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Clean up, Clean up, Everybody Clean up

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Editorial

Alzheimer's disease (AD) is a neurodegenerative disease characterized by progressive memory deficits and cognitive decline. Sleep disturbances such as sleep fragmentation, decreased sleep duration and circadian alterations not only are observed in early stages of AD, but may also appear years before the appearance of dementia. Therefore, their presence could be predictive of cognitive decline in AD. Accumulations of extracellular β amyloid protein plaques and intracellular tau neurofibrillary tangles in brain tissues, especially in the hippocampal and prefrontal areas, are the early hallmarks of AD. They begin to be deposited in the brain 2 to 3 decades before the onset of clinical symptoms. These plaques and tangles are neurotoxins that potentiate each other's destructive effects on brain cells' functions and structures, especially those responsible for memory and cognition. They work together to disrupt mitochondrial functions, destroy synapses and cause neuronal death. Extended periods of sleep deprivation, especially slow-wave sleep deprivation, lead to both impairment of normal tau metabolism and higher levels of β amyloid protein, particularly in the frontal, hippocampal and thalamic areas. The consequence is an acceleration of AD pathogenesis. On the other hand, higher levels of β amyloid protein might lead to sleep fragmentation, a worsening of sleep quality and daytime somnolence. Similarly, higher levels of abnormal tau proteins may interfere with the sleep-wake cycle. The glymphatic system, the brain network of perivascular pathways, is responsible for removing β amyloid protein and abnormal tau from brain tissues. It operates mainly when the brain is sleeping. Astrocytes are special giant cells in brain interstitial fluids that play a major role in β amyloid and tau cleanup. Their activity is increased by growth hormone and insulin-like growth factor. In adults, slow-wave sleep is the principal stage of sleep in which growth hormone secretion takes place. Thus, astrocytes are more active and more effective in clearing β amyloid and tau proteins during slow-wave sleep. As a result, slow-wave sleep insufficiency may lead to impaired peripheral clearance of β amyloid and tau proteins.

Several studies have demonstrated that early night sleep primarily improves the consolidation of declarative memories such as facts and episodes. These are hippocampus-dependent memories. Early night sleep is dominated by slow-wave sleep. On the other hand, late night sleep improves emotional and skill memories. These are amygdaloidal-dependent memories. Late night sleep is dominated by REM sleep. Altogether, the accumulated data strongly suggests that at least part of Benjamin Franklin's aphorism "early to bed, early to rise, makes a man healthy, wealthy and wise" is correct; i.e., early to bed, makes a man healthy and wise.

Alarmingly, according to the Institute of Medicine, 70 million Americans suffer from a chronic sleep disorder. The National Sleep Foundation reported that only 44 percent of Americans experience a good night's sleep every night. What we observe now in terms of incidence of AD is, at least in part, the consequence of Americans' sleep pattern 30 years ago. According to a National Health Interview Survey, the age-adjusted mean sleep duration in 1985 in US was 7.4 (SE, 0.01) hours, which significantly decreased to 7.18 (SE, 0.01) hours in 2012. The age-adjusted percentage of adults sleeping 6 hours or less was 22.3 percent (SE, 0.3) in 1985, which significantly increased to 29.2 percent in 2012. More than 70 million adults in the US reported sleeping 6 hours or less in 2012. The median bedtime of adults reported in the NHANES study of 11,951 individuals was 2:45 AM on weeknights and still later on Fridays, Saturdays and Sundays. The pattern was even worse in teenagers and young adults. The main obstacle to early bedtimes was electronic devices and social media. People who went to bed later got less slow-wave sleep than those who went to bed earlier. Given what we know about American sleep patterns and the importance of slow-wave sleep for the removal of β amyloid and tau protein, we can expect a skyrocketing incidence of AD 30 years from now.

An understanding of the bidirectional association of slow-wave sleep insufficiency, β amyloid protein, and tau protein and their pathophysiological roles in AD development (Figure 1) provides the key for primary, secondary and tertiary preventive strategies to tackle AD. Sleep insufficiency is a chronic stressor, and sleep is a modifiable risk factor and a tractable target to tackle AD pathogenesis in its early stages. Early night sleep is the daily time of brain cleanup. Otherwise, that time will be the time for building plaques and tangles for development of AD. So, let's clean up, clean up, everybody clean up!

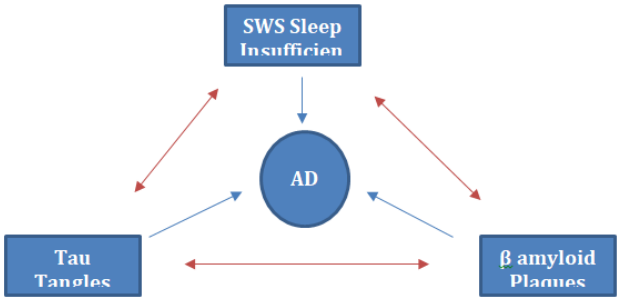

Figure 1: Bidirectional association of slow-wave sleep insufficiency, β amyloid protein, and tau protein and their pathophysiological roles in AD development. There is also some evidence of gene interactions which are not shown here.

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