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Editorial

Organophosphorus pesticide exposure and Alzheimer's disease: Recognition of a paradox

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Alzheimer's disease (AD) is increasing worldwide. It is the most common type of dementia [1]. Aging of the global population is associated with increased occurrence of the disease. Therefore, the experts believe that it is a matter of public health concern worldwide [2].

The disease is characterised by progressive neurodegeneration and deposition of beta-amyloid proteins in the nervous system [3]. The AD patients are deficient in cholinergic neurotransmission. Therefore, an attempt to increase in acetylcholine in the brain is one of the treatment options. This is done by acetylcholinesterase (AChE) inhibitors which are currently the first line treatment for AD [4]. Recent pieces of evidence also advocate the use of cholinesterase inhibitors in AD patients [5-8]. However, the clinical relevance of such an approach is not beyond criticism [9].

Standing in this factual position it can be logically concluded that cholinergic inhibition is a positive state for cognitive function. Naturally that can lead to an idea that organophosphorus (OP) poisoning (or exposure), an uncontrolled trend of the developing world is a state of palliation for AD patients because the OP pesticides are known inhibitor of the cholinesterase [10]. In fact, the erythrocyte cholinesterase is activity is considered as a biomarker of OP exposure [11]. This is a paradox and OP poisoning and AD both are public health concerns this paradox must be addressed Contrary to this logical view OP exposure is associated with cognitive decline and particularly AD [12-14].

This paradox requires recognition as OP exposure is quite common and incidence of AD is showing a rising trend. From the theoretical standpoint we feel that the following issues are to be explored to address the paradox;

1. Generally reversible AChE inhibitors employed in the management of AD but OP pesticides inhibit AChE in an irreversible manner [15].
2. OP pesticides bind to several proteins other than AChE [16-18]. It is possible that OP adducts to proteins enhance the formation of beta-amyloid protein. However, nothing conclusive is known in this regard as on date.
3. OP pesticides cause oxidative stress and apoptosis of nerve cells [19,20].

We feel that these are the major issues which if explored by focused research have the potential to explain the paradox presented here. Considering the global situation of AD and OP pesticide poisoning exploring such paradox is the need of the hour.

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