Fatigue, Performance, Errors, and Accidents

Hans P.A. Van Dongen and Steven R. Hursh

Chapter

67

Abstract

Fatigue caused by extended work hours and shift work induces cognitive performance deficits and increases risk of human error, incidents, and accidents. Fatigue results from the interaction of sleep-homeostatic and circadian drives for sleepiness with time on task and cumulative duty time effects. Hours-of-service rules typically only regulate duty hours, leaving homeostatic and circadian factors unaddressed. Human error due to fatigue is fundamentally stochastic, which has made it difficult to demonstrate the role of fatigue in specific accident cases. Self-reported, subjective sleepiness cannot be relied upon in this context, because it has a low correlation with actual performance impairment. It is posited that fatigue results in an accident when fatigue-induced, randomly occurring periods of inattentiveness coincide with high cognitive demands while the impact of human error is significant. Biomathematical models of fatigue and performance have been successfully applied to forecast the risk of accidents. Such models may thus be used to help improve performance and safety in operational settings. Clinicians should discuss the risk of errors and accidents due to fatigue with their patients and should explain that because of the stochastic nature of human error, past performance does not guarantee future performance and safety.

In large-scale correlational studies, sleepiness-inducing schedules including extended work hours and shift work have been linked to increased risk of human error, incidents, and accidents,[1-5] thus leading to reduced safety and productivity.[6-9] Yet, any given sleepy person does not simply by being sleepy make errors or cause accidents; and conversely, any person who is fully alert does not necessarily perform his or her tasks without errors. In investigations of specific accidents, establishing a causal relationship with sleepiness is often a tenuous endeavor, even if the presence of sleepiness—or "fatigue" as it is typically referred to in operational settings—is itself undisputed. There are two major reasons for this. First, fatigue is rarely the only reason for accidents to occur; multiple, diverse factors ranging from personnel shortages to equipment failures and safety check overrides typically combine with human error to lead to adverse outcomes. Second, human error due to fatigue is fundamentally stochastic. In this chapter, fatigue as a risk factor for errors and accidents is explored in terms of the sleep–wake–related regulation and expression of sleepiness.

SLEEP, CIRCADIAN, AND TIME-ON-TASK FACTORS MODULATING RISK OF ERRORS AND ACCIDENTS

Sleep homeostasis and circadian rhythmicity interact to determine sleepiness and performance capability (see Chapter 38). Sleepiness changes as a function of time awake, with longer wakefulness inducing a progressively increasing sleep drive. Sleepiness also varies as a function of time of day, with nighttime on the biological clock resulting in elevated sleep drive. As a consequence, alertness level and performance capability are reduced when working extended hours involving sleep loss and when working at night or in the early morning.[10,11] Moreover, chronic sleep loss leads to cumulative degradation of performance across days to weeks.[12,13] It has been documented that the circadian (time of day) variation in performance is associated with a circadian rhythm in accident rates and injuries.[3,14] Much less evidence is available for a relationship of accidents and injuries with homeostatic (time awake) changes in performance. However, a relationship between homeostatic sleepiness drive and accident risk might be inferred from statistics on road crashes attributed to the driver having fallen asleep.[15,16] Although definitive evidence is lacking, it would be reasonable to assume that the same interaction between homeostatic and circadian processes that increases sleepiness and decreases performance capability also increases the risk of errors and accidents.

Hours-of-service regulations and policies for operational settings tend to ignore homeostatic and circadian factors and rather focus on duty time. There are good reasons for this. Dealing with homeostatic and circadian effects would require regulating how much and when people are awake, which would be nearly impossible to enforce outside work hours. In addition, even when nominal alertness levels are high, performance deteriorates as a function of time on task.[17] This phenomenon extends to duty hours: Generally, the longer the work period, the more performance degrades.[18] This effect is evident in the effectiveness of rest breaks as a countermeasure to performance degradation.[19] Performance degradation also accumulates across consecutive work shifts,[20,21] as illustrated in Figure 67-1. Thus, restricting work hours limits performance impairment and helps to reduce incidents and accidents.[22]

Homeostatic and circadian drives for sleep interact with the time-on-task effect, however, such that when sleepiness is increased, performance degrades faster across task duration (see Chapter 65). Sleep homeostasis and circadian rhythms likewise interact with the effect of the duration of work.[20] Thus, hours-of-service regulations may be only partially effective when they ignore homeostatic and circadian factors. They can be inadequate in night and shift-work settings, while potentially being overly restrictive during normal daytime operations. In addition, hours-of-service regulations do little to protect workers from fatigue during the commute to and from work.[23-25]

Although the terminology used in the literature, in policy documents, and in jurisprudence is varied and

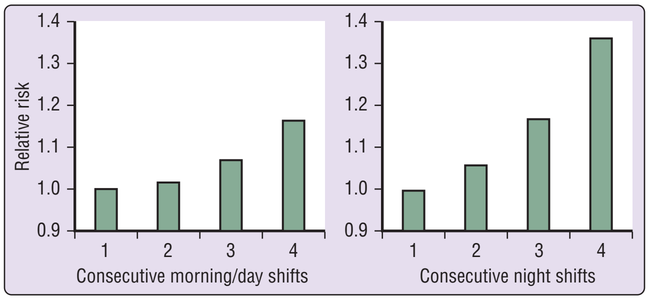

Figure 67-1 Risk of accidents and injuries across four consecutive morning or day shifts *(left)* and across four consecutive night shifts *(right),* expressed relative to the first shift in the sequence. Data were compiled from five published studies on 8-hour shift systems. (Graphs redrawn from Folkard S, Åkerstedt T. Trends in the risk of accidents and injuries and their implications for models of fatigue and performance. Aviat Space Environ Med 2004;75:A161-A167, with permission.)

inconsistent, it is useful here to define "fatigue" as the combined influence of sleep homeostasis, circadian rhythm, and time on task on performance capability. So defined, fatigue may be seen as an index of basal risk of errors and accidents. This broadly incorporates effects of sleep disorders and other illnesses as well: Many clinical conditions cause fatigue through disruptions of sleep homeostasis or circadian rhythmicity (as discussed in several other chapters in this volume).

There are additional, indirect effects of sleep loss on accident risk. Sleepiness tends to promote impulsivity and risk taking,[26-28] and impairs self-monitoring of error.[29,30] To what extent these effects contribute to incidents and accidents beyond the direct effects of sleep loss on performance is unknown, although risk taking and fatigue have been noted to be a deadly combination in young male drivers.[31,32]

A variety of other factors unrelated to sleep–wake contribute to the risk of errors and accidents in operational environments[33-35] and on the road.[36] These include task demands, prior training and experience, safety measures, risks inherent in the tasks at hand, and others, as discussed elsewhere.[37-39]

FATIGUE, PERFORMANCE IMPAIRMENT, AND WAKE-STATE INSTABILITY

It is not well understood why persons impaired by sleep loss, adverse circadian timing, or long duty hours appear to have poor awareness of their performance impairment and increased risk of error, and they do not take steps to avoid accidents and get out of harm's way. Investigations of automobile drivers about whether or not individual drivers are prospectively aware of their own deficits have yielded mixed results.[40-44] Laboratory sleep-deprivation studies have revealed that there are systematic disconnects between self-reported subjective sleepiness and objectively measured performance deficits. For instance, there are considerable individual differences in accident-proneness[45,46] and substantial, traitlike differences in the level of sleepiness and in the level of performance impairment people experience as a consequence of sleep deprivation.[47] However, the correlation between individual differences in subjective sleepiness on the one hand, and individual differences in objective performance impairment[47,48] or driving simulator accidents[49] on the other hand, is low. In addition, chronic sleep restriction leads to cumulative performance deficits, but these deficits are not tracked accurately by subjective sleepiness.[13] As a result, people cannot unequivocally be expected to assess their own risk level accurately.[50,51]

Detailed examination of moment-to-moment fluctuations in cognitive processing may yield some insight into this matter. Figure 67-2 shows reaction times on a 10-minute psychomotor vigilance test (PVT), a simple reaction time test with stimuli appearing randomly at intervals of 2 to 10 seconds. The test was administered every 2 hours during an 88-hour period of laboratory-controlled sleep deprivation.[52] In Figure 67-2, one subject's raw data at 12, 36, and 60 hours awake (i.e., at 24-hour intervals) are displayed. At 12 hours awake, reactions times are consistently short (predominantly in the range of 200 to 250 msec), representing optimal performance capability. At 36 hours awake, some reaction times are longer, indicating occasional errors of omission (lapses of attention). In addition, there are a few false starts—these reflect errors of commission.[52] At 60 hours awake there are many more errors of omission, and errors of commission are also still present. Error responses are increasingly prevalent toward the end of the 10-minute test duration, which reflects the time-on-task effect.[17,52] Note, however, that normal responses continue to be mixed in with the error responses. From moment to moment, it is unpredictable whether a response will involve an error or not. This illustrates that performance impairment due to fatigue is a stochastic phenomenon.

The random variability in performance impairment during sleep deprivation has been theorized to be due to *wake-state instability:* moment-to-moment fluctuations brought about by an "elevating homeostatic drive for sleep, resulting in rapid and uncontrolled sleep initiation, which subjects seek to resist using increasingly greater compensatory effort to perform."[52] In this view, performance lapses are caused because the brain is effectively (if not actually) falling asleep in part or in whole for a brief period[52,53] during otherwise normal (albeit effortful) wakefulness. If this explanation is correct, then a sleep-deprived person might not be aware of any performance deficits because concurrently he or she is unable to perceive any errors of omission. This could provide an explanation for the discrepancy between subjective (i.e., perceived) sleepiness and actual performance deficits from sleep loss.

In accordance with the time-on-task effect (or "vigilance decrement"[54]), performance impairment is minimal when a task is just begun, and only exposed more substantively when task duration is extended (see Fig. 67-2, middle and bottom panels). This might provide a further explanation for the discrepancy between subjective sleepiness and objective performance impairment in situations where tasks are typically brief and distinct and the time-on-task effects are limited. Such an explanation implicitly assumes that subjective awareness of sleepiness is driven by

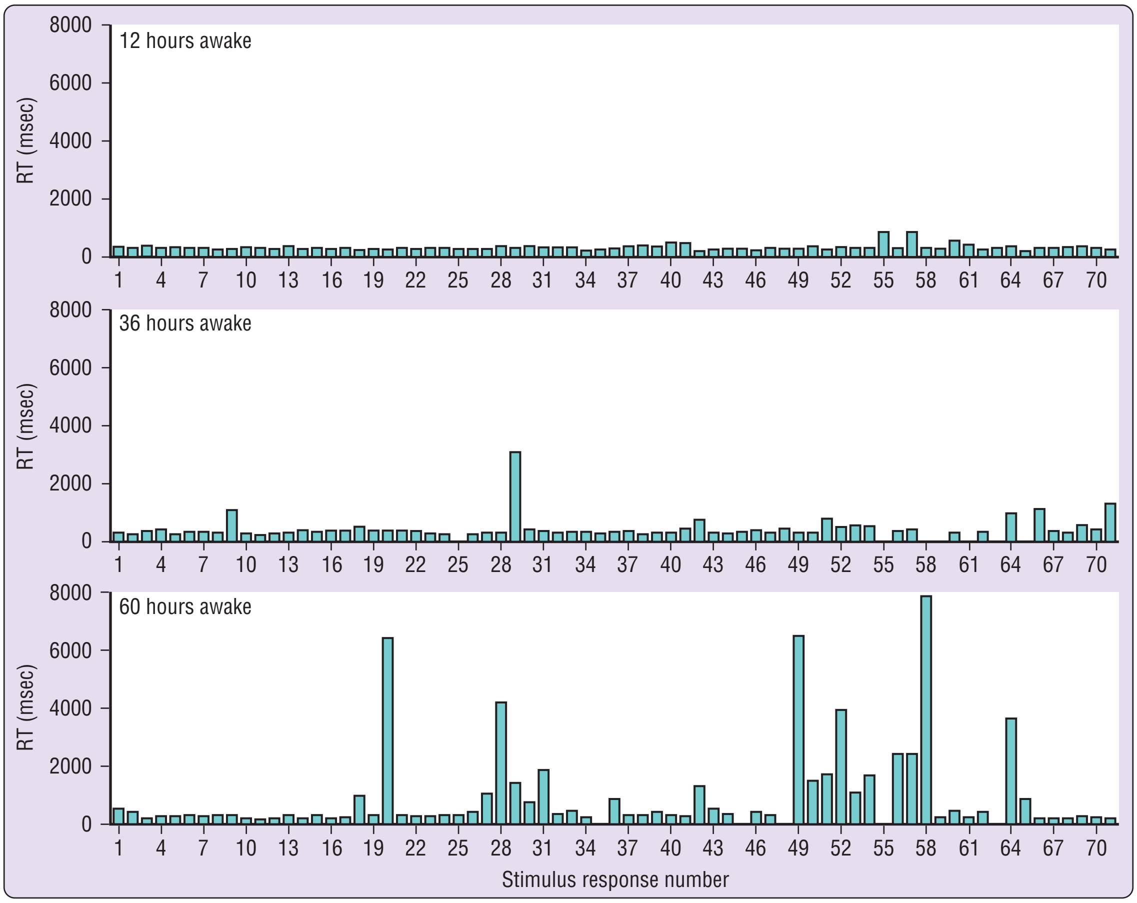

Figure 67-2 Individual reaction times (RT) in milliseconds on the psychomotor vigilance test (PVT) for a subject undergoing total sleep deprivation. The panels each show raw reaction time data as observed during a 10-minute PVT bout administered in 24-hour intervals (at 8 PM) over 60 hours of continuous wakefulness. (Graphs redrawn from Doran SM, Van Dongen HPA, Dinges DF. Sustained attention performance during sleep deprivation: evidence of state instability. Arch Ital Biol 2001;139:253-267, with permission.)

perception of one's cognitive performance. Indeed, knowledge of performance level interacts with the effects of sleep deprivation,[55] and subjects calibrate their self-reported sleepiness after receiving performance feedback (see Fig. 38-1). However, it should be noted that this recalibration is short-lived, and subjects quickly revert back to what appears to be a distinct, internal subjective state (see chapter 38).

The PVT is a task requiring sustained attention to perform, and it is therefore sensitive to lapses of attention.[54] Indeed, the effects of sleep deprivation are readily exposed on the PVT[56] (after the first 1 or 2 minutes of task duration have passed), as is illustrated in Figure 67-2. Many occupationally relevant tasks ranging from systems monitoring to threat detection also require sustained attention[57] and are likewise vulnerable to the adverse consequences of sleep loss.[58,59] In modern operational settings with high degrees of automation, such tasks are prevalent. Automation and other technological innovations have broadly improved safety, but by shifting performance demands to sustained-attention tasks, they may have simultaneously increased the likelihood of human error.[60] The paradoxical result is that serious accidents are increasingly rare, but when they do occur, their outcomes are often dramatic and costly.[61]

PREDICTING ACCIDENTS

Because accidents are rare and ostensibly randomly occurring events, it has remained difficult to predict the risk of fatigue-induced accidents despite increasing knowledge of the processes underlying fatigue. Even when considering incidents more broadly by including near-accidents ("near-misses"[62]) and other performance errors, a relationship between fatigue level and accident rate is still difficult to discern and even considered by some to be controversial.[63] Consideration of the stochastic nature of performance impairment due to fatigue, as discussed earlier, may shed some light on this issue. Using the PVT as an assay of performance impairment,[57] the series of reaction times in a test bout may be seen as a record of task inattentiveness (sampled randomly every 2 to 10 seconds). Assuming for illustration purposes that the same pattern of cognitive function could apply during a given job task, then the observed reaction times (minus ~250 msec necessary for responding even when fully alert) would indicate the intervals of inattentiveness (lapses of attention) during the task at hand. When the demands for cognitive processing are high during such an interval of inattentiveness, human error would occur. And if the impact of human error at that specific time is high, then an accident could take place.

For example, if the task involves driving a car, and an interval of inattentiveness coincides with the approach to an intersection with a stop sign, then the detection and processing of the stop sign would fail and the intersection would be crossed without braking: human error. If at the same time another car enters the intersection as well, a collision could ensue: an accident. Yet, if no other car had been nearby, or of there had been no intersection, or if the interval of inattentiveness had occurred a little earlier or later, then no accident would have occurred.

In this view of the relationship between fatigue and accidents, illustrated in Figure 67-3, it is necessary for a period of inattentiveness, high cognitive demands, and significant impact of error to all line up temporally in order for fatigue-induced impairment to actually result in an accident. Indeed, the same circumstances—same level of fatigue, same task demands, and same impact of error—can happen many times without noticeable consequences, until one day, due to a small difference in the timing of attentional lapses, suddenly a major accident occurs.

Such appears to have been the case with the crash of Comair flight 5191 at the Blue Grass Airport in Lexington, Kentucky, in the early morning of August 27, 2006. The pilots as well as the air traffic controller failed to notice that the plane, a commercial jet requiring a long runway, was erroneously positioned on a general aviation runway that was much too short for takeoff. The plane crashed about half a mile past the end of the runway, killing 49 people. Both pilots as well as the air traffic controller were fatigued due to extended work hours and the early time of day of the scheduled flight. Runway choice at the airport required considerable cognitive processing because the taxiway signage was poor at the time; the appearance of the runways is similar both from the holding point and from the beginning of the take-off roll; the entry points from the taxiway to the two runways are close together; and the taxi to the holding point is short, leaving little time for completing checklists and for orientation and decision making. Furthermore, the impact of human error was clearly substantial. These critical factors coincided and precipitated the tragic event.

Yet, the same circumstances were present at the Blue Grass Airport almost every morning, without any other crash. What exactly made the difference on the fateful morning of August 27, 2006, is impossible to ascertain. Inferring from the conceptual framework sketched out in Figure 67-3, though, it would seem that by chance the pilots' and air traffic controller's fatigue-induced intervals of inattentiveness overlapped with the critical periods of cognitive processing that should have flagged the incorrect choice of runway and prevented the accident.

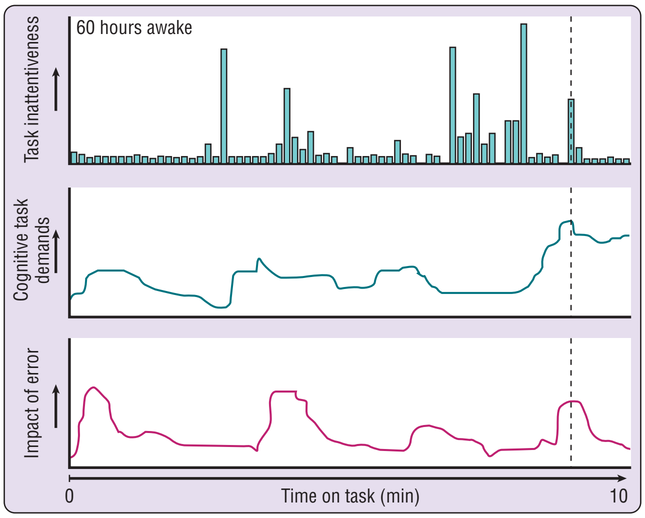

Figure 67-3 Schematic of the (speculative) mechanism by which fatigue contributes to accidents. *Top,* Stochastically occurring intervals of inattentiveness across a 10-minute span of task performance taking place at 60 hours of total sleep deprivation. *Longer bars* represent slowed responses indicating attentional lapses (taken from Figure 67-2). *Middle,* Hypothetical pattern of changing cognitive demands across the duration of the task. *Bottom,* Hypothetical level of impact a human error would have over the course of the task. In this view of how fatigue contributes to accident causation, intervals of inattention when cognitive task demands are high lead to human error, which in turn, if the impact of error is considerable, results in an accident. Thus, for fatigue to actually lead to an accident, attentional lapses must line up temporally with high cognitive processing demands and high impact of human error—in this case at the *dotted line.*

MODELING SLEEP–WAKE–WORK AND ACCIDENT RISK

Figure 67-3 may serve as a heuristic for understanding the complex relationship between fatigue and accidents. It illustrates that accident risk, while fundamentally stochastic, is proportional to both total time of inattentiveness (cumulative lapse time) and density of critical task events (i.e., prior risk or exposure[64]). Given information about the latter, it may be possible to predict accident risk by predicting cumulative lapse time. There is a strong correlation between the number of lapses of attention and their duration,[65] and so mathematical models capable of predicting PVT lapse counts could serve this purpose. Several biomathematical models of fatigue and performance based on the neurobiology of sleep–wake regulation (see Fig. 38-5) can predict PVT lapses for a number of different sleep–wake scenarios[66] and may thus be useful in this context (see Chapter 66 for a more detailed discussion).

Some successful applications of sleep–wake biomathematical models of fatigue for predicting accidents have been documented.[4,67] Even so, the usability of fatigue and performance models for predicting accident risk has been questioned.[20] A reason is that under conditions of (presumably) near-constant operational conditions, the daily peak in published trends for accident frequency in 8-hour shift systems is around midnight,[3,20] which is several hours earlier than the circadian peak in performance impairment (see Chapter 38). However, interpreting the timing of the daily peak in accident frequency in circadian terms only is not likely to be appropriate. Morning, afternoon, and night shifts are typically accompanied by different sleep–wake behavior, and thus sleep homeostasis also plays a role. Furthermore, in just about any operational setting it is difficult to establish the temporal consistency of (or correct for the temporal variation in) other factors that contribute to accident risk.

One way to circumvent the difficulties of disentangling the various factors that contribute to incidents and accidents and that are or are not related to fatigue is to predict accident risk on the basis of descriptive modeling of published incident data. This idea has led to a risk index for assessing work schedules.[3,21,68] Because this approach is purely descriptive, however, its generalizability across different shift systems is uncertain. Prediction strategies based on the neurobiology of sleep–wake and fatigue give greater confidence that findings for one shift system will generalize to another, because the underlying mechanisms involved do not change.

For the purpose of predicting accidents with a biomathematical model of fatigue and performance based on sleep–wake, accident risk may be defined as an odds ratio.[69] This odds ratio is expressed as the percentage of accidents co-occurring with a given range of predicted fatigue (incidence level) divided by the percentage of time spent working in that range of predicted fatigue (exposure level). For example, if 20% of accidents occur in a particular range of predicted fatigue while 10% of work time is spent in this range of predicted fatigue, then that specific range of fatigue is associated with a doubling (odds ratio 2) of accident risk.

A study published by the Federal Railroad Administration (FRA) applied this technique to validate accident risk predictions made with a biomathematical model of fatigue and performance.[4] The data set at hand concerned 400 human-factors accidents and 1000 non–human-factors accidents in railroad operations. The SAFTE fatigue model[70] was used to predict operator performance, on an effectiveness scale from 0 (worst) to 100 (best), at the time of the accidents. The model predictions were based solely on the work histories and estimated sleep opportunities of the locomotive crew in the 30 days leading up to each of the accidents.

The results of this analysis indicated that there was a significant, high correlation between reduced predicted crew effectiveness (i.e., increased fatigue) and the risk of a human-factors accident, as displayed in Figure 67-4. No significant relationship was expected, and none was found, for non–human-factors accidents. At predicted effectiveness scores below 70, the risk of human-factors accidents was elevated above chance level and was greater than the mean risk of non–human-factors accidents.[71] At such low levels of predicted effectiveness, accident cause codes (defined by the FRA to indicate the factors that caused the accident, such as passing a stop signal or exceeding authorized speed) were of the sort expected to be related to fatigue, which confirmed that the detected relationship between accident risk and predicted effectiveness was meaningful.

A significant relationship was also found between reduced predicted crew effectiveness (fatigue) and increased accident damage costs. Human-factors accident costs were 2.5 times greater when predicted effectiveness was below about 77.5 as compared to above 90. Figure 67-5 shows the relationship between predicted effectiveness and damage risk (i.e., the combination of the risk of human-factors accidents and their damage costs).

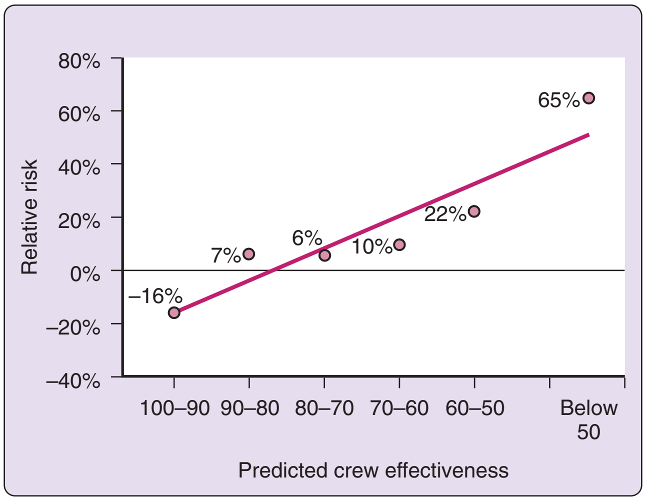

Figure 67-4 Human-factors accident risk, from aggregated data of five railroads, at specific predicted ranges of decreasing effectiveness (increasing fatigue). The *line* indicates the linear relationship between predicted crew effectiveness and relative risk ($r = -0.93$). A relative risk of 0% corresponds to an odds ratio of 1, which is the chance level of risk. Less than 0% is reduced risk; greater than 0% is elevated risk. A relative risk of 65% means that accidents were 65% more frequent than chance.

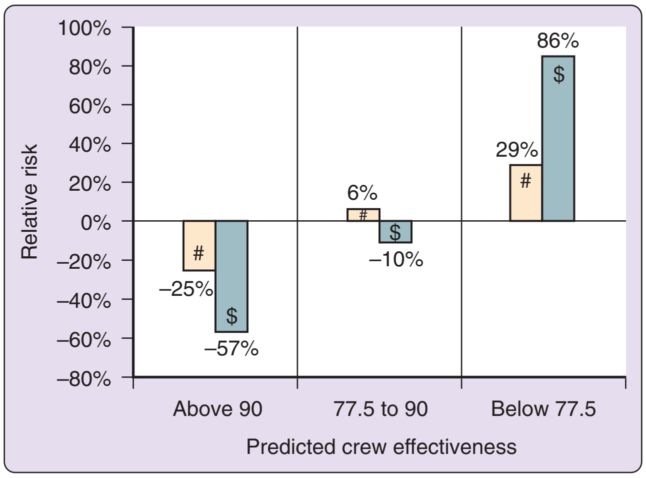

Figure 67-5 Human-factors accident risk and damage risk as a function of predicted ranges of decreasing effectiveness (increasing fatigue). The zero line represents a risk level equal to the overall (average) risk. #, frequency of accidents; $, damage costs.

The significant relationships revealed in this FRA study confirm that accident risk can be predicted, at least to some extent, on the basis of fatigue as predicted from sleep–wake–work schedules. The specific risks associated with given levels of predicted fatigue are likely to be occupation-specific and related to the demands of the job, and data from rail operations should not be used to predict risks for other occupations. Nonetheless, it is likely that systematic relationships between predicted fatigue and risk of accident and damage exist in almost any occupational setting. Initiatives to avoid work schedules that induce high levels of fatigue should thus result in a beneficial reduction in risk for any safety-sensitive job.

CONCLUSION

Fatigue is believed to be involved in hundreds of thousands of road accidents each year, and it has been cited as a contributing factor in occupational disasters such as the meltdown of the Chernobyl nuclear reactor, the grounding of the *Exxon Valdez* oil tanker, and the ill-advised launch of the *Challenger* space shuttle. Fatigue from sleep loss and circadian factors—and, by extension, fatigue from sleep disorders—combines with the effects of time on task and duty hours to induce performance impairment and thereby increase risk of errors and accidents. It should be clear that the various factors contributing to accident risk must be considered simultaneously because they interact. For example, the maximum length of a work shift considered to be safe would vary according to the circadian timing of the shift. Present-day work-hour regulations tend to consider risk factors independently,[3] and as mechanisms for promoting safety and productivity they can thus be ineffective by in some circumstances being too liberal and in other circumstances being overly restrictive.

The concepts laid out in this chapter are part of a developing science and, in part, await substantiation with further experimental data. They may nevertheless be used as a heuristic for understanding how fatigue leads to accidents, how the temporal changes in accident risk arise, and how the stochastic nature of performance impairment induced by fatigue contributes to the rare and seemingly unpredictable nature of accidents. Such information is important for implementing fatigue risk management (see Chapter 68), and for the cost–benefit or actuarial analyses that justify the allocation of resources to fatigue risk management.

At the level of individuals, the consequences of fatigue and the need to intervene remain difficult to gauge. A clinician seeing patients experiencing fatigue should have discussions with them about the risk of errors and accidents and should document this in their files. The clinician may try to determine the risk level by asking questions about the type of job, nature of job (safety sensitive, mission critical), level of exposure, history of incidents or "near misses," safety measures that could be put in place (e.g., rest breaks, ergonomic tools), habitual sleep–wake and work schedules, length of commute to and from work, and likely outcomes if an adverse event were to occur.

It is important to explain that a patient's own subjective evaluation of his or her impairment resulting from fatigue may be inaccurate and, because of the stochastic nature of human error, past performance or safety does not guarantee future performance or safety. Bringing these issues to the attention of the patients may prompt them to make behavioral changes and may thereby help to reduce their fatigue-related risk of errors and accidents.

❖ Clinical Pearl

Fatigue from sleep loss, circadian misalignment, and sleep disorders, in conjunction with time on task, duty hours, and consecutive work shifts, results in cognitive impairment and contributes to errors, incidents, and accidents. In general, individuals cannot be relied upon to accurately self-estimate their accident risk, but increased risk can be predicted using biomathematical models of fatigue and performance.

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