

IB Extended Essay

The Effects of Sleep Deprivation and Substance Abuse as Stressors on the Adolescent Brain

Research Question: How do the psychological effects of sleep deprivation and alcohol abuse on adolescents compare?

IB Subject: Psychology

Personal Code:ggr348

Session: May 2017

Word Count: 4000 Words

Abstract

This essay investigated the research question: how do the psychological effects of sleep deprivation and alcohol abuse on adolescents compare? Sleep deprivation was chosen as it is a problem not widely known or often considered unimportant. Alcohol abuse was chosen because it is considered a huge problem that significantly impacts the lives of adolescents that use it. Thus, the comparison of the two would lend itself greatly to emphasizing the effects of both on the adolescent brain and the fact that both can cause great damage. In an attempt to answer the question, the essay draws on several psychological and neurological studies that cover functional, neurotransmitter/hormonal and structural effects of both sleep deprivation and alcohol abuse. These are analyzed separately and revealed several common microbiological effects between the two. However, for the most part, the microbiological effects remained distinct between the two topics. The most significant common factors were decreases in hippocampal volume, problems in various types of memory, and interruptions of normal hormone and neurotransmitter levels. These were caused by a variety of direct effects from either sleep deprivation or alcohol abuse, including neuron death, neural reorganization, and increased or decreased hormones/neurotransmitter secretions. This led to the conclusion that sleep deprivation and alcohol abuse have very different negative microbiological effects, yet both ultimately lead to very similar large-scale brain issues and cognitive deficits. However, their differences were not relevant in terms of the extent they could harm those inflicted. Furthermore, it was concluded that sleep deprivation is an underestimated danger to adolescents, as it presents similar consequences of alcohol abuse, yet receives far less preventative measures from parents or school administrators.

272 words

Table of Contents

Abstract.....	2
Introduction.....	4
Sleep Deprivation and Alcohol Abuse in Terms of Adolescent Life and Development.....	5
The Effect of Sleep Deprivation on the Physiology on the Adolescent Brain.....	8
The Effect of Alcohol Abuse on the Physiology on the Adolescent Brain.....	11
Evaluation of Interconnections Between Sleep Deprivation and Alcohol Abuse.....	14
Conclusion.....	16
Sources.....	18

Introduction

The idea of the teenager is relatively new; an anonymous writer once said, “young people became teenagers because we had nothing else for them to do” (Jenson & Nutt, 2015, p. 17). While the psychological development of the teenager has always been there, for most of history teenagers were just considered slightly less experienced adults. This shaped the world adolescents live in; however, over the last century, the idea that it is a distinct period of psychological development has begun to take hold. This has left the world and the field of psychology only just beginning to understand how the adolescent mind works and fits into society (Jenson & Nutt, 2015, p. 17).

As mentioned, psychological study of the adolescent brain is relatively new, but some is known about it. Psychologically speaking, adolescents is a period of brain development where teenagers transition from dependent children to independent adults. However, it is not simply a period of transition- in order for this change to take place the brain goes through rapid, extreme modification that leaves emotions wired, reactions extreme, and personalities altered. The neuroplasticity required for this leaves the brain at a point of amazing potential, as well as amazing vulnerability. This is why problems that are treated as no big deal, as well as problems society seems to blow out of proportion, are actually an even bigger problem than may be thought, as they affect an adolescent brain differently than they would affect a matured one (Jenson & Nutt, 2015)(Ziegler et al., 2005). This essay will investigate elements of this issue, using two problems that commonly plague adolescents and attempt to answer the question of **how the psychological effects of sleep deprivation and alcohol abuse on adolescents compare?**

Sleep deprivation is generally considered a normal part of adolescence, as opposed to alcohol abuse, which is considered dangerous and self-deprecating behavior. However, the effect of both of these is dangerously underestimated. Evidence shows that sleep deprivation and alcohol abuse have very different negative microbiological effects on the adolescent brain. Yet, both ultimately lead to very similar large-scale brain issues and cognitive deficits.

Sleep Deprivation and Alcohol Abuse in Terms of Adolescent Life and Development

Sleep and drug use are treated very differently in school systems, despite the fact that the number of adolescents who are sleep deprived is just as worrying, if not more so than the number of those abusing alcohol. Significantly more kids get less sleep than recommended than have recent alcohol use, however, drug and alcohol abuse is treated as a more serious problem. “According to a 2006 National Sleep Foundation poll, the organization’s most recent survey of teen sleep, more than 87 percent of high school students in the United States get far less than the recommended eight to [ten] hours” (Richter, 2015). This may seem comparable to alcohol abuse at first glance, as “lifetime rates indicate that 73% of youth have used alcohol...by their senior year of high school” (Squeglia, Jacobus, & Taport, 2009). However, one is addressing the long-term problem of sleep deprivation, whereas the other is referring to alcohol use at least once in their life. When a shorter window of time is observed, it appears that fewer adolescents use alcohol on a regular basis. “More than 10 million American youth aged 12 – 20 years old report past-30-day use of alcohol. This represents more than 28% of this age group” (Ziegler et al., 2005, p. 24).

None of these statistics should be underestimated in their severity. In response to substance

abuse, especially in adolescents, the Red Ribbon program began, and it is estimated 80 million people participate each year (Red Ribbon, n.d.). This has proved wildly successful, as the move to reduce substance abuse in minors is considered crucial to many people. In comparison, a slow effort is being made to move school times forward as a means of allowing kids to get more sleep. It is still considered to be experimental, despite the fact that the first successful study was conducted in the late 1990s, and has been followed up numerous times since then. It began in Edina, Minneapolis; concluding students were less depressed, more likely to succeed, and gained about five hours of sleep time per week. Now, Start School Later.net estimates a few hundred schools in America have made a change, which at its best still represents an effect on less than 1 million high schoolers (2016). A cause of this may be the claim that changing school start times would be too drastic to pull off (O' Malley & O'Malley, 2008, pp. 90). High school aged students will naturally sleep from 1:00a.m to 11:00a.m if left to their own devices; if schools were to change to accommodate fully they would not start until noon or later (Jenson & Nutt, 2015, pp. 126). However, an interesting study done by Edward and Mary O'Malley found that while 7:30 or 8:00a.m is too early, 8:15 seemed to be a sweet spot, and starting at that time or slightly later showed a huge improvement in alertness and performance (2008, pg 90-91). Another concern is that if schools were to start late, students would respond by simply staying up later. However, a survey of a school that changed their times done by O'Malley found that not to be true, saying, "We found that student bedtimes were essentially unchanged compared with before the schedule change, but later rise times were reported such that they obtained nearly all of this time as additional sleep" (O' Malley & O'Malley, 2008, pp. 90-91). Of course, this data does not erase all potential problems of changing school start times, but it does make the prospects sound more practical and productive.

As the fights against both sleep deprivation and alcohol abuse move forward, it is important to know that adolescence is a critical time to face these problems, as the brain goes through many changes during this time. As children, humans have too much gray matter and not enough white matter; therefore, adolescence is a time of rapid pruning and growth (Squeglia, Jacobus, & Taport, 2009). These create connections in the brain and train it to live as an adult, losing skills no longer needed and developing new ones, such as independence (Squeglia, Jacobus, & Taport, 2009). These changes occur all over the brain, but it is done in concurrence with the development of specific parts of the brain that are only during adolescence beginning to fully develop. Brain maturation in humans occurs from back to front; another way of saying this is it develops from old to new. The most recently evolved human functions, such as reasoning, logic, and even some aspects of risk/reward evaluation are the newest, located in the frontal lobe, and therefore develop last (Jenson & Nutt, 2015, pg 38). An example of older and sooner developing traits is facial recognition. As a social species, recognizing other members of our own species has been critical for most of our existence, and it develops within a few days of birth (Norcia & McClure, 2012). Another event that occurs during the maturing of the brain is dendritic pruning. Dendrites are the receptors of signals sent by other neurons, and as they are pruned, the brain is more likely to misfire or mix signals of neurotransmitters until it adjusts to the new landscape of neurons. Finally, there are also hormones that can affect behavior. Researchers have discovered that puberty is initiated by a gene producing protein, kisspeptin, that eventually trigger the pituitary gland to release a storage of hormones. These are entirely new to the teenager, and again, adjustment is required for teenagers to behave normally under this influence of hormones. These three developmental processes not only leave the brain more vulnerable to any harmful agents, it also makes adolescents less likely to make wise decisions

related to their health, only increasing their susceptibility to sleep deprivation and drugs.

Between the lack of reasoning skills, mixed neurotransmitter signals, and an onslaught of mostly unfamiliar hormones, the risks and rewards of behavior is not properly evaluated (Jenson & Nutt, 2015). Additionally, interference with this process, such as sleep deprivations or illicit substance use can fundamentally change the structure many parts and functions of the brain for life, as once the adolescent period has ended, change is possible, but much harder (Squeglia, Jacobus, & Taport, 2009).

The Effect of Sleep Deprivation on the Physiology of the Adolescent Brain

In psychological studies, sleep deprivation is referred to in a range of ways, such as limiting sleep to five hours a night or skipping a night of sleep. However, based on a comment by the National Sleep Foundation, it seems that chronically getting far less than the recommended eight to ten hours is enough to cause concern and lead to potential health problems. Studies found negative effects of sleep deprivation both in limited sleep and in no sleep (Beebe, n.d.)(Richter, 2015), though total sleep deprivation would show much stronger effects. In reference to sleep deprivation of adolescents, a limited amount of sleep per night is more applicable to real problems faced by adolescents on a regular basis, therefore all studies in this analysis reference sleep deprivation of four to six hours of sleep per night.

Being sleep deprived is more than just feeling tired, it can cause irregular levels of hormones in the brain, which may lead too real, long-term brain damage. Several sleep studies show seemingly insignificant small differences in behavior when sleep deprived. One of these ‘small’ things is increased fear and anxiety at specific stressors while sleep deprived (Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010 [abstract]). This is likely indicative of increased

cortisol present in the brain, which is no small matter. Of the three major stress and fear hormones, adrenaline, cortisol, and norepinephrine, cortisol fits best. Adrenaline and norepinephrine are both immediate reactions that filter out of the body relatively easily. They are both characterized by a spike in energy, i.e the flight or fight response. Excess cortisol for unnecessarily long periods of time is the outcome in many types stressful situations, whereas the same regulation problem with adrenaline or norepinephrine is almost unheard of (Klein, 2013). The more-than-normal fear presented by subjects in the study suggest that their system already had cortisol in it and that the sleep deprivation was the cause of the increased levels, as all other factors were controlled. Because cortisol is a hormone designed for only short periods of time, long-term exposure caused by chronic sleep deprivation has negative, long-term cognitive consequences. A study done to test the effect exposure of cortisol in the brain found a reversible decrease in verbal declarative memory after just four days of exposure (Newcomer et al., 1999, [Abstract]). However, this study was done over only four days, and cannot safely be generalized to those who experience longer term sleep deprivation, and may experience more extreme and possibly permanent damage.

Ethical issues prevent experiments on humans to test hypotheses suggesting potentially permanent brain damage, though there are animal studies to test how this may affect humans. A study on tree shrews by Ohl et. al over four weeks of exposure tested the effect on hippocampal-mediated memory and hippocampal volume, and observations found a trend towards reduced hippocampal volume. The study tested both administering cortisol to the shrews, the method used in the four-day human study, as well as stressing the shrews to cause natural cortisol increases. The effects were observable sooner in the naturally occurring cortisol subjects, indicating that more extreme effects than the ones found in the human study may be present in

people with naturally occurring high cortisol levels, such as those caused by sleep deprivation. Additionally, this study was significantly longer than the study done on humans, showing that longer term cortisol exposure is more severe than exposure over just a few days. The study concluded that chronic cortisol exposure does affect hippocampal-mediated memory as well as hippocampal volume (Ohl, Michaelis, Vollmann-Honsdorf, Kirschbaum, & Fuchs, 2000). No recovery period was allowed for, so it cannot be confirmed from this study that the damage is permanent, however, such a drastic change in brain structure is less likely to reverse itself.

It is not certain that these studies can be applied to sleep deprivation. The stressed response of subjects who have been sleep deprived indicates cortisol, which indicates that lack of sleep is a stressor on the body, but it is not a guarantee. However, another study tested many of the conclusions drawn that were previously based only on extrapolation from other studies. Bruce McEwin conducted this study, and his results lead him to conclude that, “these changes in brain and body are further evidence that sleep deprivation is a chronic stressor and that the resulting allostatic load can contribute to cognitive problems” (McEwin, 2006). What he observed were changes in many aspects of bodily functions, including appetite, insulin, nerve function, blood sugar levels, etc. Relevant to the aspect of sleep deprivation being studied, memory and the hippocampi, he observed an increase in evening cortisol levels, as well as stress in the hippocampi that eventually lead to impaired memory. Many of these were observed in animals, however, McEwin was comfortable applying them to humans, saying “Structural and functional magnetic resonance imaging studies in depression and Cushing's disease, as well as anxiety disorders, provide evidence that the human brain may be similarly affected” (McEwin, 2006). This also indicates Ohl's study done on tree shrews may be generalizable to humans as well.

However, even four weeks is just a fraction of the sleep deprivation many adolescents experience throughout high school. It may seem students would not constantly be in a sleep deprived state that would contribute to such high neural stress, however, the brief relapses on weekends and school breaks are often not enough to override the losses during school. Adolescents do have an opportunity to catch up on sleep over the weekend, but it is not enough to bring them out of a sleep-deprived state. During research on adolescent sleep done in the 70s, researchers observed, “when older adolescents were restricted to just five hours of sleep a night, they would become progressively sleepier during the course of the week. The loss was cumulative, accounting for what is now commonly known as sleep debt” (Richter, 2015). This means that in theory, a student who only gets six hours of sleep on a school night would have a sleep debt of around fifteen hours by the time the weekend rolled around. In order to make up that debt as well as maintain the minimum of eight recommended hours per night, fifteen and a half hours Friday and Saturday night would be necessary. This need for so much sleep brings in other negative factors, for example, research is currently being done to investigate a possible link between excessive sleep (several hours more than recommended per night) and higher mortality rates (Youngstedt & Kripke, 2004).

The Effect of Alcohol Abuse on the Physiology of the Adolescent Brain

“According to the National Institute of Health, any alcohol use by an underage youth is considered to be alcohol abuse” (Jenson & Nutt, 2015, p. 127). Because of this, even alcohol use a few times or infrequently is considered in the studies mentioned. Results only applicable to subjects that drink heavily are often mentioned as ‘heavy’ or ‘binge’ drinking, and studies using infrequent drinking or other unusual patterns will go into detail as to what those patterns are. Nonetheless, this analysis is in keeping with the idea that all underage alcohol use is alcohol

abuse.

The effects of alcohol abuse on the teenage brain can be hard to see or misleading. The most common result of binge drinking is a blackout or memory loss, caused by an acute dysfunction of the hippocampus. It is easy to disregard as it is generally temporary, but, this is not the only possible memory problem related to drinking, just the most obvious. Blackouts of full or significant events can be indicative of serious hippocampal damage. Even without these incidences, “researchers have shown a direct correlation between hippocampal volume and the age of onset of alcohol abuse” (Jenson & Nutt, 2015, pp. 125-140). This information can be further supplemented by a study on adolescent rhesus monkeys during which the subjects were given regular, strong doses of alcohol every day. This reveals the specifics of what occurs to the hippocampus during heavy alcohol abuse; “over a period of eleven months, Taffe [the researcher] discovered a significant reduction not just in the number of neurons in the monkeys’ hippocampi but also in the number of neural stem cells...the production of neurons the monkeys’ hippocampi was slashed by more than half, and what neurons remained looked damaged” (Jenson & Nutt, 2015, pp. 125-140). The hippocampus has several important functions within our ability to remember and learn, but plasticity allows deficits like this to hide, to an extent.

While adolescents appear to bounce back from alcohol abuse very quickly, the neuroplasticity that causes this illusion is the very reason the brain of an adolescent is more damaged than the brain of an adult when exposed to substance abuse (Squeglia, Jacobus, & Taport, 2009). Two studies done by Squeglia, Jacobus and Taport’s lab combined show that the adolescent’s ability to apparently bounce back from heavy drinking is the result of subtle neural reorganization that compensates for the influence of substance abuse. They first tested spatial working memory in teens with 1-2 years of drinking and found that while the subjects performed

similarly on the tests, activation patterns in the subject's brain were different, suggesting compensation. However, it is not sustainable, and once the sensitive period for pruning in the teenage brain has passed it can be hard to recover from (Squeglia, Jacobus, & Taport, 2009). Another test done on older subjects who had been drinking longer showed the drinkers no longer performed similarly on the SWM test than their nondrinking counterparts, as well as decreased activity in the parts that had abnormally lit up for drinkers in the younger group. This reorganization done in the prime time for neuroplasticity may ingrain the addiction more deeply into the brain than if the subject started drinking when they were older, explaining why those who start drinking before the age of eighteen have higher rates of adulthood alcohol abuse disorders and relapses than those who do not (Jenson & Nutt, 2015, pp. 125-140). This failure to 'bounce back' applies to those who do not binge drink as well; even subjects who have more than three drinks annually, but did not drink every month have behavioral problems in school, decreased career prospects, and a higher rate of substance use as an adult (Ziegler et al., 2005, p. 25).

One study on the effect of alcohol abuse on hippocampal volume found that the volume of the left hippocampus decreases with binge drinking. A greater left to right hippocampal ratio is generally indicative of greater verbal learning, but in the subjects, this reduction in hippocampal volume showed impaired verbal learning. A study on verbal encoding found that binge drinkers did not utilize their working memory systems when encoding verbal information, yet they encoded only marginally fewer words (Squeglia, Jacobus, & Taport, 2009). Another example of this was explained in Jenson's book, *The Teenage Brain*: "When asked to solve a simple problem, the adolescents with alcohol-use disorders showed less activity in their prefrontal cortex than a control group and had to rely on other areas...to figure out the answers to their problems" (Jenson

& Nutt, 2015, p. 137). These phenomena show that those with substance use disorders may appear to have mostly normal functioning brains if their brain activity is not monitored as well. This change in brain functioning may be a result of the previously mentioned neural reorganization experienced within the brain of an adolescent who drinks, and only highlights how well substance use disorders can go unnoticed.

One of our most basic neural functions is the communication of different neurons via neurotransmitters. Alcohol interferes with many neural systems, specifically favoring inhibitory influences. This can cause one of the most severe forms of neural reorganization, as after long-term exposure the brain compensates for this change and tries to push the balance back towards equilibrium. This can occur in the form of reducing the number of inhibitory receptors or in the overproduction of inflammatory neurotransmitters. This can have a heavy impact on the memory systems, as they are highly complex and require the inhibition and inflammation of multiple neurotransmitter systems. A specific example of this effect can be seen in the process of long-term potentiation, which is considered critical for memory processes. “In general, LTP seems to require activation of glutamate receptors and inhibition of GABAA receptors” (Valenzuela, 1997, p.146). Two studies, Lovinger et. al and Weiner et. al have found alcohol exposure to have the opposite effect. Longer term exposure would likely cause the brain to attempt to rebalance the neurotransmitter levels, further skewing the memory storage process (Valenzuela, 1997, p.146). This shows clearly how alcohol can interfere with crucial memory processes, even on a molecular level.

Evaluation of Interconnections Between Sleep Deprivation and Alcohol Abuse

The study of both sleep deprivation and alcohol abuse show that their effects have many

similarities, however it is important to consider that sleep deprivation and alcohol abuse can affect each other: to what extent are they the causes or consequences, and what impact do social circles have on this? Alcohol has long been considered a cause of sleep deprivation, but new studies are showing it may be more complex than that. A recent study showed not only does substance abuse cause sleep deprivation, but the effect also works backward, and across people. Patterns were found among social groups of up to four degrees of separation affecting each other, showing that if a friend's friend was sleep deprived it could impact one's likelihood of substance abuse (Mednick, Christakis, & Fowler, n.d). This phenomenon is still in the early stages of investigation. Additionally, while substance abuse interrupting sleep is currently the accepted version of events, it is worth noting that sleep deprivation has been found to increase delinquent behavior, including drugs (Clinkinbeard, 2011). Both sleep deprivation and alcohol abuse have been known to cause the abuse of caffeine. Teenagers abuse caffeine to keep themselves awake during school as well as using it while drinking to keep them from passing out (Jenson & Nutt, 2016, pp. 86-102,125-140). These interconnections make it necessary to consider the possibility that some effects found in the cited studies may share causes between sleep deprivation and alcohol abuse, possibly caffeine as well, as some of the subjects may have been experiencing multiple influences. This is especially important because of the nature of what was being studied; it is unethical to cause sleep deprivation or alcohol abuse among people, so most results found on humans were gained through interview or observation of subjects who were already inflicted, making it much harder to control for other factors. Regardless, it is still relevant to observe adverse consequences of an adolescent's lifestyle in the hopes to understand how their health is affected by the normal or not-so-normal behaviors of their everyday lives.

Conclusion

In the fast paced and competitive world we live in today there are thousands of stressors that put the vulnerable changing mind of a teenager at risk. Thus, it is important for teachers, parents, and scientists to objectively identify and respond to these stressors. In order to investigate this, two stressors have been compared; one considered one of the most dangerous threats to adolescents and one largely ignored and accepted. Alcohol abuse and sleep deprivation respectively. Both were analyzed using effects on structures, functions, and hormones/neurotransmitters as factors, drawing information from several research studies published in multiple journals. To answer the research question, **how do the psychological effects of sleep deprivation and alcohol abuse on adolescents compare**, the results of the investigation found that they share many significant negative consequences and that their differences were not relevant in terms of the extent they could harm those inflicted. This lead to the further conclusion that sleep deprivation has been underestimated as a threat to adolescents' psychological health.

Through the separate analysis of both sleep deprivation and alcohol abuse it is clear to see that the two situations share remarkably similar consequences. The greatest of these appeared to be changes in hippocampal volume, interruption of normal hormone and neurotransmitter levels in the brain, and problems in various types of memory. It is also fascinating to note that these differences arose from separate microbiological factors in the brain. Both sleep deprivation and alcohol abuse cause significant alterations to the brain on both a macro-biological and microbiological scale. The macro-biological effect, or hippocampal volume loss, is caused by either cortisol exposure or neuronal death respectively. A similar case is shown in the microbiological effects, which are either excess cortisol due to allostatic stress, or an imbalance

or inhibitory and inflammatory neurotransmitters, respectively. However, these slight differences are semantics, as similar detrimental effects are observed.

Sleep aids memory, learning, and appetite, and allows the body to clean itself from the day. It is essential to health (Jenson & Nutt, 2015). Lack of it has the opposite effect- which is similar, in fact, to the effects of alcohol. Alcohol impedes memory encoding, interferes with normal bodily functions such as appetite, and clutters your body and brain (Jenson & Nutt, 2015)(McEwin, 2006)(Squeglia, Jacobus, & Taport, 2009). When the two are compared side by side, it becomes clear that they are nearly identical evils facing the youth of today.

Sources

Beebe, D. W. (n.d.). Cognitive, behavioral, and functional consequences of inadequate sleep in children and adolescents. *Pediatric Clinics of North America*, 58(3), 649-665. Retrieved from <http://www.sciencedirect.com/science/article/pii/S0031395511000186>

Clinkinbeard, S. S. (2011, July). Sleep and delinquency: Does the amount of sleep matter? *Journal of Youth and Adolescence*, 40(7).

Jenson, F. E., & Nutt, A. E. (2016). *The teenage brain: A neuroscientist's survival guide to raising adolescents and young adults*. New York City, NY: HarperCollins.

Klein, S. (2013, April 19). Adrenaline, cortisol, and norepinephrine: The three major stress hormones, explained. *The Huffington Post*. Retrieved from http://www.huffingtonpost.com/2013/04/19/adrenaline-cortisol-stresshormones_n_3112800.html

McEwin, B. S. (2006). Sleep deprivation as a neurobiologic and physiologic stressor: Allostasis and allostatic load. *Metabolism*, 55(2), S20-S23.

Mednick, S. D., Christakis, N. A., & Fowler, J. H. (n.d.). The spread of sleep loss influences drug use in adolescent social networks. PLOS.

<http://dx.doi.org/10.1371/journal.pone.0009775>

Newcomer, J. W., Selke, G., Melson, A. K., Hershey, T., Craft, S., Richards, K., & Alderson, A. L. (1999, June). Decreased memory performance in healthy humans induced by stress-level cortisol treatment. *Archives of General Psychiatry*, 56(6). Retrieved from <http://archpsyc.jamanetwork.com/article.aspx?articleid=1673779>

Norcia, A., & McClure, M. (2012, December 11). Infants process faces long before they recognize other objects, Stanford vision researchers find [Newsgroup post]. Retrieved from Stanford News website: <http://news.stanford.edu/news/2012/december/infants-process-faces-121112.html>

Ohl, F., Michaelis, T., Vollmann-Honsdorf, G. K., Kirschbaum, C., & Fuchs, E. (2000, June). Effect of chronic psychosocial stress and long-term cortisol treatment on hippocampus-mediated memory and hippocampal volume: A pilot-study in tree shrews. *Psychoneuroendocrinology*, 25(4), 257-263 Retrieved from https://www.researchgate.net/publication/12589584_Ohl_F_Michaelis_T_Vollmann-Honsdorf_GK_Kirschbaum_C_Fuchs_E_Effect_of_chronic_psychosocial_stress_and_long-term_cortisol_treatment_on_hippocampus-

mediated_memory_and_hippocampal_volume_a_pilot-study_i

O' Malley, E. B., & O'Malley, M. B. (2008). School start time and its impact on learning and behavior. In *Sleep and psychiatric disorders in children and adolescents* (pp. 79-94). Informa Health Care.

Red ribbon week fact sheet [Pamphlet]. (n.d.).

Richter, R. (2015, October 8). Among teens, sleep deprivation an epidemic [Editorial]. Retrieved August 6, 2016, from Stanford Medicine News Center website: <https://med.stanford.edu/news/all-news/2015/10/among-teens-sleep-deprivation-an-epidemic.html>

Squeglia, L. M., Jacobus, J., & Taport, S. F. (2009, January). The influence of substance use on adolescent brain development. *Clin EEG Neurosci.*, 40(1), 31-38. Retrieved from <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2827693/>

StartSchoolLater.Net. (2016). Retrieved October 27, 2016, from <http://www.startschoollater.net/>

Talbot, L. S., McGlinchey, E. L., Kaplan, K. A., Dahl, R. E., & Harvey, A. E. (2010, December).

Sleep deprivation in adolescents and adults. *Emotion*, 10(6), 831-841. Abstract retrieved from

PsycARTICLES database.

Valenzuela, F. C. (1997). Alcohol and neurotransmitter interactions. *Alcohol Health and*

Research World, 21(2), 144-147. Retrieved from

<http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.523.5258&rep=rep1&type=pdf>

Youngstedt, S. D., & Kripke, D. F. (2004). Long sleep and mortality: Rationale for sleep

restriction[Abstract]. *Sleep Medicine Reviews*, 8(3), 159-174.

<http://dx.doi.org/10.1016/j.smr.2003.10.002>

Ziegler, D. W., Wang, C. C., Yoast, R. A., Dickson, B. D., Macffree, M. A., Robinowitz, C. B.,

& Stirling, M. L. (2005). The neurocognitive effects of alcohol on adolescents and college

students. *Preventative Medicine*, 40(1), 23-32. Retrieved from [http://www.sciencedirect.com/](http://www.sciencedirect.com/science/article/pii/S0091743504002658)

<http://www.sciencedirect.com/science/article/pii/S0091743504002658>