# Did that brownie do its job? Stress, eating, and the biobehavioral effects of comfort food

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**Abstract**

Comfort eating is a widespread behavior, but does it actually work? The purpose of this review is to provide a summary of the existing research on the potentially comforting effects of comfort food. We begin by summarizing the existing non-human animal research in this area, and then summarize the human research. Based on this foundational research, we provide a conceptual model of comfort eating that can be used as a hypothesis-generating tool to guide future research. Finally, we highlight what we consider to be the most exciting future directions in comfort eating. These include (1) determining whether comfort eating is trait-like or state-like, (2) understanding the antecedents and sequelae of comfort eating, (3) elucidating the types of food implicated in comfort eating, (4) creating linkages between comfort eating and comfort drinking, (5) incorporating measures of the autonomic nervous and immune systems in addition to the current focus on the hypothalamic-pituitary-adrenocortical axis, (6) studying both short-term and long-term effects, and (7) testing the biological and psychological mechanisms of comfort eating. Given that comfort eating has been practiced for centuries, we conclude that the time is ripe to advance the science of comfort eating.

**Keywords**: Comfort eating, stress eating, emotional eating, HPA axis, cortisol, stress-response dampening, food, alcohol

**Disciplines:** Biology, health psychology, comparative psychology

"Milky Way – Comfort in every bar."

## Introduction

As suggested by food company slogans like "Open happiness," "Double your pleasure, double your fun," and the Milky Way slogan above, the idea of food as a comforting substance is widespread. Another, albeit more somber, example is that death row prisoners get to choose their last meal before their execution. Research shows that psychological stressors reliably promote *comfort eating* (Torres & Nowson, 2007), a behavior defined by increased consumption of high-fat, high-sugar, or high-calorie foods in response to negative emotion. Although the existence of comfort eating as a phenomenon is relatively well established, researchers have rarely considered whether it actually works – is comfort eating truly comforting? The answer to this question, no matter what the answer, is critically important. If comfort eating fails to comfort, this points to clear intervention targets: teaching individuals that food is an ineffective coping mechanism and providing alternative coping strategies. On the other hand, if comfort eating is in fact comforting, interventions may need to modify their existing approach of eradicating comfort eating altogether.

The purpose of this review is to provide a summary of the existing research on the potentially comforting effects of comfort food. As this is an emerging area of research, there are many important questions still to be answered. We therefore provide a conceptual model of comfort eating, designed to be a hypothesis-generating tool to guide future research, and highlight what we consider to be the most exciting future directions.

Answering the question of whether comfort food is comforting requires an interdisciplinary approach. Biological and psychological perspectives are necessary, particularly because "comfort" is a difficult term to operationalize; some research has characterized comfort

as reduction in biological stress responsivity of the hypothalamic-pituitary-adrenocortical (HPA) axis, while others have focused on decreased negative emotions. Both perspectives are necessary. Furthermore, both human and non-human animal models are necessary. On the one hand, rodent models provide essential causal data on the potentially comforting effects of comfort eating, and in particular the brain-based mechanisms of this process. On the other hand, only human models can provide comprehensive insight into emotional processes. With this in mind, we first review the extant research conducted in rodent models and then in human models.

## What evidence exists?

### Comfort eating in rodent models

To investigate comfort eating in rodents, researchers predominantly expose rats or mice to physical stressors, which involve the introduction of strain or challenge to the rodent's body. For example, a researcher may induce physical restraint stress by placing the rodent in a snug-fitting tube, which prohibits free movement, or use stressors such as warm/cold swim tasks, elevated maze tasks, novel environments, rotating platforms, or oxygen deprivation. Alternatively, rodents may be exposed to types of social-behavioral stressors, such as maternal separation or defeat to a socially dominant peer.

Physical, social-behavioral, and (in humans) psychological stressors all have an immediate effect on an organism's physiology by stimulating a cascade of hormonal secretions via the HPA axis. This begins with the release of corticotropin-releasing hormone (CRH) from the hypothalamus, which stimulates the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary gland. ACTH then circulates through the bloodstream to the adrenal cortex, where it stimulates glucocorticoid secretion.

Growing evidence from rodent models supports a salutary role of comfort food in reducing the physiological stress response to acute stressor (for reviews of the field summarized below, see work by pioneer Mary Dallman, beginning with Dallman et al., 2003). A common procedure involves feeding rodents a diet of either palatable comfort foods (e.g., high-fat lard or high-sugar sucrose) or a bland control food (chow), introducing a physical or social-behavioral stressor and assessing activity of the HPA axis. Studies applying this design have collectively revealed that comfort food functions to dampen HPA axis activation at every step of its physiological cascade. For example, in rats exposed to an acute restraint stressor, those fed a diet of comfort food versus chow in the 7 days prior showed reduced hypothalamic CRH mRNA, inhibited ACTH secretion, and decreased corticosterone (glucocorticoid) response to the stressor. Similarly, sucrose consumption has been shown to dampen corticosterone response to acute restraint stress. Furthermore, it appears that comfort food may dampen physiological responses to chronic stress. In chronically restrained rats the combined provision of lard and sucrose decreased ACTH response to repeated stress compared to chow alone. Taken together, these studies evidence what Dallman and colleagues (2003) have termed a *chronic stress response network* model, a process through which comfort eating reduces the neuroendocrine activity of the physiological stress response.

Importantly, glucocorticoids such as corticosterone that are released as a normative component of the stress response also go on to promote both stress-induced eating and the accumulation of abdominal fat (Torres & Nowson, 2007), which in turn attenuate stress responses (Dallman et al., 2003). Indeed, several studies have demonstrated strong negative feedback between abdominal fat stores and hypothalamic CRH expression. Researchers have yet to identify the particular mechanism through which abdominal fat may dampen CRH expression;

however, some have suggested that under chronic stress these fat stores may serve as a surrogate for the negative feedback signal of glucocorticoids that is normally present under acute stress conditions (Dallman et al., 2003).

In addition to dampening physiological stress responses, comfort eating has also been shown to reduce behavioral stress responses (reviewed in Finch & Tomiyama, 2014). For example, consuming palatable comfort foods decreased anxiety and depressive-like behaviors in rats exposed to chronic maternal separation stress and chronic unpredictable social defeat and overcrowding stress. Similarly, a high-fat diet has been shown to reduce behavioral anxiety in rats placed in an elevated, narrow cross-shaped platform. Interestingly, this effect may be highly dependent upon macronutrient content, as high-carbohydrate and high-protein diets did not decrease anxiety behaviors. In aggregate, these studies suggest that palatable foods function to inhibit behavioral anxiety and depressive responses to acute and chronic stress.

**Comfort eating in human samples**

Although comfort eating and abdominal fat stores consistently reduce physiological and behavioral stress responses in rodent models, only a handful of studies have tested these processes in humans. One key difference between non-human and human studies of comfort eating is the operationalization of the construct of comfort eating. Rodent models tend to focus on comfort eating primarily in response to stress, whereas human studies commonly measure *emotional eating*, which refers to comfort eating in response to a broader range of emotional states. Despite this difference, preliminary evidence in humans appears to converge with rodent findings in support of the chronic stress response network.

In a study by our group (Tomiyama, Dallman, & Epel, 2011), women with high chronic perceived stress reported greater emotional eating and had significantly greater BMI and waist

diameter compared to low-stress women. In a laboratory component of the study participants underwent a public speaking and arithmetic stress task, the gold-standard procedure used to induce psychological and physiological stress responses in humans. Following stress-induction, women characterized by chronic stress and high emotional eating also exhibited a reduced physiological response of cortisol (the human version of corticosterone) compared to women low in chronic stress and emotional eating. Although these results are consistent with rodent models, this study is limited by its reliance upon self-report measurement of emotional eating; direct measurement of eating behavior would provide improved validity.

Several human studies have directly measured food intake in the laboratory after acute stress induction. In one study, Tryon, DeCant, and Laugero (2013) assessed both food intake and total cortisol response following acute stress. In addition, the women in this sample were categorized as high or low chronic stress based on a self-reported measure. Results showed that women with high chronic stress and low cortisol reactivity to the speech/arithmetic task also consumed more calories from chocolate cake in response to the stressor. Furthermore, high chronic stress in these low cortisol reactors was also associated with greater total fat mass and regional fat percentage. Thus, in keeping with rodent models, this study demonstrates correlations between high chronic stress, high comfort eating, greater abdominal fat, and reduced cortisol response to acute stress.

In a similar experimental design, a second study exposed female students to the speech/arithmetic task and subsequently assessed total cortisol response and food intake (van Strien, Roelofs, & de Weerth, 2013). A self-reported measure of emotional eating was also administered to categorize the women as either extremely high or extremely low on emotional eating. Results revealed that emotional eating moderated the relationship between cortisol

reactivity and food intake. In high emotional eaters, low cortisol reactors consumed more food post-stressor than high cortisol reactors, whereas no such relationship was found in low emotional eaters. Thus, in this study we see again a pattern of association between reduced cortisol response to acute stress and increased comfort eating in high emotional eaters.

Only two known research groups have experimentally manipulated emotional eating behavior. Macht and Mueller (2007) conducted two studies in samples of healthy men and women to test whether consuming comfort food after viewing a sad film clip would improve mood to a greater extent than other conditions. In the first study, they found that the consumption of chocolate after the film improved negative mood to a greater extent than drinking water. In the second experiment Macht and Mueller examined whether food palatability was a necessary requirement for mood improvement. Results showed that the consumption of palatable milk chocolate after viewing a sad film clip improved negative mood to a greater extent than the consumption of non-palatable dark chocolate (70-99% cocoa) or no food. This effect was short lived, however; there were no significant differences in mood across conditions at 3 minutes post-consumption. Thus, perhaps comfort foods primarily function to improve mood in a more immediate sense.

In a series of within-subjects experiments, Wagner, Ahlstrom, Vickers, Redden, & Mann (2014) tested whether the consumption of a self-reported favorite comfort food after a negative mood induction (film presentations) would lead to significantly greater mood improvement compared to the consumption of an equally-liked non-comfort food, a neutral food, or no food at all. Examining changes in mood from immediately post-film to three minutes later, the consumption of a top-ranked comfort food did not improve mood significantly more than the consumption of a non-comfort food or no food at all. Furthermore, in a final between-subjects

study, Wagner et al. found that the consumption of chocolate before viewing the negative film presentation was not more effective at preventing a negative mood than merely receiving (but not eating) chocolate. Importantly, the authors concluded that comfort foods *did* indeed cause improvements in mood; however, these improvements were not significantly greater than those induced by other foods or by no food at all.

In sum, whereas preliminary cross-sectional human studies reliably show correlations between high chronic stress, greater comfort eating, and reduced cortisol response to acute stressors, early experimental studies of emotional eating have yielded conflicting results and further research is needed to form a conclusive understanding of causal relationships.

## Key Issues for Future Research

Given this emerging research, there are many promising future directions for the study of comfort eating. In Figure 1, we integrate the complex components of comfort eating into a comprehensive conceptual model. We designed this model to be used as a flexible tool to generate new hypotheses and study designs. By choosing one or more components from each box, researchers can generate a theoretically-based, sophisticated interdisciplinary research question that will move the field of comfort eating forward. For example, the rodent research discussed above has mainly tested a hypothesis that draws a straight line across the top components of the model in Figure 1 – stress with administration of sugar affecting biological mechanisms (specifically the HPA axis), which then dampens the stress responsivity of the HPA axis. However, there are many more hypotheses to be tested, and next we discuss the "low hanging fruit" in the area of comfort eating.

### Comforting eating: Trait or State?

A survey study (Oliver & Wardle, 1999) found that approximately 40% of respondents reported eating less when stressed, and another 40% reported eating more. In line with these findings, most of the human literature characterizes emotional eating as an individual difference variable. Commonly, studies use the Dutch Eating Behavior Questionnaire (DEBQ; Van Strien, Frijters, Bergers, & Defares, 1986) to categorize individuals into emotional eaters versus non-emotional eaters. This is in contrast with the rodent literature, in which comfort eating effects emerge across all members of the species. Future studies using quasi-experimental designs of participants categorized as emotional eaters versus not could shed light on the universality of comfort food's effects. However, self-reported emotional eating tendencies often do not describe actual eating behavior in experimental contexts. For example, Bongers and colleagues (2013) found that emotional eaters and non-emotional eaters ate the same amount in response to a negative mood induction. It may be that humans are unable to accurately report on their own eating behavior. Indeed, a study specifically designed to test whether self-report measures of emotional eating were related to eating under emotional circumstances found no differences in food consumption between self-identified emotional eaters and non-emotional eaters (Evers, de Ridder, & Adriaanse, 2009). They declared that assessing oneself as an emotional eater was "mission impossible" (p. 717).

**Antecedents and sequelae of comfort eating**

*Emotional antecedents.* Although we have used the term comfort eating throughout this paper, and the word "comfort" implies that such eating is in response to negative stimuli, in practice the operationalization of this phenomenon is inconsistent. The rodent studies are tightly focused on stress specifically as the triggering context, using well-accepted stress paradigms such as restraint stress. In human studies, however, studies of "stress-eating" and "emotional

eating" and "comfort eating" vary in their conceptualization of the triggering context. The commonly used DEBQ emotional eating scale contains only negative eliciting contexts (e.g., irritated, depressed, bored, lonely, angry, "feel bad," anxious, stressed). However, other researchers have used both negative and positive words. We recommend that the operationalization of comfort eating solely include eating in response to negative emotions, as eating in response to positive emotions has no "comfort" element, and thus is likely a theoretically distinct phenomenon from comfort eating. This is supported by Sproesser, Schupp, and Renner (2014), who found that emotional eaters eat less in response to positive emotions.

*Emotional and physiological sequelae.* What would constitute evidence of the effectiveness of comfort eating? Is dampened physiological stress reactivity sufficient, or must it also coincide with decreases in negative emotion? What if negative emotions are unaffected, but positive emotions increase? To facilitate progress in research in this area, it is critically important that researchers be precise and consistent not only in their choice of operationalization of the phenomenon of comfort eating, but also in what to accept as proof of efficacy of this behavior.

**Type of food**

Rodent studies of comfort eating predominantly use lard + sucrose mixtures as comfort food. As a result, calorie density, fat, and sweetness are confounded. Beyond the obvious question of whether one of these nutritional characteristics in particular is driving comfort eating effects, this methodological convention leaves several questions unanswered. For example, what about salty foods? Anecdotally, many individuals would name salty foods as comfort foods – foods like macaroni and cheese or mashed potatoes. Some studies (e.g., Tomiyama et al., 2012) have provided both high fat and low fat foods, crossed with sweet versus salty taste, which has yielded differential effects. These examples also highlight the confounding of nutrient properties

(such as carbohydrates) versus hedonic (such as sweetness) properties of food, either of which (or both) may be driving comfort eating effects. Further clouding the picture, at least in humans, is the issue of conditioning. Comfort food may be paired with social and emotional comforting from family members during childhood and beyond. Thus, any observed comforting effects of food might not be due to a conserved stress-buffering mechanism, but rather a conditioned effect. This could be tested in studies providing participants' most favorite comfort foods versus standard calorie-dense, sweet foods analogous to the rodent studies, to see whether there are differential effects. One study (Scherschel et al., 2014) used such a design, and found no difference between whether the food was a favored comfort food or not. That study, however, found that the comfort food condition provided no greater comfort than a no-food condition, thus calling altogether into question the idea of comfort foods as comforting.

**Comfort Eating and Comfort Drinking?**

Although not always initially thought of as food – but it is fermented sugar – alcohol is another type of food to consider in comfort eating research. While researchers have traditionally studied drinking alcohol and eating in isolation, there are several commonalities between these behaviors: each coincide with similar activation of reward pathways (Volkow, Wang, Fowler, & Telang, 2008), the chronic effects of eating too much sugar have been compared to the chronic effects of drinking alcohol (Lustig, Schmidt, & Brindis, 2012), and researchers have begun identifying commonalities between binge eating and binge drinking (see Ferriter & Ray, 2011 for a review). Additionally, as the phrase "drown your sorrows," suggests, drinking alcohol is another behavior individuals engage in to comfort themselves. Indeed, stress reduction is one of the most widely reported motives for alcohol use (Cooper, Frone, Russell, & Mudar, 1995). Social and problem drinkers, and even children who have not begun drinking alcohol, commonly

report stress reduction as an expected outcome of alcohol use (Christiansen, Goldman, & Inn, 1982).

There is a sizeable literature on the stress-dampening properties of alcohol, beginning in the 1950s when Conger put forth the tension reduction hypothesis of alcohol, spawning nearly 70 years of research on what the literature has termed *Stress-Response Dampening* (for reviews of the non-human and human animal research, respectively, see Becker, Lopez, & Doremus-Fitzwater, 2011; Becker, 2012). Therefore, it may be fruitful to link the nascent research on comfort eating to the alcohol stress-response dampening literature. Future research can answer several questions, which may benefit our overall understanding of each behavior. For instance, do comfort foods and alcohol initiate the same effects on the stress response? The research on alcohol stress-response dampening has mixed results, with alcohol decreasing sympathetic nervous system (SNS) components like cardiovascular reactivity, skin conductance response, and blinking, but sometimes increasing HPA reactivity like cortisol response to lab stressors (Jones et al., 2013). Moreover, advice from "Living Sober" from Alcoholics Anonymous states, "We can only pass on the word that thousands of us—even many who said they had never liked sweets—have found that eating or drinking something sweet allays the urge to drink," despite the fact that virtually no data exist on the effectiveness or harm of this strategy. When trying to reduce overall harm, might comfort eating be a healthier alternative for heavy drinking to alleviate stress? Are there certain situations where one behavior may be more useful for stress reduction relative to the other behavior? A final consideration is that when someone is under stress he or she may comfort eat and drink alcohol to reduce stress. What then happens at the intersection of comfort eating and alcohol stress-response dampening? Future research could disentangle the overlap and divergence between these two behaviors.

**Beyond the Hypothalamic-Pituitary-Adrenal axis**. The HPA axis is only one of two main stress-responsive pathway systems of the body. The other is the sympathetic branch of the autonomic nervous system, or the SNS. The general purpose of the SNS is to mobilize the body into action when under threat, regulating bodily components such as blood pressure, heart rate, and gut motility. Thus, one clear step for comfort eating research is to measure SNS related stress-responses after an individual eats comfort food. Just like in the alcohol stress-response dampening literature, there may be mixed findings between HPA and SNS responses after comfort eating. Further, previous research in humans has found that SNS and HPA activity can diverge in response to certain types of stressors (e.g., Frankenhaeuser, Lundberg, & Forsman, 1980). For instance, among healthy subjects exposed to the Trier Social Stress Test three times over 12 weeks, salivary cortisol levels seemed to habituate – decrease or cease to respond to stress – quickly, while heart rate response was uniform to the repeated exposures (Schommer, Hellhammer, & Kirschbaum, 2003). Therefore, the field's current narrow focus on the HPA axis may be missing important potential comforting effects of food. This may be especially relevant to examine with comfort eating in response to types of stressors that do not seem to activate the HPA axis, or stressors other than social threats (Dickerson & Kemeny, 2004).

In addition to the main stress-responsive systems of the HPA axis and SNS, the immune system is another bodily system that interacts with stress processes. The immune system responds to psychological stress by (1) increasing its activity in the short-term in the form of innate immune responses like inflammation, and (2) decreasing its activity in the long-term in the form of adaptive immune responses like creating antibodies. If comfort eating were to attenuate either one of these immune responses, long-term health could potentially be affected.

**Long-term versus short-term effects**

The small existing experimental literature on comfort eating in humans has focused on short-term effects. Rodent experimental research, on the other hand, has identified long-term fat deposition and stress dampening effects of comfort eating. Future research, therefore, should take a longer view of the potential effects of human comfort eating. Given that both stress and overeating individually contribute to poor health, long-term research would extend our understanding of the potentially divergent effects of comfort eating. First, comfort eating may provide short-term salubrious effects and function as an effective coping mechanism in response to acute stressors. However, will comfort eating used as an acute coping mechanism have long-term negative health effects? Or will its stress-dampening properties override any calorie or nutrient imbalance and ultimately contribute to better health? If so, rather than characterizing comfort eating as unequivocally harmful to health, individuals and interventionists will have to carefully weigh the benefits and costs of comfort eating. This will provide a more comprehensive understanding of health that incorporates both mind and body – necessary given that the dominant perspective currently is that comfort eating is a negative health behavior to be targeted through intervention and eradicated.

**Mechanisms**

Finally, if comfort eating is shown to be emotionally and physiologically comforting, the next step will be to identify the precise mechanisms that drive its comforting effects. Here we will highlight three likely suspects. First, the fact that comfort is a behavioral response that is conserved across multiple species suggests that the mechanism may be biological, although researchers in this area acknowledge that the precise signaling mechanism remains unidentified (Dallman et al., 2003). Nonetheless, the rodent findings indicate that when eating food there is a comfort signal that does indeed act in the brain to decrease the adverse effects of the chronic

stress response. A remarkable study by Van Oudenhove and colleagues (2011) used a gastric feeding tube (thereby ruling out the conditioning, placebo, and other psychologically-mediated effects we discuss next) to infuse participants with either fatty acids or saline. They found that those randomly assigned to receive fatty acids rated sad music and sad faces as less sad than those assigned to receive saline. Furthermore, key emotion-related brain areas were activated in concurrent brain scanning using fMRI in the fatty acid group. The authors speculated, based on the time course of the effects and the specific brain regions activated, that these findings were likely neurally mediated by cholecystokinin (a gut hormone)-induced signals traveling through the vagus nerve to the brain (Van Oudenhove et al., 2011).

Second, and as noted above, conditioning is another likely mechanism driving the comforting effects of food, as food is often paired with other comfort-inducing situations such as social interactions. A third possible mechanism is the placebo effect. Just as non-active placebos can elicit dramatic improvements in health, so too might food act as a placebo to ameliorate psychological and biological stress. Placebo pills, after all, are often *sugar* pills.

## Conclusion

In sum, there is an enormous amount of exciting research to be conducted in the area of comfort eating. We summarized this research in Figure 1, which researchers can use as a tool to generate hypotheses and study designs. For example, a researcher could randomly assign individuals to experience either an activated emotion like anger or a withdrawn emotion like sadness, then provide either standard high-fat, high-sugar comfort foods or their comfort food of choice, and examine sympathetic nervous system activity (particularly relevant here would be blood pressure, which is closely linked to anger) to look at short-term blood pressure dampening. This tool can even be used to design intervention studies. For example, a depression researcher

could test an intervention providing socially isolated (i.e., lonely) individuals high-sugar, -fat, and-carbohydrate foods before exposing them to a supportive therapist, to take advantage of conditioning mechanisms. The researcher could then test whether this dampens HPA activity (as HPA activity has been linked with depression), and additionally conduct longitudinal follow-ups to monitor any potential weight changes in the long-term.

Future research, however, will require interdisciplinary approaches that unite psychology and biology. The idea of comfort eating has existed for many centuries now, as evidenced by the Cervantes quote "All sorrows are less with bread" from *Don Quixote.* We believe it is high time to advance the science of comfort eating.

## References

Asterisks denote recommended reading.

*Becker, H. C. (2012). Effects of Alcohol Dependence and Withdrawal on Stress Responsiveness and Alcohol Consumption. *Alcohol Research-Current Reviews*, *34*(4), 448–58.

*Becker, H. C., Lopez, M. F., & Doremus-Fitzwater, T. L. (2011). Effects of stress on alcohol drinking: a review of animal studies. *Psychopharmacology*, *218*(1), 131–56.

*Bongers, P., Jansen, A., Havermans, R., Roefs, A., & Nederkoorn, C. (2013). Happy eating. The underestimated role of overeating in a positive mood. *Appetite*, *67*, 74–80.

Christiansen, B. A., Goldman, M. S., & Inn, A. (1982). Development of alcohol-related expectancies in adolescents: separating pharmacological from social-learning influences. *Journal of Consulting and Clinical Psychology*, *50*(3), 336–44.

*Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: a motivational model of alcohol use. *Journal of Personality and Social Psychology*, *69*(5), 990–1005.

*Dallman, M. F., Pecoraro, N., Akana, S. F., La Fleur, S. E., Gomez, F., Houshyar, H., … Manalo, S. (2003). Chronic stress and obesity: a new view of "comfort food." *Proc Natl Acad Sci U S A*, *100*(20), 11696–11701.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355–91.

*Evers, C., de Ridder, D. T. D., & Adriaanse, M. A. (2009). Assessing yourself as an emotional eater: mission impossible? *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, *28*(6), 717–25.

*Ferriter, C., & Ray, L. A. (2011). Binge eating and binge drinking: an integrative review. *Eating Behaviors*, *12*(2), 99–107.

*Finch, L. E., & Tomiyama, A. J. (2014). Stress-induced eating dampens physiological and behavioral stress responses. In R. R. Watson (Ed.), *Nutrition in and Prevention and Treatment of Abdominal Obesity* (pp. 189–196). New York: Elsevier.

Frankenhaeuser, M., Lundberg, U., & Forsman, L. (1980). Dissociation between sympathetic-adrenal and pituitary-adrenal responses to an achievement situation characterized by high controllability: comparison between type A and type B males and females. *Biological Psychology*, *10*(2), 79–91. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7437488

Jones, A., McMillan, M. R., Jones, R. W., Kowalik, G. T., Steeden, J. a, Pruessner, J. C., … Muthurangu, V. (2013). Habitual alcohol consumption is associated with lower

cardiovascular stress responses--a novel explanation for the known cardiovascular benefits of alcohol? *Stress (Amsterdam, Netherlands)*, *16*(4), 369–76.

Lustig, R. H., Schmidt, L. A., & Brindis, C. (2012). The toxic truth about sugar. *Nature*, *482*, 27–29.

*Macht, M., & Mueller, J. (2007). Immediate effects of chocolate on experimentally induced mood states. *Appetite*, *49*(3), 667–74.

*Oliver, G., & Wardle, J. (1999). Perceived effects of stress on food choice. *Physiol Behav*, *66*(3), 511–515.

Scherschel, H., Ahlstrom, B., Vickers, Z., Redden, J., & Mann, T. (2014). The myth of comfort food. *Health Psychology*.

Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2003). Dissociation Between Reactivity of the Hypothalamus-Pituitary-Adrenal Axis and the Sympathetic-Adrenal-Medullary System to Repeated Psychosocial Stress. *Psychosomatic Medicine*, *65*(3), 450–460.

*Sproesser, G., Schupp, H. T., & Renner, B. (2014). The bright side of stress-induced eating: eating more when stressed but less when pleased. *Psychological Science*, *25*(1), 58–65.

*Tomiyama, A. J., Dallman, M. F., & Epel, E. S. (2011). Comfort food is comforting to those most stressed: Evidence of the chronic stress response network in high stress women. *Psychoneuroendocrinology*.

*Tomiyama, A. J., Schamarek, I., Lustig, R. H., Kirschbaum, C., Puterman, E., Havel, P. J., & Epel, E. S. (2012). Leptin concentrations in response to acute stress predict subsequent intake of comfort foods. *Physiology & Behavior*, *107*(1), 34–39.

*Torres, S. J., & Nowson, C. A. (2007). Relationship between stress, eating behavior, and obesity. *Nutrition*, *23*(11), 887–894.

*Tryon, M. S., DeCant, R., & Laugero, K. D. (2013). Having your cake and eating it too: a habit of comfort food may link chronic social stress exposure and acute stress-induced cortisol hyporesponsiveness. *Physiology & Behavior*, *114-115*, 32–7.

*Van Oudenhove, L., McKie, S., Lassman, D., Uddin, B., Paine, P., Coen, S., … Aziz, Q. (2011). Fatty acid-induced gut-brain signaling attenuates neural and behavioral effects of sad emotion in humans. *The Journal of Clinical Investigation*, *121*(8), 3094–9.

Van Strien, T., Frijters, J. G. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional and external eating behavior. *Int J Eat Disord*, *5*, 747–755.

*Van Strien, T., Roelofs, K., & de Weerth, C. (2013). Cortisol reactivity and distress-induced emotional eating. *Psychoneuroendocrinology*, *38*(5), 677–84.

Volkow, N. D., Wang, G.-J., Fowler, J. S., & Telang, F. (2008). Overlapping neuronal circuits in addiction and obesity: evidence of systems pathology. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *363*(1507), 3191–200.

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**Figure Caption**. Researchers can use this tool to generate hypotheses and study designs. “Bored” appears in gray as we believe it is still unclear whether boredom constitutes an emotion. We have crossed out “Happy” because we believe it is qualitatively different from the other emotions, and happiness-induced eating likely is driven by different processes and therefore will have different sequelae. Nonetheless, we include these two emotions in our model because they can also generate hypotheses for future research. Note: carb = carbohydrate; kcal = Kilocalorie; HPA = Hypothalamic-Pituitary-Adrenal; SNS = Sympathetic Nervous System; PNS = Parasympathetic Nervous System.

*Figure 1.* Conceptual, hypothesis-generating model of comfort eating.