People with obsessive-compulsive disorder (OCD) often have sleep difficulties such as problems with initiating and maintaining sleep and a delayed sleep phase. However, it is unclear if sleep difficulties primarily result from OCD symptoms (e.g., ruminative thoughts, anxiety, compulsive behavior) or an intrinsic problem. Recent studies indicate that sleep problems in people with OCD may be more intrinsic than previously thought.

OCD is an anxiety disorder characterized by intrusive thoughts, images, or impulses (i.e., obsessions) and/or by repetitive behaviors or rituals (i.e., compulsions). The obsessions produce anxiety, and the compulsive behaviors are aimed at reducing the anxiety. A person is aware that the obsessive thoughts or repetitive behaviors are excessive or inappropriate but feels incapable of controlling them. A compulsion or obsession may interfere with the person's occupational, academic or social functioning if it is time-consuming.

Delayed sleep phase disorder (DSP) is a circadian rhythm sleep disorder in which one's peak periods of alertness, sleep and other daily biological rhythms occur later than the societal norms. The length of sleep and wake cycles are normal in people with DSP, but they are physiologically unable to fall asleep until late into the night (usually hours after midnight) and, as a result, they awaken late in the morning. If they attempt to maintain a “normal” schedule, they will have difficulty waking up in the morning and then struggle with sleepiness throughout the day. However, if people with DSP are allowed to follow the biological rhythm of sleeping and waking late, they sleep normally, awaken spontaneously, and do not struggle with daytime sleepiness.

While investigating the common association between OCD and depression, researchers have noted that some OCD subjects apparently have DSP. However, the studies could not determine whether the delayed sleep phase resulted from DSP or from difficulty initiating sleep due to ritualistic behaviors, obsessions or depression. In 2001, Bobdey and colleagues proposed that the results of some of the studies may have been uncertain because they combined populations of depressed subjects without OCD with non-depressed and depressed OCD subjects.

Therefore, they assessed the sleep characteristics of patients with OCD only, patients with major depression only, patients with both OCD and depression, and healthy controls (i.e., no OCD or depression). They found that the sleep characteristics of the OCD-only patients resembled those of people who did not have OCD, and the sleep characteristics of depressed OCD patients resembled those of patients who had major depression only. The researchers concluded that OCD itself does not impair sleep. However, the study did note that a small subgroup within the OCD-only group had a sleep phase shift.

With this in mind, British researcher Suman Mukhopadhyay and colleagues in 2008 investigated the prevalence of DSP in people with chronic, severe, treatment-resistant OCD. Their study involved 187 OCD hospital inpatients. Of these patients, 17.6 percent had DSP, 31 percent had other types of sleep disturbances, and 51.3 percent had no sleep disturbances. The 17.6 percent prevalence of DSP in OCD patients is 17 times greater than current prevalence estimates for DSP in the general population, which range from 0.17 to 0.72 percent. Mukhopadhyay concluded that a more substantial number of people with OCD may have DSP than was previously thought.

The Mukhopadhyay study, however, had some drawbacks. The data they collected was not intended for research purposes, and standard sleep equipment was not used to assess the patients’ sleep. Instead, hospital nursing staff subjectively assessed whether a person was asleep or awake. This may have affected the results.

Jo Turner and colleagues used more extensive questionnaires to assess sleep in relation to an OCD patient’s degree of obsession, depression and disability. These tools included the Pittsburgh Sleep Quality Index, the St. George’s Hospital Medical School Insomnia Questionnaire, the Yale–Brown Obsessive–Compulsive Scale (Y–BOCS), and the Montgomery–Asberg Depression Scale. Based on these questionnaires, Turner found that nearly half (42 percent) of the patients had DSP. OCD patients with DSP tended to be younger and to have more severe OCD symptoms than OCD patients without DSP.

A delayed sleep phase is not the only type of sleep disturbance noted in people with OCD. Arriga and colleagues found that people with OCD have both difficulty initiating sleep and problems with early awakenings. Thomas Insel and colleagues noted a short rapid eye movement (REM) sleep latency, decreased amounts of REM sleep, decreased amounts of slow wave sleep (SWS), and decreased amounts of total sleep time in OCD patients. In contrast, Fritz Hohagen and colleagues reported normal amounts of REM sleep and SWS. However, Hohagen confirmed Insel’s finding of a lower sleep efficiency. Michael Kluge and colleagues found that patients with OCD have significantly less SWS than healthy controls. They noted that a small subset of the OCD patients had a short REM sleep latency, and that OCD symptoms in this small group were worse than in the OCD patients who had a normal REM sleep latency.

The conflicting results found in some studies may reflect the existence of more than one subtype of OCD. If this is true, then different treatment approaches may improve sleep in various subtypes of OCD patients. For example, OCD patients with DSP may benefit from the addition of bright light therapy to other interventions used to treat their OCD symptoms.

Bright light therapy involves brief, daily exposure to light at an intensity that is greater than sunlight. The exposure is scheduled at certain times of the wake cycle to shift (and then

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maintain) one’s sleep/wake cycle at a certain time. For example, bright light exposure soon before bedtime shifts a person’s sleep/wake cycles to a later time (i.e., phase delay), while bright light exposure soon after awakening shifts a person’s sleep/wake cycles to an earlier time (i.e., phase advance). Bright light therapy successfully induces a phase advance in some people with DSP. This allows the person to be able to sleep and wake at more socially acceptable times. No studies have investigated the use of bright light therapy to improve sleep in people with OCD and DSP.

Current treatment approaches for OCD involve psychotherapeutic techniques such as cognitive behavior therapy, drug therapy with medications such as antidepressants, or a combination of drug therapy and psychotherapeutic techniques. Cognitive behavior therapy (CBT) is effective at alleviating symptoms in many people with OCD. CBT involves either teaching a person techniques (e.g., relaxation, distraction, imagery) to counteract obsessive thoughts or exposing the person to anxiety-producing stimuli with the goal of reducing the anxiety and compulsive behavior. Tricyclic antidepressants (TCAs) such as clomipramine and selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, paroxetine, fluvoxamine, and sertraline, are effective at reducing symptoms in some people with OCD. This effect may be related to how TCAs and SSRIs impact the transmission of serotonin in the brain. (Altered transmission of certain serotonergic pathways in the brain may play a role in OCD and other anxiety disorders.)

Physiological factors may link OCD and sleep disturbances since some of the same serotonergic pathways involved in anxiety are also involved in sleep. For example, the dorsal raphe nuclei and the median raphe nuclei in the brainstem play a role in REM sleep. These nuclei have projections to the limbic system, which plays a role in emotions and mood. In animal studies, lesioning these nuclei can result in anxious behavior. However, some recent research indicates that certain drugs used to treat OCD—in particular, fluvoxamine—may induce DSP. Turner cites a study by Hermish in which OCD patients developed DSP while taking fluvoxamine, which resolved on withdrawal from the drug. Because this is a recent finding, future studies are needed to determine the extent that DSP in OCD patients may be induced by the treatment used to alleviate OCD symptoms.

Future studies also may determine if bright light therapy could help advance sleep/wake cycles in OCD patients in whom DSP is not induced by rituals, obsessions or drug therapy. The sleep-inducing hormone melatonin is another treatment that may be helpful in improving sleep in OCD patients. However, scientists have not yet explored this option. Much of the research evaluating sleep in people with OCD has focused on the impact of depression on the sleep of OCD patients. Scientists are now beginning to investigate other factors that could be involved in disrupted sleep in people with OCD. Such research could potentially lead to more options for improving sleep in these patients.

REFERENCES


