Review of: "Hypocholinergic Stress and Neuronal Pruning in Alzheimer's Disease"

M. A. Bendary[1]

1 King Abdul Aziz University

Potential competing interests: No potential competing interests to declare.

Dear Editor-in-Chief,

Thank you for giving me the opportunity to evaluate this review titled "Hypocholinergic Stress and Neuronal Pruning in Alzheimer's Disease"

The document did not offer new and original insights on the topic of Alzheimer's disease. The manuscript has the ambitious goal, and the empirical data does not fit the aim of the study.

There is a flawed theoretical framework and internal discrepancies; for example,

on page 2, the author has mentioned that nicotinic receptors are located mainly in the synaptic cleft,

on page 3, the author has mentioned that neural networks are produced through competition.

On page 3, the author has mentioned that as a result acetylcholine is a key regulator of…… as a result of is not convenient with the sentence context

On page 3, a sentence needs to be revised ……The significance of this neurodegeneration is recognized by its inclusion alongside amyloid deposition and neurofibrillary tangles as a biomarker for Alzheimer's disease by the National Institute on Aging and the Alzheimer's Association in their recent research framework for diagnosing Alzheimer's disease (Jack et al., 2018).

On page 4, the author has mentioned wrongly…..low hypocholinergic levels of acetylcholine

In page 4, there is a contradiction in the sentence…..Hypocholinergic states, such as those found during development, stimulate neuronal growth, but without eventual positive feedback through successful synaptogenesis, result in apoptosis. Similarly, prolonged hypocholinergic stress likely reduces positive feedback in mature neural networks, thereby promoting growth and apoptosis (Russell, 1996).

In page 5, there is a contradiction in the sentence….A hypocholinergic state in the nervous system likely destabilizes neuronal integrity by activating developmental processes and promoting axonal growth and synaptogenesis.

The manuscript has not been written well and is hard to understand. The document is presented in an unorganized way with many biases and no consequent link between most sentences. Even the references list did not cover the relevant

literature adequately and needs updating. Besides, the conclusion and objectives don't cross.

Unfortunately, in its current state, I cannot recommend accepting this paper.

Yours,

The reviewer, Prof. MA. Bendary

Professor of Clinical Physiology, King Abdul Aziz Medical College, Saudi Arabia