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Battling Fatigue in Aviation: Recent Advancements in Research and Practice

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Despite knowledge gained through decades of research, fatigue due to insufficient sleep remains an ingrained part of military and commercial aviation and represents a major threat to the health, safety, and effectiveness of aircrew. Long duty periods, high workloads, circadian disruptions, and insufficient recovery time between flights ensure sleepiness is a continued problem for both civilian and military aircrew. The majority of our knowledge concerning the effects of fatigue is gained from acute, total sleep deprivation laboratory-based studies which describe results in terms of the average individual's response to total sleep loss. However, in operational environments, limited sleep over many days, termed chronic sleep restriction, is more commonly experienced than acute, total sleep deprivation, casting some doubt on the operational applicability of many previous studies. Furthermore, recent studies have identified strong individual differences in fatigue resistance. Our understanding of the effects of chronic sleep restriction and the individual differences in response to fatigue is currently limited in comparison to that of acute sleep deprivation. In this review, we identify the substantial progress made over the last 2 decades in closing these gaps. Advances in understanding the effects of chronic sleep restriction, the recovery timeline associated with sleep loss, and individual responses to sleep loss represent a critical step in the improvement of current, and the formulation of future, countermeasures in the aviation environment. Adjustments to duty rotation and crew scheduling, refinement of biomathematical models of fatigue, and application of currently available countermeasures are the most immediate of these improvements.

Key words: sleep deprivation, sleep restriction, aviation, individual differences, recovery

INTRODUCTION

The detrimental effects of sleep loss in aviation have been documented for over 60 years.[1,2] Fatigue has been on the National Transportation Safety Board's (NTSB) list of most-wanted aviation safety improvements since 1989,[3] and has been recognized as a leading contributor to military mishaps.[4-6] Still, fatigue due to insufficient sleep remains an ingrained part of military and commercial aviation. The lack of adequate sleep represents a major threat to the health, safety, and effectiveness of aircrew and passengers. Between 1993 and 2008, fatigue was a factor in 7 U.S. aviation accidents which resulted in 250 fatalities and 52 serious injuries.[7] Similar trends have been noted in military aviation in each of the 3 service branches. Yet, long duty periods, high workloads, circadian disruptions, and insufficient recovery time between flights ensure that sleepiness will continue to be a problem for both civilian and military flight operations.[8-10]

While the body of research regarding fatigue effects and countermeasures is vast, the majority of information is gained from acute, total sleep deprivation-based laboratory studies in which subjects must maintain wakefulness from 24 to 88 hours or more. These studies have proven invaluable in highlighting the average performance deficits associated with inadequate sleep. However, operational experience shows that chronic inadequate sleep is more commonly experienced than acute, total sleep deprivation, and both elicit a wide range of detrimental effects on performance. Our understanding of the effects of chronic sleep restriction is limited in comparison to our understanding of the effects of acute sleep deprivation, as is our understanding of the effects of individual differences in response to fatigue in general. In this review, we identify the substantial progress made over the last 2 decades in closing these knowledge gaps. Specifically, we briefly review the literature concerning the effects of acute, total sleep deprivation and chronic sleep restriction on performance, the recovery timeline associated with both types of sleep loss, and individual responses to these insults. We conclude with a brief summary of currently-used fatigue countermeasures in the aviation environment.

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14. ABSTRACT

Despite knowledge gained through decades of research, fatigue due to insuffi cient sleep remains an ingrained part of military and commercial aviation and represents a major threat to the health, safety, and effectiveness of aircrew. Long duty periods, high workloads, circadian disruptions, and insuffi cient recovery time between fl ights ensure sleepiness is a continued problem for both civilian and military aircrew. The majority of our knowledge concerning the effects of fatigue is gained from acute, total sleep deprivation laboratory-based studies which describe results in terms of the average individual?s response to total sleep loss. However, in operational environments, limited sleep over many days, termed chronic sleep restriction, is more commonly experienced than acute, total sleep deprivation, casting some doubt on the operational applicability of many previous studies. Furthermore, recent studies have identifi ed strong individual differences in fatigue resistance. Our understanding of the effects of chronic sleep restriction and the individual differences in response to fatigue is currently limited in comparison to that of acute sleep deprivation. In this review, we identify the substantial progress made over the last 2 decades in closing these gaps. Advances in understanding the effects of chronic sleep restriction the recovery timeline associated with sleep loss, and individual responses to sleep loss represent a critical step in the improvement of current, and the formulation of future, countermeasures in the aviation environment. Adjustments to duty rotation and crew scheduling, refi nement of biomathematical models of fatigue, and application of currently available countermeasures are the most immediate of these improvements.

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Overview of acute, total sleep deprivation versus chronic sleep restriction

Research concerning the effects of acute, total sleep deprivation on neurobehavioral performance has proliferated, possibly due to the relative ease in its study and magnitude of its effects. Acute, total sleep deprivation is simple to manipulate in a laboratory setting (keep participants awake), easily defined (continuous wakefulness), and readily quantified (time spent awake in hours). Furthermore, its neurobehavioral effects are clear and pronounced; impairment of various functions occurs quickly and increases in a relatively linear fashion, but is modulated by the circadian rhythm. Numerous reviews provide a comprehensive assessment of these effects, including an increase in involuntary miscrosleeps, attentional instability, and judgment errors with a simultaneous decrease in response speed, response accuracy, learning, task-shifting ability, and situational awareness.[11-14] These effects are so consistent under acute total sleep deprivation conditions that Lim and Dinges[12] present meta-analytic evidence of a common, accuracy-based theoretical framework underlying the effects of acute, total sleep deprivation. They suggest that sustained wakefulness results in a build-up of homeostatic sleep pressure; the longer the time awake, the greater the physiologic urge to sleep. This propensity or drive for sleep increases until the initiation of sleep processes begin intruding on waking behavior, in the form of microsleeps.[15] Increases in sleep propensity coincide with increases in subjective sleepiness as well.[15]

Many of the effects of chronic sleep restriction are similar to those of acute, total sleep deprivation, though less thoroughly catalogued. Like acute, total sleep loss effects, performance on measures of reaction time, accuracy, and vigilance-mediated tasks suffer increased errors as additional wakefulness accumulates during chronic sleep restriction. For example, when the performance of subjects receiving either 4, 6, or 8 hours of sleep per night for 2 weeks was compared with that of subjects who were fully sleep deprived for 88 hours, the data indicated performance degradation during all conditions.[16] Specifically, all groups exhibited neurobehavioral response degradations as a result of sleep loss, such as an increase in the number of lapses on the Psychomotor Vigilance Task (PVT; i.e., response times greater than 500ms) and higher levels of subjective sleepiness. However, for the chronic sleep restriction conditions, these changes were most evident among subjects who were restricted to 4 or 6 hours time in bed in comparison to those who had 8 hours time in bed each night, demonstrating a significant dose-response effect. Further, although both acute, total sleep deprivation and chronic restricted sleep loss led to changes in neurobehavioral response, the steepest decline was noted among subjects who were totally sleep deprived. Other studies in which nightly time in bed was reduced to 6 or fewer hours revealed increased response times and decreased cognitive performance;[17,18] these decreases were more rapid with reduced amounts of nightly time in bed.[16,19]

Advancements in characterizing the physiologic processes that underlie acute, total sleep deprivation and chronic sleep restriction have led to more understanding of their similarities and differences. For instance, new information regarding the interplay of sleep-promoting and wake-promoting neurons during sleep loss has led to substantive improvements in biomathematical models of performance while fatigued.[20] Biomathematical models of performance were developed to predict cognitive performance degradation during acute, total sleep deprivation with the goal of identifying unsafe levels of performance. The aviation community was particularly invested in models to help with risk assessment of long missions. The early models were based on acute, total sleep deprivation data and were fairly accurate in predicting performance under such circumstances. However, efforts to apply these models to real-life situations led to recognition of their imprecision when predicting performance under chronic sleep restriction conditions. In a discussion of this imprecision, Van Dongen and Hursh[21] review the addition of a slow, time-constant homeostatic process to the traditional 2-process model of alertness[22] in order to account for the differences in decline and recovery between acute and chronic fatigue. Acute, total sleep deprivation results in fast depletion of resources, followed by a correspondingly fast recovery after normative sleep (i.e., 8 hours). Chronic restriction results in a similar, though slower, trade-off, but only when sleep is restricted to 3 hours or less per night. The rate of recovery never matches the rate of depletion, and performance suffers in a slow, but continuously increasing, fashion. However, when sleep is restricted to 4 to 6 hours per night, the rate of recovery eventually catches up to the rate of decline, resulting in steady, but depressed, performance.[23] This 4 hour mark is referred to as a homeostatic "set-point" in which the cycle of performance depletion during wakefulness and restoration during sleep reaches a suppressed equilibrium.[24] That equilibrium can theoretically be maintained without further decrement until the set-point is breached (i.e., by returning to the nominal average of 8 hours sleep per night for recovery or by dropping to 3 or fewer hours per night for resumed ac-

cumulation of deficit). Previous models of fatigue did not account for the homeostatic set-point; thus, the lack of success of older acute, total sleep deprivation models in predicting the effects of chronic sleep restriction.

While advancements in understanding recovery from different types of sleep loss have improved the ability to predict performance, there is still room for improvement in modeling the effects of fatigue. For instance, the pattern of recovery not only varies with type of sleep loss, but also from person to person. Recent advances in the understanding of these 2 factors – recovery patterns and individual differences – are reviewed next.

Recovery from sleep deprivation

Individuals recover from acute, total sleep deprivation relatively quickly, with results from most studies showing evidence that performance returns to baseline levels within 2 nights of recovery sleep of at least 8 hours.[14] This is not the case with chronic sleep restriction, where returning to well-rested baseline performance can take multiple days for some tasks and at least a week for others.[25] For example, Dinges and colleagues[26] concluded that 8 to 10 hours of sleep for 2 nights allowed recovery of performance on the PVT following a week of sleep restricted to 5 hours time in bed per night. However, other researchers indicated that recovery did not occur on numerous cognitive measures even after 3 nights of 8 hours sleep.[17,19] Instead, initial "recovery" manifests as a new, depressed baseline of performance,[16,19] which is indicative of adaptation to the restricted amount of sleep.

For example, in a well-controlled study of sleep restriction and recovery, participants were tested over 7 days with sleep restricted to either 3, 5, 7, or 9 hours per night.[19] Following this sleep restriction period, 8 hours of recovery sleep were scheduled for 3 days. Data indicated that for subjects who were restricted to 7 or fewer hours of sleep, 3 days with 8 hours of sleep per night were insufficient to restore performance on the PVT to pre-restriction baseline levels. These results contrast the recovery pattern noted in the Dinges *et al.* study[26] described above. However, these studies clearly indicate that recovery from chronic sleep restriction occurs at a slow rate, with the resulting sleep debts affecting neurobehavioral functions, such as alertness and attention.

One possible explanation for the lack of recovery after 3 days is that participants were not fully sleep-satiated prior to the experimental period. Rupp *et al.*[18,27] assigned subjects to either 10 hours time in bed (extended group) or their habitual time in bed (mean of 7 hours) for one week. All subjects were then tested across 7 nights of sleep restricted to 3 hours time in bed, followed by 5 nights of recovery sleep of 8 hours time in bed. The results indicated that for subjects in the extended sleep group, performance on the PVT returned to pre-restriction levels after the first recovery night, but the same recovery was not observed for subjects in the habitual time in bed group. Performance on a math test improved across the sleep restriction period, but the extended group showed better performance on the last 4 recovery nights compared with the habitual group. The authors attributed this difference to the extended sleep group's improved ability to learn the task during the recovery phase of the study. Overall, reduction of the existing sleep debt prior to sleep restriction allowed faster recovery from sleep restriction compared to those who were not pre-loaded with sufficient sleep.

Banks and colleagues[17] extended this line of research in a study to determine whether recovery followed a "dose-response" curve. Participants were provided with 10 hours time in bed for 2 baseline nights prior to 5 nights of sleep restricted to 4 hours time in bed. Following the sleep restriction nights, participants were assigned to 1 of 6 recovery sleep conditions: no sleep, 2 hours, 4 hours, 6 hours, 8 hours, or 10 hours for 1 night. Although the results indicated that higher amounts of recovery sleep were associated with better recovery, statistical modeling of the recovery function revealed that 1 night of 10 hours in bed was not sufficient to restore measures of behavioral alertness to baseline levels.

While evidence from research clearly indicates that recovery from sleep restriction is slow compared to recovery from acute, total sleep deprivation, the exact reason for these results is not fully understood. A possible explanation given for this slow recovery difference is the increased basal forebrain receptivity to the inhibitory neurotransmitter adenosine (AD) following chronic sleep restriction.[14] AD contributes to sleep onset by inhibiting wakefulness-promoting neurons in the basal forebrain. In acute, total sleep deprivation, AD levels in the basal forebrain increase as time awake increases. This build-up is reversed with recovery sleep, with no lasting change to the relative receptivity of the basal forebrain to AD. However, results from research using a rat model have indicated that when rats are exposed to an extended schedule of chronic sleep restriction (i.e., 5 or more consecutive days) the number of AD-A1 receptors increases, likely in response to elevated levels of extracellular AD. When recovery sleep is obtained from chronic sleep restriction, down-regulation of AD-A1 receptors to normal levels may take several days or even a week.

Individual differences in response to sleep loss

As our understanding about the effects of chronic sleep restriction has increased over the past decade, so has the importance of understanding individual susceptibility to the effects of sleep loss. These individual differences in sleep-related behavior, referred to as trototypes, are stable, trait-like inter-individual differences in susceptibility to fatigue[28] and are not yet widely recognized in operational settings. Specifically, workers are still assigned arduous schedules with the misunderstanding that all people are equally susceptible to fatigue. The importance of identifying these susceptible individuals was noted by Van Dongen[29] who reported that the majority of workplace accidents occurring during the night shift were caused by a small number of night shift workers. Moreover, Van Dongen and colleagues[30] systematically demonstrated that individual differences are robust, stable traits. Other studies have found trait-like characteristics which reflect differences in the degree of impaired performance as a result of either sleep deprivation or sleep restriction.[16,18,30,31]

Because these individual differences have been viewed as stable traits, some authors have suggested that one contributing factor may be genetics.[32-34] For example, Landolt[35] described research which revealed that there were significant differences between subjects with the G/A polymorphism of the adenosine deaminase (ADA) gene and the G/G polymorphism. During both baseline assessment and recovery sleep after acute total sleep deprivation, those with the G/A genotype exhibited greater levels of low-frequency delta activity during non-REM sleep and slow-wave sleep than did subjects with the G/G genotype. Similar research has been reported by Viola and colleagues[36] who found that fatigue susceptibility may be related to the PERIOD3 (PER3) gene which previously had been linked to morningness / eveningness traits and considered a circadian, rather than homeostatic factor. Specifically, during the circadian trough, subjects who were sleep deprived for 40 hours and had the 4 repeat allele of the gene (i.e., $PER3^{4/4}$) exhibited superior cognitive performance in comparison to subjects who had the 5 repeat allele (i.e., $PER3^{5/5}$). However, in a sleep-restriction paradigm, Goel, Banks, Mignot, and Dinges[37] found no evidence that the PER3 polymorphism influenced fatigue susceptibility, suggesting that the PER3 gene only influences cognitive performance under total sleep deprivation, but not during sleep restriction. However, under conditions of more extreme chronic sleep restriction (3 hours time in bed per night for 7 nights), Rupp and colleagues[38] found results consistent with those of Viola *et al*.[36] Taken together, these findings indicate that PER3 polymorphisms likely play a role in neurobehavioral vulnerability to sleep loss. Another possible source for these trait-like individual differences is the human leukocyte antigen DQB1*0602. Specifically, polymorphisms of the DQB1*0602 antigen have been linked to differences in sleep architecture and fatigue during chronic sleep restriction, possibly providing evidence of a genetic biomarker for predicting susceptibility to the influence of sleep restriction. Further research with larger sample sizes is needed to investigate this possibility.[39]

Another endogenous trait which has been linked to trototypes is variation in rested baseline neural activity as measured by functional magnetic resonance imaging (fMRI). Caldwell and colleagues,[40] using fMRI, showed that individuals who demonstrated high cortical activity while rested performed better on tasks during 37 hours of continuous wakefulness than did those with lower levels of cortical activity. Mu and colleagues[41] found similar results in a study in which 10 subjects identified as resilient to sleep deprivation showed more brain activation during an fMRI than did subjects identified as vulnerable to sleep deprivation. However, brain activation differences may be related to other measures. Chuah, Venkatraman, Dinges, and Chee[42] kept subjects awake for 24 hours to examine how inter-individual differences in fatigue susceptibility were related to one's ability to inhibit responses on a go / no-go task. Their results indicated that subjects who were more vulnerable to fatigue demonstrated poorer inhibitory efficiency as well as higher levels of cortical activation.

Cortical activation may also explain differences in personality type and therefore, differences in response to sleep deprivation. Results from research using positron emission tomography (PET) indicated that introverts typically demonstrate greater cortical arousal than do extroverts.[43] Killgore, Richards, Killgore, Kamimori, and Balkin[44] found that when subjects were deprived of sleep for 77 hours, extroverted subjects demonstrated greater performance impairment than did introverted subjects. Rupp, Killgore, and Balkin[38] extended these findings in a study to include social experience to the personality factor in their investigation of factors affecting individual response to sleep deprivation. Results indicated that extroverts exposed to a socially-enriched environment were more vulnerable to sleep deprivation than when they were exposed to a socially-impoverished environment. In contrast, variations in social environment did not affect the response of introverts. The authors concluded that so-

cial exposure combines with personality trait to modulate vulnerability to sleep deprivation via effects on levels of brain activation.

Another factor identified as important to an individual's susceptibility to the effects of sleep loss is age. Results from research using chronic sleep restriction has revealed that response times for older subjects declined at a slower rate than those of younger adult subjects; however, performance of the younger adults recovered more quickly than did that of the older adults.[18,45] Further, the research results from Rupp and colleagues[18] showed that younger adults were less aware of the degree of their impairment than were older adults. When combined with the increased rate of performance degradation, these results suggest that young adults are at greatest risk for fatigue-related mishaps.

In addition to the physiologic traits discussed above, researchers have identified that occupation may be associated with a given individual's level of fatigue susceptibility. Research has focused on individuals with highly-demanding jobs such as military pilots. One study examined the fatigue susceptibility of skilled F-117 pilots compared with that of non-pilots in response to 38 hours of continuous wakefulness.[40,46] In addition to concluding that there were significant inter-individual differences in performance as a result of sleep deprivation, this research found that pilots were in general more fatigue resistant than non-pilots. Another study from Previc *et al.*[47] examined the performance of Air Force pilots who completed a series of flight simulations over the course of 34 hours of continuous wakefulness. As expected, most subjects in the study demonstrated deteriorated flight simulation performance over the course of the study, with some subjects' performance decreasing as much as 30%; however, the performance of several other subjects actually improved. Thus, although military pilots may be more fatigue resistant than are age-matched non-pilots, they are still vulnerable to the effects of sleep deprivation.[48]

Though not a stable trait, another factor associated with resistance to the effects of sleep loss is recent sleep history.[49] A study by Harrison and colleagues[33] utilized a 36-hour total sleep deprivation paradigm to compare subjects' performance with the amount of sleep they obtained in the 2 nights prior to study participation. Subjects who regularly slept more were generally more fatigue resistant than were subjects who slept less. Likewise, another study found that increasing subjects' nightly time in bed to 10 hours per night prior to a week of sleep restriction reduced the deleterious effects on performance noted among subjects who received their normal amount of sleep.[18] Contrary to both of these studies however, research from Van Dongen, Baynard, Maislin, and Dinges[30] showed that individual differences in performance were not related to sleep history, but rather to other individual differences which could not be controlled.

The findings from this research indicate that there are certain factors which predict individual differences in fatigue susceptibility. Moreover, the large differences in performance deterioration between fatigue resistant and fatigue vulnerable individuals suggest that there may be important benefits to identifying those who are least fatigue resistant and who should thus use special caution when participating in sustained operations or working night shifts. These differences also indicated that both pilots and the general work force could benefit from the development of a tool or technique which could effectively identify those individuals who are most susceptible to the effects of fatigue.[32,50,51] For example, researchers in Naval aviation have studied the use of non-invasive eye-tracking metrics, such as saccadic velocity, to identify those pilots who are severely fatigued, and to potentially predict those who are most susceptible to the effects of fatigue.[52,53] Preliminary laboratory and field tests of these metrics have demonstrated their ability to effectively explain individual variability in fatigued performance when combined with existing fatigue modeling software already in use by military schedulers. This approach – the informed combination of predictive modeling with real-time, non-invasive physiologic screening – may represent a viable approach to capturing individual differences in fatigue response. Though promising, further research is necessary before this type of approach can be implemented as an operational prediction tool for cognitive performance.

DISCUSSION

Advances in understanding the effects of chronic fatigue, the process of recovery, and individual differences represent a critical step in the improvement of current, and the formulation of future, countermeasures in the aviation environment. Adjustments to duty rotation and crew scheduling, refinement of biomathematical models of fatigue, and application of currently available countermeasures are the most immediate of these improvements.

Duty rotations and crew scheduling

In the recently released rules and regulations for duty and rest requirements for aircrew, the FAA now recog-

nizes the fluctuations in alertness and performance due to circadian rhythms, and has changed rules to allow flightcrew members to work longer hours during the day than during the night. Furthermore, the new rules also allow consideration of changes in time zone acclimation.[54] Recognition of cumulative fatigue led to changes in the amount of time allotted for rest; aircrew are now required 10 hours of rest between duty periods, with 30 continuous hours off during a 7-day duty period. Fatigue Risk Management Systems (FRMS) are beginning to replace prescriptive duty hours and standard crew rotation methodologies. The International Civil Aviation Organization (ICAO) recently adopted fatigue management recommendations which include methods of monitoring and managing fatigue based on current science.[55] While the rules for duty and rest requirements mandated by the FAA and the FRMS recommendations by the ICAO include recognition of sleep need, circadian rhythms, and to some extent, recovery, recognition of individual differences is still lacking. The new management and mitigation techniques recognize the difference between acute and chronic fatigue, but assume an equal response to fatigue across individuals. The next step for FRMSs may be to further tailor these systems to take these factors into account, adjusting rest and recovery times according to an individual's fatigue susceptibility profile and the type of fatigue experienced. However, the former will require more advances in knowledge of what individual difference factors account for the greatest variability in fatigue susceptibility.

Biomathematical models of fatigue and performance

Integration of fatigue type, recovery, and individual difference data into the FRMS framework requires improvements in biomathematical models of fatigue and performance. The original 2-process model of sleep has incorporated some of this information into its prediction methods, resulting in the Sleep / Wake Predictor model.[22] Other modelers have also integrated chronic fatigue information in their algorithms. The Civil Aviation Safety Authority provided an overview of 6 fatigue models. Of these 6 models, 5 included chronic sleep restriction components into their algorithms.[56] Of the models which included chronic sleep restriction in their parameters, the Sleep, Activity, Fatigue, and Task Effectiveness (SAFTE) model was developed based on results from the study by Belenky *et al*.,[19] and later validated on a 14-day sleep restriction study. The Circadian Alertness Simulator (CAS) model parameters were developed with data sets of 2-4 weeks in duration from real-world transportation workers who slept a variety of different lengths and schedules. However, because the scientific knowledge of chronic sleep restriction effects and recovery from chronic sleep restriction are still developing, model estimation of performance under these conditions requires ongoing validation.

The next step in improving prediction accuracy of fatigue and performance is to include individual variability since model output generally represents the average individual. Building confidence intervals around the averaged performance predictions is one way to quantify individual differences in fatigue susceptibility. While this method captures some of the variability in individual performance, it still lacks specificity to the individual, which is desired by operational communities. Modeling individual crew members will advance the usefulness of model predictions in specific environments. By using situation- and individual- specific models to predict performance, followed by corresponding FRMS implementation of duty, rest, and recovery schedules, it will be possible to improve the most effective fatigue countermeasure available: prevention.

Practical application of current countermeasures

When adequate sleep is either impossible or impractical, such as in some military operations, there are a number of countermeasures that can be implemented.[8,57,58] In-flight countermeasures such as cockpit napping, breaks, and light are all available to help increase alertness and performance. Napping has been shown to enhance alertness and performance in sleep-deprived individuals,[58-61] and specifically in sleepy pilots.[62-64] While the FAA does not allow pilots of US carriers to participate in cockpit napping on domestic flights, other countries do allow this behavior (e.g., Canada, New Zealand, Great Britain, Australia). Cockpit napping is safe and effective, but is not a replacement for good sleep practice prior to the flight, or for in-flight bunk sleep scheduled during long-haul operations.

When sleep is not an option, there are other countermeasures which are effective in increasing alertness. Taken under appropriate circumstances, breaks as short as 10 minutes can interrupt the boredom associated with monotonous tasks. Time away from a task has been shown to increase attention and alertness.[65,66] Light exposure is also effective for maintaining pilot alertness in the cockpit. The flight deck environment is dark and thus conducive to promoting sleepiness, so increases in light even to 100 lux could be beneficial for increasing alertness[67] with short wavelength light showing the greatest

effect.[68]

Pharmacological alertness aids are beneficial when behavioral countermeasures are no longer sufficient. Results from most studies show that caffeine (which is available in various forms e.g., coffee, tea, gum) improves alertness and performance, especially in those individuals who do not consume high doses on a regular basis.[69] Prescription alertness aids (i.e., stimulants) are allowed in some US military operations when proper scheduling has still not allowed adequate sleep. All 3 US services allow some form of prescription alertness aid, either dextroamphetamine or modafinil, during certain operations to enhance alertness and safety during a mission. Use of prescription alertness aids during military operations is strictly controlled and implemented under a Performance Maintenance Plan which requires individual consent. Proper scheduling of crew and missions is the goal, with stimulant use authorized only when periods of unavoidable sleep loss occur.[70]

SUMMARY

As knowledge is gained concerning differential recovery patterns following chronic versus acute, total sleep deprivation and the manner in which individuals respond to sleep loss, countermeasures to offset the effects will continue to be developed and implemented in aviation practices. The way forward includes improvement in duty rotations and scheduling by giving greater consideration to circadian factors, and improvements in biomathematical models that predict performance effects of chronic sleep restriction. As information accrues from ongoing research, the effects of individual differences and countermeasures will be incorporated in these models. Additional research is needed to investigate the extent to which countermeasures affect performance and alertness during chronic sleep restriction. Additional research also is needed to determine how individuals respond to countermeasures based on their individual vulnerability to sleep loss. Aviation technology continues to push the limits of pilots. Continued advances in the science of fatigue are required to keep pace with such technology in order to improve safety in the aviation environment by enabling targeted, more effective fatigue countermeasures.

DISCLOSURE

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, nor the U.S. Government.

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