Sleep

Insufficient sleep is a key lifestyle factor which may well determine whether or not you will develop Alzheimer's disease. Even moderate reductions in sleep for just one week disrupts blood sugar levels so profoundly that you would be classified as pre-diabetic and short sleeping increases the likelihood of your coronary arteries becoming blocked and brittle, setting you on a path towards cardiovascular disease, stroke, vascular dementia and congestive heart failure, all circulatory problems identified by the British Heart Foundation.

It is no coincidence that countries where sleep time has declined most dramatically over the past century, such as the U.S., the UK, Japan, South Korea, and several other nations in western Europe, are also those suffering the greatest increase in rates of the aforementioned physical diseases and mental disorders. The two most feared diseases throughout developed nations are dementia and cancer. Both are related to inadequate sleep. Lack of sleep is connected to cancer but it is also becoming recognised as a key lifestyle factor determining whether or not you will develop Alzheimer's disease (1). Sleep quality - especially that of deep, NREM or non-rapid eye movement sleep, which is the sleep we have in the first part of the night - naturally deteriorates as we age, which is linked to a decline in memory. However, if you assess a patient with Alzheimer's disease, the disruption of deep sleep is far more exaggerated. More telling, perhaps, is the fact that sleep disturbance precedes the onset of Alzheimer's by several years, suggesting that it may be an early warning sign of the condition, or even a contributor to it.

Alzheimer's disease is associated with the build-up of a toxic form of protein called beta-amyloid, which aggregates in sticky clumps, or plaques, in the brain. The amyloid plaques kill brain cells. But the amyloid plaques only affect some parts of the brain and not others, for reasons which remain unclear. The location in the brain where amyloid accumulates during the early stages of Alzheimer's and most severely in the later stages is the middle of the frontal lobe - which is essential for the generation of deep, non-rapid eye movement sleep. The more amyloid deposits in the middle regions of the frontal lobe, the more impaired the deep sleep quality. Whether this particular 'dent' in sleeping brainwave activity represents an early identifier of those who are at greatest risk of developing Alzheimer's disease, years in advance, is being explored (1).

Further studies in mice show that a lack of deep sleep also affects what can be called a night-time power cleanse, where cerebrospinal fluid bathes the brain to flush out metabolic waste. Without sufficient sleep, this does not happen, creating a vicious cycle where amyloid plaques prevent deep sleep, and in turn a lack of deep sleep prevents brain cleansing, leading to more build-up of plaques. From this comes a prediction: getting too little sleep across the adult life span will significantly raise your risk of developing Alzheimer's disease. As a note, it interesting that both Margaret Thatcher and Ronald Reagan - two leaders who were very vocal, if not proud, about only sleeping four to five hours a night - both went on to develop the disease. Donald Trump, the current occupant of the White House and also a vociferous proclaimer of sleeping just a few hours each night.

Digital devices such as fitness trackers can track the beat-to-beat changes in heart rate, known as heart rate variability (HRV), which fluctuate as you transition between light sleep, deep sleep, and REM sleep stages. Whilst asleep each night, the body typically goes through several sleep cycles that last on average 90 minutes. Each cycle alternates between two types of sleep, Light Sleep and Deep Sleep, with less brain activity than REM sleep. Non-rapid eye movement (NREM) includes the stages of light sleep and deep sleep. Periods of deep sleep are typically longer early in the night. Rapid Eye Movement (REM) Sleep. the stage associated with vivid dreams, REM sleep periods are typically longer as the night goes on.

How does memory work? How is it possible, for example, that you can keep on learning new things, day after day, and still remember them weeks, months or years later? How does the brain, which is constantly acquiring information when awake, manages to retain it. It was found that sleep is absolutely crucial to the process, both before learning (to prepare the brain for making new memories) and after learning (to cement those memories and prevent forgetting). It is already known that for fact-based information, such as memorising someone's name or a new phone number, the hippocampus - a long, finger-shaped structure tucked deep on either side of the brain offers a short-term reservoir, or temporary information store, for accumulating new memories. Unfortunately, the hippocampus has a limited storage capacity, almost like a roll of camera film. How, then, does the brain deal with this limited storage — and is sleep part of the process? Memory refreshment happens during lighter, stage 2 NREM sleep (nonrapid eye movement), and specifically short, powerful bursts of electrical activity called sleep spindles. The more spindles during the nap, the greater the restoration of learning. Sleep-spindle bursts of activity are a strikingly reliable loop of electrical current pulsing throughout the brain that repeated every 100 to 200 milliseconds. The pulses kept weaving a path back and forth between the hippocampus, with its short-term, limited storage space, and the larger, long-term storage site of the cortex, an electrical transaction occurring in the quiet secrecy of sleep. In other words, a shifting of fact-based memories from the temporary storage depot (the hippocampus) to a long-term secure vault (the cortex). Sleep had cleared out the hippocampus, replenishing this short-term information repository with plentiful free space so that the learning of new facts could begin again. The more sleep spindles an individual has at night, the greater the restoration of learning ability come the next morning.

The second benefit of sleep for memory is that it effectively clicks the 'save' button on those newly created files. Countless experiments in the past 100 years have shown that sleep provides a memory retention benefit of between 20 and 40 per cent, compared with the same amount of time awake not a trivial amount when you consider studying for an exam, or, in an evolutionary context, being able to remember the locations of food, water and predators. We obtain most of our deep NREM (non-rapid eye movement) sleep early in the night. In contrast, we get most of our rapid eye movement (REM) sleep the stage in which we dream - and lighter NREM sleep late in the night. The different sorts of sleep were discovered in the Fifties, and experiments in which participants were allowed to sleep only for the first or second half of the night were clear: for fact-based memory, it was early-night sleep, rich in deep non-rapid eye movement, that provided better memory retention. Investigations in the early 2000's arrived at a similar conclusion. Using MRI scans, investigators have since looked deep into the brains of participants to see where memories are being retrieved from before sleep, and how that compares with from where they are retrieved after sleep. Those information packets were being recalled from very different geographical locations within the brain at the two different times. Before having slept, participants fetched memories from the short-term storage site of the hippocampus - that temporary warehouse. But things looked very different by the next morning. The memories had moved. After the full night of sleep, participants were retrieving that same information from the neocortex - a region at the top of the brain that serves as the long-term storage site for fact-based memories - where they can live safely, perhaps in perpetuity. This transaction takes place each night when we sleep. The slow brainwaves of deep NREM serve as a courier service, shifting memory packets from a temporary storage hold (hippocampus) to a more secure, permanent home (the cortex). However, his process only works if you get a good night's sleep the same day that you've learned something. If you miss out on that sleep, the memory will not be kept in the same way, even if you try to catch up on subsequent nights. In terms of memory, sleep is not like the bank. If you accumulate a debt, you cannot pay it off later. Recent studies show that sleep also helps you regain access to memories that you could not retrieve before you went to sleep. Like a computer hard drive where some files have become corrupted and inaccessible, sleep offers a recovery service at night, so that you awake the next morning able to locate and retrieve those once unavailable memory files with ease and precision. That's the, 'Ah yes, now I remember' sensation that you may have experienced after a good night of sleep.

Patients with Alzheimer's often suffer from disturbed sleep-wake patterns (circadian rhythm) including poor sleep continuity, increased activity at night and daytime sleepiness. These alterations are more obvious than those observed in normal ageing and have been shown to correlate with the degree of dementia (3). The prediction of Vascular Dementia seems to be particularly strong for daytime sleepiness (7). Increased aggregation of amyloid β has been shown to disrupt sleep cycles suggesting a reciprocal relationship between sleep disturbance and amyloid β deposition. In addition, altered sleep patterns, ie decreased REM sleep is also shown in those with MCI who are at greater risk of developing Alzheimer's (4). Even so, it has not been shown that sleep disturbance in itself predicts the development of dementia in otherwise healthy individuals (2).

How disturbed sleep becomes associated with dementia is not entirely clear. Relatively little is known about the Alzheimer's associated changes to the molecular and cellular components of the circadian clock although macular and optic nerve degeneration is common in older individuals and reduced light input may contribute to the disruption of sleep-wake rhythms. The cholinergic system experiences the greatest deficits in Alzheimer's, impaired cholinergic neurotransmission contributes to the memory dysfunction visible in the early course of the disease whereas normal signalling protects neurons from amyloid β accumulation. It has been proposed that functional cholinergic disconnection between brain regions together with reduced oxygen supply levels contribute to memory loss in Alzheimer's (5, 6).

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