Biological Psychiatry

Review

A Framework for Developing Translationally Relevant Animal Models of Stress-Induced Changes in Eating Behavior

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ABSTRACT

Stress often affects eating behaviors, leading to increased eating in some individuals and decreased eating in others. Identifying physiological and psychological factors that determine the direction of eating responses to stress has been a major goal of epidemiological and clinical studies. However, challenges of standardizing the stress exposure in humans hinder efforts to uncover the underlying mechanisms. The issue of what determines the direction of stress-induced feeding responses has not been directly addressed in animal models, but assays that combine stress with a feeding-related task are commonly used as readouts of other behaviors, such as anxiety. Sex, estrous cyclicity, circadian cyclicity, caloric restriction, palatable diets, elevated body weight, and properties of the stressors similarly influence feeding behavior in humans and rodent models. Yet, most rodent studies do not use conditions that are most relevant for studying feeding behavior in humans. This review proposes a conceptual framework for incorporating these influences to develop reproducible and translationally relevant assays to study effects of stress on food intake. Such paradigms have the potential to uncover links between emotional eating and obesity as well as to the etiology of eating disorders.

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The goal of this review was to develop a conceptual framework to guide the development of reproducible and translationally relevant assays to study effects of stress on food intake. Stress represents a challenge to homeostasis. In the broadest terms, it occurs when a biological system detects a failure to control a variable that is critical for survival and/or reproductive success (1). To maintain energy balance, organisms have evolved robust mechanisms to integrate interoceptive signals of fuel availability with predictions about the caloric value of foods that are available (2). Food seeking and consummatory behaviors involve risks, such as getting sick from spoiled or poisonous food, exposure to extreme temperatures, and lurking predators. Strong evolutionary pressures fostered the development of systems that respond to challenges to energy homeostasis in a manner that maximizes benefits while minimizing risk (3). When external stressors pose an imminent threat to survival, interoceptive signals of hunger that promote food seeking and consummatory behaviors are suppressed.

The relationship between a severe stress and feeding behaviors is preserved in present-day society, where stresses such as job loss, divorce, or death of a loved one are usually accompanied by anhedonia and weight loss (4,5). On the other hand, in the context of a chronic threat, an organism will die if it is not motivated enough to take the risk to find food. Therefore, it is important that the drive to seek food overcomes fear in a state of negative energy balance and that nutrient intake occurs in the most efficient way possible through the consumption of calorically dense foods (3,6). The tendency to eat junk foods in response to stress is well documented in clinical and epidemiological studies (7–9).

The relative strength of signals relaying information about internal fuel availability, perceived threats in the environment, and the reward value of the food item determines whether food intake is increased or decreased in response to mild and moderate stressors (10). Yet, studies that examined the effect of stress on eating attitudes or intake reported mixed results (7,11,12). This led to the hypothesis that individual differences in the perception of external stressors and reward value of food underlie this heterogeneity (11,13–15). Identifying physiological and psychological factors that determine the direction of eating responses to stress has been a major goal of epidemiological and clinical studies. However, challenges of standardizing the stress exposure in humans hinder efforts to uncover the underlying mechanism.

This relationship between stress and feeding in rodents is best studied in the context of pathophysiological eating behaviors. Rodent paradigms to model binge-eating behavior and activity-based anorexia produce a consistent and robust increase or suppression of food intake, respectively [reviewed in (16,17)]. Standardization of these assays has permitted comparisons of findings between laboratories and across species that are critical to establish relevance to humans. Several key elements of the neural signature of individuals with anorexia nervosa have been recapitulated in the activity-based anorexia model, such as disrupted reward signaling (18–20) and reduced serotonin signing (21–23), supporting construct

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validity. The ability to perform neurogenetic manipulations of discrete circuits and pathways in these models is accelerating progress in the understanding of mechanisms driving pathological eating behaviors (20,24,25). However, because they are designed to induce a specific type of outcome (i.e., anorexia vs. binge eating), they cannot be used to identify determinants of stress-induced increases versus decreases in food intake.

While the relationship between stress and feeding in a nonpathophysiological context has not been explored directly, paradigms commonly use stress-induced suppression of appetitive behaviors as a surrogate measure of anxiety- or depression-like behaviors in rodents [reviewed in (26,27)]. We initially set out to mine the literature for studies that combine stress with measurements of food intake, with the goal of developing a predictive model of stress-induced changes in food intake; we identified 57 publications that fit these criteria. Unfortunately, the lack of standardization across key features of the paradigms precluded meaningful comparisons.

The purpose of this review is to provide a framework for developing standardized assays to study determinants of stress-related eating behavior that are translationally relevant to humans. First, we consider major influences on feeding behavior—sex, estrous cyclicity, circadian cyclicity, caloric restriction, palatable diets, elevated body weight, and properties of the stressors—and discuss the degree to which they are conserved between humans and rodents. We summarize evidence that implicates each factor in feeding responses to stress from studies in humans and rodent models. Then we discuss the degree to which the selected rodent studies recapitulate conditions that are relevant for humans. Finally, we present recommendations for incorporating these influences into the design of paradigms to evaluate the effects of stress on food intake.

METHODOLOGY

We performed an extensive search of the PubMed database for assays involving stress and feeding-related tasks. Studies using these assays usually focus on anxiety- and depressive-like behaviors, so ancillary effects on food intake are rarely described in the abstract. While we used keyword searches to identify relevant articles, we manually inspected the contents of each article and selected those that quantified caloric intake. Keywords included the following: “food intake,” “stress,” “chronic stress,” “early life stress,” “novelty-suppressed feeding,” “novelty-suppressed hypophagia,” “hyponeophagia,” “sucrose intake,” “sucrose preference,” “fast-refeeding,” “circadian cyclicity,” “sex,” “estrous,” “estrogen,” “age,” “palatable diet,” “chronic high fat diet,” “adiposity,” “caloric restriction,” “social isolation.” After excluding activity-based anorexia and binge-eating models that are designed to achieve a targeted outcome, we identified 57 studies that met our criteria (Table S1). Some used one or more assays that measure acute intake in response to acute, subchronic, or chronic stress: fast/refeeding (n = 7), hyponeophagia (n = 1), novelty-suppressed feeding (n = 12), novelty-induced hypophagia (n = 4), food choice (n = 1), and sucrose preference (n = 15). Some also reported intake patterns over hourly, daily, or weekly time scales (n = 29). We searched for patterns across these studies with the goal of identifying aspects of the paradigms that determine whether stress increases or decreases intake.

EFFECTS OF SEX IN PARADIGMS INVOLVING STRESS AND FEEDING

In humans and rodents, males and females experience stress-induced changes in food intake, with sex differences in the impacts of distinct types of stress (4,13,28–30). For example, in both mice and humans, females preferentially decrease energy expenditure during caloric restriction, and males consume more when access to food is restored after caloric restriction (30,31).

In humans, anxiety, depressive symptoms (32), eating disorders, and subclinical disordered eating behaviors (33,34) are more prevalent in females. The earliest studies of stress-induced eating behavior reported stronger effects in women (11). These observations fostered an overall bias toward stress-induced increases in intake in females that is reflected in subjects chosen for clinical and epidemiological research, as well as the lay press. As the number and size of studies examining stress-induced feeding behaviors increased, findings of sex differences were inconsistent (35). Sex differences in responses to different types of stress could contribute to the variability in the findings (4,13,36,37). For example, women are generally more sensitive to interpersonal and emotional stress, while men are more sensitive to ego-threatening stressors (13,14).

In rodent models, reports of sex biases in studies of stress-related feeding behaviors are similarly heterogeneous. While some studies showed no significant sex differences in the effects of stress (38–42), others reported reduced intake in females during the stress paradigms (43–46). As seen in humans, sex differences in responses to the type of stress incorporated in the study could confound interpretation of the results. For example, experimental paradigms that involve substantial movement, such as open field tests, elicit higher locomotor activity in female rodents (28,29,47,48). On the other hand, males spend more time engaged in social interaction when paired with conspecifics (28).

In the selected studies, almost half (47%) used males exclusively, while 16% used only females (Figure 1). The rest included both sexes in their design. This demonstrates the strong bias of animal studies toward studying males. The growing appreciation of the importance of characterizing sex differences (49,50) and the National Institutes of Health mandate to include sex as a biological variable (51) have spurred a recent increase in the number of studies in our analysis that incorporate both sexes (38% of studies in the 2010s vs. 0% in the 1990s).

Recommendation

The fact that sex differences were observed in some contexts across species (11,43–46) suggests that studies should be powered to detect sex-specific differences in sensitivity to the type of stress that is incorporated into the paradigm. If true, it may be necessary to develop sex-specific variations of a paradigm.

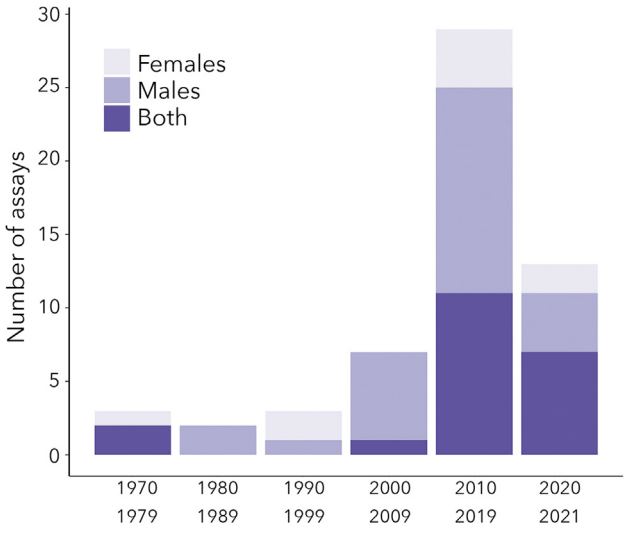

Figure 1. Sex. Numbers of publications that used males exclusively, used females exclusively, or incorporated both sexes by decade.

EFFECTS OF ESTROUS CYCLICITY IN PARADIGMS INVOLVING STRESS AND FEEDING

In both human and rodent females, food intake is influenced by hormones that fluctuate across the ovarian cycle (52–54).

In humans, individual behaviors are highly predictable (4,7), suggesting that these responses are primarily driven by invariant factors. However, there are significant associations between levels of ovarian hormones and likelihood of engaging in emotional eating, and these are exacerbated in conditions of stress [reviewed in (55)].

In rodent models, ovariectomy leads to increased food intake that is reversed by estradiol replacement, consistent with an anorexigenic action of estrogen (56). Potential effects of gonadal hormones on food intake have been used to justify the exclusion of females (57). Yet, studies that explicitly examined stress-induced feeding behaviors in female rodents across the estrous cycle do not support the idea that these fluctuations are a major source of variability (41,58–60). However, there are some contexts when differences in the levels of gonadal hormones exert a strong effect on food intake, such as a severe fast (61) or adolescent stress (62).

Of the selected studies that included females, 33% reported estrous phase.

Recommendation

Considering that levels of gonadal hormones can affect the outcome of the assay in some contexts (61,62), differences across the estrous cycle should first be assessed. As collecting vaginal smears and frequent handling of the mice can affect stress levels, developing assays that do not vary across the estrous cycle is preferable.

EFFECTS OF CIRCADIAN CYCLICITY IN PARADIGMS INVOLVING STRESS AND FEEDING

In both humans and rodents, systems regulating feeding behaviors fluctuate across the circadian cycle, with orexinergic pathways gradually increasing the homeostatic drive for feeding and arousal across the inactive phase, peaking at the onset of the active phase [reviewed in (63)].

Humans are active and usually eat during the light cycle. Studies in shift workers show that working at night is associated with increased intake of junk food or foods with a high carbohydrate content (64,65) and with dysregulated eating behaviors (66,67).

Rodents ingest the majority of their daily food intake during their active phase, similar to humans, but their active phase is at night (68,69). Yet, most studies using rodent behavioral assays are performed during the day. Direct comparisons of the active and inactive phases demonstrate that appetitive behaviors and motivation to eat are significantly higher at the onset of the dark period (69–71), regardless of the length of the fast (68,69).

Of the assays analyzed here that evaluated acute feeding behaviors ($n = 41$) (as opposed to daily intake), 82% were in the inactive phase, 13% were in the active phase, and only 5% assessed both the active and the inactive phases (Figure 2).

Recommendation

While the outcome of assays modeling other aspects of behaviors such as anxiety or social behaviors may not be affected (72), feeding is tightly regulated by influences of circadian cyclicity in both humans and rodents. Evaluating dark phase behavior is necessary to translate food intake–related findings to humans.

EFFECTS OF CALORIC RESTRICTION IN PARADIGMS INVOLVING STRESS AND FEEDING

In both humans and rodents, the state of negative energy balance created by caloric restriction influences the drive to eat (73–75).

In humans, reducing caloric intake to lose weight has been shown to modulate the effects of stress on eating in both laboratory-based and questionnaire-based studies. People who are dieting are more likely to report stress-induced hyperphagia, while nonrestrained eaters are more likely to report

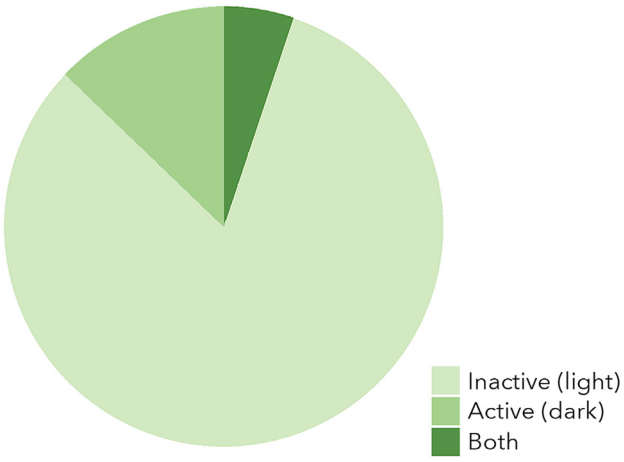

Figure 2. Circadian cyclicity. Percentage of assays that evaluated food intake in the active phase (13%), the inactive phase (82%), or both (5%).

stress-induced hypophagia, regardless of sex (7,76,77). Although fasting also promotes subsequent eating in humans, most studies are based on self-reported measurement, without explicit consideration of acute prandial state, and generally focus on eating in the absence of hunger (78).

In rodents, caloric restriction is used as a way to achieve binge eating–like behaviors in rodents when associated with chronic stress and access to palatable diets (79–82), but the effects of caloric restriction on nonpathological feeding behaviors were not thoroughly studied. A prolonged (24–48 hours) fast is more commonly used to trigger food intake in various assays (i.e., fast/refeed, novelty-suppressed feeding, sucrose preference test). This approach implicates a metabolic and psychological stress in rodents (83,84) and can change behaviors in rodents (85).

Of studies analyzed here that evaluated acute feeding behaviors, 58% were performed after a fast (overnight or longer), 5% were performed after a short food deprivation (several hours), and the remainder were performed in the random fed state (Figure 3). Only one of the studies we analyzed examined the effects of stress in the context of weight loss (79).

Recommendation

Shifting studies to the active phase (as decribed above) eliminates the need for a fast, which is stressful (83,84) and not relevant to humans. Conversely, the effect of chronic caloric restriction on stress-induced changes in feeding behaviors needs to be evaluated more thoroughly in rodents, as it is commonly observed in humans (7,77,86).

EFFECTS OF PALATABLE DIETS IN PARADIGMS INVOLVING STRESS AND FEEDING

In humans and rodents, palatable diets activate reward circuits in the brain that regulate motivated behaviors (87–89).

In humans, both chronic self-reported stress and acute laboratory stress increase consumption of high fat, palatable snack foods in males and females, with no impact on overall caloric intake (7,76).

In rodent models, although stress is often linked to the consumption of "comfort foods" (90,91), this is not the case for all stressors. For example, in males, social isolation (92,93) and chronic mild stress (94–98) can decrease intake of sucrose.

Of the selected studies, only 27% involved palatable diets, and 37% used sucrose solutions. The composition of the palatable diets varied widely; 17 out of 26 assays involved distinct combinations of palatable foods.

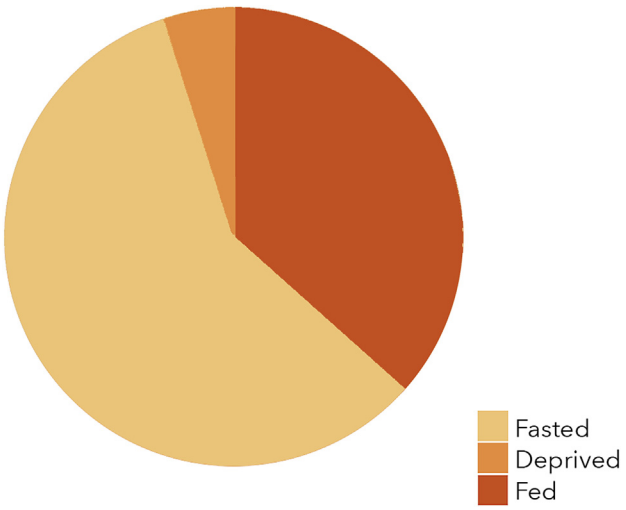

Figure 3. Prandial state. Percentage of assays evaluating the effects of stress on food intake after a fast (58%), after food deprivation (5%), or in the fed state (37%).

Recommendations

Palatable diets are usually an option for humans and seem to play a critical role in the feeding response to stress (7,76). Therefore, they should be incorporated into rodent paradigms. However, the degree to which differences in macronutrient composition (i.e., high fat vs. high sugar) and whether the diet is presented in liquid or solid form should be considered, and diets should be standardized to permit comparisons between studies.

EFFECTS OF ELEVATED BODY WEIGHT IN PARADIGMS INVOLVING STRESS AND FEEDING

In humans and rodents, diet-induced obesity is associated with changes in systems regulating reward and motivation to eat (99–103).

In humans, elevated body mass index is consistently associated with eating in the absence of hunger in both children (104) and adults (14,105). High body mass index and chronic stress are also strongly associated with unhealthy eating habits in shift workers (106,107). Increased cravings for highly palatable foods in satiated individuals with obesity (105,108) in the presence of dampened dopaminergic reward circuits (99,100) are proposed to drive excessive caloric intake.

In rodents, the most common model of obesity is chronic exposure to a high fat diet (HFD) or high fat/high sugar diet. As observed in humans with obesity, prolonged HFD exposure weakens signaling in dopamine reward circuits in male rats (101–103). Mice consume more calories overall, and chronic exposure devalues subsequent intake of standard chow after a fast in both sexes independent of body weight gain (109). The relationship between chronic HFD exposure and stress-induced feeding behavior in rodents is complicated. It seems to magnify existing tendencies for stress-induced hyperphagic (110,111) or hypophagic responses (112–114). However, because each study here involved a different length of HFD exposure and type and duration of stress, it is hard to draw conclusions that are more definitive.

Of the selected assays, only 23% incorporated elevated body weight into the paradigm, mostly through diet-induced obesity.

Recommendations

Considering the importance of the relationship between elevated body weight and stress eating in humans (14,107,108), incorporating diet-induced obesity in rodent models would increase translatability and could help to parse effects of chronic versus acute exposure to palatable diets.

CONSIDERATION OF STRESSORS THAT ARE INCORPORATED INTO PARADIGMS INVOLVING STRESS AND FEEDING

In both humans and rodents, variability in the type of stress incorporated into the study design hampers efforts to uncover determinants of stress-related feeding behaviors. The most consistent findings are that people are more likely to eat less as the severity of the stress increases (4,5), and when they engage in emotional eating, it usually involves consumption of palatable foods (8,9,76,115). This general pattern is conserved in rodents (112,116–121).

We classified the selected studies according to the type of stressor (physical, psychological, social), the length of the stress (acute, subchronic, chronic) and the timing (early life, adolescence, adulthood). In addition to the intended stressors, we noted two additional stressors that are unintentionally imposed in many experiments in rodents: fasting and single housing. As discussed above, fasting is often used to motivate consumption of a chow diet during the day. It also increases the level of circulating stress hormones (83,84) and food-seeking behavior (85). Measurement of individual food intake in rodents usually necessitates single housing, which has sex-specific effects on the function of the hypothalamic-pituitary-adrenal axis (122–124) and feeding behaviors (93). In the studies analyzed here, 63% housed the animals individually, and only 7% compared single housing with group housing (Figure 4). There were no consistent patterns in the combination of stressors used. Of the 48 assays that involved an intentional stress, we identified 35 different combinations of experimental conditions when sorted by the acute assay, type of stressor, prandial state, and housing status. Although the variability in the study designs did not permit us to identify general determinants of the direction of feeding responses to stress, we highlight aspects of stressors that should be considered when designing assays.

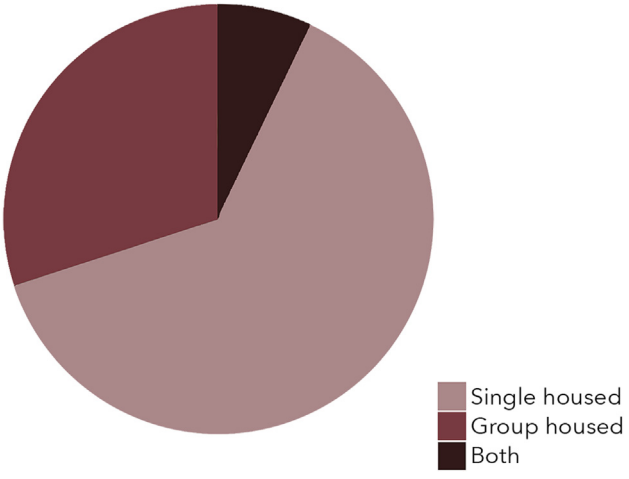

Figure 4. Social isolation. Percentage of assays evaluating the effects of stress on food intake in single-housed animals (63%), group-housed animals (30%), or both (7%).

Type of Stress

It is not possible to make general claims about the effect of social stress or physical stress on food intake because stressors of the same class can have different effects on food intake in rodents. For example, in males, social isolation and overcrowding decrease feeding (92,93,125), while chronic social defeat stress promotes hyperphagia (111,126). In females, depriving access to maternal care can have opposite effects, depending on the severity of the manipulation. Maternal separation (3 hours for 14 days) increases consumption of a palatable diet, while maternal deprivation (no access for two 24-hour periods) decreases it (127).

Timing of Stress Across the Life Span

Exposure to stress across the life span can impact the outcome of the stress response on food intake in a sex-, age-, and diet-dependent manner in both humans [reviewed in (128)] and rodents (44,45,91,127,129–132). In rodents, stress exposure throughout gestation does not affect baseline food intake in males (91,129), but it decreases baseline intake in females (91) and reduces sucrose preference in both sexes (129,132). Combining stress exposures in two developmental periods can produce synergistic effects. For example, male mice exhibit anhedonia when exposed to stress in both gestation and adolescence, but not when the manipulation is limited to one time period (42). Of the experiments that imposed developmental stressors, 6 were gestational, 8 were early postnatal, and 4 were adolescent. Even when study designs are similar, comparisons are hindered by differences in the reported outcome measures.

Age of Acute Test

The age of the animals at the time of the assay can influence its outcome. In rodents, chronic social defeat stress tends to increase sucrose preference in adult males (126), while it decreases it in adolescent males (133). Even when studies are performed in "adult" animals, the differences in ages used in the studies we analyzed (range 21–630 days, median 70 days) can affect stress-induced feeding behaviors. Performing the test in older adult male rodents (>1 year) is more likely to result in decreased consumption of chow compared with younger adult animals (6–10 weeks) (134).

Recommendations

Ideally, the development of a consensus on several standardized assays that could be used as a battery to evaluate stress-related feeding would permit comparisons between groups, enhancing the rigor and reproducibility of the research, as shown for models of anorexia-like and binge-eating behaviors [reviewed in (16,17)]. Until that happens, there are several ways to increase the impact of studies by individual groups. First, the impact of varying the length and/or the severity of the stressor could be assessed. In addition, analyses at the individual level, instead of providing group averages, could permit comparisons between susceptible and resistant individuals that could uncover important determinants of feeding behavior. This strategy has been used successfully to study factors that promote susceptibility to

binge eating in rats (135,136) and anorexia-like behaviors in mice (24,137).

TECHNICAL CONSIDERATIONS IN MEASURING FEEDING BEHAVIORS

Because some of the assays were designed to study anxiety-like behavior, the standard reported measures of food intake are not adequate to draw meaningful conclusions about appetite drive (57). In assays involving intake of palatable food/drink, rodents are typically acclimated to the novel substance for a few days, without confirming that they reached a stable baseline (138). If the test involves consumption of the diet in a manner to which the rodent is not accustomed (i.e., in a weight boat or pipette), they should be trained for 5 to 7 days beforehand. It is critical to ensure that baseline food intake is stable, particularly when introducing novel foods as part of the assay. Individuals that do not train to eat or drink in the test conditions should be excluded. Intake at baseline and in the stress condition should be reported. Paradigms in which the main outcome measure is latency to eat often report intake for 5 to 10 minutes after the start of the test. Measurements of food intake should begin after the first bite/lick and not at the start of the test. Finally, food intake should be recorded for at least 30 minutes (57,138).

SUMMARY AND RECOMMENDATIONS

Influences of sex, estrous cyclicity, circadian cyclicity, caloric restriction, palatable diets, elevated body weight, and properties of the stressors are conserved across species, supporting the use of rodent models to study stress-induced changes in food intake. The sheer number of factors that shape stress-related eating behaviors complicates efforts to uncover key pathways and therapeutic targets. We present some recommendations to foster efforts to develop reproducible and translationally relevant assays to study stress-induced feeding behaviors (Figure 5):

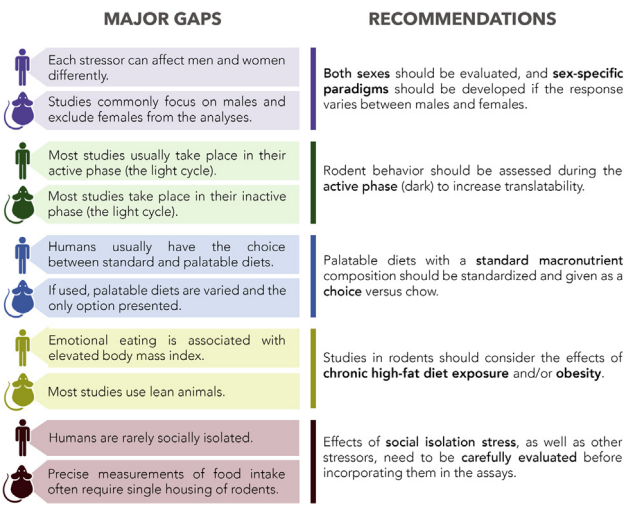

Figure 5. Recommendations to increase translatability of rodent studies of stress-related eating behaviors.

- The research community would benefit from establishing a battery of assays to examine stress-induced feeding behavior with standardized protocols (including housing conditions and antecedent chronic stressors) and feeding-related outcomes measures.
- Studies should be powered to detect sex differences. If the same stressor produces opposite effects on food intake, developing sex-specific paradigms may be needed. Similarly, potential effects of estrous cyclicity should also be considered, and adaptations to the protocol can be made to eliminate these effects if needed.
- Assays should be performed in the active phase; this enhances translational relevance and avoids stresses from caloric restriction.
- Palatable diets should be used in the acute test.
- Studies of stress-induced overeating should include models of chronic exposure to obesogenic diets and/or caloric restriction to recapitulate observations in humans.
- It is important to appreciate that measurement of food intake almost always involves single-housing of animals, which exerts opposite effects on feeding behaviors in males and females. Inclusion of group-housed control animals should be considered, as people are usually not socially isolated.

FUTURE DIRECTIONS

Recent technical advances make it possible to perform unbiased analyses to identify brain regions and molecular pathways during a specific behavioral task. The development of translationally relevant assays of stress-related eating behaviors is needed to fully exploit these cutting-edge tools. Applying these new approaches to study the effects of stress on food intake has the potential to uncover links between emotional eating and obesity as well as links to the etiology of eating disorders.

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ARTICLE INFORMATION

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