

Comparative Analysis of Systemic Redox Imbalance in Ovalbumin-Induced Asthma and Aluminum Chloride-Induced Alzheimer's Disease

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Asthma drives significantly greater systemic oxidative stress than early-stage Alzheimer's disease, positioning pulmonary redox control as a potential neuroprotective strategy along the lung-brain axis

INTRODUCTION	METHODS	RESULTS	CONCLUSIONS	REFERENCES
Chronic respiratory inflammation may accelerate neurodegeneration through a proposed “lung-brain axis,” as midlife asthma is linked to higher dementia risk (Rusanen et al., 2013). Oxidative stress bridges both conditions: pulmonary ROS and cytokines enter systemic circulation, compromise the blood-brain barrier, and activate microglia, while intermittent hypoxia from uncontrolled asthma promotes amyloid-β production via HIF-1α/BACE1 signalling.	Fifteen female Albino Wistar rats (250–300 g) were randomly assigned to three groups (n=5): Control, AD-induced (aluminium chloride, 175 mg/kg for 28 days), and Asthma-induced (ovalbumin/aluminium hydroxide sensitisation on days 1, 7 and 14, then 1% ovalbumin challenge biweekly for 28 days via nebuliser). AD induction was confirmed by neurobehavioural assessment and serum acetylcholine/acetylcholinesterase levels. Serum SOD, CAT and GPx activities and MDA concentration were measured. Ethical approval: University of Benin CMS/REC/2024/741. Data were analysed by ANOVA and Student's t-test (GraphPad Prism 9.0); p<0.05 was considered significant	SOD, CAT & GPx: significantly reduced in the asthma group versus both control and AD groups (p<0.05); no significant change in the AD group versus control (p>0.05). MDA: significantly elevated in the asthma group versus control (p<0.05), with no significant change in the AD group versus control (p>0.05).	Asthma induced a more profound systemic oxidative stress response than the early-stage AD model, evidenced by depleted antioxidant enzymes and elevated lipid peroxidation. These findings support pulmonary oxidative stress management as a potential intervention point for preventing downstream neurodegenerative complications and underscore the clinical relevance of the lung-brain axis.	Rusanen M, et al. Curr Alzheimer Res. 2013;10(5):549–555. Comhair SA, Erzurum SC. Am J Physiol Lung Cell Mol Physiol. 2002;283(4):L709–L727. Erickson MA, Banks WA. Immunol Rev. 2018;282(1):122–141. Praticò D. Trends Pharmacol Sci. 2008;29(12):609–615. CONTACT Emmanuel.aloamaka@uniben.edu +234 703 041 3444 ORCID: 0000-0003-4771-3553 Dept. of Physiology, University of Benin, Nigeria  ALZHEIMER'S ASSOCIATION 