CHAPTER 40

EAT, DRINK, AND BE SEDENTARY

A Review of Health Behaviors' Effects on Emotions and Affective States, and Implications for Interventions

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There is a large body of work showing that emotions and emotion regulation shape health behaviors (DeSteno, Gross, & Kubzansky, 2013; see Chapter 36, "Emotions and Health," this volume). In turn, many health behaviors have potent effects on how we feel. When feeling emotional, many people often turn to unhealthy behaviors to modulate or dampen their emotions. It is not surprising then that health behaviors are often conceptualized as a way to cope with stress. Standard coping scales include, in addition to emotional and problem-focused coping responses, the common behavioral responses to stress such as eating, drug use, drinking, watching television, or sleeping more than usual. Behavioral responses such as these are a form of passive coping or mental disengagement, reflected in our cultural idioms "drown your sorrows," "numbing out," using "food as therapy," or "sleeping it off." While the more common behavioral responses to "negative" emotions, like sadness and anxiety, tend to be "negative" health behaviors, some people lean toward exercise to feel better. Positive health behaviors such as exercising and sufficient sleep may also impact affective states, both dampening negative but also increasing positive affect and related specific emotions. Clearly, then, this relationship is bidirectional, although much less attention is on the path from health behaviors to affect.

Why should one care about the transient emotional responses to a bout of exercise, for example? It may be that within such phenomena lie the critical levers to promoting positive habits. The link between how health behaviors shape emotional responses is sorely underexamined. Emotional responses that are induced by health behaviors predict a wide range of outcomes that can then feed back to further shape decision making and other health behaviors. New initiatives to understand behavior change, such as the National Institutes of Health Science of Behavior Change (SOBC) initiative's attempt to find commonalities in mechanisms of behavioral change across types of behaviors, in order to promote more effective in-

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terventions (National Institutes of Health, 2015). Promoting adherence to health behaviors is one of the toughest issues in public health, and the role of emotional responses may provide one window into understanding individuals' choices to engage or terminate health behaviors.

Here we review the primary health behaviors that are relevant to most people as they age, regardless of health status, including positive behaviors—*exercise* and *good sleep*—and consummatory behaviors—*overeating* and *drinking alcohol*—and what is known about their effects on emotion and affective states, and stress-related processes, including physiological responses to stress. We touch upon emotion regulation when there is research directly assessing regulation (see the section "Eating"). Health behaviors can affect default or basal emotional states, anticipation of stressors, and then appraisal and evaluation. They also impact neurobiological processes that maintain or modulate affective states and discrete emotional experiences *after* a stressor has occurred. Further, the affective and emotional responses during and immediately after engaging in a health behavior may affect a person's decision to continue to engage in or maintain the behavior, although this is less studied.

This chapter lays initial groundwork for a better understanding of the behavior–affect link. In addition, we pose the question of whether health behaviors at the right dose and intensity can promote more positive emotional responses, which may predict better adherence to the healthy behavior (Figure 40.1). This is a nascent area of research, applied mainly to exercise so far.

Acute versus Chronic Effects

One obstacle to understanding health behavior's effects on emotions is that there is not a programmatic literature with a common language and conceptual paradigm. How can we best learn from the studies that have been done thus far? Here we organize studies based on the exposure (chronic/regular or acute health behavior). If an individual is regularly engaging in a health behavior, we can consider *chronic effects* of the health behavior on emotion and emotion regulation processes. We review how emotional experiences differ in people who vary on physical activity, sleeping habits, eating patterns, and alcohol consumption. To varied extents, each behavior has been studied in its chronic form and how it impacts discrete emotions and affective states, in psychiatric conditions, general emotional experiences of everyday life, and in the laboratory in response to stress induction. We also review the small but important experimental literature on the impact of acute health behaviors on emotions. *Acute effects* of a health behavior can be studied through one-time or short-term repeated manipulations of the health behavior, by using either standardized conditions, such as in the lab, or by using more naturalistic examination in daily life, with repeated ecologically valid measurements, such as those afforded by ecological momentary

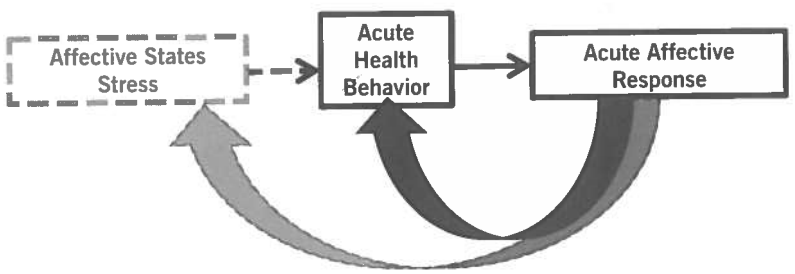

FIGURE 40.1. Health behaviors can acutely alter affective experience—emotions (valence, arousal), emotion regulation, and stress appraisal. This can affect both longer-term affective states, and we propose may affect frequency of the health behavior. Using the example of exercise, a positive emotional response to a bout of exercise can predict better adherence (Williams et al., 2008). This may extend to other health behaviors where changes in negative and positive emotional balance could be critical determinants of future adherence or abstinence from that behavior, and predict treatment success.

assessment (EMA). And while each behavior has been studied in a vacuum, we present suggestions for a more programmatic focus on health behaviors and their impact on emotions that envelops common strategies and language to uncover common, underlying neurobiological mechanisms situated within a broader conceptual model.

Physical Activity

First, it is important to be specific about definitions of activity, since they may work differently. Physical activity, exercise, and sedentary behavior are typically used interchangeably, yet incorrectly (Caspersen, Powell, & Christenson, 1985). *"Physical activity" is defined as any skeletal muscle movement that results in energy expenditure.* Physical activity can occur at home during household activities, at work, during leisure time, and sports, for example. Exercise, on the other hand, is structured, deliberate, and repetitive physical activity that has the planned outcome of improving physical fitness. Researchers also differentiate between type, duration, and intensity (light, moderate, and vigorous) of physical activity. Activity in the moderate and vigorous intensity zones is typically considered an "exercise bout" since these levels of intensity are typically only reached during structured and deliberate exercise. Recent research also differentiates between physical activity and sedentary behaviors (television watching, sitting at work, spending time at a computer), as there is growing evidence that they independently predict health and disease (Chomistek et al., 2013; Katzmarzyk, 2010).

It is apparent from the literature that depression and physical activity are intimately connected. Depressed and anxious individuals are less likely to be physically active (Camacho, Roberts, Lazarus, Kaplan, & Cohen, 1991; Goodwin, 2003; Stroehle, 2009) and remain more sedentary (Teychenne, Ball, & Salmon, 2010). Prospectively, physical inactivity appears to increase the risk of developing depression (Farmer et al., 1988; Lampinen, Heikkinen, & Ruoppila, 2000; Motl, Birnbaum, Kubik, & Dishman, 2004). Several meta-analyses and reviews suggest that exercise interventions promote reductions in depressive and anxious symptoms in healthy and clinical populations (Blumenthal et al., 2007; Brosse, Sheets, Lett, & Blumenthal, 2002; Conn, 2010a, 2010b; Rethorst, Wipfli, & Landers, 2009; Wipfli, Rethorst, & Landers, 2008). Randomized trials demonstrate that exercise interventions considerably benefit depressed individuals, with similar effects to pharmacotherapy (Barbour, Edenfield, & Blumenthal, 2007; Blumenthal et al., 2007; Brosse et al., 2002), and that depressed individuals who start an intervention and maintain regular exercise over the long term also maintain the therapeutic affective effects of exercise up to 1 year later (Babyak et al., 2000; Hoffman et al., 2011). Physical activity, thus, appears to have antidepressant effects.

Here we examine research that assesses the emotion- and affect-altering effects of physical activity by examining how physical activity and exercise can help people "feel less worse" and "feel better." Much of the work to date rests in the area of affective states, general states of positive affect or negative affect, or pleasure and displeasure. We highlight evidence from observational and experimental studies that examines differences in affective states that occur naturally and in the laboratory between physically active people and those who are less active. We also highlight the work on affective states from naturalistic and experimental studies that occur after a bout of exercise in everyday life and in response to a standard exercise bout in the laboratory. We complete the section with a focus on the potential neurobiological mediators of these effects.

Regular Physical Activity

Regular Physical Activity, Emotions, and Affect

From a broad emotions perspective, it has been of considerable interest to examine whether becoming physically active makes people feel better (i.e., boost positive affect) or makes people feel less worse (i.e., reduce negative affect). Reviews and meta-analyses abound addressing these studies. A review of the literature indicates that physical activity is more likely to be related to changes in positive affect than to negative affect. For example, the long-term benefit of an exercise intervention on older adults' quality of life is mediated by positive affect alone, not negative affect (Elavsky et al., 2005). In fact, a meta-analysis (Reed & Buck, 2009) suggests that participants who are randomized to an exercise program increase in positive affect, with a Cohen's *d* effect size of 0.57. Importantly, those who start preintervention with lower positive affect scores or who exercise more than three times a week are especially more likely to increase in their positive affect ratings. However,

it is unclear which discrete emotions are altered or whether the impact of exercise is more general.

Regular Physical Activity and Emotional and Stress Reactivity

Studies in the laboratory have, however, examined the response of discrete emotions to novel stressors in active individuals compared with those less active. Physical activity buffers individuals' emotional responses to acute and novel stressors. Active individuals are less likely to experience psychological stress or increased negative emotions after a laboratory-induced stressor than inactive individuals. For example, Rimmele and colleagues (2007, 2009) demonstrated that while all individuals experience heightened state anxiety and decreased calmness in response to stress induction in the laboratory, athletes are seemingly less emotionally responsive to stressors than sedentary, untrained individuals. Similarly, individuals with high levels of regular physical activity have smaller neuroendocrine and autonomic responses to a stressor compared with those who are sedentary (Puterman et al., 2011; Rimmele et al., 2007, 2009; Traustadóttir, Bosch, Cantu, & Matt, 2004).

Acute Physical Activity

Acute Physical Activity, Emotions, and Affect

While regular physical activity is psychologically beneficial, perhaps via alterations in positive affect, less is known about how an acute bout of exercise impacts trajectories of positive and negative affect or discrete emotions. Diary studies, including EMAs, have opened the door to understanding the relationships between acute episodes of physical activity and positive and negative affective states on a daily basis. Giacobbi, Hausenblas, and Frye (2005) examined the relationships between self-reported physical activity and affect on a daily basis in 106 college students utilizing end-of-day 24-hour recall self-reports. They demonstrated that on days that students were physically active, reported positive affect was higher whereas negative affect was lower, compared with the days these students were inactive. However, to eliminate the effects of random events that occurred, Giacobbi and colleagues (2005) covaried actual positive and negative events that the participants' experienced on each day and found only positive affect was related to reporting being active, whereas negative affect was no longer related. These findings corroborate previous research on daily activity and positive affect (Steptoe, Kimbell, & Basford, 1998), although there are also null findings with similar methods (Ready, Marquez, & Akerstedt, 2009).

To better assess acute effects of activity naturalistically, it is necessary to examine repeated assessments of mood in order to get at changes before and after activity. Using such an exemplary study method as EMAs, Wichers and colleagues (2012) examined the lagged relationships between physical activity and affect in over 500 female twins by measuring self-reported physical activity and negative and positive affect at 10 time points throughout the day for 5 consecutive days. Fluctuations in negative affect within individuals were not related to engaging in physical activity on a daily basis nor did physical activity decrease negative affect after an increase in activity. On the other hand, positive affect increased immediately after a bout of activity for as long as 180 minutes in women with no history of depression, and for 90 minutes in women with a history of depression.

The question of whether a bout of exercise makes people "feel better" (i.e., increase in positive affect) or "feel less worse" (i.e., decrease in negative affect) has received wide attention beyond daily experiences, and has a longer history in laboratory experiments. Wichers and colleagues' (2012) study suggests that activity makes people feel better, but experimental studies suggest that this might be the case because people in their daily lives self-select types of activities at intensity and duration levels that *make* them feel better. In a series of studies that span the past decade, Ekkekakis, Hall, and Petruzzello (2008), as well as others, have demonstrated that while most people feel better *after* exercising (typically when affect is measured in EMA studies), not everyone feels better *during* exercise—in fact, many people feel worse.

One might wonder why exercise seems pleasant for some people and torturous for others. One's fitness level and the intensity of a bout of activity seem to play crucial roles in this equation (Reed & Ones, 2006). One's physiological fitness states, including ventilatory and lactate thresholds, are key to understanding when physical activity switches from a positively to negatively valenced effect on emotional state. The ventilatory threshold is the point at which breathing becomes disproportionately high in comparison to how much oxygen is actually being consumed. The lactate threshold is the point at which lactic acid starts to accumu-

late in the blood, forcing the organism to switch to anaerobic respiration (Caiozzo et al., 1982). While these thresholds are reached at slightly different intensities, based on nutrition and overall fitness of the individual, a wide range of studies suggest that below these thresholds, physical activity is widely experienced as pleasurable, with some interindividual variability. Above these thresholds, exercising switches from an experience of positive affect to negative affect (Ekkekakis et al., 2008; Ekkekakis & Petruzzello, 1999; Hall, Ekkekakis, & Petruzzello, 2002; Welch, Hulley, & Beauchamp, 2010). Furthermore, in those who remain subthreshold, positive affect seems to peak at 30 minutes and starts declining soon after (Woo, Kim, Kim, Petruzzello, & Hatfield, 2009). Last, during recovery from exercise, there is another burst of positive affect (Woo et al., 2009).

This trajectory—positive affect subthreshold, negative affect above threshold, and positive affect after a bout of exercise—may differ for individuals based on factors such as how active individuals are in their daily lives. For example, some evidence suggests that *inactive* women tip toward negative affect even before the ventilatory threshold is reached, and bounce back toward a positively valenced experience after around 10 minutes after a bout of exercise (Welch, Hulley, Ferguson, & Beauchamp, 2007). In contrast, active women typically bounce back immediately after a bout of exercise that surpasses the ventilator threshold.

Temperament also seems to influence the experience of affect during exercise. Individuals who tend toward reward seeking or high arousal states experience greater positive affect during exercise than those who are more driven toward low arousal states or avoiding pain (Legrand, Bertucci, & Thatcher, 2009; Schneider & Graham, 2009). Additionally, those who feel higher levels of mastery during activity (Hu, Motl, McAuley, & Konopack, 2007; Jerome et al., 2002) and autonomy over the choice of intensity (Lind, Ekkekakis, & Vazou, 2008; Parfitt, Blisset, Rose, & Eston, 2012; Parfitt & Hughes, 2009) seem to receive greater boosts in positive affect during a bout of exercise. For example, overweight and obese women *forced* to exercise 10% greater than their preferred or self-selected intensity level are more likely to experience negative affect during a 20-minute bout of exercise than normal-weight women (Ekkekakis & Lind, 2006).

These studies may be of critical importance in understanding adherence to—versus dropping out—from exercise programs. In one study, greater positive affective response to one acute bout of moderate intensity exercise predicted maintenance of a fitness regimen up to 1 year after assessment (Williams et al., 2008). As a result of these findings, clinical researchers are increasingly recommending that individuals without current fitness routines base their new activities on what feels good to them, and less on their maximum heart rate target zones. Target zones might surpass the thresholds in unfit individuals, in turn promoting negative affect sooner into a bout of exercise (Welch et al., 2010) and thus, an increased likelihood to drop out of an exercise routine (Ekkekakis, 2009b). A recent study increased expectations of a positive mood after exercise in one group of volunteers. Both groups exercised for 10 minutes, and the group with the positive expectation induction indeed had significantly higher positive affect and greater behavioral intentions to exercise again (Helfer, Elhai, & Geers, 2015). Manipulating positive expectations, in order to promote more positive affect, may be yet one more important lever that can be used in promoting better adherence.

Acute Physical Activity and Affective and Stress Reactivity

The affect-moderating effects of activity in response to stressors are not limited to only those who are active versus inactive but extend to a single bout of exercise as well (Anshel, 1996; Mata, Hogan, Joormann, Waugh, & Gotlib, 2013; Smith, 2013). In a study of nondepressed and previously depressed individuals, Mata and colleagues (2013) induced a sad mood twice in a 20-minute period and monitored mood states continuously. Prior to the sad mood induction, however, half of the participants exercised for 15 minutes at self-selected intensities while the other half rested comfortably. All increased in negative affect after the first mood induction; however, only those with histories of depression who did not exercise for 15 minutes prior to the mood-induction task continued to increase in their negative affect after the second induction, whereas all the other participants did not. Thus, those with tendencies toward depression may be sensitive to the positive effect of exercise on negative emotional processing.

Neural Mechanisms of Physical Activity and Affective Experience

Research from animal and human studies has identified neurobiological underpinnings that may

partially mediate the affective responses experienced during a bout of exercise. One of a rodent's favorite pastimes is to run on a wheel. Fortunately for the rodent, wheel running appears to be mood enhancing and stress buffering. Rodents provided a running wheel have amplified expression of several neurotransmitters (e.g., serotonin, brain-derived neurotrophic factor [BDNF], galanin) that increase cognitive flexibility and learning and reduce arousal of the endocrine and autonomic nervous activity, systems that play a role in the experience of affect and stress (Dishman et al., 2006; Matta Mello Portugal et al., 2013; Sciolino, Dishman, & Holmes, 2012; Sciolino & Holmes, 2012). In a recent mouse study, immersing mice in a cold-water swim stress task—much more of a stressor than an exercise opportunity—provoked a large stress response and neurogenesis in the region of the hippocampus that plays a role in heightened emotion processing (ventral hippocampus). However, mice that were provided free access to running wheels for several weeks prior to immersion in the cold-water swim stress task calmed much sooner. These exercise related effects on reducing behavioral signs of anxiety were accounted for by increased gamma-aminobutyric acid (GABA)-releasing neurons in the ventral hippocampus that inhibited the excitatory neurons (Schoenfeld, Rada, Pieruzzini, Hsueh, & Gould, 2013).

In humans, such detailed examinations of the neurobiological effects of chronically exercising are either not explored as of yet or currently impossible to test. However, physical activity in humans changes brain activity in ways that may affect stress appraisal and emotion regulation processes, and possibly underlie antidepressant responses. Recent advances in cerebral hemodynamics support previous studies of electroencephalogram (EEG) research demonstrating differential activation of the frontal and prefrontal areas of the brain at increasing levels of exercise intensity. Many of these changes correlate with the experience of pleasure or displeasure during exercise, and the transition from the former to the latter at the ventilatory threshold (Ekkekakis, 2009a; Fumoto et al., 2010; Hall, Ekkekakis, & Petruzzello, 2010; Kop et al., 2011; S. Schneider et al., 2009; Tempest, Eston, & Parfitt, 2014; Woo, Kim, Kim, Petruzzello, & Hatfield, 2010). In individuals not pushed to their ventilatory threshold, however, it is believed that the required neural activity for movement and balance during exercise, coupled with the finite metabolic resources of the brain that limit neural activity in nonmovement essential areas, allows for the decreased stimulation of the prefrontal and limbic regions. These regions are typically overly active in anxiety and depressive disorders, and, in part, these alterations in the brain that occur during exercise may partly underlie some of the benefits of physical activity to those suffering mood disorders (Dietrich & Audiffren, 2011).

Summary and Future Directions

Regular physical activity is associated with less major depression, anxiety disorders, and greater positive affect in general. Acute exercise is associated with a trajectory of affective responses—more positive emotional response in the low and moderate intensity zones—and if it reaches the ventilator threshold and an anaerobic state, a short-lived negative affective tone until the exercise bout ends.

In recovery from exercise, EMA studies suggest that a positive affective state is induced that can last for up to 3 hours. If this type of quantification of standard affective effects was performed reliably across studies and types of health behaviors, we could better examine how affective responses predict future health behaviors and modifiers of these effects. As shown in Figure 40.1, the dynamics of the affective response may be critical for understanding individual differences in new exercisers—or any new health behavior for that matter—who take to their new lifestyle and those who drop out quickly. There are hints that a positive affective response to a bout of exercise predicts long-term adherence to a program. Thus, making exercise pleasurable by, in part, keeping people in a subthreshold zone is key to long-term maintenance in new exercisers. Past depression, temperament, and fitness level are important moderators of affective response.

It is clear from previous research that being physically active has affect-altering effects, and that perhaps, the immediate effects of a single bout of exercise build over time to secure a general better mood for individuals. Much less is known about discrete emotional experiences, whether experienced naturalistically, in the laboratory after a bout of exercise, or in response to a laboratory stressor. Whether the affect-altering effects are universal remains unknown. For example, the physical activity-depressive symptomatology association was apparent only in adolescent girls with the met-allele BDNF polymorphism (not val/val polymorphism) in a prior study (Mata, Thompson, & Gotlib, 2010). Future research directed toward

the genetic, social, or life history underpinnings of who benefits affectively from interventions is thus of particular interest. To date, few exercise-based intervention studies combine daily process methods and laboratory evaluations of affect and emotion pre- and postintervention, so it is unclear exactly how affect experience is altered in naturalistic and experimental conditions.

Sleep

The link between sleep and emotion is intuitive. One only needs to pull an all-nighter, either in the service of extending fun or by necessity, and then reflect on how one's mood was disrupted the next day to appreciate that sleep loss can dramatically affect the emotional system, including one's expression and regulation of emotions. Taken to its extreme, chronic sleep disturbances, such as prolonged difficulties in falling or staying asleep, can constitute psychiatric conditions in their own right (e.g., primary insomnia) and co-occur with many other psychiatric conditions (e.g., mood and anxiety disorders; Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010). This co-occurrence is so common that sleep disturbance has been proposed as a potential transdiagnostic factor in psychiatric illness (Harvey, Murray, Chandler, & Soehner, 2011). The relation between sleep and emotion is complex and bidirectional, and accruing evidence suggests that sleep and emotion are in "obligate symbiosis" (Walker & Harvey, 2010). Here we focus primarily on one direction of this dynamic system, namely, on how the quantity and quality of sleep serves to modulate emotional health, by studying affect in people with chronic sleep conditions, and by examining how acute sleep deprivation influences affective experience.

Chronic Sleep Disturbances

Chronic sleep disturbance is a significant predictor of onset and recurrence of several psychiatric conditions, especially major depressive disorder (MDD; Baglioni et al., 2010, 2011; Tsuno, Besset, & Ritchie, 2005). Further, sleep complaints often remain after the MDD remits (Iovieno, van Nieuwenhuizen, Clain, Baer, & Nierenberg, 2011). Accordingly, sleep interventions have emerged as promising treatment strategies in mitigating affective disorders. Chronic sleep problems also negatively affect emotional functioning in nonclinical samples. For instance, rotating shift workers, who by the nature of their jobs have disrupted sleep, show elevated rates of emotional exhaustion as well as mood disorders compared with nonrotating night-shift workers and day-shift workers (Drake, Roehrs, Richardson, Walsh, & Roth, 2004; Jamal, 2004). Similarly, police officers who screened positive for a sleep disorder, including obstructive sleep apnea and insomnia, were at significantly greater risk of making cognitive errors on the job, falling asleep while driving, and missing work. However, what was particularly alarming was that these officers were also significantly more likely than non-sleep-disordered officers to display uncontrolled anger toward a citizen or suspect and receive citizen complaints (Rajaratnam et al., 2011). The impact of sleep on emotion generation and regulation is complex. To get a better handle on the link between sleep and emotion, researchers have turned to more experimental paradigms.

Acute Sleep Disturbances

Acute Sleep Disturbance, Emotions, and Affect

The effects of sleep disturbance on emotions can be large, and in some cases more robust than sleep-induced decrements in cognition (Pilcher & Huffcutt, 1996). Experimental paradigms employing sleep deprivation serve as useful tools for elucidating these effects (Hall, Levenson, & Hasler, 2012; Kahn, Sheppes, & Sadeh, 2013; Vandekerckhove & Cluydts, 2010). In general, findings from this literature demonstrate that experimental acute sleep deprivation is associated with increases in a bevy of negative emotions. For instance, participants subjected to 1 week of partial sleep deprivation (5 hours of sleep per night) reported increases in daily emotional difficulties and negative mood states (Dinges et al., 1997). Elevations in reports of anxiety and depression have been observed in several other studies employing sleep deprivation in otherwise healthy adults (Babson, Trainor, Feldner, & Blumenthal, 2010; Franzen, Siegle, & Buysse, 2008; Sagaspe et al., 2006). These sleep-related impairments in emotion are thought to be particularly salient to adolescents, who as a group are experiencing developmental brain changes that affect sleep and emotional processes (Dahl, 2004). For example, 1 week of partial sleep restriction (6.5 hours in bed per night for five nights) produced greater reports of anxiety, anger/hostility, fatigue, and confusion in adolescents, and increased parental reports of oppositional behavior by their adolescent child as compared with the

same young adults (ages 14 to 17) under a week of "healthy sleep duration" (10 hours in bed per night for five nights; Baum et al., 2014).

There is also growing evidence that positive affect is tied to sleep (Ong et al., 2013; Steptoe, O'Donnell, Marmot, & Wardle, 2008), a relationship that is often independent of negative affect. In a recent naturalistic study of 100 midlife adults who underwent eight consecutive end-of-day telephone interviews and 7 consecutive days of actigraphy to measure sleep, greater trait levels of positive affect were associated with feeling more rested and overall better-quality sleep. However, greater positive affect reactivity in response to daily events was related to overall poorer self-reported sleep efficiency (Ong et al., 2013). While this study cannot determine the directionality of the relationship between positive emotions and sleep, a recent report found that acute sleep loss modulates the neural processing of positive stimuli. In this regard, Gujar, Yoo, Hu, and Walker (2011) demonstrated that acute sleep deprivation led to interpreting emotional stimuli as more positive, which in turn was correlated with exaggerated activity in the mesolimbic circuitry.

Positive affective responses may be partly dependent on the stimulus. For instance, in a longitudinal study of medical residents, researchers employing EMA found that sleep loss intensified negative affect from a disruptive daily event, while sleep loss attenuated positive affective responses to goal-enhancing events (Zohar, Tzischinsky, Epstein, & Lavie, 2005). This study stresses the importance of context when considering the effects of sleep on affect. In another recent study, adolescents undergoing a 2-night partial sleep-deprivation protocol showed a decrease in the ratio of positive to negative affect—but this was solely due to a decrease in positive affect alone, and no significant change in negative affect following sleep deprivation (Dagys et al., 2012). Endogenous diurnal sleep rhythms may also matter—those who had an evening chronotype (went to bed much later) had a lower positive to negative ratio even when rested.

Chronotype also appears to modulate diurnal patterns of positive affect, and has been implicated in the onset of mood disorders such as depression (Hasler, Allen, Sbarra, Bootzin, & Bernert, 2010). In a recent analysis, participants with an evening chronotype displayed lower amplitude and delayed peak in daily levels of positive affect as compared with other chronotypes (Miller et al., 2014). This underscores the importance of accounting for circadian factors when investigating links between sleep and affect.

Acute Sleep Loss and Affective Reactivity to Stress

Sleep disturbance may lower one's threshold for emotional reactivity, enhancing one's emotional sensitivity to stressors. In this regard, Minkel and colleagues (2012) showed that following a night of total sleep deprivation participants reported greater subjective distress, anxiety, and anger in response to a low-stress cognitive task compared with normal sleepers; however, no differences in affect were observed between groups following a high-stress task (Minkel et al., 2012). This suggests that sleep-deprived individuals may have a lower threshold for what they perceive as stressful. Concordant with the line of thinking, participants under a night of sleep deprivation showed enhanced systolic blood pressure reactivity following an acute laboratory stressor (Franzen et al., 2011). In terms of chronic effects, poorer global subjective sleep quality enhanced the effects of acute stress exposure on proinflammatory cytokines (Heffner et al., 2012; Prather, Puterman, Epel, & Dhabhar, 2013).

Neural Mechanisms of Sleep and Affective Experience

Studies of sleep restriction have also demonstrated changes in several objective markers of emotional functioning, which further strengthens the argument that sleep is intimately related to the processing and production of affective experience. Franzen, Buysse, Dahl, Thompson, and Siegle (2009) employed pupillography as an indicator of affective processing and found that sleep-deprived participants showed greater pupil dilation to negative emotional stimuli compared with normal sleepers. Advances in neuroimaging have further illuminated the effects of sleep on the emotional brain. In a seminal study, Yoo, Hu, Gujar, Jolesz, and Walker (2007) showed that 1 night of sleep deprivation resulted in a 60% increase in reactivity of the amygdala to negative emotional stimuli compared with a rested control group. Moreover, sleep-deprived participants showed less functional connectivity between the amygdala and medial prefrontal cortex, a brain region important in exerting top-down regulatory control of the amygdala. This is consistent with a recent study that found similar decrements in functional connectivity between these

brain regions and that the declines were associated with higher reports of state anxiety (Motomura et al., 2013). Habitual sleep quality has been significantly related to stronger links between amygdala reactivity and negative affect (Prather, Bogdan, & Hariri, 2013), while longer durations were associated with stronger resting state connectivity between the medial prefrontal cortex and the amygdala (Killgore, 2013). Taken together, these data suggest that sleep loss results in greater threat reactivity, possibly due to impaired emotion regulation. Consistent with this, a recent study found that poorer overall sleep quality was related to greater difficulty utilizing cognitive reappraisal strategies in response to a sadness-inducing film clip (Mauss, Troy, & LeBourgeois, 2013).

There is also mechanistic understanding of why sufficient sleep enhances emotional functioning (Goldstein & Walker, 2014), focused on the role of rapid eye movement (REM) sleep. In this regard, REM sleep has been shown to be central to emotional memory consolidation (see Walker & van der Helm, 2009, for a review). Additionally, REM sleep is proposed to aid in resolving the strong emotions associated with challenging memories, serving as a sort of "overnight therapy" (Walker & van der Helm, 2009). REM sleep has also been implicated in recalibrating the emotional brain before the next-day emotional events, thus restoring appropriate emotional reactivity (Goldstein & Walker, 2014). The centrality of REM sleep in emotional functioning is consistent with the observed REM abnormalities associated with daily affect (Vandekerckhove & Cluydts, 2010) and REM alterations commonly observed in psychiatric samples, particularly MDD and posttraumatic stress disorder (PTSD; Baglioni et al., 2010).

Summary and Future Directions

There continues to be disagreement over the purpose of sleep and why it is conserved across species despite drastically varied predatory environments. However, it is clear that one function may be to maintain adaptive emotional functioning—by resetting the limbic machinery each night for greater connectivity between aspects critical to emotion regulation, such as the medial prefrontal cortex and amygdala. Chronically disrupted sleep leads to greater negative mood, less positive mood, and in the extreme, affective disorders. In some cases, acute and chronically disturbed sleep predicts greater reactivity to an acute lab stressor, such as in terms of blood pressure and proinflammatory responsiveness. Acute sleep deprivation appears to shape affective experience as well, although few studies have actually assessed the dynamics of emotional experience across the day. Therefore, it remains unclear whether acute sleep loss affects the diurnal rhythmicity of positive and negative mood, which tends to change significantly from waking to bedtime. There is some evidence to suggest that circadian rhythms play a role, particularly with regard to positive affect. Research methods using both standardized lab stimuli, employing state-of-the-art emotional probes, and EMA to examine the architecture of affective experience of a day would shed light on the complex dynamics between sleep and affect. Sleep can vary across several distinct dimensions, including type (REM, non-REM), ability (initiation, maintenance), form (amount, structure), and occurrence (timing, variability; Walker & Harvey, 2010). The extent to which each dimension contributes to the generation and regulation of emotion remains to be discovered.

Another open question in sleep research is whether improvements in sleep can promote long-term changes in emotion. Indeed, behavioral sleep interventions are remarkably effective in improving sleep quality among individuals with clinical sleep disorders (e.g., insomnia; Edinger & Means, 2005), and while affective processes are not primary outcomes in trials testing the efficacy of sleep interventions, there is accruing evidence that improvements in symptoms of depression, anxiety, and overall quality of life often co-occur with resolving sleep (e.g., Thorndike et al., 2013). Unfortunately, a sophisticated exploration of the affective processes that may underlie some of these findings, as well as identification of individual differences that may contribute to success of the sleep treatment for some but not others, has been lacking. Accordingly, there is unique space for affective and sleep scientists to work together to explore these processes and improve health and well-being.

Eating

Culturally speaking, the power of food to comfort us has been assumed across many centuries, as early as 1615 in the first modern novel *Don Quixote*, in which Cervantes penned the line "All sorrows are less with bread" (p. 537). The rigor-

ous study of how eating behavior affects emotions is, however, sparse. The effects of emotions—in particular, negative emotional states or stress—on eating are summarized in several reviews (Adam & Epel, 2007; Greeno & Wing, 1994; Sinha & Jastreboff, 2013; Wardle, Chida, Gibson, Whitaker, & Steptoe, 2011), and several of the most commonly used scales to measure eating behavior contain emotional eating subscales (Stunkard & Waterland, 1997; Van Strien, Frijters, Bergers, & Defares, 1986). Emotion is clearly important in triggering eating behavior, but does this eating behavior have consistent effects on changing emotional experience? If so, for who, and for how long? What are the long-term consequences?

Chronic Eating Behaviors

Chronically Disordered Eating, Emotions, and Affect

Individuals with eating disorders (anorexia nervosa and bulimia nervosa) report higher emotion regulation difficulties and have attentional biases toward negative emotional stimuli (Harrison, Sullivan, Tchanturia, & Treasure, 2010). Emotion regulation is particularly relevant to binge-eating disorder, with negative affect being the most commonly reported triggers for binge episodes (Polivy & Herman, 1993). In a clinical sample of treatment-seeking patients with binge-eating disorder, for example, emotional eating was related to fewer emotion regulation strategies, lack of emotional clarity, and difficulty accepting emotions (Gianini, White, & Masheb, 2013). Although studies of emotion regulation in eating-disordered populations are often framed as emotion regulation failure causing the disordered eating, many of these studies are cross-sectional, and therefore the converse—that eating disorders yield negative emotion regulation strategies—should also be considered.

Chronic Dietary Restraint and Dieting

Dieting is a common health behavior that can affect emotional states as well. Here we review studies on (1) diet interventions, which lower caloric intake; (2) self-reported dieting status (e.g., "Are you currently on a diet?"); and (3) chronic *dietary restraint*, which is not necessarily associated with lower caloric intake.

The negative emotional sequelae of severe caloric deprivation itself have been documented since the 1950s, beginning with Ansel Keys's seminal Minnesota Starvation Experiment using conscientious war objectors (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). In that study and others, negative emotional symptoms following dieting interventions included depressive symptomatology, anxiety, nervousness, weakness, and irritability (Stunkard & Rush, 1974; Stunkard, 1957). In experimental studies, dieting can cause depressive symptomatology in both animals (Chandler-Laney et al., 2007; Jahng et al., 2007) and humans (Wadden et al., 2004). Other human experimental data has indicated that dieting causes negative emotion after eating (Hetherington & MacDiarmid, 1993), as well as self-blame and negative views of one's character (Jeffery, French, & Schmid, 1990). In one of the few studies designed to examine the stress of dieting, in a randomized, controlled experiment, participants assigned to a 1,200-kilocalorie diet for just 3 weeks increased in both perceived stress and diurnal salivary cortisol levels, thereby providing causal evidence of dieting as an elicitor of the stress response (Tomiyama et al., 2010).

However, many intervention studies also find positive emotion effects of dieting. A paper that reviewed 10 studies of the mood effects of behavioral weight loss therapies found six out of 10 studies (separate from those above) showed an improvement in mood posttreatment while the other four showed no significant change in mood (Wing, Epstein, Marcus, & Kupfer, 1984). One short-term experimental study also observed improvements in depressive symptomatology (Bryan & Tiggemann, 2001).

In terms of self-reported dieting, depressive symptomatology appears to be one of the most common correlates of dieting in cross-sectional human studies (Ackard, Croll, & Kearney-Cooke, 2002; Cachelin & Regan, 2006; Crow, Eisenberg, Story, & Neumark-Sztainer, 2006; Gillen, Markey, & Markey, 2012; Isomaa, Isomaa, Marttunen, Kaltiala-Heino, & Bjorkqvist, 2010; Wadden, Stunkard, & Smoller, 1986). Other correlates of self-reported dieting status include anxiety symptoms (Isomaa et al., 2010). Similarly, higher dietary restraint is associated with greater depression (Cachelin & Regan, 2006), higher perceived psychological stress (McLean & Barr, 2003), and higher urinary levels of the stress hormone cortisol (McLean, Barr, & Prior, 2001).

Acute Eating Behavior

In terms of acute/experimental effects of eating on emotion and emotion regulation, several stud-

ies have examined effects of acute stress reactivity and negative mood on eating during recovery from stress. While some studies have shown that acute high cortisol reactivity predicts greater snacking after stress (Epel, Lapidus, McEwen, & Brownell, 2001; Newman, O'Connor, & Conner, 2007), other studies have found important moderators that suggest people with more stress eating may have underlying profiles of cortisol hyporeactivity. One study found that emotional eating moderated the relationship between cortisol responses to a standardized laboratory stressor (Trier Social Stress Test) and subsequent food intake, such that high-emotional eaters with a blunted cortisol response to the stressor ate more food (van Strien, Roelofs, & de Weerth, 2013). Tryon, DeCant, and Laugero (2013) found that chronically high-stress women with low cortisol reactivity ate more food in response to the stressor and had higher total fat percentage. It is unclear whether blunted stress response is a marker for vulnerability to emotional eating, or if emotional overeating dampens stress reactivity chronically, or both. In rodent research, chronic access to palatable food leads rats to develop abdominal fat, which then functions to dampen physiological stress responses at every level of the hypothalamic–pituitary–adrenal (HPA) axis (Dallman et al., 2003)—a phenomenon termed the "chronic stress response network" (Dallman, 2010; Dallman et al., 2003, 2004). Human research on this phenomenon is sparser, but one cross-sectional study found positive correlations among high chronic stress, emotional eating, and abdominal obesity, where high-stress women demonstrated dampened HPA axis activity (lower cortisol responses to a standardized stressor as well as lower daily cortisol levels; Tomiyama, Dallman, & Epel, 2011).

A small number of studies have examined food's effect on affect, rather than on stress response. Macht and Mueller (2007) found that compared with water, chocolate (but only palatable chocolate) ameliorated negative mood, although the effects were short-lived on the order of 3 minutes. In a different study by this group (Macht & Dettmer, 2006), however, both chocolate and apple consumption showed positive mood effects as long as 90 minutes after consumption. Interestingly, these positive mood effects were concurrent with guilt effects, particularly in the case of chocolate. Hetherington and MacDiarmid (1993) observed that positive mood effects of food occur only during the moment of eating, with negative emotions returning soon thereafter. Bongers, Jansen, Havermans, Roefs, and Nederkoorn (2013) found a positive correlation between calories consumed and mood improvement on a visual analog scale, regardless of whether the eating was induced by a negative, neutral, or positive mood-eliciting film clip. Finally, a study of adolescent young adult females found that the relationship between stressful life events and perceived stress was attenuated in self-reported emotional eaters (Finch & Tomiyama, 2015). However, this attenuation was not observed in those high in depressive symptoms, pointing to the complexity of the effect of eating behavior along the many different dimensions of affect.

Clearly, emotions must be measured frequently to capture acute effects of eating, and palatability of food may matter. Across three well-controlled lab studies, Wagner and colleagues (2014) sought to determine whether "comfort" food had comforting properties. Participants in these studies watched negative emotion-inducing film clips and were fed their top-ranked comfort food (including their desired brand and flavor) or a control food (a low-ranked noncomfort food), a neutral food (granola bars), or no food in respective studies. Comfort foods did indeed lead to significant improved mood, but no more so than any of the control conditions (which included no food at all), prompting the authors to determine that comfort food was a "myth." It is possible that for some people, when facing naturalistic life stressors that cause larger affective responses than the lab-based stressor, comfort eating might promote quicker recovery from negative affect through distraction or neural mechanisms of reward. It may also be that eating motives shape emotional response. Parker, Parker, and Brotchie (2006), based on their literature review, conclude that eating to feel better prolongs dysphoric mood, whereas eating to satisfy cravings provides hedonistic reward and improvements in mood.

Nutritional scientists have come at this question with a different lens by identifying chemical properties of food that might have psychoactive properties. Food contains substances such as carbohydrates, protein, fat, caffeine, tryptophan (the precursor to serotonin), and theobromine (a stimulant that is relatively high in chocolate), all of which can affect emotions (Rogers, 1995). These emotion effects are sometimes observed only when the relative ratio of one substance to another is altered. For example, food that is high in carbohydrates but low in protein can increase the ratio of tryptophan to other large neutral amino acids

and therefore affects central serotonergic function (Markus et al., 1998; Wurtman, Hefti, & Melamed, 1980). Beyond chemical substances, the orosensory properties of food (i.e., sweetness as opposed to sucrose content) can also have emotion effects. Administering sugar (vs. water) to infants reduces crying (Ramenghi et al., 2002; Smith, Fillion, & Blass, 1990), and these effects can be seen with nutritionally insignificant amounts of sucrose, implicating orosensory effects. These effects are likely mediated through opioid peptides (Kirkham & Cooper, 1988). Rogers's (1995) review of the chemical properties of food highlight other notable inconsistencies in the literature, such as the fact that mood-altering chemicals in chocolate occur at much higher levels in foods not often used for mood effects (Rogers & Smit, 2000), and note the importance of culture, learned preferences, and moderators such as dietary restraint. Examining key mediators and moderators, as well as nutritional and orosensory components, may provide a fuller picture of the effects of eating on emotions.

Neural Mechanisms of Eating and Affective Experience

The neural mechanisms that govern eating are complex and deeply intertwined with affective and emotional experience, and beyond the scope of this chapter. Here we highlight a few key models that specifically address affective and emotional *responses* to eating behavior, as that is the focus of this chapter. The aforementioned chronic stress response network as put forth by Dallman and colleagues (Dallman, 2010; Dallman et al., 2003) describes decreased basal corticotropin-releasing factor (CRF) in the hypothalamus of rodents that have consumed "comfort foods" under conditions of chronic stress. In this model, comfort foods also lessen chronic stress-induced dopamine inhibition (by preventing inhibition of dopamine output and increasing dopamine transporter activity) in the nucleus accumbens seen under chronic stress. Furthermore, comfort-eating behavior promotes intra-abdominal fat stores, which go on to act as a surrogate negative feedback signal, suppressing hypothalamic CRF expression (Dallman et al., 2004). These neural mechanisms, however, have yet to be confirmed in humans. In their reward-based stress eating model, Adam and Epel (2007) describe the ability of palatable foods to stimulate endogenous opioid release in the brain, which in turn function to attenuate the HPA stress response. Stress further sensitizes the brain to the rewarding value of palatable food, leading to a chronic drive for palatable food, and chronic suppression of the stress response. In our unpublished data from a large weight loss trial, we find that women with a blunted cortisol response to a stressor have both a greater drive to eat and a significantly greater likelihood to regain weight earlier, underscoring the tight links between eating and reactivity. Causality is unknown but the rodent studies suggest that palatable food consumption can precede the blunted reactivity.

Summary and Future Directions

Eating is a complex behavior, with many factors to consider such as overeating versus dieting, nutrient content versus orosensory properties of food, and time course of emotion effects due to eating behavior—the latter particularly in the context of acute eating given the conflicting literature. Furthermore, the potential negative emotional consequences of dieting, which is typically considered "healthy," should not be overlooked. Conversely, the positive emotional effects of being on a diet should be better understood. We must understand how dieting can promote both positive and negative emotion. What type of restricted eating behavior promotes positive emotion, and for what type of person? Can we tailor interventions so that people have positive emotion as a reinforcement and less of the "stress of dieting," and are thus more likely to adhere?

Alcohol

"Drown your sorrows" is an adage that captures the emotion-dampening role of alcohol. For centuries, humans have turned to alcohol in response to emotions (Sayette, 1993). How does imbibing alcohol, in turn, affect emotion? Of the health behaviors discussed in this chapter, the theory and literature on alcohol's effect on emotions is the most long-standing. The dampening effect of alcohol on negative emotion is one of the key examples of Hull's (1943) drive reduction theory of motivation from the 1940s. An often-cited study from this decade showed, for example, that cats provided ethanol demonstrated reduced tension and cat neurosis (Masserman & Yum, 1946). Conger (1956) then formally characterized the tension reduction hypothesis of alcohol in the 1950s, which

has spawned nearly 70 years of research on what the literature has termed "stress-response dampening" (SRD; Levenson, Sher, Grossman, Newman, & Newlin, 1980; Sher & Walitzer, 1986) resulting from alcohol consumption (Sayette, 1993).

Chronic Alcohol Use

The effects of chronic alcohol use and alcoholism on emotion have been studied during use and in response to withdrawal. Anxiety and depression are often comorbid with alcoholism, although in some cases a pathological absence of anxiety (externalizing psychopathology) is also observed (Heilig, Egli, Crabbe, & Becker, 2010). Increased anxiety is a hallmark of alcohol withdrawal, and poses a risk for relapse during treatment (Heilig et al., 2010). In later stages of alcohol withdrawal, heightened stress sensitivity (Lovallo, Dickensheets, Myers, Thomas, & Nixon, 2000) and upregulated HPA axis responsivity (Heilig & Koob, 2007) are observed. In recently abstinent alcohol-dependent individuals, however, hyporesponsivity of the HPA axis is observed (see Adinoff, Junghanns, Kiefer, & Krishnan-Sarin, 2005).

Acute Alcohol Use

The acute effects of alcohol use on stress reactivity have been widely studied in the SRD literature. Stress has been operationalized using a variety of measures. Rat models demonstrate a direct SRD effect, wherein ethanol administration dampens basal corticosterone levels (Brick & Pohorecky, 1985) as well as corticosterone responses to foot shock, restraint, and tail pinch stress (Brick & Pohorecky, 1982). In humans, acute alcohol consumption has been shown to dampen self-report measures of anxiety and distress, such as the State/Trait Anxiety Inventory (Spielberger, 2005) and physiological measures such as galvanic skin response and cardiovascular reactivity as measured by heart rate (Sayette, 1993). Despite these and other empirical studies, notable inconsistencies in the literature are evident (Wilson, 1988). For example, researchers have observed a "crying-in-your-beer" effect, wherein alcohol consumption can exacerbate negative emotions (Steele & Josephs, 1988). Indeed, for every paper indicating SRD effects in self-report, physiological, and behavioral stress, roughly equal numbers of studies fail to find effects in these very outcomes (Sayette, 1993). Even within studies examining physiological outcomes, alcohol effects can diverge, with significant findings for some measures and not others (Stritzke, Lang, & Patrick, 1996). A number of theoretical approaches have been advanced to reconcile these conflicting findings. Most notable are the Steele and Josephs's (1988, 1990) attention allocation model (later renamed the alcohol myopia model), Sayette's (1993) appraisal disruption model, and Hull's (1981) self-awareness model.

Noting that none of the existing models of alcohol and emotion was able to account for the many divergent findings, Stritzke and colleagues (1996) outlined several methodological issues to consider, including lack of consistent operationalization of "stress," both in terms of experimental manipulations and outcome measures, variation in time sampling, and variation in data analytic procedures. They called for multilevel, multidimensional models of emotion/affect that simultaneously take into account, for example, appetitive versus withdrawal motivations, positive and negative valence, and primary motivational systems versus higher cognitive functions. They also called for experimental designs that do not confound the physiological effects of alcohol with the outcome measures. For example, alcohol pharmacologically increases heart rate, and therefore using heart rate to index emotional arousal would be unproductive (Stritzke et al., 1996).

The phrase "drown your sorrows" suggests that dampening negative emotion is the predominant reason for alcohol use, but individuals also use alcohol to experience positive emotion. Among recovering alcoholics, the reasons cited for relapse are not only reducing negative emotions but also actively seeking euphoria (Marlatt & Gordon, 1980). Individuals also use alcohol acutely to increase positive emotions, particularly when fatigued or underaroused (Wills & Shiffman, 1985), or when drinking with social motives (Cooper, Frone, Russell, & Mudar, 1995).

Neural Mechanisms of Emotional Response to Alcohol Consumption

Alcohol, unlike food, is processed centrally by the central nervous system (CNS). The stimulant/depression hypothesis of alcohol accounts for both the positive and negative emotions that arise out of alcohol consumption (Tucker, Vuchinich, & Sobell, 1982). At low to moderate levels, alcohol is presumed to act as an emotional stimulant and euphoriant (or "elatant"), whereas at higher levels,

alcohol acts as an emotional depressant (Stritzke et al., 1996). Another conceptualization that takes into account the arc of CNS metabolism of alcohol is the slope of intake; rather than simple blood alcohol levels, the "rising limb" of the blood alcohol content curve is presumed to be stimulating and elating, whereas the "falling limb" of intoxication acts as an emotional depressant (Newlin & Thomson, 1990; Stritzke et al., 1996). In support of this, euphoria ratings and EEG alpha activity were closely tied to the rising blood alcohol curve in one study, but only at moderate (not low) doses (Lukas, Mendelson, Benedikt, & Jones, 1986).

Summary and Future Directions

Taken together, substantial research supports the existence of a relationship between alcohol and changes in emotional experience. However, the literature is extremely conflicted, pointing to the existence of many dispositional and situational moderators (summarized in Sayette, 1999). Regardless of whether drinking actually elicits positive or negative emotion, it is clear that individuals *expect* changes in emotional states—and these expectations may be the most fruitful area for intervention. In the validation sample of the widely used Alcohol Expectancies Scale (Brown, Christiansen, & Goldman, 1987), "physical and social pleasure" and "relaxation and tension reduction" were among the subscales with the highest endorsement (Brown, Goldman, Inn, & Anderson, 1980). Indeed, interventions that target these expectancies have had success in the short term (Darkes & Goldman, 1993; Scott-Sheldon, Terry, Carey, Garey, & Carey, 2012).

Future research should identify individual differences that govern differential responses to alcohol, with careful calibration of blood alcohol content given the physiological actions of alcohol. Do emotional effects on a person predict vulnerability to addiction, or treatment response? For example, those who evidence subjective sensitivity to the stimulating properties of alcohol (e.g., those who feel "elated" and "excited"; Martin, Earleywine, Musty, Perrine, & Swift, 1993) relative to the sedating properties of alcohol (e.g., those who feel "down") are at greater risk of developing alcoholism (Ray, Mackillop, & Monti, 2010). Finally, as alcohol contains calories, the fields of eating behavior and alcohol consumption should intersect, and future studies should also examine the two behaviors in tandem as they relate to emotional experience.

General Conclusions and Future Research Directions

Our emotional life, starting early in life, shapes our pattern of health behaviors in ways that are difficult to change. While the transactional process between emotions and health behaviors has been a focus of some scientific inquiry, one angle that has received little attention is whether engagement in health behaviors shapes emotional processes, and if this, in turn, has consequences for future health behaviors. This review shows that health behaviors have potent effects on immediate affective and emotional responses, longer-term mood states, appraisal of stressful situations, and stress reactivity.

While our review also highlights specific areas in the brain that play a role in modulating emotional states and affective responses to stress in response to each specific behavior, it is important to move beyond the idea that there is any type of one-to-one mapping of brain region and function. While each has been studied in isolation, recent reviews highlight common underlying brain regions for many health behaviors and emotions (Barrett & Simmons, under review; Lenard & Berthoud, 2008). Barrett and Simmons (under review) and Lenard and Berthoud (2008) highlight the key role of the limbic region to gauge and modulate glucose storage and usage in the body, and to promote behavioral modifications (e.g., increased or decreased food intake, exercise, sleep) accordingly to access more or use fewer nutrients (or vice versa). Emotions are the interoceptive experiences of the incongruence between the nutrient needs and usage of the body and memories from previous experiences. Emotions can thus result from or promote/hinder behavioral engagement. Within this context, for example, the findings that exercising beyond our physiological thresholds stimulates negative affective states (Ekkekakis et al., 2008; Ekkekakis & Petruzzello, 1999; Hall et al., 2002; Welch et al., 2010) makes biological sense. The negative affective state that is reached when one surpasses his or her threshold is perhaps the interoceptive experience resulting from a physiological imbalance that attempts to provoke disengagement of the activity.

We propose that a deeper understanding of the behavior–affect trajectory, in part by adopting a transdisciplinary framework, may lead to powerful insights into how to tailor interventions for better adherence and ultimately mobilize this informa-

tion for health promotion at the individual and population level (Figure 40.1). We further propose that the study of chronic health behavior effects would be best performed through long-term interventions that examine the process of change, as well as an examination of dose–response effects, and a closer examination of affect valence and arousal dynamics (Kuppens, Tuerlinckx, Russell, & Barrett, 2013).

There is tremendous potential to learn about the development and maintenance of health behaviors by applying an emotion research lens. Emotion research provides a fresh perspective on the old but important questions such as Why do some people adopt and adhere to health behaviors, both salutary (sufficient exercise and sleep) and damaging (overeating and alcohol abuse)? It also raises new questions at the heart of the emotion–health behavior processes, such as How can we capitalize on the finding that, in many cases, doing what is good for us feels good? Further, how do we reconcile that harmful appetitive behaviors, like overeating, make us feel good in the moment but often promote more negative affect than positive in the long term? Using the methodology of emotion research for a more mechanistic view of how health behaviors affect emotional and affective experiences and regulation will provide a large step forward in this field. Such information might be applied to promote more effective interventions.

We call for a new generation of research that takes on understanding the health behavior–emotion relationship with greater granularity. Specifically, we believe that it is critical that research investigate the emotional antecedents and consequences that bookend both salubrious and deleterious health behaviors, with attention to both valence and arousal, and their interrelationship (Kuppens et al., 2013; see Figure 40.1). Factors that increase postbehavior positive affect or reduce postbehavior negative affect should be identified and capitalized on. For example, manipulating positive expectations appears to have at least a short-term effect on positive affect and intentions to engage in the behavior again.

The first steps in laying a foundation in this area would be a fuller examination of emotional experience throughout the health behavior process—before onset of health behavior, during, in recovery, and long-term effects. For people who do not have the habit of a behavior (e.g., not a regular exerciser) what is the valence, arousal, and duration of positive and negative emotions arising from a bout of a new health behavior (e.g., a bout of exercise)? Do emotional effects last across a day or spill into subsequent days, and what determines duration? Does the health behavior impact the balance of positive and negative valence, evoke specific types of emotions, and/or alter appraisal processes? Such studies will require both laboratory experiments, EMA methods in naturalistic environments, and randomized behavioral interventions. **Without a within-person examination of behavior and affect dynamics, intervention data will be of limited utility in explaining individual differences in uptake and adherence of new behaviors.**

There is also a need to identify important individual differences in the behavior–emotion relationship, which may be behavior dependent. As noted above, both fitness level and dietary restraint appear to matter for exercise and for eating, respectively. Genetic propensities, personality, or emotional differences are likely important as well. It will also be important to more closely examine the mechanisms through which the health behavior modulates and improves emotional responses. For instance, the specific emotion regulation processes underlying exaggerated stress sensitivity and how they shift after behavioral interventions are key to our understanding of the emotional benefits of behavior change.

The science of emotion and behavior change hold great promise for improving individual and population health. It is clear that investigations of chronic and acute effects of specific health behaviors provide important information about emotions, affective states and regulatory processes, and their underlying neurobiology. Innovations in physiological assessment, including neuroimaging, and in sampling daily experience, provide windows into health behavior–emotion processes once hidden from view. Future research focused on teasing apart and carefully tracking the dynamic and recursive processes linking emotion and health behaviors will, without question, advance our science of both, and thus our ability to promote lifelong health at its roots.

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