

## **The Relationship between Sleep and Exceptional Behavior: A Literature Review**

Every stage of our lives, from infancy to old age, is punctuated by bouts of unconscious brain activity called “sleep.” We know that sleep is good for our physical health, but we often overlook sleep’s enormous effects on our mental health. The amount of sleep we get has both short term impacts on cognition and long term impacts on mental health. The goal of this paper is to compile these various findings about sleep’s relation to long term and short term mental health and present them in a manner that is useful for future research. Sleep’s relation to developmental disorders, adult mental illnesses, and structural changes in the brain are highlighted and suggestions about the implications of these findings are made.

### ***What is Normal Sleep?***

The characteristics that define sleep change throughout the human lifetime. For example, a split between active and restful periods is first noticed just 8 weeks after conception. However, this kind of “sleep” bears little resemblance to the sleep of children and adults, or even the kind of sleep exhibited later in gestational development. The determination of what sleep is during the gestational period is based solely on observations of physical movements of the fetus’s limbs and eyes through the use of real-time ultrasonography. Later (after birth) researchers record EEG, respiration, heart rate, body temperature, and behavioral data to answer questions involving when and how sleep is occurring. It has been found through the use of these methods that sleep is, indeed, a brain-state that differs over the

course of human development. Therefore, to answer the physiological question “What is normal sleep?” we must provide a response in terms of physical development (a representation of the development of sleep can be seen in Figure 1).

As mentioned previously, the first observations of sleep-like activity are recorded just 8 weeks after conception. At this time in development the fetus oscillates between an active state and a restful state. This means that there is only one “sleep stage” in the earliest period of development. However, around 25 weeks after conception sleep and wakefulness become more defined and sleep itself develops into a two stage process. These two sleep stages are called “Active Sleep” and “Quiet Sleep.” During sleep the fetus experiences these stages in cycles lasting from 40 to 60 minutes long. At 35 weeks of development, a fully formed sleep cycle emerges. Birth occurs soon after, generally between 38 and 42 weeks after conception.

The sleep that occurs in humans from birth through 6 months of age is believed to be limited by the physical development of the brain and central nervous system. In fact, many researchers believe that the development of sleep across the human lifetime is in concert with – and potentially determined by – the physical development and aging of the brain and central nervous system. Directly after birth through 6 months of age the cortex is unable to sustain the type of high-frequency and high-amplitude brain waves that are recorded during adult sleep. Infants at this stage also do not display REM (rapid eye movement) sleep. Instead, three different types of sleep states have been recorded from these infants. They are called “Active Sleep,” “Quiet Sleep,” and “Indeterminate Sleep” (Gomez, Newman-Smith, Breslin, & Bootzin, 2011).

“Active Sleep” is characterized by constant electrophysiological activity throughout the neocortex and can reach 70  $\mu\text{V}$  (if occurring before the “Quiet Sleep” stage) or between 20 and 50  $\mu\text{V}$  (if occurring after the “Quiet Sleep” stage).<sup>1</sup> Interestingly, the brain activity of Active Sleep with amplitudes between 20 and 50  $\mu\text{V}$  is indistinguishable from the brain activity that occurs while the infant is awake (Gomez, Newman-Smith, Breslin, & Bootzin, 2011).

The second kind of sleep, “Quiet Sleep,” is not characterized by waves as is Active Sleep. Instead, Quiet Sleep is characterized by an EEG recording of a background pattern of “noise” with alternating “bursts” of delta and theta waves (which have amplitudes as high as 200  $\mu\text{V}$ ).<sup>2</sup> Within the delta and theta waves there are also intermittent periods of alpha and beta activity (which have amplitudes between 50 and 70  $\mu\text{V}$ ). The dynamic nature of Quiet Sleep has been coined “trace alternant” and “discontinuous sleep.” Quiet Sleep is also known to show synchronization of electrical activity across both hemispheres of the brain (Gomez, Newman-Smith, Breslin, & Bootzin, 2011).

Indeterminate Sleep is the term used to describe the disorganized EEG signals that comprise infants’ transitions between Active and Quiet Sleep. Total amount of sleep for infants at this age is normally between 12 and 17 hours a day. This total amount of sleep is split into periods of sleep lasting between 3 and 4 hours with frequent arousals, called polyphasic cycles.

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<sup>1</sup> “ $\mu\text{V}$ ” is the unit of “micro-volts” and refers to the amplitude of brain waves recorded by EEG. When referring to EEG data, amplitude indicates the number of volts and frequency (“Hz”) indicates the number of waves occurring within a second.

<sup>2</sup> Sleep is characterized by four different types of waves recorded by EEG. These waves are called alpha, beta, delta, and theta. Wave types are differentiated based on ranges of wave frequency. Delta waves are those with a frequency up to 4 Hz, theta waves have frequencies between 4 Hz and less than 8 Hz, alpha waves have frequencies between 8 and 13 Hz, and beta waves have frequencies greater than 13 Hz and less than or equal to 30 Hz.

Cycles between Active and Quiet Sleep last about 50 minutes each during sleep (Gomez, Newman-Smith, Breslin, & Bootzin, 2011).

The transition to adult-like sleep begins at about 6 months of age. Both REM and non-REM sleep develop between 6 months and 1 year after birth. This is partly due to the child's new ability to sustain higher amplitude brain waves. Non-REM sleep develops from the former trance alternate pattern of Quiet Sleep. It contains constant, fluid delta waves. K complexes and spindle activity also appear during this time period. K complexes are brain waves that occur during the second stage of non-REM sleep and consist of an initial negative, high-voltage wave that is then followed by a positive wave (Gomez, Newman-Smith, Breslin, & Bootzin, 2011). Sleep spindles are bursts of brain wave activity that also occur during non-REM sleep. Sleep spindles have been associated with integration of information about complex tasks that occurred before the sleep period (Chokroverty & Sahota, 2011).

Sleep continues to become more similar to adult sleep between 1 and 5 years of age. Children at this age are becoming more nocturnal; however a modified form of the polyphasic sleep cycle (in the form of naps) is still needed until they reach 5 years old. By 5 years of age the polyphasic cycle has ended and, though many children may still enjoy one nap during the day, naps are no longer needed. Also at about 5 years old nocturnal sleep is normally between 10 and 12 hours long. During this stage in children's lives the amount of REM sleep they experience begins to steadily decline until it reaches its adult levels later in life (Gomez, Newman-Smith, Breslin, & Bootzin, 2011).

By late childhood (7 to 12 years old) total nocturnal sleep has dropped to between 8.5 and 10.5 hours per night. Napping is essentially non-existent and bedtimes begin to be consistently delayed with increasing age (though this delay occurs in concert with delayed wake times, leaving total time awake during the day unchanged). At this point in development REM sleep takes up about 18.5% of total sleep compared to the 25% of sleep it once dominated when the child was one year old (Gomez, Newman-Smith, Breslin, & Bootzin, 2011).

Adult-type sleep cycles (four sleep stages and 90 minute sleep cycles) are achieved by adolescence; however, sleep homeostasis becomes unstable at this time in life. Sexual maturation that occurs during puberty shifts sleep preferences towards later bedtimes and later wake times. However, at this age there are numerous obligations that decrease the opportunity for sleep. More schoolwork, earlier school start times, and more extracurricular and social activities often force adolescent individuals to sacrifice sleep. Sleep homeostasis in adolescence is so affected by these factors that some researchers have claimed that the typical adolescent is chronically sleep deprived (Gomez, Newman-Smith, Breslin, & Bootzin, 2011).

### ***What determines when and how sleep occurs?***

Research suggests there is a two-process model for the temporal regulation of sleep (Durmer & Dinges, 2005) (Gomez, Newman-Smith, Breslin, & Bootzin, 2011). This model exemplifies the interplay between internal and external forces on the individual in relation to temporal sleep regulation. The two processes of this model are called Process S and Process C and they describe the drive for sleep related to homeostasis and the biological mechanisms

relating to sleep (respectively). Process C includes the functioning of the suprachiasmatic nucleus, regulation and release of neurotransmitters and hormones, and regulation of body temperature (Gomez, Newman-Smith, Breslin, & Bootzin, 2011). Process S is the degree of the feeling of need to sleep, which is dependent on Process C. When the drive for sleep reaches its threshold, sleep is triggered. When it falls below a certain threshold, wakefulness occurs (Durmer & Dinges, 2005). These two processes relate heavily to the suggestions made later in this paper, so we will now look into them with slightly more detail.

Process S involves everything that affects individuals' desire to go to sleep. The subjective feelings of being tired or wakeful are just two of the factors that are involved in Process S. These feelings are regulated by the mechanisms behind Process C and will therefore be described in the following paragraph. However, it is common knowledge that simply feeling tired enough to go to sleep does not always trigger sleep. We have the ability to delay sleep until a more convenient or appropriate time. We do this because of and by using different factors that play into Process S. People may need to fall asleep later than they would like or wake up before they are fully rested in order to accommodate school schedules, work hours, or social obligations. To achieve this, people engage in compensatory actions such as (but certainly not limited to) assuming an uncomfortable posture or attempting to sustain bodily movement. These compensatory actions can temporarily decrease the individual's drive for sleep, but eventually the physical need for sleep will override all other factors involved in Process S (Durmer & Dinges, 2005).

Process C includes all of the biological mechanisms behind the individual's circadian rhythm and the complex interplay between them (Gomez, Newman-Smith, Breslin, & Bootzin, 2011). Due to the nature and purpose of this paper, it is necessary that this section be brief. However, the reader should be aware that entire courses could be taught about the mechanisms about to be described and therefore all descriptions are simplistic and do not represent all of the importance or functions of the various biological mechanisms mentioned here.

Of the various biological mechanisms involved in sleep regulation, the suprachiasmatic nucleus is one of the most influential. The suprachiasmatic nucleus is a brain structure involved in the timing of many circadian rhythms besides sleep and is moderated by other parts of the brain and environmental cues. In particular, the suprachiasmatic nucleus influences the timing of the sleep cycle based on how much light is entering the individual's eyes. In general, when there is a lot of light entering the eyes sleep is delayed and when little to no light entering the eyes sleep is advanced (Chokroverty & Sahota, 2011).

Another brain structure involved in sleep regulation is called the "ascending reticular activating system" and contains neurons (brain cells) that produce acetylcholine when stimulated. Acetylcholine is a neurotransmitter that is involved in wakefulness, neuroplasticity, attention, and reward. The ascending reticular activating system also has connections through the thalamus (a deep brain structure involved in the relay of sensory information across the brain). These connections allow the ascending reticular activating system to cause widespread

changes in neuron excitability. This system is also involved in the creation of brainwaves that indicate the onset of REM sleep (Barker & Barasi, 2008).

Melatonin is a hormone and another member of the group of chemicals involved in drowsiness and wakefulness. Melatonin has been used to reset the timing of circadian rhythms. It is believed that these chemicals do not *cause* the timing of the sleep cycle but are instead related to our physiological experience of the sleep cycle (Chokroverty & Sahota, 2011) (Barker & Barasi, 2008).

Other structures involved in sleep regulation include various parts of the brain stem including the raphe nuclei and the locus coeruleus. These structures help regulate the various stages of sleep as well as to balance sleep and wakefulness. Overall, the brain structures involved in producing the sleep cycle are located deep in the brain and function outside of awareness (Barker & Barasi, 2008).

### ***Why is sleep necessary?***

Currently there are only hypotheses that address why sleep is necessary. However, it is known that our bodies and minds suffer without it. Generally, we know that sleep plays an important role in the strengthening of memories formed during the day. However, most of our knowledge centers on what happens when sleep does not occur, rather than what happens because sleep has occurred. We gain knowledge about why sleep is needed by learning what happens to the mind and body when it is not attained in the normally functioning individual.



We also know that sleep is affected in certain psychological disorders and cognitive exceptionalities. Determining the relationship between sleep and these disorders gives us clues about the nature of sleep as well as the etiology of these disorders and exceptionalities. As we have discussed, sleep changes throughout the lifetime. Therefore we will be addressing normally functioning individuals by age group as well as cognitive disorders by age group.

### ***Importance of Sleep in Childhood Years through Adolescence***

It is estimated that 10 to 30% of children experience some kind of significant sleep disturbance (Chorney, Detweiler, Morris, & Kuhn, 2008). Sleep deprivation in normally developing children is perceived to be particularly detrimental. Childhood years contain irreplaceable opportunities to learn as the learning that goes on during this time will lay the groundwork for the child's adult cognitions. Sleep is believed by many to be an essential time for memory consolidation, one of the fundamental aspects of learning, as well as a time for cell growth and repair (which is possibly related to neural plasticity and neurogenesis, two prominent processes of childhood). Sadeh, Gruber, and Raviv found in 2002 that, not only does sleep quality and duration affect neurobehavioral functioning such as attention, memory, concentration, and reaction times in children it also appears to have a stronger affect on younger children (Sadeh, Gruber, & Raviv, 2002). The researchers found the youngest age group to be most affected by sleep quality and duration when comparing neurobehavioral functioning across 2<sup>nd</sup> grade students, 4<sup>th</sup> grade students and 6<sup>th</sup> grade students. This is particularly interesting because the brain generally has a higher degree of plasticity when younger and is therefore more readily shaped for future cognitions. If sleep does indeed affect

younger children more than older children then it is also possible that sleep irregularities as a youngster could have much more detrimental effects on cognition than what we already know occurs in the adult population.

What about the learning of even younger children? One study looked at how one nap could affect the amount of learning retained by 15-month-old infants (Gomez, Bootzin, & Nadel, 2006). In this study the infants were exposed to an artificial language for 4 hours and then some of the infants were allowed to nap for about an hour and a half while others were not. The infants who were allowed to nap showed a greater degree of abstraction of the knowledge they had gained about the language. For example, they applied the rules of the language to strings of words they had not been exposed to during the 4 hour session. The infants who did not nap did not show this kind of abstraction, but they did show memory of the specific word pairings that they had been exposed to previously (Gomez, Bootzin, & Nadel, 2006). This study is important for two reasons. The first is that none of the children in this study were sleep deprived or experienced any kind of irregularity in their normal sleeping pattern. The experimenters made sure to test the infants at times when they would not be expecting a nap. This places the focus of the study more on what sleep does *for* the brain rather than what the lack of sleep does *to* the brain. The second is that this study is showing the importance of the polyphasic sleep cycle for young children. Children learn a large amount of information and skills in a very short amount of time, particularly during their earliest years. Naps may make this high degree of learning possible. Any disruption of these naps could result in large-scale differences later in life.

Sleep studies involving children can raise questions about validity because these studies use data from children with disrupted sleep in their natural environment (because it would be unethical to systematically deprive a child of sleep, especially due to the implications for that child's development). There are legitimate fears that there may be other variables causing the children's cognitive performance, behavior, and their sleep disruption. However, in 2002 a study was published that put some of these questions to rest (Bates, Viken, Alexander, Beyers, & Stockton, 2002). This study sampled low income preschool children and compared information about family stress, family management, and the child's sleeping behaviors with behavioral reports written by their teachers. When the researchers controlled for the child's family stress and family management, it was still found that sleep irregularities had a significant effect on the child's behavioral adjustment in school such that more sleep irregularities resulted in less adjusted behavior in the classroom (Bates, Viken, Alexander, Beyers, & Stockton, 2002). This is important research because often we hear about the achievement gap between low income students and middle to high income students. While income and family stress do play a role in the child's academic performance and behavioral adjustment, this study suggests that perhaps there is an even more profound achievement gap between the children who are able to achieve quality sleep and those who do not get the amount and quality of sleep they need at night.

### ***Sleep and Disorders Present in Childhood and Adolescence***

The study that was previously discussed suggests a relationship between sleep and maladjusted behaviors. However, there is a newly blossoming field of research that is drawing connections between developmental and psychiatric disorders and the kind and quality of sleep that children with these exceptionalities achieve.

Sleep is known to be affected in various psychological disorders, though the directionality of this relationship is still contested. A study conducted in 2007 of 128 children and adolescents with anxiety disorders showed that 88% of the participants also had at least one sleep-related problem and more than half of the participants had three or more sleep-related issues. Another study (published in 2005) followed 943 children that showed that sleep disturbances in early childhood and found that these symptoms were related to the presence of anxiety disorders in the individuals over 20 years later (Chorney, Detweiler, Morris, & Kuhn, 2008). Some believe that the association between sleep and depression in children is stronger than the relationship between sleep and anxiety in children, though the research in this area is still lacking. A 1987 study of 187 children and adolescents with major depression found that 74% of them also reported symptoms of significant insomnia (Chorney, Detweiler, Morris, & Kuhn, 2008). A later study published in 1996 found that over the course of a 3.5-year period, adolescents with at least one sleep disorder had almost a doubled risk of developing depression (Chorney, Detweiler, Morris, & Kuhn, 2008). A study involving over 5000 adolescent participants found that sleep disturbances were strongly associated with fatigue, mood disturbance and suicidal ideation. This finding was significant even when age, gender, and

socioeconomic status were controlled for (Roberts, Roberts, & Chen, 2001). However, while research has shown there is a connection between sleep disturbances and psychiatric disorders, little is known about why this connection exists. Some researchers believe that psychiatric disorders and sleep disturbances can feed into and intensify each other, creating a vicious cycle of behavior and cognition. Others believe that the overlap of similar symptoms is the cause of a perceived comorbidity between these disorders and perhaps an overdiagnosis of psychiatric disorders may be caused by their similarities to sleep disturbances and sleep deprivation (Chorney, Detweiler, Morris, & Kuhn, 2008). Figure 2 represents the overlapping symptoms of major depressive disorder, ADD, and sleep deprivation.

Recently researchers have taken an interest in the connections between sleep and developmental disorders. Recent research on preschool-aged children with autism and developmental delay found that both groups of children had more variable sleep patterns than typically developing children of the same developmental age. Interestingly, the researchers also found that children with autism awoke less often during the night but slept less during a 24 hour period than both children with developmental delay and typically developing children. Young individuals with autism also napped less frequently and had naps with shorter lengths than both children with developmental delay and typically developing youngsters. Children with developmental delay were found to awaken much more frequently during the night and had nighttime awakenings of much longer duration than children with autism. Since these studies lasted for 6 months we can be fairly certain that they represent the sleep characteristics of autism and developmental delay for this age group (Schwichtenberg, Iosif, Goodlin-Jones, Tang, & Anders, 2011) (Anders, Iosif, Schwichtenberg, Tang, & Goodlin-Jones, 2011).

ADD and ADHD have also had their relationship to sleep examined by research. Some who believe these disorders are over-diagnosed blame sleep issues for part of this problem. These critics point to research that suggest that children who get more sleep are more motivated and receptive to their teachers in school, less likely to be bored, and more likely to be able to control aggression. Since these findings represent the opposite states of the symptoms of sleep deprivation and appear to also be the opposite of symptoms of ADD and ADHD, these critics blame sleep deprivation for many of the cases of ADD and ADHD rather than an actual neurological disorder (Plaford, 2009).

### ***Conclusions about Research on Children and Adolescents***

The field of study connecting children and adolescents to sleeping behaviors is one that needs to grow. The little evidence we have now has big implications for how we as individuals, families, and as a society need to treat sleep in youngsters, especially youngsters with exceptionalities. The developmental differences of sleep for children and infants compared to sleep for adolescents and adults inform us that we can no longer believe in good faith that the research on adult sleep can be generalized to children. Also, based on the studies cited above, we know that with some developmental disorders sleep matches the developmental age of the child but with other developmental disorders sleep matches the chronological age of the child. Research needs to be done which seeks to determine whether it is more beneficial to have sleep development match chronological age or developmental age (or whether there is no difference in outcome between the two). Longitudinal studies that draw connections between

atypical sleep in childhood and psychiatric disorders in adulthood suggest that we as a society and as leaders of families can help prevent children from one day experiencing a psychiatric disorder if we can help them experience quality sleep on a regular basis. We should be extra vigilant in this respect for children with developmental disorders since research has shown that many developmental disorders also have a high rate of comorbidity with psychiatric disorders and actions that may be able to reduce this already heightened risk need to be fully explored by the research community and the public. Learning and behavior studies suggest that children's school performance can be drastically improved if they can get a full night's rest without multiple awakenings. Again, these are areas that are particularly difficult for some children with exceptionalities and fully understanding the sleep mechanisms behind these improvements could perhaps lead to understanding about the mechanisms behind these functions in the exceptional mind. Overall, sleep is an inextricable part of life and therefore it is also an inextricable part of life with an exceptionality. It can provide us clues to further understanding of the expression of different developmental and psychiatric disorders in the brain and perhaps one day suggest ways to make life with these exceptionalities a little easier.

### ***The Importance of Sleep in Adulthood***

Much more is known about the effects of sleep deprivation on adult individuals. Sleep deprivation has been linked to a host of physiological conditions. Sleep deprivation increases the risk of cardiovascular conditions, gastrointestinal issues, weight gain, weakened immune system, and endocrine system malfunction (Chokroverty & Sahota, 2011). These findings have

particular importance to adults whose professions require some degree of sleep deprivation such as commercial vehicle drivers, aviation workers (such as pilots and air traffic controllers), medical residents, and college students. These groups are often used as subjects in research studies about sleep deprivation and its effects on their respective job performance (Chokroverty & Sahota, 2011).

New and exciting research has shown that new neurons are produced in the brain during adulthood. However, sleep research has shown that sleep deprivation has the ability to slow or even stop the growth of these new cells. These findings carry with them the weight of the unknown purpose of these new neurons. New brain cells produced during adulthood have been found in the hippocampus and the subventricular zone of the lateral ventricles. Researchers hypothesize that the new neurons in the hippocampus are related to memory processes and regulation of emotion and therefore disruption of the growth of these cells could be a contributing factor to cognitive dysfunction and mood disorders that appear later in life (Meerlo, Mistlberger, Jacobs, Heller, & McGinty, 2009) (Lucassen, et al., 2010). Research to support this claim has found that neurogenesis in the hippocampus can be stimulated by antidepressant drugs (Lucassen, et al., 2010). However, the mechanisms of adult neurogenesis, how sleep is able to affect this cell growth, and why antidepressant drugs are able to partially ameliorate this situation remains a mystery (Lucassen, et al., 2010) (Meerlo, Mistlberger, Jacobs, Heller, & McGinty, 2009).



### ***Sleep's Relation to Mental Disorders of Adulthood***

Research on major depression in adults has shown EEG sleep abnormalities. People with depression have been shown to have shorter REM cycles, reduced slow wave sleep (non-REM stages 3 and 4), decreased sleep time and sleep efficiency, and an increased experience of early morning awakening. In all, between 80% and 90% of people with major depressive disorder have symptoms of disordered sleep (Chokroverty & Sahota, 2011).

PTSD patients have also been found to have quantifiable sleep issues. People with PTSD often have difficulty falling asleep and staying asleep as well as frequent nightmares. However, PTSD's interaction with sleep goes beyond these expected difficulties. Some individuals with PTSD have shown to have impaired skeletal muscle inhibition during REM sleep which results in the patients acting out their dreams while not awake. Individuals with PTSD also have a higher likelihood of experiencing sleep-disordered breathing and movement disorders such as restless leg syndrome which often lead to sleep deprivation and its associated cognitive and physical deficits (Chokroverty & Sahota, 2011).

The research community is also interested in the sleep abnormalities found in schizophrenia. Interestingly, sleep abnormalities are not a defined symptom of schizophrenia but many sleep issues have been found in these patients. Individuals with schizophrenia are frequently observed to have issues with insomnia and nightmares. Also, abnormal EEG patterns have been found such as decreased slow wave sleep, decreased time spent in REM sleep, increased REM sleep density, and shortened total sleep time. Of particular interest are the recent findings that suggest that the decrease in slow wave sleep have an inverse relationship

with the size of brain ventricle volume. This relationship suggests that as brain ventricle volume increases (as is characteristically observed in these patients) slow wave sleep decreases (however the directionality of this relationship is not fully known) (Sakar, Katshu, Nizamie, & Praharaj, 2010) (Chokroverty & Sahota, 2011).

### ***Conclusions from the previous Sleep Research***

Research evidence pointing to sleep deprivation and poor quality sleep as possible mechanisms behind the development of various physiological and psychological disorders as well as the exacerbation of disorders already present in individuals is enough cause for alarm and increased funding for research in this field. One can legitimately wonder how much money, time, and quality of life could be spared by a greater focus on healthy sleep in both adults and children. The evidence presented above suggests that mental health costs could be spared by ensuring children and adolescents get adequate amounts of sleep. But, more importantly, working towards lessening sleep difficulties present in children can prevent them from ever having to experience a psychiatric disorder. In adults, a greater respect for the need to sleep could one day be adopted by drivers, medical institutions, and schools so that less accidents are caused by drowsy driving, less medical errors are the result of sleepless medical residents, and more school systems can better promote learning and behavior by allowing children to sleep when it is biologically appropriate.

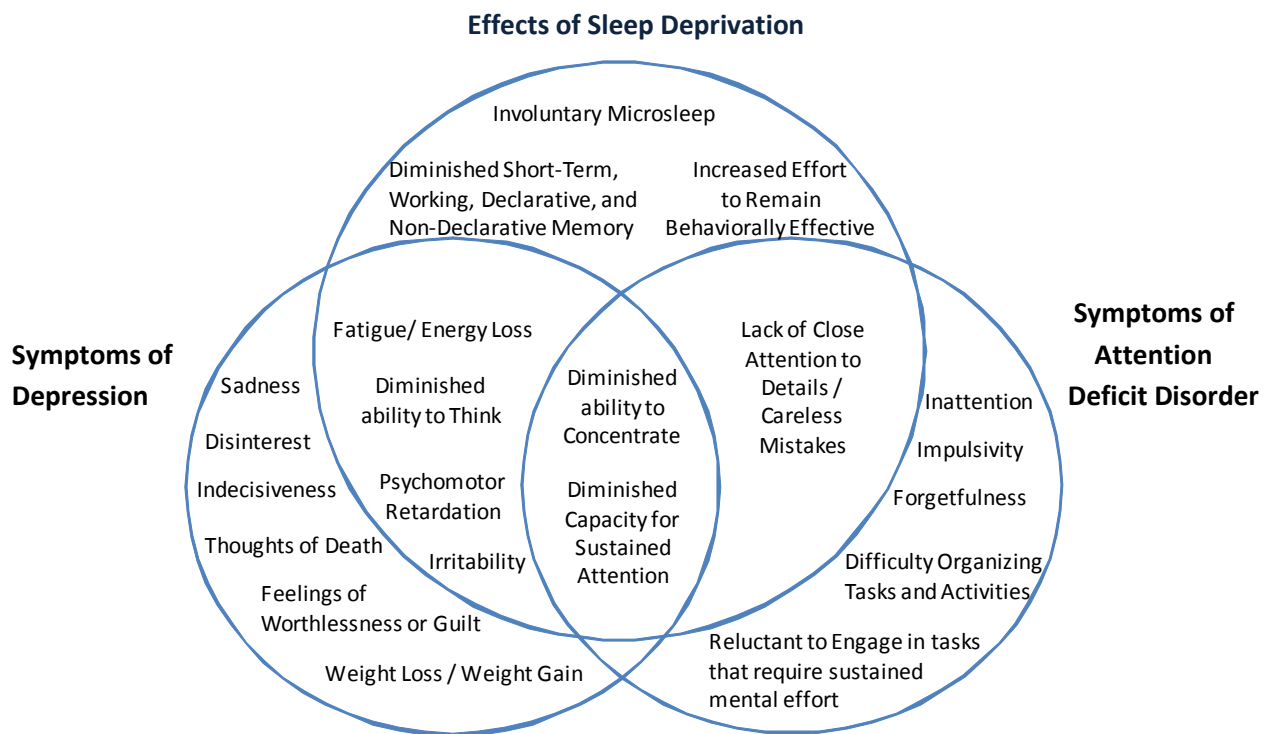
Sleep research also gives us vital clues as to the nature of mental illnesses. It is not a coincidence that children with sleep issues are more likely to develop psychiatric issues that

have sleep disturbances as a defining characteristic. Research into how sleep plays a role in the brain changes that eventually result in mental illness and worsen mental illness may one day provide the necessary information for new treatments and prevention programs.

Finally, the research that shows that sleep abnormalities and deprivation are somehow affecting the actual structure of parts of the brain is perhaps the most alarming and most deserving of more study. The idea that sleep issues are somehow related to the expansion of the ventricles in schizophrenic brains and the inhibition of adult neurogenesis in the hippocampus is both intriguing and horrifying. There is little doubt that these very recent findings will spur new research on the effects. However, it is impossible to know where research on these topics and the others mentioned in this review will lead us in the future and what kinds of benefits they could hold for society.

**Figure 1: The Development of Sleep**

### Figure 2: The Similarities between Sleep Deprivation and Common Psychological Disorders



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