have investigated the potential medicinal properties of HE have found that various neuroprotective effects are exerted when following consumption[(3)]. The aim of this review is to systematically investigate the neuroprotective pathways impacted by dietary supplementation of HE, determine specific bio-compounds responsible, and highlight the importance of continued research to determine the true potential relevance of this therapeutic treatment for AD.

Electronic databases were systematically searched for studies investigating the relationship between HE and AD. For inclusion in this review, human studies must have been of a clinical design involving adults >30 years that are healthy, have mild cognitive impairment, probably AD or memory deficits. Animal studies were required to involve interventions that directly impact AD-related mechanisms. The exclusion criteria involved any observational studies that involved participants with other neurological implications, or any studies that noted existing interventions (i.e., medications, post-surgery, dietary intervention). A study quality assessment was conducted for all qualified studies using Cochrane RoB2 tools. Data extraction was undertaken according to PRISMA guidelines.

A total of 16 studies (including 3 human clinical trials and 13 animal model studies) met the criteria for inclusion. All studies included either behavioural, biochemical, ophthalmic, and neuroimaging assessments which demonstrated to be directly influenced by HE intake and highlighted key mechanisms previously associated with neurohealth promotion or neuropathological decline. Behavioural and biochemical clinical trial results revealed statistical differences ($p < 0.05$) between result comparison between HE and control groups and various week intervals. Animal model behavioural and biochemical assessments demonstrated positive findings. Histological assessments of AD-induced rodents following HE administration revealed statistically significant results ($p < 0.05$) in cholinergic transmitter and NGF concentrations, β-amyloid peptide plaque accumulation, and microglia and astrocyte activation compared to control groups.

Evidence suggests that an intake of HE, specifically the compound erinacine-A may be an appropriate and relevant candidate for the future therapeutic treatment for the prevention and delayed progression of AD. Application of HE demonstrated numerous improvements in AD-related behaviour, biomarker parameters, histological features, and physiological mechanisms while neuroprotective and neurotrophic properties were also clearly established. Nevertheless, the review highlights the necessity for continued research specifically human clinical trials to contribute continued evidence surrounding the use of HE for AD, to provide direction for future research and provide constructive methods for the possible future targeting of AD populations.

References

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