Deficient sleep is ubiquitous and has been associated with a broad range of psychopathology (Harvey, Murray, Chandler, & Soehner, 2011). Difficulties with emotions and emotion regulation have similarly been identified as a potential transdiagnostic process across various forms of psychopathology (Kring & Sloan, 2010). Recently, this has led to an increased interest in the relationship between sleep and emotional experiences. The aim of this chapter is to provide a framework for understanding how sleep and emotions relate to one another. Because the relationship between sleep and emotions appears to be moderated by the presence of psychopathology (see Chapter 11), we focus on studies looking at individuals without a current psychiatric disorder. Understanding the relationship between sleep and emotions in the absence of psychopathology could provide insight into how such processes might be altered following the development of psychopathology.

In evaluating the relationships between sleep and emotional functioning, we must further elucidate and parse our definition of sleep deficiency along the following three dimensions: acute-chronic, intrinsic-extrinsic, and subjective-objective. For instance, insomnia is characterized by chronic intrinsic and subjective sleep deficiency, whereas sleep deprivation is characterized by acute extrinsic and objective manipulation of sleep opportunity, usually in the laboratory. The effect of sleep deficiency on emotional functioning and its role in the development and/or maintenance of psychopathology might vary depending upon each of these dimensions.
For instance, the effect of acute sleep deficiency on emotional functioning might be a proximal factor contributing to the etiology of emotional disorders. In contrast, chronic sleep deficiency might be a maintaining factor for emotional disorders largely independent of their etiology (i.e., regardless of when the sleep deficiency emerged relative to the onset of the psychopathology). There is no reason to assume that these effects are dependent on one another. For a subset of individuals, sleep deficiency might play an etiological role, but not a maintaining role. For another subset of individuals, sleep deficiency might not be involved in the etiology of their psychopathology, but it might have become a strong maintaining factor. Although the extant literature is still relatively new, we hope that increasing conceptual and definitional clarity in the literature may help further elucidate the precise nature of such complex, dynamic relationships between sleep and emotional experiences and psychopathology.

**EMOTIONS AND EMOTION REGULATION**

Emotions are functional and adaptive phenomena that help organize and motivate behavior in the service of an organism’s goals. Emotions can interrupt ongoing cognitive or behavioral processes, direct attention to stimuli directly relevant to goal pursuit, and trigger action tendencies supporting the individual’s goals (Frijda, 2007; Gross, 2014; Levenson, 1999). We consider emotions to be emergent phenomena, arising from a dynamic interaction of input across multiple modes of experience, including cognition, behavior, and physiological sensations (Barrett, 2013; Barrett, Mesquita, Ochsner, & Gross, 2007).

Conceptually, we consider emotion regulation as the attempted modification of any aspect of an emotional experience that can occur at various levels of conscious awareness and utilize varying degrees of effortful control. Broadly speaking, the act of emotion regulation may be aimed at amplifying, attenuating, maintaining, or even preventing emotional responding and can occur in the context of motivated behavior intended to promote or prevent desired or undesired future states, respectively. Gross (1998, 2014) provides a useful conceptual framework for understanding the dynamic process of emotional experience and its regulation. His model takes the emotion-generative process as its base, highlighting the dynamic nature of the process and delineating how emotion regulation efforts can be initiated at any point during the process of emotion generation. The emotion-generative process, which he terms the modal model of emotion, begins with some psychologically relevant situation that can be external (e.g., encountering a bear in the woods) or internal (e.g., a fleeting thought about a past or potential future failure). Attention is then brought to bear on specific aspects of the situation, and appraisals of the situation
are generated based on their presumed relevance and impact upon an individual’s currently salient goals. The emotional response generated by this unfolding process then helps to coordinate responses across multiple systems (i.e., behavioral, cognitive, physiological) in service of an individual’s current motivationally salient goals. From this perspective, emotion regulation can occur at any point during the emotion generative process. For instance, emotion regulation can occur prior to encountering a given situation (e.g., turning down an invitation to a party to avoid social interaction), by altering the situation in some way to reduce its emotional impact (e.g., avoiding making eye contact when someone else initiates a conversation), by focusing or redirecting attention to alter a situation’s emotional impact (e.g., focusing on what one will say next in order to avoid saying something “stupid”), by generating alternative appraisals of the situation to alter its emotional impact (e.g., “I don’t care what Sally thinks about me anyway”), or altering one’s response to the situation itself (e.g., resisting the urge to end the conversation and escape the situation). Any such attempts to modify the emotional response will alter the psychological situation as the dynamic emotion-generative process continues to unfold over time. Although this model has not yet been directly evaluated in the sleep and emotion literature, we believe that it provides a useful heuristic for evaluating the extant research, and we return to the modal model later in the chapter.

SLEEP AND ITS IMPACT ON AFFECT AND AFFECT REGULATION

A number of studies have utilized sleep restriction (reduced amount of total sleep) or sleep deprivation (no sleep) paradigms to evaluate the impact of sleep duration on affective functioning in healthy samples. As such, these studies evaluate the effect of acute, objective sleep deficiency attributable to external sources. The extent to which findings from these studies might generalize to other types of sleep deficiency or individuals with psychopathology remains unclear. However, a number of studies suggest that acute, objective, external sleep deficiency increases negative emotions and emotional reactivity among healthy participants.

Using a within-subject design, Larson, Durocher, Yang, DellaValla, and Carter (2012) found that one night of sleep deprivation led to increased subjective reports of state anxiety and increased cardiovascular reactivity to a mild stressor compared to a night of normal sleep, although increases in subjective anxiety were not correlated with increases in cardiac reactivity. Increases in stress reactivity following sleep curtailment may depend on the intensity of the stressor. Minkel et al. (2012) randomized healthy adults to receive either 48 hours of sleep deprivation or 9 hours of
sleep opportunity while being continuously monitored in the sleep laboratory. The authors then examined each participant’s subjective affective response to high- and low-stress experimental tasks using the Profile of Mood States. Compared to those provided with ample sleep opportunity, participants who were sleep deprived showed large increases in anger and anxiety ($d = 0.91$, $d = 0.81$, both $p’s < 0.01$) and moderate increases stress and depression ($d = 0.61$, $d = 0.56$, $p < 0.05$, $p = 0.07$) when exposed to a low-stress but not to a high-stress experimental task. This finding suggests that insufficient sleep might lower the threshold for emotional reactivity by impairing the emotional appraisal process, potentially sensitizing individuals to the effects of events that may have otherwise been discounted as minor.

Further support for this hypothesis comes from the literature on post-traumatic stress disorder (PTSD). Bryant, Creamer, O’Donnell, Silove, and McFarlane (2010) assessed individuals during a hospital admission immediately following a traumatic event and found that self-reported sleep deficiency occurring during the 2 weeks prior to the traumatic event was associated with a threefold increase in new incident cases of psychiatric disorders after the trauma. Thus, it is possible that the relationship between poor sleep and emotional reactivity could follow a curvilinear trend, such that sleep deficiency increases emotional reactivity to low- and very high-intensity stressors, and reactivity to more moderate stressors remains comparable.

A seminal study by Zohar, Tzischinsky, Epstein, and Lavie (2005) examined the effects of externally imposed sleep loss on emotional reactivity to goal-enhancing and goal-disruptive events among medical residents. Reduced total sleep duration was associated with elevated negative affect in response to goal-disruptive events, but not in the absence of such events. That is, basal negative affect did not vary depending upon sleep the previous night. However, the opposite pattern was observed for positive affect. Reduced total sleep duration was associated with elevated basal positive affect (i.e., in the absence of a goal-enhancing event), but the affective benefit of goal-enhancing events was attenuated following nights with less sleep. An effect of reduced sleep duration (acute, objective, extrinsic sleep deficiency) on negative affect was only observed when a stressor with motivational significance was encountered, whereas reduced sleep duration directly impacted positive affect. One intriguing implication of this finding is that reduced sleep duration exerts its influence on affect, at least partially, through motivational mechanisms, potentially by increasing sensitivity to punishment or negative consequences. Given the intimate link between motivation and emotions (Frijda, 2007), one intriguing implication of this finding is that poor sleep might exert an indirect effect on psychopathology via its impact on motivation. It is possible that poor sleep impairs motivation and ultimately functioning
by increasing the motivational salience of goal-disruptive events and reducing the motivational salience of goal-enhancing events. Unfortunately, relatively less literature has examined the motivational consequences of sleep disruption.

The three studies described so far suggest that insufficient sleep might exert relatively larger effects on emotion-regulatory and self-regulatory processes than on state or trait levels of affect. There is some additional evidence supporting an effect of sleep deficiency on emotion regulation. Yoo, Gujar, Hu, Jolesz, and Walker (2007) used functional magnetic resonance imaging (fMRI) to examine emotional responses to negative visual stimuli under conditions of sleep deprivation (one full night) compared to normal sleep. Consistent with previous findings that insufficient sleep leads to increased emotional reactivity, they found increased amygdala activation following exposure to negatively valenced pictures among individuals in the sleep deprivation group compared to a normal sleep control condition. They also found evidence for reduced downregulation of negative emotional reactions. Compared to the normal sleep controls, individuals in the sleep-deprived group evidenced reduced functional connectivity between the medial prefrontal cortex and the amygdala. This suggests the intriguing possibility that not only does insufficient sleep lead to increased emotional reactivity, thus increasing the chances that relatively minor stressors will lead to maladaptive emotional responses, but it is also associated with failure to downregulate the negative emotional experience, thus possibly exposing the individual to prolonged amygdala activation.

Baum et al. (2014) utilized an experimental design to examine the effect of sleep on affect and affect regulation among healthy adolescents (aged 14 to 17 years). They manipulated sleep across 2 weeks, following a baseline week, using a counterbalanced, crossover design. Participants were randomized to a sleep restriction (6.5 hours in bed a night for five consecutive nights) or a sleep extension (10 hours in bed a night for five consecutive nights) condition and then crossed over to the other condition the following week. At the end of each week, adolescents and their parents rated the adolescent’s affect and affect regulation difficulties over the previous week. Adolescent’s subjective ratings of anger and anxiety were significantly higher, and ratings of positive affect were significantly lower following sleep restriction, compared to sleep extension but ratings of depressed mood did not differ between the two conditions. Importantly, both adolescent and parent ratings of emotion regulation difficulties were significantly higher following sleep restriction compared to sleep extension. The study by Baum and colleagues is among the first to directly evaluate the effects of both restricting and extending sleep on emotion regulation. Moreover, the findings suggest that chronic partial sleep deprivation at a level naturally experienced on school and work days is detrimental to emotional experiences that could be part of the pathway linking stress to
the development of psychopathology. More broadly, findings such as this suggest multiple pathways for sleep deficiency to impact emotions and possibly psychopathology.

Insufficient sleep might also impact emotions and psychopathology via synergistic effects with other vulnerability factors. A recent imaging study found evidence that insufficient sleep might amplify the impact of a trait vulnerability factor (i.e., trait anxiety) on emotional responding (Goldstein et al., 2013). The study examined anticipatory responding to negative and neutral stimuli under conditions of certainty or uncertainty following a night of sleep deprivation or normal sleep. Participants were provided with a cue indicating whether the stimuli would be negative or neutral, or the cue presented was ambiguous so that it did not indicate the valence of the stimuli that was to follow. Consistent with the findings from the above-described study conducted by Yoo et al. (2007), sleep deprivation increased emotional reactivity (as indexed by the magnitude of bilateral amygdala response) independent of cue type. Furthermore, following sleep deprivation, the right anterior insula, which under normal circumstances evidences increased activation to uncertainty or ambiguous cues, showed elevated responding under the two certainty conditions (negative and neutral cues) but not following ambiguous cues. In support of the idea that sleep deprivation synergistically interacts with other vulnerability factors, evidence of increased activation in the right anterior insula in response to uncertainty or ambiguous cues was the strongest among individuals high in trait anxiety.

Collectively, these findings suggest multiple pathways by which insufficient sleep might impact affective functioning. First, acute, objective, external sleep disruption can directly increase negative affect and reduce positive affect. Second, acute, objective, external sleep disruption impairs emotion regulation, potentially reducing resources for coping with increased emotional reactivity. Third, acute, objective, external sleep disruption might reduce the individual’s ability to contextualize an emotional response, resulting in further amplification of emotional reactivity independent of current cue or context. It is noteworthy that a disconnect between the magnitude of emotional response and the current context represents a key clinical feature of a wide range of psychopathology, including anxiety disorders, depressive disorders, and insomnia.

**AFFECT AND ITS IMPACT ON SLEEP**

Compared to the extensive literature evaluating the effect of sleep on affective functioning, relatively fewer studies have evaluated the effect of affective functioning on sleep parameters. In general, research examining the relationship between sleep and emotion has conceptualized emotion
as varying along a valence and an arousal dimension (Watson & Tellegen, 1985). The available literature does strongly indicate that arousal has an adverse effect on a range of sleep parameters, although operational definitions and uses of the term arousal vary substantially in this body of literature. As discussed earlier, emotions are multifaceted constructions, comprised of thoughts, physical sensations, and action tendencies. Similarly, arousal can refer to increased physiological and/or cognitive activity, both of which we consider to be aspects of emotional arousal.

Stress can be thought of as generating a range of emotions with negative valence and high arousal. Etiological theories have long acknowledged the potent role of stress in the pathogenesis of insomnia disorder (Spielman, Caruso, & Glovinsky, 1987), and the relationship between stress and poor sleep is well documented in the literature. Stress leads to increases in sleep latency and number and duration of awakenings, decrease in sleep efficiency, and alteration in sleep architecture, such as decreased percent time in rapid eye movement (REM) sleep and slow-wave sleep (SWS; Kim & Dimsdale, 2007). This literature suggests that sleep fragmentation (increased awakenings) is the most common consequence of stress (broadly defined to include both acute and chronic stress), whereas sleep latency is particularly responsive to acute stressors.

Additional support comes from studies that have used caffeine as an analog for examining the effects of physiological arousal on sleep. Caffeine offers a nice paradigm for examining the effects of physiological arousal, separate from valence. Using a within-subjects design, Bonnet and Arand (1992) examined the effects of thrice daily caffeine administrations on subjective (self-report) and objective (EEG) sleep parameters. For both self-report and EEG assessments, they found that acute caffeine ingestion decreased total sleep time, SWS (EEG only), sleep quality (self-report only), and sleep efficiency, while it increased sleep latency and nocturnal awakenings, supporting the notion that arousal adversely impacts sleep. Physiological arousal did not show any impact on latency to REM sleep or percentage of total sleep spent in REM, suggesting differential effects of arousal and valence on sleep architecture. However, valence is typically not directly assessed or accounted for in studies utilizing caffeine as a proxy for physiological arousal, leaving open the question of whether valence plays a role in sleep disruption above and beyond arousal.

Unfortunately, relatively fewer studies have examined the effects of valence on sleep parameters. A notable exception is a recent study that used polysomnography to examine the effects of a negative-emotion induction on sleep parameters (Vandekerckhove et al., 2011). Healthy controls were randomized to receive either neutral or failure feedback on a task presented as a test of general intelligence. The negative-emotion induction significantly reduced sleep efficiency, total sleep time, and percentage
REM sleep, while significantly increasing number of awakenings, total time awake, number of awakenings from REM, and latency to SWS.

Tang and Harvey (2004) conducted a pair of experiments to further explore the relationship of anxious arousal on sleep assessed via subjective reports and actigraphy. This study is noteworthy because they attempted to separately manipulate anxiety and arousal in order to examine how valence and arousal might uniquely impact sleep. In the first experiment, good sleepers were randomized to an anxious arousal group (told they would have to give a speech in front of an audience upon waking from a nap), a nonanxious cognitive arousal group (told they would have to write an essay upon waking from a nap), or a no manipulation group (just asked to take a nap). Importantly, manipulation checks supported the dissociation of anxiety and arousal across the three groups. Participants were then provided with a 60-minute nap opportunity, and their sleep was assessed with actigraphy and retrospectively by subjective report following the nap opportunity. Importantly, consistent with the hypothesized manipulations, the anxious arousal group was the only group to evidence a significant increase in anxiety, while cognitive arousal did not differ between the two arousal groups prior to the sleep opportunity. The anxious arousal group reported longer time to fall asleep and shorter total nap times than the no manipulation group (although interpretation of the total nap time finding is limited because total nap time was artificially restricted by the 60-minute sleep opportunity provided, and it is a direct function of time to fall asleep). There were no significant differences between the nonanxious cognitive arousal and control groups on sleep latency or total nap time, suggesting that valence exerts an effect on sleep above and beyond arousal, at least for cognitive arousal. In a second experiment, Tang and Harvey randomized good sleepers to an anxious arousal group (the same speech threat paradigm), a nonanxious physiological arousal group (caffeine pill), or a placebo control group. Both the anxious and physiological arousal groups reported taking longer to fall asleep and having shorter total nap times than the placebo control group; however, only the anxious arousal group reported poorer sleep quality than the control group.

Taken together, these findings suggest that valence and arousal might both contribute to poor sleep. Valence might be more likely to affect appraisals of the previous night’s sleep and lead to subjective complaints of poor sleep, whereas arousal might be more likely to impair sleep latency and duration. Furthermore, valence and arousal appear to differentially affect sleep architecture, with valence exerting a relatively larger influence on REM sleep (Vandekerckhove et al., 2011) and arousal exerting its influence by reducing SWS (Bonnet & Arand, 1992). However, no study has directly examined the potential differential effects of valence and arousal on sleep architecture using polysomnography. A study utilizing
polysomnography to extend the findings from Tang and Harvey (2004) would provide direct examination of this hypothesis.

AFFECT REGULATION AND ITS IMPACT ON SLEEP

Affective influences on sleep are not restricted to the individual’s current affective state. Consistent with the modal model of emotions discussed earlier, emotions are generated and unfold over time. As they unfold, they are susceptible to implicit and explicit attempts to modify their expression and subjective experience (Gross, 2014). Such attempts to modify the experience and expression of emotions, which we consider to be emotion regulation, are also likely to affect sleep parameters. Certain emotion regulation strategies have been found to have more adverse consequences than others. For instance, attempts to suppress emotional responses or avoid the subjective experience of particular emotions altogether have been shown to have a range of maladaptive sleep consequences, whereas strategies promoting nonjudgmental acceptance of current emotional states seem to promote more positive outcomes. These are reviewed below.

A number of studies have evaluated the consequences of overreliance on maladaptive strategies such as worry and rumination to manage emotions. Thomsen and colleagues examined the relationship between the self-reported habitual tendency to engage in rumination following stressful events and self-reported sleep quality in a sample of undergraduates (Thomsen, Mehsen, Christensen, & Zachariae, 2003). Rumination was significantly correlated with self-reported sleep quality, and this relationship remained significant after adjusting for current negative mood, suggesting that emotion regulation strategies might impact sleep above and beyond current mood.

Another study extended this finding by inducing rumination following a stressful event (Guastella & Moulds, 2007). Following a midterm exam, undergraduates scoring high and low on a trait measure of rumination were randomly assigned to either a rumination or distraction condition. Prior to going to bed, individuals in the rumination condition were instructed to reflect on the test they took earlier that day and think about how they felt during the exam, how they performed, and the potential consequences of their performance, whereas individuals in the distraction condition were provided with neutral statements to think about. The next morning, all participants completed questionnaires assessing sleep quality. In the rumination condition, high-trait ruminators reported significantly worse sleep quality than low-trait ruminators. There was no significant difference in subjective sleep quality ratings for high- and low-trait ruminators in the distraction condition, suggesting that individual
differences in how individuals respond to and attempt to manage negative emotions might contribute directly to poor sleep.

A recent study by Vandekerckhove et al. (2012) used polysomnography to examine the effect of two different emotion regulation strategies on various sleep parameters among healthy controls. Following an adaptation and baseline night in the lab, participants completed a series of cognitive tasks that were presented as a test of general intelligence, following which they received feedback that they had performed poorly (well below average) on the tasks. After being provided with an appropriate rationale, and just prior to bed, participants were randomized to write about the task and their performance on the task either using an “experiential” strategy (where they were asked to focus on emotions brought up by the task and their performance) or an “analytic” strategy (where they were asked to focus objectively and analyze their performance and its potential causes and consequences). Participants in the experiential strategy group took longer to fall asleep, but experienced significantly fewer awakenings, longer total sleep time, and higher sleep efficiency compared to the analytic strategy group. This suggests that processing an emotion prior to going to sleep might make it more difficult to fall asleep, but might help downregulate emotions enough so that they are less likely to disrupt sleep during the night, leading to increased sleep duration, reduced sleep fragmentation, and improved sleep quality.

There are a number of ways in which affective functioning can impact sleep. Cognitive and physiological arousal can lead to poor sleep by increasing sleep latency and sleep fragmentation, as well as decreasing sleep quality and total sleep time. Negatively valenced emotions have also been found to increase sleep fragmentation and reduce sleep quality, sleep efficiency, and total sleep time. Negative valence and high arousal appear to have differential effects on sleep architecture, with arousal reducing SWS and negative valence adversely affecting REM sleep. In addition to direct effects of valence and arousal on sleep, attempts to modify the expression or subjective experience of emotions also appear to affect sleep. It is possible that strategies aimed at reducing or “getting rid of” emotional responses appear to be associated with reduced sleep quality, more frequent awakenings, shorter total sleep time, and lower sleep efficiency compared to strategies promoting acknowledgement and an acceptance of an individual’s current emotional state.

**SLEEP AND AFFECT: BIDIRECTIONAL RELATIONS**

The studies reviewed so far have primarily investigated unidirectional relationships, with sleep predicting affect or vice-a-versa. Comparatively fewer studies have directly examined the hypothesized bidirectional or
reciprocal relationship between sleep and affect. Longitudinal evaluation of patterns of sleep and affective experience within individuals across time will help to shed light on the dynamic relationship between these two constructs. A number of the studies that have been conducted have relied upon mood ratings taken immediately upon waking and prior to bedtime; however, such an assessment schedule is likely to offer inflated estimates of the relationship between mood and sleep. Experience sampling methodology (ESM) is an alternative technology that might offer a more reliable estimate of the sleep-mood relationship than once-a-day mood measurements. ESM takes random samples of affective experience throughout the day. Aggregations of these repeated measurements will offer a more reliable and potentially more representative picture of daytime mood.

A recent study by de Wild-Hartmann et al. (2013) used ESM and a sleep diary to examine the relationship between nighttime sleep and daytime mood among 553 woman without current psychopathology. This study is different from previous studies described because the authors examined the covariation between naturally occurring variation in night-to-night sleep (as opposed to strong manipulations reducing sleep opportunity) and variations in day-to-day affect. Multiple indicators of poor subjective sleep (lower sleep quality, shorter total sleep time, longer sleep latency, increased sleep fragmentation) were prospectively associated with reduced levels of next day positive affect. In contrast, only lower sleep quality and longer sleep latency were prospectively associated with higher next-day negative affect. Given the potential buffering effects that positive affect can have upon negative affect (Wichers et al., 2007), poor sleep’s deleterious effect on positive affect might represent another way in which poor sleep might indirectly influence negative mood and ultimately increase risk for psychopathology. The authors also evaluated prospective relationships of daily positive and negative affect on sleep parameters but found only weak associations. The only significant finding was that lower daily positive affect was prospectively associated with reduced sleep quality. Daily negative affect did not significantly predict any of the sleep parameters in this sample of healthy controls.

The de Wild-Hartmann et al. (2013) study found strong support for the hypothesis that poor sleep predicts reduced levels of positive affect and more modest support for the hypothesis that poor sleep predicts increased negative affect. It is noteworthy that, when the authors accounted for daily relationships between sleep and positive affect, negative affect did not predict any of the sleep parameters examined. The only significant prospective relationship between affect and sleep was the finding that reduced positive affect predicted poorer sleep quality. It is possible that the relationship between affect and sleep is strongest for emotions with high negative valence and high arousal, such as following stressful events. It is
also possible that, in the absence of significant stressors, affect is much more likely to be impacted by than to impact sleep. This suggests that, in addition to direct effects, affect and sleep might have a synergistic effect on functioning following a stressor.

**DISCUSSION**

Sleep and emotions are related in a complex and dynamic way. Increasing evidence suggests that sleep impacts affective functioning in multiple ways. Insufficient and poor sleep appear to have a robust inverse relationship with positive affect. However, the relationship between sleep and negative affect is less clear, with a number of studies failing to find an association. Insufficient sleep (either acute or chronic partial sleep deprivation) has been shown to have a strong direct effect on negative affect, but this effect is not observed when examining daily variations in sleep quality or duration. However, multiple indirect pathways seem to lead from deficient sleep to negative emotion. Insufficient and/or poor sleep might indirectly increase negative affect by reducing the potential buffering effect of positive affect, altering the motivational salience of goal-relevant events, increasing sensitivity to punishment or barriers to goal attainment, and increasing emotional reactivity more broadly. Insufficient sleep also appears to impair emotion regulation and the individual’s ability to interpret stressors in the context of current salient goals. Thus, a likely consequence of insufficient sleep is a reduced ability to cope with ongoing stressors and an increased likelihood of reacting negatively to relatively neutral or mildly stressful events, both of which might increase vulnerability to developing psychopathology.

An inherent difficulty in synthesizing the literature on the impact of poor or insufficient sleep on affect is the lack of coherence among definitions of affect. Indeed, there is an ongoing debate in the emotion-science literature about how to define affect. The modal model of emotions (Gross, 2014) might be particularly helpful for framing the complex relationship between sleep and affect because it highlights the process by which emotions are generated and unfold over time. According to this model, the emotion-generative process begins with the situation, broadly construed to capture the internal experience or construction of the situation, which could be based on external or internal cues. Attentional resources are then allocated to particular aspects of the situation and appraisals are assigned to help interpret and make meaning of the current situation. The appraisal results in an emotional response that could be experienced and/or expressed across multiple modes (cognition, behavior/action tendencies, and physiological sensations). The emotional response, which may or may not be subjectively and/or objectively observable, then feeds back
into and modifies the current situation, and the process continues (Gross, 2014). This process is graphically depicted in the bottom half of Figure 3.1.

Sleep can impact emotional experiences at different stages of this modal model of the emotion-generative process (see Figure 3.1). Insufficient and poor sleep can lead to reduced energy and activity levels, which can influence the types of activities individuals engage in and the kinds of situations they encounter. For instance, insomnia patients often respond to the feeling of low energy by canceling obligations or avoiding activities that are usually enjoyable in favor of “resting” and conserving energy. Activity reduction (particularly reduced social contact) and failure to fulfill roles or obligations can each contribute to negative emotions. Insufficient sleep also affects attentional processes (Lim & Dinges, 2010), including working memory, as well as simple and complex attention. Such attentional impairments might contribute to preferential processing of negative or “threat-relevant” information, although this later point has not yet been directly evaluated (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). In that way, insufficient and poor sleep could also affect the appraisal process. Insufficient sleep might also lead individuals to adopt more conservative, generalized interpretations (which would typically be more catastrophic or negative in tone than when well rested) in order to prioritize conserving energy and maintaining safety. As reviewed above, insufficient sleep is known to amplify emotional reactivity (particularly to neutral and mildly negative events), directly increasing the intensity of emotional responses, independent of sleep’s impact on earlier stages.
of the emotion-generative process. The modal model of emotions can be useful in the study of how sleep quantity and quality impacts emotional functioning. It will be important to understand the effect of sleep at different points of the emotion-generative process because these effects might be different. Increased understanding of how sleep might affect the temporal dynamics of emotional responding might help shed further light on the complex, dynamic relationship between sleep and affect.

Affective functioning also influences various sleep parameters, although relatively less literature has examined the influence of affective states on sleep. Two widely agreed upon dimensions of emotion, valence and arousal, appear to have similar effects on global sleep parameters (increased sleep latency and fragmentation, decreased sleep quality and total sleep time) but potentially unique effects on sleep architecture, with arousal relating more strongly with SWS and valence with REM sleep. This is consistent with recent conceptual models emphasizing the importance of REM sleep for emotional processing (cf., Goldstein & Walker, 2014). Emotion regulation efforts also appear to influence sleep parameters, with the habitual tendency to rely on particular maladaptive strategies producing alterations to global sleep parameters. In particular, strategies that promote a hyperfocus on or overelaboration of emotional states (Guastella & Moulds, 2007) or detached processing of emotionally salient events (Vandekerckhove et al., 2012) appear to contribute to reduced quality sleep, increased fragmentation, and shorter total sleep time.

A significant challenge in understanding how affective functioning influences sleep is disentangling the unique contributions of arousal and valence. Studies that have examined the impact of affect on sleep have largely not examined the potentially unique effects of valence and arousal (with Tang and Harvey (2004) a notable exception). Some preliminary evidence suggests that valence and arousal might differentially impact sleep architecture; however, additional studies utilizing polysomnography are necessary to examine this hypothesis. One way to begin to disentangle the unique impact of arousal and valence would be to examine manipulations of both high-arousal positive (e.g., excitement) and high-arousal negative (e.g., anxiety) emotions on sleep parameters. Such data would provide direct evidence for the hypothesis that arousal and valence have differential effects on sleep. Such data might also help clarify some of the discrepancies in the literature examining the impact of stress on sleep (Kim & Dimsdale, 2007).

A growing literature suggests that emotion regulation might also have an impact on sleep parameters. Individual differences in the habitual use of particular strategies (such as rumination and worry) appear to be associated with sleep deficiency. Importantly, this relationship appears to be present above and beyond the influence of state affect. This suggests that interventions directly targeting maladaptive emotion regulation strategies
might show improvements in sleep parameters. Indeed, some evidence supports this hypothesis. McGowan and Behar (2013) randomized participants with high levels of trait worry to 2 weeks of either a stimulus control (consisting of a daily 30-minute worry period occurring at an a priori time and place) or focused worry intervention (consisting of instructions to not avoid naturally occurring worry, because doing so might paradoxically increase worry and anxiety). Stimulus control resulted in significant reductions in worry, anxiety, general negative affect, and symptoms of insomnia.

Evidence suggests that, although processing negative emotions prior to sleep initially increases sleep latency, such efforts to downregulate negative emotions might be effective in reducing sleep fragmentation and improving sleep duration and sleep quality. If replicated, this finding could support utilizing interventions targeting emotion processing such as expressive emotional writing (Smyth, 1998) or even stimulus control for worry (McGowan & Behar, 2013) prior to a traditional “buffer zone” recommendation in sleep hygiene or stimulus control for insomnia instructions.

The empirical literature consistently supports a strong relationship between sleep deficiency and reduced positive affect and increased negative affect and difficulties with emotion regulation. Increasing evidence supports an impact of emotions and emotion regulation on sleep parameters and sleep architecture as well. Such data might have important clinical implications in treating individuals with deficient sleep or disorders characterized by emotion-processing deficits. Key areas for future research include studies examining (a) how chronic, intrinsic sleep deficiency (as is commonly seen among individuals with insomnia) impacts emotional functioning, (b) how arousal and valence might uniquely impact sleep architecture, and (c) the particular conditions under which emotion regulation influences sleep, independent of the impact of state affect.

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