

2014

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The Relationship between Stress and Dementia:

An Investigation of Physiological and Psychological Connections across the Lifespan

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Abstract

While many studies have examined the relationship between stress and dementia, there is no general consensus on what the relationship is, or even whether such a relationship exists at all (Fountoulakis, 2011; Wang, 2012). This literature review consequently considers a broad range of hypotheses on the topic. The central focus of this review was to identify what aspects of stress – throughout life – increase the likelihood of dementia in the elderly. The available research indicates that there is indeed a relationship between stress and dementia, and that stress interacts with dementia risk in multiple ways. There is, first of all, significant evidence to support the hypothesis that chronic stress leads to excessive cortisol release, and that excessive cortisol release damages the brain in ways that can lead to dementia. There is, secondly, significant correlation evidence to suggest that stress interacts with low intellectual stimulation, and that both stress and low intellectual stimulation predict the onset of dementia. Both of these pathways to dementia present multiple opportunities for clinical intervention. This literature review therefore directs some focus toward what aspects of stress would seem to be conducive to treatment. It was found that there are a number of stress interventions that could and should be implemented in large segments of the population. This review, though, fails to completely isolate the effects of stress, and consequently can only suggest a few general interventions. There is still much research that could be done in the area and more specific interventions to be discovered.

The Relationship between Stress and Dementia:

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A recent systematic review carried out by The Alzheimer's Association concluded that, in 2010, worldwide, 35.6 million people were living with dementia. They projected that this number will increase to 65.7 million by 2030 and 115.4 million by 2050 (Alzheimer's). Despite the growing magnitude of the dementia problem, there is – as of yet – no cure for Alzheimer's, frontotemporal dementia, or any of a number of the forms dementia can take. Dementia has proven to be a complicated condition, brought on by a multitude of conspiring factors. Research on such a complicated disease tends to identify – at best – the factors that correlate with incidence, and medical response to correlation information is inherently very tentative. Nonetheless, researchers have recently managed to unearth information about dementia that promises to inform medical intervention with reasonable confidence. While many researchers have maintained noble efforts to seek out mechanistic explanations for each of the various types of dementia, other researchers have identified immediately applicable interventions that can greatly reduce one's risk of getting dementia (of any type). Until dementia cures begin emerging, the medical community ought to place the utmost importance on these practical interventions. One such set of interventions involves targeting stress – more specifically distress – across the lifespan. Chronic and severe stress have both been linked to dementia, and stress could be an effective target for future clinicians, whether their patients are middle-aged, elderly, or actually on the verge of dementia onset.

Findings and Discussion

Stress can be construed in many ways. It is often thought of as a subjective feeling, best determined from self-report. Johansson, with this understanding of stress, chose to use self-

reported stress as a measure of stress in a cohort of middle-aged women. Stress levels for the women were determined at six year intervals. Johansson found that women who reported stress more often were more likely to get dementia (Johansson 2010). Hui-Xin Wang, with a more objective approach, chose to infer workplace stress by looking at the nature of the work itself. He proposed – in accordance with the Karasek strain model (Carayon 1993) – that a job will be most stressful when the worker is faced with heavy demands and possesses little control (high job strain). On the other hand, a job is expected to be least stressful when the worker is faced with light demands and possesses significant control (low job strain). Wang showed – as expected – that individuals who experienced high job strain for the bulk of their career were more likely to get dementia in later life, and that those who experienced low job strain were less likely to get dementia in later life. The explanatory variable in this study was – perhaps surprisingly – not “difficulty of workplace demands,” but instead “level of control”; individuals with heavy demands and high control (active job strain) were less likely to get dementia, whereas individuals with light demands and low control (passive job strain) were more likely to get dementia (Wang 2012). These results indicate that difficult work does not necessarily cause detrimental, dementia-inducing stress, or that, if it does, the detrimental effect of the stress is mitigated by a “high-control” environment. One possible explanation for the protective effect of a “high-control” job could be that “high-control” jobs require more critical thinking and thus allow for more intellectual stimulation. The protective action of intellectual stimulation throughout life is an effect which has been shown (Hultsch 1999) and which may be intimately related to the stress-dementia relationship.

In support of the theory that intellectual stimulation has a protective effect, it has been shown that greater work complexity lowers one’s risk for acquiring dementia (Karp 2009).

Because the relationship between work complexity and dementia is largely explained by the education level of the subjects (Karp 2009), one possible conclusion is that it is education level – and not work complexity – that directly influences dementia susceptibility; low-control, low-complexity jobs might result from or simply coincide with low education. This would be a reasonable conclusion. The importance of education in lowering dementia risk has been shown repeatedly (Ngandu 2007). Using data from the Sao Paulo Aging and Health Study, researchers found that birthplace (whether rural or urban) was more predictive of dementia than both occupation and income, and that among rural-born subjects, those without significant education were twice as likely to become demented. All this would suggest that one's predisposition towards dementia is often determined by education and upbringing early in life, before the age of twenty-five. Yet there is still the possibility that other influences, such as work complexity, acting beyond the age of twenty-five, mediate and explain the predisposition. In the same Sao Paulo study, it was found that occupation is more predictive of dementia than literacy rate, and that when occupation is considered in combination with literacy rate and income level, it is more predictive still, predicting over fifty percent of dementia cases. It appears that if one is able to find an intellectually satisfying job, despite his illiteracy, he can expect significant protection against dementia. High income level or high education level, in the absence of an intellectually stimulating job, appears to offer little protection (Scazufca 2010). Even the little dementia protection high income level does offer may simply be mediated by the generally low stress levels one would expect to come subsequent to financial stability. Stress, in this construction, constitutes a more direct influence on dementia risk than does education, income level, or even occupation; general tendency toward low stress would constitute an explanation for why both educated individuals and individuals in high-complexity jobs are less likely to get dementia.

All of the above interrelated variables which seem to predispose people towards dementia can be seen – in a broad light – as products of socioeconomic status. It is therefore not surprising that socioeconomic status – albeit not directly – appears to be one of the strongest influences on dementia risk. Goldbourt showed that low socioeconomic status corresponds to high dementia risk (Goldbourt 2007). Fotenos showed that high socioeconomic status can delay the onset of dementia, even when brain atrophy is present (Fotenos 2008). A stress theory of dementia – rather than supplant the explanatory variable of low socioeconomic status – would offer stress (and intellectual stimulation) as intermediates in the relationship between socioeconomic status and dementia. Such a theory would add to our understanding of dementia's causes. Whether low intellectual stimulation causes stress – or whether these two negative influences simply tend to coincide – would be difficult to determine; it may be that the two are inherently inseparable. At any rate, the research seems to suggest that early life events – and especially one's birthplace – significantly impact one's dementia risk, but only because these factors often predetermine many aspects of adult life.

In line with the conclusion that early life events are only of secondary importance in considering dementia risk, there is evidence that an elderly person's dementia risk is unaffected by distal life events, unless some chronic life condition, such as chronic stress, arose from the life event. Ravona-Springer, for instance, found that Holocaust survivors were less likely to become demented, despite their traumatic and stressful experience (Ravona-Springer 2011). This result is quite surprising. Holocaust survivors have been shown to be especially susceptible to Post Traumatic Stress Disorder (PTSD) (Kuch 1992), Holocaust survivors with PTSD have been shown to experience unusually fast memory decline in old age (Golier 2002), and memory decline is a key criterion for dementia diagnosis (DSM-IV); it would thus be expected – from

these three studies – that first-hand experience of the holocaust would predict dementia. But this is not the case. One explanation for the apparent contradiction is that PTSD only promotes dementia because it acts chronically: it infuses one's entire life with stress, often into old age. The isolated Holocaust event – though incredibly stressful – by itself has little impact on late life cognition, and may even endow the survivor with a cognitive advantage, as Ravona-Springer suggests. In a similar study to Ravona-Springer's, Qureshi found that war veterans with both a Purple Heart and post-traumatic stress disorder were more likely to get dementia than veterans with only a Purple Heart (Qureshi 2010), even though both groups were presumed to have had equally stressful war experiences. The explanatory difference between the groups may be that only the former group (Purple Heart and PTSD) experienced stress throughout life as a result of the war. The requirement that stress be chronic in order to impact dementia risk would explain the importance of occupation, which constitutes a chronic influence. Also, chronic stress secondary to a traumatic event could partially explain why abused children are more likely to have impaired cognition later in life (Bremner 1999). We can infer that abuse in childhood, much like a lack of education, would send detrimental reverberations across the entire adult life and thereby exert a chronic influence of both stress and intellectual stimulation inhibition. These reverberations would be exaggerated if the abuse had resulted in neurological damage. Reverberations from abuse may sometimes take the form of PTSD, but even if not, it may also be that other types of long-term stress that result from traumatic events that occur before the conclusion of child development are sufficient to explain an increased dementia risk.

It would likely be an oversimplification to say that PTSD modifies dementia risk because it constitutes chronic stress. PTSD, though, does certainly render many of the detrimental effects associated with chronic stress, suggesting a deep connection. Yaffe unearthed many of these

connections between PTSD and chronic stress, most of which involve cortisol. As in the Qureshi study, Yaffe showed that PTSD increases dementia risk. But Yaffe additionally showed, interestingly, that PTSD is accompanied by damage to the hypothalamic-pituitary-adrenal axis, a structure intimately related to chronic stress and to cortisol. The hypothalamic-pituitary-adrenal (HPA) axis consists of the hypothalamus, the anterior lobe of the pituitary gland, and the adrenal cortex of the adrenal gland. The HPA axis responds to stress by releasing cortisol. Cortisol then prompts a number of secondary effects, among them the release of epinephrine and norepinephrine. Cortisol in this way elicits a stress response which lasts longer and is less reversible than the response elicited by epinephrine or norepinephrine alone. Cortisol release, like most human physiological functions, is typically a tightly regulated and beneficial response to stress. But in cases of both PTSD and chronic stress, excessive cortisol release can have a damaging effect (Mayo). If PTSD and chronic stress can be equated regarding dementia risk – as seems to be the case – it is because both are intimately related to excessive cortisol release, which can negatively impact memory in multiple ways.

Excessive cortisol release – first of all – can cause damage to the HPA axis itself, leading to either cortisol deficiency or unregulated cortisol release. Peavy showed that an abnormally low cortisol awakening response (CAR) often accompanies the decline from normal cognitive function to mild cognitive impairment (Peavy 2012). The cortisol awakening response is the natural spike of an individual's cortisol level following nighttime sleep. A low CAR could make an individual less capable of responding appropriately to stress throughout the day, leading ultimately to an increase in stress and possible brain damage. In another study, Tata showed that excess cortisol can result in the loss of synapses within the hippocampus, an important brain component responsible for storing short-term memories as long-term memories (Tata 2006).

Other studies have shown that excessive cortisol release can result in decreased hippocampus volume (Sapolsky 2000), and that high cortisol levels can sometimes explain poor memory in the elderly (Newcomer 1994). Not only would loss of hippocampus function lead directly to memory decline, but – because the hippocampus itself modifies the action of the HPA axis – hippocampus loss could lead to further dysfunction of the HPA axis, initiating a detrimental downward spiral. Even without hippocampus damage, excessive stress could give rise to an unresponsive HPA axis, yielding what is sometimes called a “defeat response.” Because cortisol itself plays a role in memory formation, a “defeat response” could decrease memory capability, blunt cognition, and make the victim less responsive to his environment (Maglione-Garves). Whatever its cause, excessive cortisol release certainly seems to cause dangerous changes in the brain – changes that could lead to dementia.

Cortisol levels, though, do not fully predict the acquisition of dementia. Peavy, for instance, found that ineffective cortisol release does not explain the decline from mild cognitive impairment to full dementia; this stage of decline is instead influenced by current and recent stress, independent of cortisol levels (Peavy 2009). It may thus be that while chronic stress throughout life can weaken brain structures and predispose an individual towards dementia, it is specifically stress in old age, coupled with low intellectual stimulation, which precipitates the final collapse into dementia. But because stress and mental activity in old age are likely functions of stress and mental activity throughout life, we can presume that intellectually active individuals are more likely to retain intellectual habits of mind into old age, and that those who cope with stress well in early life are less likely to fall into an apathetic decline in mental responsiveness at late life. It thus remains the case that socioeconomic status and mental tendencies throughout life can – at least in part – explain the final cognitive collapse into dementia. On the other hand, the

importance of late-life stress insures that even the socioeconomically privileged and intellectually capable are susceptible to dementia. Certain traumatic events in late life may be able to facilitate cognitive collapse, even when, at earlier times in late life, mental activity was high and mental stress manipulation deft. Caring for a spouse with dementia, for instance, may facilitate a cognitive collapse. Often over a short caretaking period, during which brain atrophy would be negligible, dementia caregivers acquire dementia, despite any habits of intellectual discipline they may have possessed at the beginning of the caregiving period. Norton and Vitaliano both showed that dementia caregivers are significantly more likely to get dementia (Norton 2010; Vitaliano 2011). This result is not surprising; without even resorting to physiological explanations, the mental collapse of such an individual can be easily imagined.

First of all, a caregiving spouse is likely to experience depression simply because of the condition of his or her spouse, and depression has been shown to mediate cognitive decline (Vitaliano 2009). A mediating effect of depression would furthermore explain why even non-caregiving spouses are more likely to get dementia when their spouse has dementia (Norton 2010). Depression can then become compounded with the time necessary for caregiving, leaving spouses with little time or willingness to interact with others. This can lead to isolation and loneliness, thereby lowering intellectual discipline and exasperating the depression. Severe depression, when coincident with chronic stress, has been linked to cognitive dysfunction (Steffens 2006). Moreover, all of this has the potential to reduce the amount of quality sleep, which would add yet more detrimental influence into the complex cycle of cognitive decline. Even without physiological damage to the hippocampus during the caregiving phase, the retention and exercise of complex cognitive processes throughout this period would be incredibly difficult. It may be that high-degree intellectual activity, which is often and maybe inevitably

coupled with stress control, is powerless to operate effectively in such a hopeless situation. The caregiver's situation is made all the more dire if the death of the care recipient is expected; such anticipatory grief has been shown to greatly increase caregiver burden. In light of all this, it is not surprising that Alzheimer's disease caregivers exhibit decreases in memory (Mackenzie 2007) and decreases in verbal fluency (Lee 2004). Such losses, without effective compensatory action by the brain, will only hasten the departure from rigorous, disciplined, and coherent thought. Significant recovery from such a collapse may be impossible, given the weak infrastructure of the brain in old age. It is thus evident that stress and its counterparts can not only weaken the infrastructure of the brain throughout life, but can precipitate the final collapse into irreversible dementia.

Conclusion

Stress can be thought of as detrimentally impacting the human brain in two ways. Firstly, chronic stress can result in excessive cortisol release and damage to a vital memory component of the brain: the hippocampus. Such damage can obviously lead directly to memory loss and therefore dementia; it can also predispose the brain to dementia later in life. Secondly, stress can become coupled to minimally-satisfying intellectual activity, which can make an individual more likely to succumb to depression and cognitive apathy, and therefore more susceptible to dementia. These two impacts of stress – direct brain damage and indirect brain activity reduction – are not independent of each other. For instance, hippocampus damage – as from child abuse – could lead to chronic stress, which could then lead to low cognitive stimulation throughout life. Conversely, chronic stress could lead to neurological changes that could in turn lead to low cognitive stimulation. Also, brain activity reduction late in life – resulting from proximal stress events – could have an exaggerated effect if coincident with a brain that has been structurally and

neurologically damaged by a lifetime of high stress and high cortisol release. In any case, it is a combination of predisposition and the immediate effect of stress (and its counterparts) which eventually causes the full decline into dementia. Therefore, as one might expect, stress-induced cognitive decline can be observed most poignantly in dementia caregivers, in whom a number of factors coalesce to encourage the onset of dementia. In these individuals, cognitive decline is accelerated if chronic stress has already weakened brain structures, or when chronic stress has encouraged minimal interpersonal interaction and low brain stimulation (Vitaliano 2011).

The relationship between stress and dementia is obviously complex and somewhat convoluted. Nonetheless, there are some practical interventions which may be effective in protecting against dementia, and which therefore should be implemented when possible. First of all, immediate action should be taken any time stress or depression causes an individual to become withdrawn; maintaining high intellectual and social stimulation throughout life is very important. For the same reason, any career that is not intellectually satisfying and which gives the worker little control should be viewed as dangerous, to be supplemented with intellectually stimulating activities that are not stressful. Also, cortisol levels should be monitored in individuals who are experiencing chronic stress. If cortisol levels are excessively high, therapeutic measures should be taken to reduce stress levels. Active meditation exercises have been proven to be effective in this capacity (Lavretsky 2013). Such meditation exercises should also be employed by any elderly individuals at risk for dementia. All elderly individuals, it should be noted, are to some degree susceptible to dementia. With age, the neurological infrastructure of every human weakens, regardless of what preventative measures have been taken throughout life. Clinicians should therefore encourage even their healthiest elderly patients to make efforts to deal with stress appropriately, to maintain active and hopeful minds, and to,

above all, retain healthy relationships with a number of other people, lest they slip into isolation.

None of these measures are counterintuitive, but their importance is often underestimated.

Individuals who want to live dementia-free would be wise to give an especially high priority to some of these simple interventions.

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