Bidirectional Causality in Anxiety and Sleep Disorders

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Introduction

This paper explores bidirectional causality in anxiety and sleep disorders. While clinicians and scientists have long recognized the role of anxiety as a cause of insomnia, research now points to a more reciprocal relationship. Our review of relevant literature concludes that disruptions in sleep and anxiety disorders interact in a bidirectional or cyclical manner, and therefore that therapists working with anxious clients should consider sleep disorders as they search for the causes of anxiety and develop a therapeutic approach and vice versa.

Our review of pertinent literature includes three discussions. First, "Sleep Disorders as a Cause of Anxiety—An Evolving Perspective," provides an overview of changing views of the causal relationship between anxiety and sleep disorders: from (1) the relationship is unidirectional, anxiety leads to sleep disorders; to (2) the relationship is unidirectional, but sleep disorders can exacerbate anxiety, to (3) there is a bidirectional relationship between anxiety and sleep disorders; each is capable of initiating and influencing the other. Second, "Sleep, Circadian Rhythms and Anxiety," explores two distinct but interacting physiological systems involved in sleep (Circadian Rhythms and Sleep Debt) and describes biologically how disruptions in Circadian Rhythms relate to symptoms commonly associated with anxiety. Third, "Sleep Disorders as an Underlying Cause of Anxiety" reviews two strains of research that link disruptions in circadian rhythms to anxiety disorders. The first suggests that an underlying genetic or biological vulnerability related to circadian rhythms can predispose humans to anxiety disorders. The second links circadian rhythm disorders to the experience of anxiety disorders. Given the research that demonstrates a bi-directional relationship between sleep disorders and anxiety, we conclude with implications for therapists.

Sleep Disorders as a Cause of Anxiety—an Evolving Perspective

For millennia, observers of the human condition have recognized that anxiety disrupts sleep. In Ecclesiastes 5:12, the ancient sage writes, "The sleep of a hard-working man is sweet, but the worries of a rich man will not allow him to sleep." Similarly, after Shakespeare's Macbeth murders the king, he comes to the realization that never again will he experience untroubled sleep, "Methought I heard a voice cry, 'Sleep no more! Macbeth does murder sleep,' the innocent sleep" (2.2.34-35).

Today, it is an established canon of psychology and medicine that anxiety causes insomnia and other sleep disorders. Lichstein's review of fifty epidemiological studies of sleep (Lichstein, 2004, p. 38) reveals that the relationship between anxiety and sleep disorders is often viewed unidirectionally, that disturbed sleep is considered a secondary and direct consequence of primary psychiatric disorders such as anxiety or depression. In his classic book, *The Promise of Sleep*, Dement (1999) identifies psychological, emotional and psychiatric problems as a major cause of persistent insomnia (p. 144). These precipitating causes include "phobias, anxieties, and neuroses" (p. 144). Supporting a unidirectional understanding of the relationship between anxiety and sleep disorders, the American Psychiatric Association (American Psychiatric Association *[DSM-IV-TR]*, 2000) locates insomnia as either primary or secondary to other disorders. Both post-traumatic stress disorder (PTSD) and generalized anxiety disorders (GAD) have sleep disturbance as a diagnostic criteria. Interestingly, however, the *DSM-IV* does not list anxiety as diagnostic criterion of primary insomnia. Is the relation of anxiety to sleep disorders simply one way—anxiety causes insomnia?

Some causal models of the relationship between anxiety and sleep disorders that maintain a unidirectional influence from anxiety to sleep disorders at least allow that sleep disorders can exacerbate the presentation, course or outcome of anxiety. This is the position of Chanin (2010),

who begins his discussion on the relationship between sleep and psychiatric disorders emphatically, “There is no evidence that sleep disorders are a cause of psychiatric disorders” (p. 1). Nevertheless, he goes on to assert that difficulties with sleep can make psychiatric disorders worse by making the person confused or frustrated, as well as more sensitive to pain and other medical problems. This also appears to be the position taken by Bourne (2005), who omits sleep disorders from his discussion of the major causes of anxiety (pp. 30-52), but later includes “Sleeplessness (Insomnia)” in his discussion of health conditions that may contribute to anxiety (pp. 350-356). Such “contributors” are “common physical conditions that can aggravate anxiety or tax your system and make you more vulnerable to its effects.” (p. 332).

The movement away from understanding the causal relationship between anxiety and sleep as unidirectional (with or without exacerbating influences) to bidirectional, in which anxiety contributes to the development of sleep disorders and sleep disorders contribute to the development of anxiety, has gained momentum through numerous studies that demonstrate the co-morbidity between anxiety and sleep disorders. Ohayon & Roth (2003) found that in those with no previous history of anxiety disorders, insomnia occurred before the current anxiety disorder in 18% of the cases and appeared at about the same time in 39%. In 43% of the cases anxiety appeared before the insomnia. In a long-term study that followed children from three years of age to adults 26 years of age, Gregory, Caspi & Eley (2005) found that sleep problems before nine years of age were strongly predictive of later anxiety disorders, but not depression. In their review of the National Comorbidity Survey Replication, Roth, Jaeger & Jin (2006) found that insomnia was observed in 32.5% of those who had anxiety disorders. Those having anxiety disorders were four times more likely to have insomnia than those without these disorders. Based on data collected from 25,130 adults from two general health surveys conducted over a ten-year

period, Neckelmann, Mykletun & Dahl (2007) found significant associations between the long-term course of chronic insomnia and the development of anxiety disorders and depression. Those subjects who reported that they had insomnia during the initial survey had a higher risk of developing an anxiety disorder during the second phase of the study conducted 10 years later. The findings held up even when factors such as the patients' age, gender, and educational level were taken into consideration. Neckelmann et al. concluded that insomnia may be a trait marker for those at risk for the development of anxiety disorders.

After reviewing of some of the more important studies on the bidirectional relationship between anxiety and sleep disorders, Dahl, A. A. & Bjorvsatn (2009) conclude, "the most parsimonious interpretations of these findings is that insomnia can predispose for anxiety and vice versa" (p. 44). They then describe four temporal/causal hypotheses, currently the subjects of on-going research, that seek to explain the bi-directional relationship between sleep disorders and anxiety: (1) the "Predisposition or Vulnerability Model," in which either anxiety or a sleep disorder is a risk factor for the development of the other; (2) the "Common Cause Model," in which sleep disorders and anxiety have a common aetiology arising from a common core liability; (3) the "Pathoplasthy or Exacerbation Model" in which insomnia and anxiety are not causally related but the presence of one disorder influences the presentation, course or outcome of the other; and (4) the "Complication or Scar Model," in which residual effects associated with either insomnia or anxiety, which has remitted, influence the course or presentation of the other. According to the authors, research is currently favoring the first two hypotheses, although none are mutually exclusive (Dahl, A. A. & Bjorvsatn, 2009, pp. 44-45).

Sleep, Circadian Rhythms and Anxiety

One thread of research into the bidirectional relationship between sleep disorders and anxiety focuses on the disruption of circadian rhythms and anxiety. In this section, we focus on the role of circadian rhythms in facilitating normal sleep, the circadian biochemical processes that induce sleep and wakefulness, the biochemical relationship between normal sleep and anxiety, disruptions in circadian cycles and how such disruptions might induce anxiety.

Intuition tells us that we sleep when we are tired, that peak periods of alertness would occur when we first awaken and would decrease gradually during the day, until, at the end of it, our sleep debt or tiredness causes us to fall asleep. This is only half the story. Normal sleep involves two opposing processes (Dement, 2000, pp. 102-124; Foster & Kreitzman, 2004, pp. 177-200; Goldbeter, 1997, pp. 460-464; Kalat, 2009, pp. 259-268): first, the "homeostatic drive for sleep" is the intuitive process just described in which the drive for sleep increases the longer an individual stays awake; and second, a circadian driven rhythm that promotes initial wakefulness in the morning continues throughout the day, keeping us alert, and then decreases the drive for wakefulness during sleep. These two processes interact to consolidate sleep. Homeostasis maintains the duration and intensity of sleep, while the circadian rhythm determines the timing and propensity to sleep (Foster & Kreitzman, 2004, p. 185).

Like most animals and plants, human beings have circadian rhythms (from Latin *circa* meaning "around" and *deum* meaning "day") that keep physiological systems working in harmony. Human circadian rhythms create approximately a 24-hour biological cycle that affects body temperature, appetite, hormone secretion, alertness and sleep. Three characteristics define human circadian rhythms (Goldbeter, 1997, p. 460): (1) they are endogenous, that is, they persist internally even in the absence of external cues such as constant light or darkness; (2) they are

sensitive to light and can be phase-shifted or entrained by exposure to light; and (3) they maintain their periodicity independent of temperature changes. The last two are required if an organism is to adapt to the natural light-dark cycle regardless of temperature.

Human circadian rhythms are driven by the suprachiasmatic nucleus (SCN), a tiny group of genetically powered neurons in the hypothalamus that sits directly above (*supra*) the optic chasm (Kalat, 2009, p. 264). A small branch of the optic nerve, known as the retinohypothalamic pathway, extends from the retina to the SCN. This pathway allows light to alter SCN settings and thereby sets and resets the biological clock. Two genes called *Period* and *Timeless* produce the proteins *Per* and *Tim*, which underlie the SCN's regulation of the sleep/wake cycle. Within the SCN, Per and Tim start out in small amounts in the morning and increase during the day. By evening they reach a level high enough to induce sleepiness. This high level also feeds back to the genes to shut them down, thus decreasing Per and Tim throughout the night until their low levels induces wakefulness (Kalat, 2009, p. 266).

Stimulated genetically by Per and Tim, the SCN induces morning wakefulness, daytime alertness, bedtime sleepiness and nighttime sleep by stimulating the production of hormones periodically throughout the day and night. At night the SCN stimulates the pineal gland to release melatonin, which induces sleep. In the morning, the SCN stimulates awakening through two separate processes. First, through the normal hypothalamo-pituitary-adrenal (HPA) pathway, the locus coeruleus (in the brain stem) signals the adrenals to produce cortisol and norepinephrine, which produces wakefulness. Since continual exposure to cortisol and norepinephrine causes brain neurons to lose sensitivity to them, the SCN's circadian clock stimulates the adrenals to release cortisol and norepinephrine periodically throughout the day to maintain alertness (Kalat, 2009, p. 272). A second overlaying circadian pathway, called the

Cortisol Awakening Response (CAR), is specifically linked to awakening. This pathway originates not in the hypothalamus but in the hippocampus, the brain structure that translates short term memory into long term memory. Through this rhythm, the hippocampus prepares the normal HPA Axis to face the anticipated stress of the day. The CAR produces a sharp 38-75% (average 50%) increase in blood cortisol in about 77% of healthy adults, which rises to a peak about 20-30 minutes after awakening (Wüst et al., 2000; Fries, Dettenborn & Kirschbaum (2009).

Since circadian patterns related to the production of cortisol and norepinephrine play an important role in morning awakening and daytime alerting and since these hormones also play significant role in supporting anxiety and the stress/fight/flight response, the question arises whether correlations can be made between the circadian timing of their release and increased symptoms in patients suffering with anxiety. Does the circadian production of alerting hormones add to a pre-existing pool of hormones already supporting anxiety to exacerbate symptoms at certain times during the day?

Cortisol production peaks in the morning and declines throughout the day. Then, around 3 pm, another burst of alerting hormones revs up the body's energy, helping us counter the effects of sleep debt and achieve a high level of functioning during the evening. Dement calls this process "Clock Dependent Alerting" (1999, pp. 83-85). If there is a correlation between the production of alerting hormones and an increase in symptoms related to anxiety, we might expect to see this occur when the production of alerting hormones is high or is increasing, that is, upon awakening, in the mid-afternoon with the retriggering of these hormones and in the evening when these hormones reach another peak. Such an inquiry is supported by Mitrovic (2007):

"Excessive concentrations of cortisol in blood can cause insomnia and strikingly increase or decrease mood" (p. 478).

Is anxiety worse in the morning? Anecdotally, many people report morning anxiety. An internet search of the phrase "early morning anxiety," produced over 2.6 million hits. There is a significant and increasing body of research on the effects of the Cortisol Awakening Response (CAR) and anxiety that points to a connection. Greaves-Lord et al. (2007) tested the association between cortisol levels of a large population of 10-12 year olds at three points during the day and found that individuals with persistent anxiety problems had higher morning cortisol levels and a higher cortisol awakening response. In a similar study of 230 late adolescents, Adam et al. (2010) found that clinical diagnoses of stress related diseases were predicted from CAR functioning gathered one year earlier. Age and gender, health and health behaviors, baseline neuroticism, exposure to stressful life events and past episodes of mood and anxiety disorders were included as covariates.

Does the production of alerting hormones at other times of the day correlate to an increase in symptoms of anxiety? In a study of 86 patients with anxiety disorders (63 with panic disorders or agoraphobia with panic attacks), Cameron, Lee, Kotun & McPhee (1986) found that anxiety symptoms tended to be more severe in the afternoon or evening than in the morning, with no abnormalities of heart rate or oral temperature, while depressive symptoms were higher upon awakening. Participants rated their symptoms at five time periods during the day (7:00, 11:00, 15:00, 19:00 and 23:00). The worst symptoms appeared at 15:00 and 19:00 (Cameron et al., 1986, p. 215), when we would expect them to be worst based on circadian production of alerting hormones during these periods. The authors of this study however did not attempt to correlate their findings to the production of alerting hormones.

In another study of cortisol patterns, stress, symptoms of depression and anxiety in working parents, Kirina, Schneider, & Waite (2004) found a correlation between severe symptoms of anxiety and significantly higher average cortisol levels in men (p. 53).

Even if daily periodic exacerbation of anxiety through the influence of circadian production of alerting hormones can be established, it is another matter to ask if disruptions of circadian rhythms and consequent dysregulation of alerting and/or sleep inducing hormones can cause anxiety. There are many ways that sleep can be disrupted. Dement (1999, pp. 523-527) lists 78 different kinds of sleep disorders, most of which involve a dysregulation of sleep hormones. When alerting or sleep inducing hormones are produced at the wrong time, or in amounts that exceed or are less than normal, so that one sleeps too much or too little or at the wrong time, can this produce anxiety? The next section reviews literature that points to a causative relationship between sleep disorders and anxiety. Before proceeding to it, in an excursus, we consider whether research that demonstrates a causative relationship between sleep disorders and depression/mood disorders is relevant for this discussion.

Excursus: The Relation of Research Related to Sleep Disorders and Depression to the Inquiry into the Relationship of Sleep Disorders and Anxiety Disorders

Much research supports the idea that sleep disorders and disruption of circadian rhythms can cause depression and mood disorders. Sleep disturbances are commonly observed in patients with depression. Research supports that circadian and sleep disturbances may play a critical role in the physiology of mood disorders (Germain & Kupfer, 2008). In animal models, sleep restriction results in changes in serotonin and cortisol-releasing hormone receptor systems, as well as changes in neuroendocrine reactivity that are similar to changes seen in depression. This suggests a role for sleep disturbances in sensitizing individuals to depression (Novati, Roman &

Cetin, 2008). Insomnia lasting two weeks or longer predicted subsequent major depressive episodes among young adults across twenty years of follow-up (Buysee et al., 2008). In women, reproductive hormones modulate the timing between different components of the circadian system. It is hypothesized that hormonal flux may destabilize circadian rhythms and thereby contribute to the development of mood disorders in predisposed women postpartum (Parry & Newton, 2004; Ross, Murray & Steiner, 2005; Parry, Meliska & Sorenson 2008*;* Goyal, Gay & Lee, 2009; Posmontier, 2008). Moreover, manipulations of sleep and circadian rhythms can alleviate depressive symptoms, providing support for a positive relationship between mood and sleep, as well as potential intervention strategies (Ross, Murray & Steiner, 2005).

Is this research relevant for our discussion about the bidirectional relation of sleep disorders to anxiety? Ten years ago British psychiatrist P. Tyrer (2001) wrote: “Every psychiatrist and general practitioner diagnoses mixed anxiety and depression readily. The combination of typical depressive symptoms, such as low mood, lassitude and pessimism about the future and anxious ones, such as tension, insomnia and irritability is so common that about one in seven of the population is experiencing them at any one time in the UK” (p. 191). He then puts forth evidence from genetics, neurobiology, epidemiological studies, treatment studies and outcome studies to support the interrelatedness of these disorders. A similar position on this issue is taken by Bourne (2005) who relates Seasonal Affective Disorders to Panic Attacks and GAD and concludes, “It is not surprising that this is so, because the same systems of the brain that contribute to the neurobiological basis of depression, the noradrenergic system and the serotonin system, are also implicated in anxiety disorders, particularly panic disorder, GAD and obsessive-compulsive disorder” (p. 348).

Mixed Anxiety-Depressive Disorder was included in the *DSM-IV-TR* as a disorder for further research (*DSM-IV-TR*, p. 780) suggesting that there is a need to reflect the fact that there is a group of individuals who experience both depression and anxiety symptoms, and that the categorical distinction between mood and anxiety disorders may not be clear and firm. It appears now that a new diagnostic category, "Mixed Anxiety Depression," will appear in the 2013 edition of the *DSM-V* (www.dsm-5diagnosis.com).

Sleep Disorders as Underlying Causes of Anxiety

Given the body of research that confirms a bidirectional relationship between sleep disorders/disruptions in circadian rhythms and depression and given the common genetic and neurobiological basis of depression and anxiety disorders, it can be concluded that such research at least suggests that inquiry into the impact of sleep disorders on anxiety might prove fruitful. That this is the case is demonstrated in this section. Here we focus on two strains of research. The first suggests that an underlying genetic or biological vulnerability related to circadian rhythms can predispose humans to anxiety disorders. The second links circadian rhythm disorders to the experience of anxiety disorders.

Circadian genetic or biological vulnerability as a cause of anxiety disorders.

Several research studies suggest that an underlying genetic or biological vulnerability related to circadian rhythms can predispose humans to anxiety disorders.

Sipilä et al. (2010) conducted a genetic association analysis on 13 circadian-clock-related genes. The study included 321 individuals diagnosed with an anxiety disorder and 653 matched healthy controls from a Finnish population-based cohort. The results showed that nucleotide polymorphisms in two circadian genes show evidence for association to social phobia; four genes show evidence for association with generalized anxiety disorder and two genes show

evidence for association with a pooled group of all anxiety disorders. These findings lead to the conclusion that circadian genes play a role in genetic predisposition to anxiety disorders (pp. 1169-1170).

Vreeburg et al. (2010) examined whether HPA Axis dysregulation represents a biological vulnerability for depressive and anxiety disorders. Based on data gathered from the Netherlands Study of Depression and Anxiety, among those without a lifetime diagnosis of depression or anxiety disorders, they distinguished three groups: (1) 180 people without parental history, (2) 114 with self-reported parental history, and (3) 74 with CIDI-diagnosed parental history. These groups were compared with people with major depressive disorder or panic disorder with agoraphobia. Salivary cortisol samples were obtained upon awakening, and 30, 45 and 60 minutes later. The results demonstrated that compared with unaffected participants without parental history, unaffected individuals with diagnosed parental history of depression or anxiety showed a significantly higher cortisol awakening curve that was similar to that observed in the participants with depression or anxiety disorders. This suggests that a higher cortisol awakening curve reflects a trait marker, indicating an underlying biological vulnerability for the development of depressive and anxiety disorders.

In a similar study, Lai & Wan (2009) examined the relationship between trait anxiety and multiple indices of the CAR in a group of 79 healthy university students. Saliva tests were collected from participants four times (0, 15, 30, and 45 minutes after awakening) to determine the cortisol curve with reference to zero (the AUC Score) and the increase of Cortisol from immediately after wakening to the last post-awakening sample (the Mnlnc Score). They found that higher trait anxiety was associated with a larger Mnlnc score and concluded that an

increased rise in cortisol in the post-awakening period correlates with trait anxiety or chronic stress (p. 4).

In a longitudinal study that included four assessments of infant cortisol reactivity to stressful events in a non-clinical population, Tollenaar, Beijers, Jansen, Riksen-Walraven & de Weerth (2011) found that early intrauterine environmental factors can shape the development of the HPA Axis potentially creating a biological predisposition to anxiety. General and pregnancy-related feelings of stress and anxiety, as well as circadian cortisol levels, were measured in 173 mothers in the last trimester of pregnancy. Infant cortisol reactivity was measured at 5 weeks to a bathing session, at 8 weeks to a vaccination, at 5 months to a stressful mother-infant interaction (still face procedure), and at 12 months to a maternal separation (strange situation procedure). The results showed that maternal prenatal fear of bearing a handicapped child was a consistent predictor of infant cortisol reactivity. Therefore, pregnancy-specific anxieties predicted infant cortisol reactivity in the first year of life.

According to Dement (1999, p. 87), the typical pattern of circadian clock dependent alerting is an idealized "average" that represents a range of alerting schedules that humans experience. In reality, within the human community, there are different chronotypes—some of us are "larks," who perform best in the morning and others are "owls," who perform best in the evening. Vardar, E., Vardar, S. A., Molla, Kaynak & Ersoz (2009) examined the psychological symptoms and sleep quality in young subjects with different chronotypes: morning types, evening types, and intermediate types. Their study of 79 female and 63 male medical students between the ages of 17 and 23 years revealed that there were significant differences in the psychological symptoms of anxiety, obsessive-compulsiveness, hostility and phobia among the groups. Positive symptom scores were higher in the evening types, which correlated with poorer

sleep quality and daytime sleepiness in the evening types. The finding suggest that evening chronotypes suffer more anxiety, obsessive compulsiveness, hostility and phobia symptoms and sleep problems than other chronotypes.

Circadian rhythm disorders as a cause of anxiety disorders.

Circadian Rhythm disorders are a family of sleep disorders that affect the timing of sleep (Dement, 1999, pp. 528-529). A number of intrinsic and extrinsic causes of circadian rhythm disruption can result in a dysregulation of hormones related to the sleep-wake cycle, creating problems with the timing of sleep, with falling asleep or awakening, with staying asleep or with the quality of sleep. Circadian rhythm sleep disorders are categorized in four distinct types (*DSM-IV*, pp. 623-624): (1) Delayed Sleep Phase Type is characterized by delay of circadian rhythms, including the sleep-wake cycle, relative to the demands of society; (2) Jet Lag Type affects people who travel over several time zones and then experience a mismatch between desired and required hours of sleep and wakefulness; (3) Shift Work Type affects people who work nights or on rotating shifts and experience a conflict between their normal pattern of circadian sleep and wakefulness and the pattern of sleep and wakefulness required by their shift work; and (4) Unspecified Type includes other patterns of circadian sleep disturbances (e.g., advanced sleep phase, non-24-hour sleep-wake pattern, or irregular sleep-wake pattern). In our discussion below, to these types we add Seasonal Affective Disorder (SAD), Winter Type and Summer Type, in which disruptions of circadian rhythms due to lack of light during winter months lead to depression and anxiety. What follows reviews research that links these sleep disorders to anxiety.

The Delayed Sleep Phase Type circadian disorder is characterized by nightly sleep that is delayed until long after the desired sleep time. This results in a sleep-onset disorder and

trouble waking at the desired time (Dement, 1999, p. 529). The disorder is often associated with adolescents and the elderly. Shirayama et al. (2003) administered the Yatabe-Guilford test, Minnesota Multiphasic Personality Inventory, Picture-Frustration study and Rorschach test to two groups, one of patients with DSPS (case group) and the other a control group consisting of people without psychiatric symptoms or insomnia. The results of the test indicate that patients with DSPS showed emotional features such as nervousness, depression and lack of control of emotional expression. Specific personality traits included introspection, defensiveness, compulsivity, unawareness of impulsiveness toward immediate gratification and reduced cognitive ability. Shirayama concludes that a definite psychological profile for patients with DSPS includes an excessive defense mechanism that increases nervousness capable of developing into anxiety disorders.

Symptoms of the **Jet Lag Type** may include malaise, decreased strength and efficiency, decreased memory, decreased concentration, gastrointestinal disturbance, headache, irritability, loss of appetite, tiredness during the day, and sleeplessness at night. Katz, Knobler, Laibel, Strauss & Durst (2002) demonstrated that the dyschronism of circadian rhythms caused by long range travel across several time zones also causes major psychiatric morbidity, including increased anxiety. Between 1993 and 1998, Katz et al. conducted a study of long-distance travelers hospitalized at the Jerusalem Mental Health Center, Kfar Shaul Hospital. Patients were divided into two groups based on the number of time zones crossed in the trip to Israel: (1) seven times zones or more and (2) three time zones or less. After controlling for demographic and religious background, past psychiatric history and diagnosis upon admission, the results demonstrated that the dyschronism of circadian rhythms and jet lag played a significant role in the exacerbation of anxiety disorders.

Shift Work Sleep Disorder (SWSD) can occur when a person's sleep schedule is consistently disrupted by changing work hours. For a person to receive the diagnosis there must be evidence that the individual is experiencing a constant or recurring pattern of sleep disruption that causes insomnia or excessive sleepiness. With SWSD there is an increase in potential for accidents on the job, impaired problem-solving, motor reflexes and movements, irritability, concentration difficulties and mood problems. Munakata, Ichi & Nunokawa (2001) studied 18 healthy nurses engaged in a rapid shift rotation system to determine if neuroendocrine responses related to altered sleep states correlated with psychological disturbances, including confusion, depression, anger-hostility and anxiety. After the night shift, scores for anxiety were among the highest. In another study of shift work and operator performance in Remotely Piloted Aircraft conducted by the United State Air Force, Thompson (2006) found that operators working a rapid shift rotation experienced chronic fatigue, "The combination of partial sleep deprivation and the influences of homeostatic and circadian systems significantly increased the risk for diminished work effectiveness for crewmembers on the night shift and on the rapid shift rotation schedule" (p. 29). The study reported that such changes in sleep/wakefulness cycles resulted in increased feelings of tension, anxiety, irritability depression and anger, based on objective analysis and subjective reporting (p. 31).

Circadian Rhythm Disorder, Free Running Type (CRSD, FRT) is a disorder in which the endogenous circadian rhythm is not entrained to the 24 hour schedule. Brown, Quan & Eichling (2011) reported the case of a 67 year-old male who experienced a progressively delayed sleep time so that he would cycle around the 24-hour clock approximately every 30 days. The patient's CRSD was associated with severe depression, anxiety and agoraphobia. The case was meticulously documented each night over the course of 22 years. Through a treatment regimen

that included melatonin, light therapy and increased sleep structure, entrainment and stabilization of his circadian rhythm was accomplished.

Seasonal Affective Disorder is a mood disorder driven by disruptions in circadian rhythms caused by diminished light levels during the winter months. The Mayo Clinic (2011) recently identified a variant pattern in which SAD is experienced in the spring and summer months. This variant has anxiety as a primary symptom. Bourne (2005, p. 348) notes that many individuals with SAD also experience anxiety disorders during the late fall and winter. Along with depression, panic attacks and generalized anxiety increase.

Bidirectional Causality in Anxiety and Sleep Disorders: Implications for Therapists

The research presented in this paper supports bidirectional causality in sleep and anxiety disorders. Influenced by recent research in this area, in its rationale for proposed changes to the Circadian Rhythm Sleep Disorder section in the upcoming *DSM-V* (American Psychiatric Association *DSM-V* Development, 2011), the APA acknowledges the bidirectional influence of sleep and anxiety disorders: “Alterations in circadian rhythms can have profound effects on sleep and mental health, and similarly abnormalities in sleep and circadian rhythms are often observed in patients with depressive disorders, schizophrenia, bipolar disorder and anxiety disorders.”

A comprehensive treatment of the implications of this conclusion for therapists is not part of the scope of this paper. Still our conclusions suggest that revisions to current therapeutic approaches that treat these disorders in isolation should be reevaluated and perhaps amended. We end with three points for further consideration. First, since sleeps disorders in general and disruptions in circadian rhythms specifically are risk factors for anxiety disorders and anxiety disorder are risk factors for sleep disorders, early therapeutic intervention when one or the other is diagnosed could have a preventative effect. Second, recognition of the co-morbidity of anxiety

disorders and sleep disorders could lead to changes in treatment strategy. Third, rather than treatment focused solely on anxiety disorders or sleep disorders, a therapeutic approach focused on both disorders might be more effective and efficient.

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