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Psychosocial stress and impaired sleep

by Torbjörn Åkerstedt, PhD¹

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This review demonstrates that stress is closely related to impaired sleep in cross-sectional studies. In particular, the anticipation of high demands or effort the next day seems important. Sleep recordings show that stress is associated with shortened sleep, fragmentation, and possibly a reduction in sleep stages 3 and 4. Shortened or disturbed sleep causes increases in levels of traditional stress markers (eg, cortisol) and may thus exacerbate the effects of stress. Much knowledge is still lacking, however, particularly about the effects of real-life work stress. The latter requires longitudinal studies in real-life situations.

Key terms burnout; cortisol; polysomnography; review; work.

It is common-sense knowledge that stress disturbs sleep. Stress causes increased physiological and psychological activation as a response to increased environmental demands (1, 2), and this activation should be incommensurate with the deactivation that is a main characteristic of sleep. However, there is relatively little empirical knowledge about stress and sleep, and the topic of sleep has not caught the interest of occupational health researchers or practitioners, with a possible exception of shiftwork-related issues. This paper attempts to discuss the evidence of a connection between stress and sleep.

Sleep in relation to health and safety

Before turning to the main issues, however, I will seek to justify this focus on sleep with a short review of current knowledge on the relation between impaired sleep and health and safety. Recent research seems to suggest a rather strong relation. For example, epidemiologic cross-sectional studies have shown that especially depression (and other psychological disorders) (3), but also cardiovascular disease, high blood pressure, migraine, lung disorders, and others, are connected to disturbed sleep (4–8). Disturbed sleep is also related to higher levels of sickness absence (7, 9–11), with the burnout syndrome (12, 13), and with increased health care consumption (14). It has also been suggested that disturbed sleep may be part of the link between socioeconomic status

and health since disturbed sleep is more common in lower socioeconomic groups (15).

Also in longitudinal studies, cardiovascular disease (16–19) and diabetes are predicted from prior sleep disturbances (20, 21). Obesity also seems to be implicated in sleep loss (22). This pattern of disease is similar to the pattern of diseases related to stress, sometimes labeled the “metabolic syndrome” (23). Sleep apnea (organic sleep disturbance due to obstruction of the airways during sleep) has recently been listed as one part of the metabolic syndrome (24). Sleep disturbances also increase the risk of subsequent depression (25–27).

With respect to mortality, both reduced and increased sleep duration are important predictors (28). In a long sequence of studies on sleep deprivation in rats it has been demonstrated that total sleep loss leads to death after approximately 5 weeks (29). The cause of death is still debated, but a collapse of thermoregulation seems to be involved, even if sepsis has also been identified (30) that suggests a breakdown in the immune system. In humans, a series of studies of the so-called fatal familial insomnia syndrome (a hereditary gradual necrosis of the sleep-generating nuclei in the thalamus) leads to death over a period of 1–5 years (31).

In addition, the risk of occupational accidents is higher in persons with disturbed sleep (14, 32, 33). It has also been demonstrated in a prospective study that disturbed sleep is a predictor of fatal occupational accidents when physical work characteristics, gender, and stress are controlled for (34). Similarly, several studies

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have demonstrated that shortened or disturbed sleep is closely related to driving accidents (35, 36). Reported sleepiness is also closely related to accident risk (35), as well as to reduced subjective work performance (37).

The aforementioned results show that sleep is essential for physiological balance and long-term health and mental functioning. Long-term impairment of sleep can have serious consequences, and it seems reasonable to consider sleep impairment through stress as an interesting issue in relation to occupational and public health issues.

Reports of stress and disturbed sleep

When the physiological activation involved in the stress response is considered, it seems logical to expect a connection with sleep. In fact, stress is considered the primary cause of persistent psychophysiological (or primary) insomnia (38). In the new international classification of sleep disorders (ICSD2) (39) "adjustment insomnia" represents the effects of acute stress, whereas "primary insomnia" represents the long-term consequences of mainly adjustment insomnia. The empirical evidence is, however, modest, at least in terms of studies of causal connections.

Nevertheless, several cross-sectional epidemiologic studies have shown a strong link between stress and sleep (4, 40–43). Of these, Kalimo et al (44) used the Helsinki heart study and found that high work strain (high demands + low influence) was associated with a 30% prevalence of disturbed sleep versus 5% in the low strain group (low demands + high influence at work). Work strain was defined as high demands and low influence at work according to the "demand-control" index (45). Åkerstedt et al (43) used the same demand-control index but looked at the two dimensions separately. They found that only the demand index had a significant relation to disturbed sleep, that the strongest item of the demand index was "having to exert a lot of effort at work"—not simply "having too much to do", for example. It was also found that, when "not being able to stop thinking about work in the evening" was added to the regression, this variable took over part of the role of work demands as a predictor.

Recently, Fahlén et al (46) showed that also the effort-reward index was significantly related to disturbed sleep. It was also found that immersion (overcommitment) was important. This index included "not being able to stop thinking about work" and four more items, including "start thinking of work immediately when I wake up". This index showed a fourfold increase for the risk of reporting disturbed sleep. These observations

suggest that it may not be work demands per se that are important, but rather the preoccupation with worries about them. This possibility agrees with the results of several of the polysomnographical studies discussed below.

An additional group in this context is comprised of people suffering from burnout, the definition of which requires exposure to long-term stress (47). The characteristic clinical symptoms of the condition are excessive and persistent fatigue, emotional distress, and cognitive dysfunction (47–49). It constitutes a growing health problem in many Western countries (50). In Sweden, burnout is estimated to account for most of the doubling of long-term sickness absence that has occurred since the mid-1990s (51). Self-reports of disturbed sleep are pronounced among people scoring high on burnout (12, 13). In two diary studies, people with high burnout scores showed pronounced sleep disturbances each day across a 2-week period of sampling (52, 53). In these studies sleepiness and fatigue were also measured in 3-hour intervals across each day. In both cases, the burnout group showed a much higher level of burnout sleepiness and fatigue.

Lack of social support at work is also a risk indicator for disturbed sleep (43). Poor (general) social support has been associated, for instance, with sleep complaints among Viet Nam war veterans (6), even if the amount of work available is rather limited.

It should be emphasized that all of the aforementioned studies are cross-sectional and based on self-reports of stress and sleep. Therefore, causation cannot be inferred. The first prospective study seems to be the one by Ribet et al (41) who studied more than 21 000 persons in France, using a sleep disturbance index and logistic regression analysis. It was found that shift work, a long workweek, exposure to vibration, and "having to hurry" appeared to be the main risk factors, when age and gender were controlled for.

Another prospective self-report showed a prospective relation between reported "negative psychosocial work conditions" and insomnia, with an odds ratio of 2.15 for 5 years (54).

In a retrospective study of reported life events, Cernovsky et al (55) demonstrated a clear increase in negative life events before an outbreak of insomnia. In addition, a qualitative approach used in a burnout study showed that an important factor before seeking medical attention for burnout was a period of extreme work overload and a decrease in the time allotted for sleep (to permit more work) (56).

In summary, there is a close relation between reports of stress and disturbed sleep. Anticipation or preoccupation with work seems to be a key component in this relation, but conclusive evidence from longitudinal studies is still lacking.

Stress and polysomnography

Self-reports are important, but a major question in the present review is whether stress affects physiological indicators of sleep. By the latter, I am referring to electroencephalography (EEG) for describing brain waves, electroculography (EOG) for describing eye movements, and electromyography (EMG) for describing muscle tension. The three measures together constitute "polysomnography" (PSG), which is used to classify sleep into stages 1–4 and rapid eye movements (REM) (57). Stage 1 is the stage of transition from being awake to sleep and does not involve any recovery. Stage 2 represents basic sleep and constitutes 50% of the total amount of sleep. Stages 3 and 4 represent deep sleep, with large EEG waves, known as slow wave sleep (SWS), and what appears to be a high level of recuperation. REM sleep is similar to stage-1 sleep, but it also contains rapid eye movements, as well as a complete loss of muscle tone. Stage REM is where fully developed dreams occur.

Unfortunately, there is no detailed consensus on what constitutes objectively poor sleep (58). However, impairment on tests of psychomotor or cognitive performance is closely related to total sleep time in experimental studies (59, 60), and also with the degree of sleep fragmentation (61, 62). Sleep reduction studies suggest that sleep for less than 7 hours will show a gradual impairment across days. Executive functioning is also very sensitive to short or fragmented sleep (63, 64). With respect to sleep stages, it has been suggested that SWS may be of particular importance, but conclusive evidence is lacking (65).

Considering the common-sense notion of stress as a cause of disturbed sleep, there have been rather few studies of the effects of everyday work stress on sleep, possibly because recording sleep may add to an already burdensome situation for a person, and it is often the case that real-life stress is difficult to predict ahead of time and to fit into operating schedules of recording teams.

However, in one field study on stress, it was shown that SWS was decreased in merchant marine officers during nights with on-call duty (but without calls) compared with nights off (66). This reduction in SWS was interpreted as anticipation of the possible alarm or call. Heart rate was also significantly increased during on-call sleep, and this finding suggests an anticipatory stress response.

In another study, aircrew had their sleep recorded before an unpleasantly early morning flight (rising around 0430) and before more normal start times (rising around 0700). Sleep was shortened, and SWS was reduced before the early morning flight, but it was also

found that those who rated their apprehension of the early morning flight as high showed a stronger reduction in SWS (67, 68). This study also suggested anticipation as an important factor.

Among nonwork-related real-life studies, several have concerned sleep before university examinations. In one of these, Holdstock & Verschoor (69) found reduced total sleep time, but it had no effect on the sleep stages. However, this reduced total sleep time was due to changes in sleep behavior (late bedtime) due to studying. Lester et al (70) mainly found reduced SWS. Becker-Carus & Heyden (71) only found a small increase in REM time in a similar situation. In neither of these studies was the stress level (apprehension) associated with the examination measured, and it is possible that the examinations were not seen as important stressors.

Edéll-Gustavsson et al (72) studied patients 2 days before coronary bypass and found that involuntary thoughts predicted sleep fragmentation. No control situation was used, so it is not clear whether the operation actually caused the fragmentation. Beaumaster et al (73) found no effect on inexperienced sky divers the night before their first jump.

A stress situation of sorts is also the first night in a sleep laboratory. It often, but not always, shows less sleep continuity, as well as an increased sleep latency (74–80). Using an interindividual approach, Drake et al (81) developed an index for sleep vulnerability to stress and showed that those who reported habitual sensitivity to stress had a lower first-night sleep efficiency and longer sleep latency than those with low scores.

Some early studies of stress and sleep used an experimental approach and showed, for example, unpleasant films before sleep. This approach did not, however, seem to have much effect other than intensifying the REM sleep (82–84). It is likely, however, that artificial stress without much significance to the individual may not be expected to affect sleep. It may also be significant that this type of experimental study does not seem to involve any anticipation, unlike the other studies discussed.

It was previously mentioned that burnout is caused by long-term exposure to stress. One would then expect sleep to be disturbed. One study of this phenomenon focused on young adults employed in information technology and with high burnout scores (52). Sleep was recorded in the homes of the participants. The results showed that those scoring high on burnout had more microarousals than their age- and gender-matched low-burnout colleagues. The number of arousals was, furthermore, closely related to reported work stress.

In another study, patients on long-term sick leave for burnout were shown to have a high level of sleep fragmentation and reduced sleep efficiency, as well as

reduced SWS and increased sleep latency, as compared with age- and gender-matched normal persons (53). It was suggested that the burnout patients presented a picture of disturbed sleep that should be sufficient to start an accumulation process for fatigue, as suggested by the partial sleep deprivation studies of Van Dongen et al (60). This suggestion will need to be supported empirically, however.

Posttraumatic stress is another well-established cause of disturbed sleep, even if many of the more common indicators of sleep quality (sleep latency, efficiency of sleep, total length of sleep, and the amount of stages 3 and 4) are relatively moderately affected (85–88). Instead, it appears that the greatest part of the effect is connected with a disturbance in REM sleep, in particular in the form of an increased or reduced amount, or intensity, and a great number of awakenings. Unpleasant dreams also lead to a strong negative conditioning to the sleeping situation—one tends to avoid it.

The polysomnographical studies suggest that situations involving an anticipation of increased demands interfere with sleep. A reduction of SWS seems to be common, but fragmentation and other disturbances also occur. There is a lack of longitudinal studies that describe the polysomnographical response to periods of increased stress, particularly in relation to workload, and nothing is known about the effects of longer periods of increased load. Another problem is that most studies assume that a certain situation is perceived as stressful, but no systematic measurement of stress was used.

Stress and sleep physiology

Another way of looking at the stress–sleep intersection is to compare normal or manipulated sleep physiology with that of stress physiology. The latter involves activation of the sympatho-adreno-medullary (SAM) and the hypothalamo-pituitary-adrenocortical (HPA) systems, with increased levels of catecholamines, cortisol, ACTH (adrenocorticotrophic hormone), and CRH (corticotropin releasing hormone), as well as increased activation of the cardiovascular system (89, 90). These stress systems interact with the endocrine, gastrointestinal, and immune systems through complex stimulatory and inhibitory feedback pathways (91–94). At hypo- or hyperactivation the effects on cognitive performance, behavior, and emotions may be negative (95, 96). With respect to cognitive effects, it seems that high cortisol levels lead to impaired memory function through negative effects on neural structures, which have been observed particularly in the hippocampus (97), and it is rather obvious that sleep would be negatively affected by activation of the SAM and HPA systems (90, 92–94,

98). The immune system also responds to stress. Thus the autonomic nervous system turns on genes that activate the innate part of the immune system (99), among them proinflammatory cytokines.

People with disturbed sleep seem to exhibit much the same physiological changes as those under stress. Thus people with disturbed sleep exhibit increased levels of cortisol, heart rate, body temperature, and oxygen consumption (100–105). As a matter of fact, insomnia is currently considered to be caused by an increase in metabolic rate, probably due to prior upregulation of the SAM and HPA systems (106). Glucose tolerance is also increased in sleep disorders (107), as are the levels of proinflammatory cytokines (108, 109). In the study of young burnout participants mentioned earlier, the frequency of micro-arousals was significantly related to cortisol levels, systolic blood pressure, heart rate, and low-density lipoproteins (110).

With respect to the experimental manipulation of sleep, awakenings result in increased autonomic activation (111), as well as bursts of cortisol (112). Partial sleep reduction across some days increases cortisol levels the next day (113, 114). Furthermore, morning awakening is influenced by increasing ACTH levels (115). Thus a person who expects to be awakened unusually early will show an earlier peak of ACTH. If the predetermined awakening is set for a later time, the peak will occur later.

Infusing CRH reduces SWS during the sleep period, and REM during at least a part of the sleep (116). An infusion of ACTH results in an increased sleep latency, lowered SWS, and increased fragmentation (117). The effects of both CRH and ACTH are thus apparent sleep disturbances. Cortisol given during the day, however, results in increased SWS and decreased REM (116), probably through negative feedback via the hippocampus. Another observation is that the activation of mineral corticoid receptors increases NREM (nonrapid eye movement) sleep, while activation of glucocorticoid receptors increases wakefulness or REM (118).

Sleep loss also reduces glucose clearance (114, 119, 120), and blood sugar remains at a high level during sleep instead of falling as is normal during wakefulness. (121–123). This effect is due to the effect of growth hormone on the ability of insulin to promote the uptake of glucose by cells. Lipid levels are also increased in connection with sleep loss (114, 124). Experimental sleep loss also leads to an increased ghrelin level and a decreased leptin level, resulting in increased appetite (125).

Another aspect of sleep and stress markers is that, during normal sleep, cortisol is strongly suppressed during the early phases of sleep, as is thyroid-stimulating hormone during most of the sleep episode (126, 127). In contrast, sleep is a time of increased secretion of, for

example, growth hormone (128, 129) and testosterone (130) and a time of lowered metabolism and blood flow (131). Thus sleep appears to function as the opposite of and antagonist of stress.

The contribution of sleep loss is not only important for helping to explain fatigue and cognitive impairment in relation to stress. It may also serve as a stressor when performance capacity due to sleep loss makes it difficult to live up to demands, and lowered mood is a common observation in sleep deprivation.

The available data show that sleep loss has almost the same effects on metabolic stress markers as stress does. This finding implies that stress probably has secondary effects on metabolic stress indicators, exacerbating the direct effects on health and possibly interfering with subsequent sleep. The latter notion needs to be demonstrated empirically, however.

Animal studies of stress

Animal studies may contribute to the understanding of the association between stress and sleep. An example of such knowledge is that, during stress through immobilization or “social degradation” in mice, SWS and REM decrease during exposure, but increase during recovery sleep (132–134). Exposure to stress that is terminated, therefore, leads to more and probably better sleep. The stress also changes the daily rhythm of corticosterone. CRH (the releasing hormone of ACTH) seems to mediate part of the disruption of sleep due to stress (135). The stress-related increase of ACTH is linked to an increase in REM and partly to an increase in SWS.

The effects of acute stress seem to be centrally mediated since adrenalectomy does not modify it (136). It appears that CRH works as a neurotransmitter in the locus coeruleus and increases the activity in noradrenergic neurons, which increases REM sleep (133, 137). Different types of chronic stress (intermittent electric shocks or learned helplessness) show increased REM during restitution (138). If the acute conditions of, for example, immobilization is prolonged, the increase of SWS and REM disappears (136). Since adrenalectomized mice show a normal restitution of SWS and REM and dexamethasone-treatment re-creates the stress-effect, the peripheral corticosterone should be the mediating factor (136, 139).

Both acute and chronic stress in rodents result in a subsequent restitution of REM and SWS, both of which are suppressed during the period of exposure. It is plausible that the effect is central and CRH-mediated since adrenalectomy does not influence the reactions. It

appears, however, that chronic stress leads to disturbed sleep through increased levels of corticosterone.

Discussion

Although the picture is not complete, the evidence indicates a close link between stress and sleep. Thus stress involves increased psychological and physiological activation in response to demands (140), and an activated HPA system seems incompatible with normal sleep. It seems likely that the resulting sleep impairment causes further increases in the HPA system and thus promotes a vicious circle of stress and insomnia. One might also assume that prolonged stress involves an allostatic up-regulation of the HPA system (140) with its links to the hippocampus, the HPA system, and fatigue (141).

Psychologically, the anticipation of stress may be a key factor. Worry and arousal are antithetical to optimal sleep (142), cortical arousal is the primary feature of insomnia (143), and most insomniacs attribute their problems to cognitive arousal (144). Cognitive arousal at bedtime correlates closely with increased sleep latency (145). Furthermore, an increase in cognitive arousal in good sleepers before bedtime increases objective sleep latency (146, 147). This occurrence is probably related to “rumination” or having intrusive thoughts. Hall et al (148) showed increases in alpha and beta power and reduced delta power in insomniacs with intrusive thoughts (stress-related). It should be emphasized that disturbed sleep in itself may raise worries about being able to sleep the next night, which will contribute to sleep problems the next night, a vicious circle thus being created (149). Disturbed sleep then becomes a stressor in itself.

While available data give some indications of the important role of sleep in relation to psychosocial stress and its long-term effects, much of the necessary knowledge is still missing. The most obvious is the lack of longitudinal studies. Such studies are necessary for describing the natural development of the stress effects on sleep and determining what the first physiological and psychological effects of stress-impaired sleep are. The specific organizational causes of stress-induced sleep problems should also be investigated in longitudinal approaches, even if cross-sectional studies give some indication. An important question also concerns the role of impaired sleep in the development of stress-related diseases—is disturbed sleep part of this development? This review suggests a clear connection, but the different pieces have never been put together in the same study.

The longitudinal approach would also include answers to the question of the degree and duration of stress

necessary to cause impaired sleep. Longitudinal studies are also necessary to provide information on when acute insomnia becomes more or less chronic and which persons are susceptible to stress-related sleep impairment.

Another type of study needed is intervention with sleep advice in organizations or groups with a high risk of stress-related sleep disturbances, including burnout. If sleep is as important as suggested, this type of intervention could serve as a countermeasure. This type of study should also look at the combined effects of work stress and family stress. Little is known about the relative importance of the two.

In summary, stress and sleep seem intimately connected, but there is much need for more systematic research.

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