



# A narrative review of stress, food choices, and eating behavior: Integrating psychoneuroendocrinology and economic decision-making

Grant S. Shields<sup>a,\*</sup> , Trey Malone<sup>b</sup> 

<sup>a</sup> Department of Psychological Science, University of Arkansas, USA

<sup>b</sup> Boehlje Chair in Managerial Economics for Agribusiness, Department of Agricultural Economics, Purdue University, USA

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## ABSTRACT

Perhaps all of us have heard of the term “comfort eating,” and many of us likely engage in the practice occasionally. Despite an intuitive understanding of at least one influence of stress on food choices, the influence of stress has largely gone unconsidered in economic models of food choice and consumer behavior. A growing body of literature, from a wide array of disciplines, has highlighted the importance of considering stress within food choices and consumer behavior. In this review, we survey this growing literature, focusing as narrowly as neurobiological mechanisms linking stress to food choices and as broadly as population-level studies that have examined such influences. Considered together, the literature suggests that even a mild nationwide stressor may alter food consumption patterns. Such effects have nontrivial implications, ranging from production considerations for major snack manufacturers to public health for policymakers. We conclude with recommendations for future work on the subject, including work aiming to understand food choices on a national scale.

## 1. Introduction

The “double burden” of malnutrition has become a heightened global policy concern: Estimates indicate that one in three people are directly affected by some food deficiency or food-related problem, such as underweight, stunting, wasting, nutrient deficiencies, overweight, obesity, and diet-related non-communicable diseases (Fattore et al., 2021; International Food Policy Research Institute, 2024). Developing an understanding of the factors that influence food choices thus has profound public and economic implications. Such an understanding, for example, might permit identification of numerous effective points for intervention and elucidate unintended consequences of prior interventions at both the individual and policy levels (Li et al., 2022; Turner et al., 2024).

Despite the importance of understanding the cognitive decision mechanisms underlying food choice, prior literatures most relevant to public policy have largely overlooked stress (i.e., a situation or set of contextual demands that requires an organism to expend an atypical amount of resources in order to adapt to or cope with those demands; various forms of stress will be defined and discussed in greater detail below) as a key factor that may influence health outcomes. In response,

studies—especially in the fields of psychology, psychoneuroendocrinology, nutrition, and, recently, economics—have linked greater stress to changes in food choices, eating behaviors, and obesity and overeating (Doan et al., 2022; Doom et al., 2020; Okumus & Ozturk, 2021; Torres & Nowson, 2007). With the development of these literatures comes a need for an integrated, policy-relevant approach to the role of stress in food choices. Given the gaps in policy research, there is value in developing a conceptual framework of stress-induced food choice based on an in-depth review of the neurocognitive and hormonal connections between stress-induced cognitive function and decision-making related to nutrition. To that end, this review surveys and synthesizes the work on this topic, highlighting important potential points for intervention and additional research.

We begin this review by describing stress and its well-established physiological effects. From there, we turn to the field of economics on stress and food choices or consumption patterns. Although less work has been done in this area than in psychoneuroendocrinology, we survey this nascent body of work, highlighting changes in food consumption patterns that occur during or follow major stressful events. Throughout this review, we emphasize gaps that might be filled by a deeper

Systematic review data and syntax for effect sizes are given on the Open Science Framework: [https://osf.io/2ymgb/?view\\_only=5327e6951dfc432ba50a5f640d4bf890](https://osf.io/2ymgb/?view_only=5327e6951dfc432ba50a5f640d4bf890)

\* Corresponding author at: 480 N. Campus Walk, Fayetteville, AR USA.

E-mail address: [gshields@uark.edu](mailto:gshields@uark.edu) (G.S. Shields).

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understanding of the individual-level neurobiological processes that might lead to the causal outcomes of interest.

Next, we synthesize the relevant research in psychoneuroendocrinology on stress-related, modifiable processes involved in food choices—namely, those that underlie self-regulation. Then, we describe how stress influences self-regulation and the presumptive effects that those influences have on food choices. Next, we survey other pathways through which psychological research has shown that stress influences food choices and eating behavior, such as direct effects on taste sensitivity and hunger hormones.

Following the above, we present the results of a systematic review of all studies that have experimentally induced stress and examined its effects on food consumption within the laboratory, highlighting emergent trends on macronutrients and specific foods from that review.

Next, we integrate these findings into a broader conceptual model of stress and food choices. We also provide some testable predictions from our model and speculate about the importance of considering stress in future work that attempts to forecast consumer behavior.

Finally, we describe future directions in this field for both psychoneuroendocrinology and economics before summarizing and concluding.

### 1.1. Stress

Although stress is something with which we all have subjective experience, it can be difficult to define (Epel et al., 2018). In stress literature, *stress* refers to a subjective appraisal or experience (e.g., that one's environmental or situational demands exceed one's current resources), whereas a *stressor* is an objective circumstance that, on average, produces a stress response. Experiences of stress are typically differentiated by their timing: An *acute* stressor refers to a discrete, time-delimited event with a clear beginning and endpoint (e.g., a car accident, an argument with a significant other, etc.), whereas a *chronic* stressor refers to an enduring difficulty that often lacks clear a beginning or endpoint (e.g., debilitating neck pain following a car accident, discrimination, etc.) (Monroe & Slavich, 2016; Shields & Slavich, 2017; Slavich, 2020; Slavich & Shields, 2018). Acute and chronic stress reflect the corresponding subjective experiences that accompany said events when they are appraised as such. These distinctions are not perfect—some chronic stressors are perhaps little different from repeated acute stressor exposures (e.g., regular and ongoing arguments with a significant other is considered a chronic stressor, but each argument is considered an acute stressor)—but they are generally useful. Importantly, though, utility of this distinction may break down in some cases over the long-run in real-world settings: Sufficient repeated acute stressor exposure can produce a physiological profile of chronic stress (Lam et al., 2019) for reasons related to the physiological acute stress response, described below.

Acute stress differs from anxiety, anger, and other high-arousal, negative-valence affective states (Lebois et al., 2016; Shields, 2020; Shields & Slavich, 2017; Slavich, 2020) and is identifiable via a unique physiological response (Shields, 2020). Production of that physiological response is quite predictable, and this response is highly conserved across species: The acute stress response occurs when an organism perceives its environment to contain a threat to their safety (in social animals, this extends to social safety), either immediately or eventually, and when the demands of that environment are perceived to exceed one's resources or abilities (Epel et al., 2018; Lazarus, 1982). In other words, change is required in order to survive a stressor. The acute stress response, then, is referred to as an allostatic response, whereby stability (i.e., life) is maintained through change.

The acute stress response can be summarized as a series of systemic changes that collectively function to provide energy and improved function to muscle tissue, as well as recovery from injury or infection, should they occur (Dhabhar, 2002; Sapolsky et al., 2000; Shields & Slavich, 2017; Silverman & Sternberg, 2012; Slavich, 2020; Taborsky

et al., 2021). Like simple negative affect, the stress response activates the sympathetic-adrenal-medullary (SAM) axis, thereby increasing heart rate and improving blood flow via increases in circulating epinephrine and norepinephrine (i.e., adrenaline and noradrenaline, which are referred to as the “fight-or-flight” hormones). Unlike negative affect, the stress response also activates the hypothalamic-pituitary-adrenal (HPA) axis, which promotes the synthesis and release of glucocorticoids (e.g., cortisol) into the bloodstream (Sapolsky et al., 2000; Hellhammer et al., 2009; Allen et al., 2014). Glucocorticoids' primary functions are energetic—the “gluco” in “glucocorticoids” is for glucose, foreshadowing the role that this class of hormones appears play in food choices (Moeller et al., 2016). These hormones act to increase circulating glucose and alter glucose uptake by various tissues, such as increasing relative glucose uptake in muscle tissue (Munck & Koritz, 1962; Munck et al., 1984; Sapolsky et al., 2000). The stress response further activates the hypothalamic-pituitary-gonadal (HPG) axis (Lennartsson et al., 2012; Meethal & Atwood, 2005), facilitating muscular function and recovery via the actions of testosterone and estradiol. Finally, although this description is far from an exhaustive list of changes, stress activates the immune system via increasing synthesis and production of signaling proteins known as proinflammatory cytokines (Slavich & Irwin, 2014), and this preemptive activation of the immune system facilitates healing from injury or infection, should they occur (Dhabhar, 2002).

Although the acute stress response is adaptive in the short-term, it has long-term consequences (Lupien et al., 2009; McEwen, 1998; McEwen & Gianaros, 2011; Monroe, 2008; Monroe & Slavich, 2016; Quinn & Shields, 2023; Silverman & Sternberg, 2012). This wide-ranging breach of homeostasis via activation of numerous physiological systems can be thought of as borrowing resources from the future to survive the present. When it occurs in one isolated instance, its effects are minimal, the acute stress response produces physiological “wear-and-tear” that will eventually lead to dysfunction in stress-responsive systems (Lupien et al., 2009; McEwen, 1998; McEwen, 2005; McEwen, 2007; McEwen & Wingfield, 2003). Just as repeated administration of drugs of abuse produces tolerance to their effects, so too does repeatedly exposing neurons and immune system cells to glucocorticoids, ultimately reducing glucocorticoid receptor sensitivity and thus glucocorticoids' abilities to exert effects (Pariante, 1999; Silverman & Sternberg, 2012; Slavich, 2020; Slavich & Irwin, 2014). Collectively, the physiological profile of chronic stress is largely one of insensitivity to stress-responsive systems—blunted glucocorticoid responses to acute stress, immune insensitivity to the anti-inflammatory effects of glucocorticoids, chronic inflammatory activity, and biological aging (Burani et al., 2022; Capitano & Cole, 2015; Cole et al., 2009; Furman et al., 2019; Juster et al., 2010; Lupien et al., 2009; McEwen, 1998; McEwen & Gianaros, 2011; Nusslock & Miller, 2016; Silverman & Sternberg, 2012; Slavich & Irwin, 2014; Sloan et al., 2007). Chronic stress is further characterized by disrupted diurnal rhythms in stress-responsive physiological systems and greater susceptibility to a host of diseases (Deer et al., 2018; Furman et al., 2019; Knauff et al., 2025; Shields et al., 2020; Silverman & Sternberg, 2012; Slavich, 2020).

The above description has a few important caveats. First, it does not describe the entirety of the acute physiological stress response, and it should not be taken to do so. Indeed, the acute stress response also consists of many other components, including changes in the central nervous system that are not stress-specific (e.g., altered activity in regions supporting executive functions, emotional states, and reward sensitivity; Arnsten, 2015; Henckens et al., 2012; Hermans et al., 2014; Hermans et al., 2011; Shields et al., 2021; Shields et al., 2019), as well as other stress-specific changes, such as other forms of hormonal reactivity (e.g., dehydroepiandrosterone, or, DHEA; Shields et al., 2016), other changes in immunity (e.g., a specific timecourse of changes to natural killer cell activity; Schedlowski et al., 1993), and more. Second, acute physiological stress responses do not occur in every individual in exactly the same way, and factors such as prior stress exposure can alter acute stress responses (Kudielka et al., 2009; Lam et al., 2019; Luettgau et al.,

2018). Third, absolute levels of these markers are not indicative of stress—as described above, cortisol’s function is primarily energetic, not stress-dependent—as these markers are also, or even primarily, influenced by other factors, such as eating and sleep (Holloway & Lerner, 2024; Kudielka et al., 2009). Fourth, the above does not describe the physiological profile of chronic stress (described below), which markedly differs from the acute physiological stress response (McEwen, 2019; Russell & Lightman, 2019). Nonetheless, acute stress physiology is remarkably consistent: Every acute stressor will produce the aforementioned physiological responses at the level of a population average (i.e., mean-level changes that differ from zero) (Hermans et al., 2025; McEwen, 2019; Russell & Lightman, 2019; Selye, 1950). This unique acute physiological response permits differentiation of stress from negative affective states (e.g., anxiety, anger) within humans and across species (Epel et al., 2018; McEwen, 2019; Russell & Lightman, 2019; Selye, 1950; Shields, 2020).

There are two more takeaways from the above that are relevant to the current review. First, it would be inaccurate to simplify stress to cortisol alone, and we therefore do not review the isolated influences of cortisol on food choices or other relevant work (e.g., Hirata et al., 2012; Jaszczak & Jaszczak, 2021; Moeller et al., 2016; Reynolds, 2013; Rose et al., 2010; Serfling et al., 2019; Spencer & Tilbrook, 2011) within this review. Second, the unique conjunction of changes that form the physiological acute stress response is distinct from the changes elicited by other negative affective states (e.g., anxiety, anger, sadness), and laboratory manipulations that failed to elicit mean-level changes in all examined stress-specific biomarkers (e.g., cortisol reactivity, DHEA reactivity, systemic cytokine reactivity, etc.) cannot be said to have induced acute stress, at least as stress is operationalized within stress research (McEwen, 2019; Russell & Lightman, 2019; Shields, 2020). This is an important consideration for understanding studies examining the effects of stress on outcomes of interest to this review, to which we now begin to turn.

## 2. Stress and food choices: the perspective of economics

There is a growing acceptance that health outcomes and economic impacts are interrelated, which requires updated approaches to modeling nutrition interventions (Cawley, 2015; Dötsch-Klerk et al., 2023; Rohde et al., 2017). This need for biologically consistent economics research is critical, as public policies can create unintended consequences if they target the wrong mechanisms. For example, policies focused on improving public health by reducing consumer access to “comfort foods” are unlikely to achieve their desired goals under some circumstances, as consumers are more likely to choose novel foods during times when they are making a life change (Stacy, 2010). Similarly, implementing a soda tax can increase soda consumption for at-risk populations due to social reactance to the tax (Debnam, 2017). As such, studies emphasizing the connections between stress and eating behavior require added scrutiny to determine the causes that lead to negative outcomes (Groesz et al., 2012).

Economic factors are commonly linked to food consumption decisions via a handful of common mechanisms (Zasimova, 2022). For example, global recessions can cause changes in healthy food access due to relative price changes (Bai et al., 2021). The impact of these recessions on food consumption decisions is particularly pronounced for people who become food insecure due to their altered financial situation (Watson et al., 2020). This impact is largely consistent with traditional microeconomic theory, as the marginal utility changes caused by a price change will be larger for lower-income households. Price changes are not the only way consumer purchases might be altered by economic factors: Job loss can lead to increased drinking and body mass index (BMI) (Caroli & Godard, 2016; Deb et al., 2011; Nizalova & Norton, 2021). More generally, the stress induced by poverty has also been identified as an explanatory variable for unhealthy food choices, leading to higher rates of obesity (Offer et al., 2010; Salmasi & Celidoni, 2017;

Thomas, 2022). Similarly, public policies associated with the COVID-19 pandemic also increased BMI, though the mechanisms with which these increases occurred remain understudied (Sung et al., 2024). Mental stress is one possible mechanism that might link job loss to changes in healthy behavior, although a prior meta-analysis indicates that the effects are small (Wardle et al., 2011). Importantly, however, effects that are small in magnitude can produce a large problem for public health if these effects are consistent across the entire population.

Just as losing work can lead to overeating, so too can occupational burnout and the stress it entails (Chui et al., 2020; Nevanperä et al., 2012; Okumus et al., 2019). Similarly, caregiving stress can also alter consumer demand for snacks (Cozac et al., 2023). One possible mechanism driving these impacts is heightened cognitive load, which has been tied to significant increases in unhealthy food choices (Lien & Zheng, 2018). These stress impacts are not limited to the individual but can also spill over to the household’s overall health (Bauer et al., 2012; Kong et al., 2021).

In addition to macro-economic stressors such as unemployment, the behavioral economics literature also offers insight into a range of stress-inducing contexts that shape decision-making. Generally speaking, heightened cognitive load can mirror the effects of acute stress by impairing self-control and increasing preference for immediate gratification. For instance, time pressure, uncertainty, social evaluation, and even a large number of choice options might induce stress responses that affect consumer behavior (Malone & Lusk, 2017; Malone & Lusk, 2019). Moreover, stress from social exclusion or identity threat can alter risk preferences and food consumption patterns in ways consistent with behavioral economic theories of affective forecasting and reference dependence (DeDecker et al., 2022; Richards et al., 2014).

It is important to distinguish that much of the evidence described in the economics literature reflects correlational associations, rather than experimental tests of causal mechanisms. For example, much of the literature focuses on links between economic factors, such as job loss, and health outcomes, such as increased BMI or consumption of less nutritious foods. While these studies offer valuable insights into real-world patterns, they often cannot isolate the independent effect of stress from other confounding variables, such as income loss or access constraints. This highlights a key opportunity for future work incorporating validated stress manipulations or longitudinal quasi-experimental designs to establish causal pathways better.

Together, these findings suggest that, at the level of broader economies, stress is associated with differences in food choices. The mechanisms underlying these associations, however, are not clear. It is for this reason that we now turn to the work from psychoneuroendocrinology, which can speak to the cognitive, hormonal, and neurobiological pathways through which stress may influence food choices.

## 3. Stress and food choice: the perspective of psychoneuroendocrinology

### 3.1. Self-Regulation and food choices

Psychologists have long recognized (Birch & Deysher, 1985; Dassen et al., 2018; Hall & Fong, 2007; Shields et al., 2017; Tomiyama, 2019) that food choices are often acts of *self-regulation* (i.e., the process[es] by which we act in alignment with long-term goals when faced with attractive decisions to forego those goals). For example, we may have a long-term goal of being healthy, but such a goal often faces conflict with aspects of or items within immediate environments: Opening the freezer to retrieve a bag of frozen vegetables often prompts a consideration of the delicious ice cream bars that are then in view as well. Indeed, healthy foods are often less appetizing than unhealthy foods (Mai & Hoffmann, 2015); choosing unappetizing but goal-relevant (i.e., healthy) foods over appetizing but unhealthy foods requires self-regulation (Aguirre-Rodriguez et al., 2021; Allom & Mullan, 2015; Juergensen & Demaree, 2015; Yokum & Stice, 2013). Understanding

how stress influences self-regulation, then, should help to understand how and why stress influences food choices.

At the core of virtually every prominent theoretical model of self-regulation is the idea that self-regulation is underpinned by a top-down process, typically referred to as *executive function*, which exerts control over bottom-up influences on decisions (e.g., reward sensitivity and negative affect). Each of these processes may play a unique role in food choices. For example, better executive function predicts greater consumption of healthy foods (Allom & Mullan, 2014; Hall, 2012), whereas both greater negative affect (Heatherston & Wagner, 2011; Macht, 2008) and altered reward sensitivity (de Decker et al., 2016; Loxton, 2018) predict greater consumption of unhealthy foods. Depending upon how stress influences these component processes involved in self-regulation, stress may lead to nuanced effects on food choices that are functions of self-regulation.

In addition to the above, it is also possible—indeed, some evidence suggests—that stress may influence processes more directly involved in food choices, such as taste perception itself (e.g., Ileri-Gurel et al., 2013). Therefore, stress may influence food choices through multiple, at least partially independent routes. We thus now turn to a discussion of these routes.

### 3.2. Stress influences processes underlying self-regulation

Stress influences processes that underlie self-regulation, and these effects may help to explain the influence of stress on food choices.

#### 3.2.1. Stress and executive function

Stress impairs executive function (Quinn & Shields, 2023; Shields et al., 2016; Shields et al., 2024). For example, at the meta-analytic level, we have shown that stress impairs performance on virtually every type of executive function task outcome (Shields et al., 2016)—the one exception being “response inhibition,” which stress, surprisingly, facilitates (Chang et al., 2020; Dierolf et al., 2017; et al., 2013; Shields & Yonelinas, 2024; Shields et al., 2025). Stress correspondingly alters neural activity in regions supporting executive function, such as various subregions within the prefrontal cortex (PFC) (Berretz et al., 2021; Qin et al., 2009). This stress-induced impairment of executive function appears to be independent of affective or motivational factors related to executive function (Shields et al., 2024), suggesting an impairment of control itself.

A stress-induced impairment of executive function may mechanistically reduce healthy food choices. As described above, executive function is thought to be important in healthy food choices (Shields et al., 2021; Yang et al., 2019; Yang et al., 2018; Yang et al., 2019; Yang et al., 2020), perhaps via maintaining activation of long-term goals (e.g., being healthy) despite external distractions (Dassen et al., 2018; Houben et al., 2016; Whitelock et al., 2018). Indeed, interventions that improve executive functioning lead to healthier food choices (Yang et al., 2019), suggesting that stress, via impairing executive functioning, may decrease the number of healthy food choices that individuals make.

#### 3.2.2. Stress and negative affect

Stress increases negative affect (Denson et al., 2009; Kelly et al., 2008; Wiemers et al., 2013). People are poor self-reporters of stress—people will say they are stressed when they are not, and people will not say they are stressed when they actually are (e.g., Hunter & Shields, 2024; Moons & Shields, 2015; Shields et al., 2020; Shields et al., 2016)—but the enduring stress-induced increase in negative affect is observable across methods used for measuring negative affect (e.g., objective displays of affect, speech patterns, physiological changes, and self-reports; Blasberg et al., 2023; Buchanan et al., 2014; Mayo & Heilig, 2019; Richer et al., 2024). Similarly, stress alters neural activity in regions involved in negative affect, such as the amygdala (Henckens et al., 2012; McGaugh, 2015; Shields et al., 2021; van Stegeren et al., 2010). In short, an overwhelming quantity of research has shown that stress increases

negative affect.

A stress-induced enhancement of negative affect may mechanistically increase unhealthy food choices. Greater negative affect predicts greater use of maladaptive coping strategies, including “comfort eating” (Bemania et al., 2021; Chiu & Tomiyama, 2023; Dubé et al., 2005; et al., 2021; Klatzkin et al., 2019; Reese et al., 2016; Schüz et al., 2015; Sproesse et al., 2011; Veilleux et al., 2021; Wansink & Payne, 2007). These findings thus suggest that stress-induced negative affect may increase comfort eating, which typically means an increase in unhealthy food choices.

Because individuals who have the capability to regulate their behavior may nonetheless choose to engage in comfort eating if they see comfort eating as the most effective coping mechanism for stress-induced negative affect, then if decreasing unhealthy food choices is the goal, decreasing negative affect (i.e., removing the antecedent and proximal cause of stress eating) may thus be more beneficial than improving executive functioning. However, executive function and negative affect are not the only processes underlying self-regulation that stress influences. We now turn to a third such process.

#### 3.2.3. Stress and reward processing

Reward processing contributes in complex ways to self-regulation (Shields et al., 2017). For example, both blunted and heightened reward sensitivity can impair self-regulation, depending upon whether the rewarding stimulus is goal-relevant or not (Elliott et al., 2023; Goto & Kusumi, 2013; Shields et al., 2017). Therefore, unlike the above, the contribution of reward processes to self-regulatory success is nuanced, and the influence of stress on reward processes may not influence food choices in *a priori* expected ways. The influences of stress on reward processes are also complex and nuanced. However, there are some general trends within these effects.

As described above, stress alters reward processing in multiple ways (Burani et al., 2022; Lamontagne et al., 2018; Lataster et al., 2011; Mather & Lighthall, 2012; Oei et al., 2014; Pegg et al., 2019; Shafiei et al., 2012; Trainor, 2011; Voulgaropoulou et al., 2022; Zareyan et al., 2020). For example, stress alters neural activity in reward-related regions, such as the striatum, during reward anticipation and receipt (Kumar et al., 2014). A full review of these nuanced effects is outside of the scope of the current review. However, a relatively consistent effect of stress is that, at both a behavioral level and a neural level, stress tends to blunt reward sensitivity (Burani et al., 2022; Burani et al., 2021; Zhang et al., 2020).

Importantly, the substance use literature suggests that blunted reward sensitivity can contribute to greater consumption of highly pleasurable substances in order to obtain feelings of reward (Heitzeg et al., 2015; Joyner et al., 2019; Luijten et al., 2017). It is tempting to speculate, then, that stress, via blunted reward sensitivity, may increase unhealthy food choices. Such speculation would certainly be consistent with findings that abstinent individuals with substance use disorders are at high risk of excessive weight gain during recovery (Gottfredson & Sokol, 2019; Krotter et al., 2024) and findings that individuals are at high risk of substance use initiation following bariatric surgery (Li & Wu, 2016). However, the nuanced role of reward processing within self-regulation (e.g., finding the completion of goal-directed actions to be rewarding can facilitate self-regulation without the need to exert control, whereas exerting self-control can heighten reward sensitivity for goal-incongruent rewards and impair subsequent self-regulation; Kelley et al., 2019), coupled with the nuanced effects of stress on reward processing (e.g., amplifying sensitivity during reward anticipation while blunting sensitivity to obtained rewards; Kumar et al., 2014), make inferences related to this potential mechanism through which stress may influence food choices more tenuous than inferences made through the mechanisms described above.

In short, stress influences multiple processes involved in self-regulation, and it most consistently does so in such a way that would be expected to confer a pattern of fewer healthy food choices and more

**Table 1**  
Studies from the systematic review that met our inclusion criteria.

Refs.	N	Design	% Women	Must Ps. eat post-manip.?	Food given for a reward or thanks?	Was a plausible cover story told so Ps. did not know eating was the outcome?	Stressor	Effect size $d$ , unhealthy food eaten	Effect size $d$ , healthy food eaten	Effect size $d$ , total consumed
Clauss & Byrd-Craven, 2019 <sup>a</sup>	168	Between	54	No	No	Yes	Multiple	3.28	-0.34	-
Chaput et al., 2008	14	Within	100	No	No	No	CoABS	-	-	0.99
Zellner et al., 2006	34	Between	100	No	Yes	Maybe	CoABS	0.75	-0.70	-
Siervo et al., 2018	72	Between	0	No	No	No	CoABS	0.68	-	0.54
Lyu & Jackson, 2016	44	Between	100	No	No	Yes	CPT	0.65	-	-
Kistenmacher et al., 2018	14	Within	0	No	Yes	Maybe	TSST	-	-	0.50
Hitze et al., 2010 <sup>b</sup>	10	Within	0	No	No	No	TSST	0.42	-	-
Born et al., 2010	9	Within	100	Yes	No	No	CoABS	-	-	0.38
Wingenfeld et al., 2017	143	Within	100	No	No	Yes	TSST	-	-	0.35
Rutters et al., 2009	129	Within	50	No	No	No	CoABS	0.14	-	0.18
Masih et al., 2019	25	Within	76	No	Yes	Maybe	TSST	-	-	0.10
Lemmens et al., 2011	42	Within	62	Yes	No	No	CoABS	-	-	0.08
Petrowski et al., 2014	31	Within	45	Yes	No	No	TSST	0.07	-	-
Herhaus et al., 2018	56	Within	54	Yes	No	No	TSST	0.05	-	-0.03
Hamm et al., 2021	29	Within	100	Yes	No	No	TSST	0.02	-	-
Lemmens et al., 2011	38	Within	50	Yes	No	No	TSST	-	-	0.01
Lattimore & Caswell, 2004	40	Within	100	Yes	No	No	Multiple	-	-	-0.02
Herhaus & Petrowski, 2021 <sup>c</sup>	50	Within	48	Unclear <sup>d</sup>	No	No	TSST	-0.06	-	-0.07
Herhaus et al., 2020 <sup>c</sup>	72	Within	50	Yes	No	No	TSST	-0.09	-	-0.06
Geliebter et al., 2012	20	Within	100	Yes	No	No	CPT	-	-	-0.08
Klatzkin et al., 2024	26	Within	100	Yes	No	No	TSST	-	-	-0.11
Raspopow et al., 2010	65	Between	100	Yes	No	No	TSST	-	-	-0.16
Lustermans et al., 2024	110	Between	100	Yes	No	No	TSST	-	-	-0.24
Zellner et al., 2007	36	Between	0	No	Yes	Maybe	CoABS	-0.82	-0.26	-

Ps. = Participants; CoABS = Cognitive or Achievement-Based Stressor; CPT = Cold Pressor Task; TSST = Trier Social Stress Test. Effect sizes reported are Cohen's  $d$ . Higher values indicate greater consumption under stress. Studies are arranged in descending order of effect size when averaged across the three columns.

<sup>a</sup> Effect size calculated using their dataset linked to their article.

<sup>b</sup> Effect size from means and  $SEs$  of sweet foods in their Table 2.

<sup>c</sup> These two studies may contain some of the same participants.

<sup>d</sup> Few details are given, but this seems to follow the protocol of the Herhaus et al., 2018, study that required participants to eat.

unhealthy food choices. With these effects on self-regulation in mind, we now turn to other pathways through which stress may influence food choices, such as its effects on taste perception, neural responses to food, and appetite-related hormones.

### 3.3. Stress influences perceptual, hormonal, and neurobiological dynamics underlying eating

In addition to the self-regulatory processes underlying food choices, stress also influences dynamics directly related to eating. For example, stress increases sensitivity to sugar and salt, lowering the minimum detectable level for tasting a food as sweet or salty (Ileri-Gurel et al., 2013; Sawai et al., 2019; though see Al'absi et al., 2012). Interestingly,

at least one study has further found that a lower minimum detectable salt threshold is related to higher BMI, suggesting—speculatively—that one potential mechanism linking chronic stress to excess weight may, in part, occur through this pathway (Ileri-Gurel et al., 2013). These perceptual changes are consistent with other work showing that stress influences lower-level perception-related processes (Miller et al., 2015), representing a relatively direct pathway through which stress may alter food choices.

Stress also influences food-related activity in the striatum—a reward-related region—and the amygdala (Born et al., 2010; Nie et al., 2024; Rudenga et al., 2013). Without reverse inference, it is impossible to know whether these neurobiological effects are more related to the subjective experience of eating itself (i.e., perceptual) or to

self-regulation. Regardless, these findings clearly illustrate that stress influences the neurobiology of eating.

Another way in which stress influences dynamics underlying eating is that stress decreases the extent to which satiation influences eating (Hartogsveld et al., 2020; Quaedflieg et al., 2019; Schwabe & Wolf, 2011; Schwabe et al., 2011; Schwabe et al., 2012; Smeets et al., 2019; Soares et al., 2012; van Ruitenbeek et al., 2021). This finding is somewhat akin to continuing to snack on an open bag of chips in front of you; when satiated, we naturally decrease our choices to consume palatable food, but this influence of satiation is reduced under stress (Pool et al., 2015). Satiation does not lead to devaluation under stress in Pavlovian conditioning paradigms that require eating like satiation does under nonstressful conditions (Pool et al., 2015; Schwabe & Wolf, 2012). Put simply, when stressed, we are less likely to stop mindlessly eating even when we are full.

Perhaps underpinning some of the effects described above, stress may influence concentrations of the appetite-related hormones leptin and ghrelin. Although some well-powered studies with strong methods from well-established laboratories with a number of published prior studies that used stress manipulations successfully have not found such effects (e.g., Tomiyama et al., 2012), some work has found that stress decreases leptin concentrations and increases ghrelin concentrations (e.g., Bouillon-Minois et al., 2021; Bouillon-Minois et al., 2021). Because leptin suppresses appetite and ghrelin stimulates it (Chiu & Tomiyama, 2023; Tomiyama, 2019), if stress truly does decrease leptin and increase ghrelin, this would suggest that stress decreases the influence of satiation on eating, and increases feelings of hunger, through direct hormonal routes.

Finally, although not an internal mechanism, we briefly note that—as described in the economics section above—many forms of stress (e.g., financial) directly limit or influence the options available for food choices and thus affect choices and consumption via limited resources (Brinkman et al., 2010; Holm et al., 2020; Loibl et al., 2022).

The evidence reviewed thus far supports a model wherein stress should increase consumption of hyperpalatable foods but decrease consumption of healthy foods via modulation of multiple processes (e.g., diminished self-regulation, direct modulation of taste sensitivity, hormonal modulation of appetite, etc.). We now turn to the experimental literature that has tested whether—and if so, how—stress influences food choices.

### 3.4. Stress affects food choices: randomized experiments

The field of economics has become increasingly experimental, often requiring laboratory and field experiments to explore decision-making (Buckert et al., 2017; Buser et al., 2017; Cahliková & Cingl, 2017; Cetolin et al., 2020; Veszteg et al., 2021). However, our review of the literature suggests that the studies that employ validated acute stress manipulation, such as those that assess physiological responses such as cortisol reactivity, are still predominantly situated within the psychoneuroendocrinology literature. This is likely because inducing and verifying acute stress responses requires meeting specific hormonal and systemic physiological criteria (e.g., activation of the HPA axis), historically the domain of psychology, neuroscience, and endocrinology. Although the experimental studies we review in this section are interdisciplinary, we group them here due to their shared reliance on these physiological stress protocols, which align more closely with psychoneuroendocrinological methodologies than traditional economic experiments. This organization is intended to reflect the methodological

**Table 2**

Studies from the systematic review that would have met inclusion criteria but either failed to induce stress or did not report the effect of stress on food choices.

Refs.	N	Design	% Women	Stressor	Reason for exclusion
Appelhans et al., 2010	34	Within	100	TSST	Unreported effect of stress on eating
Chang et al., 2022	28	Within	50	MAST	Unable to calculate effect size <sup>b</sup>
Klatzkin et al., 2018	35	Within	100	TSST	Unreported effect of stress on eating
Klatzkin et al., 2019	43	Within	100	TSST	Failed manipulation
Laessle & Schulz, 2009 <sup>a</sup>	48	Within	89.5	TSST	Failed manipulation
Leow et al., 2021	23	Within	43	TSST	Unable to calculate effect size <sup>c</sup>
Schmalbach et al., 2021	38	Within	100	TSST	Failed manipulation
Schulz & Laessle, 2012 <sup>a</sup>	71	Within	100	TSST	Failed manipulation
Vitt et al., 2021	196	Between	100	CoABS	Failed manipulation

MAST = Maastricht Acute Stress Test; TSST = Trier Social Stress Test; CoABS = Cognitive or Achievement-Based Stressor.

<sup>a</sup> These two studies may contain some of the same participants.

<sup>b</sup> Reported statistics were median and IQR, with nonsignificant *p* values for tests of stress differences. Depending upon the outcome, participants in the combined stress conditions appeared to slightly eat more (e.g., kJ intake from unhealthy foods, relative energy intake) or slightly less (e.g., % intake from unhealthy foods, total energy intake).

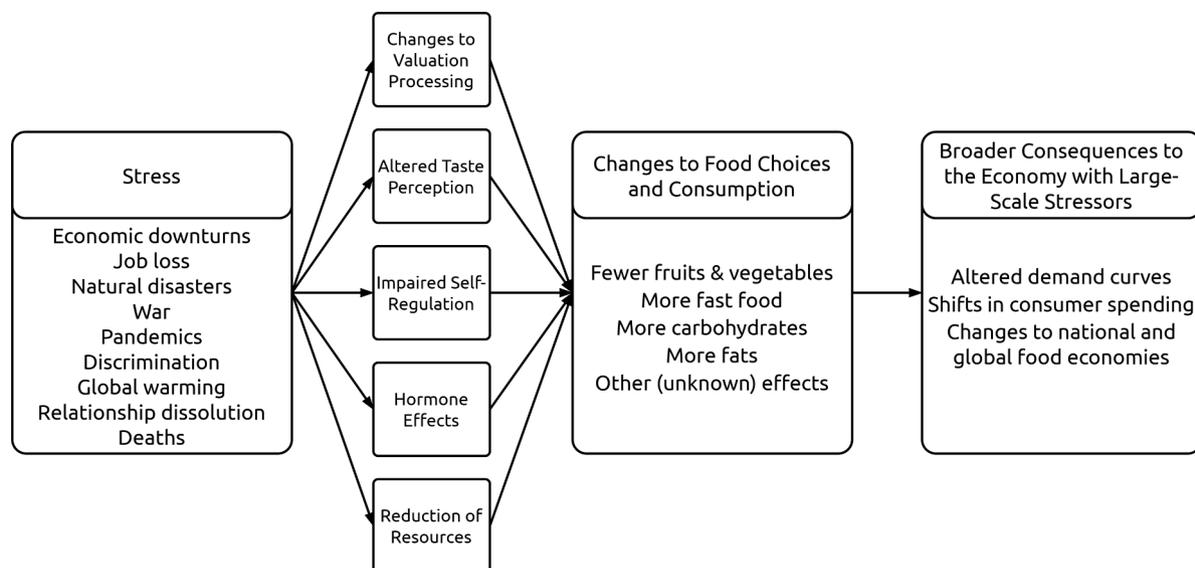
<sup>c</sup> Reported statistics were solely nonsignificant *p* values for group and visit effects. No directional inferences could be made, even numerically, from the reported data.

convergence of this body of literature rather than to reinforce disciplinary boundaries.

To comprehensively describe prior work on the causal effects of stress on real food choices, we conducted a systematic review of PubMed and Web of Science for experimental manipulations of stress with actual food intake as the outcome.<sup>1</sup> All studies that used a validated stress manipulation (validated in that study or another) in adults, with random assignment to stress or control conditions and subsequent assessment of actual food consumption, are presented in Table 1. Studies with a fixed order of stress and control conditions for within-subjects designs (i.e., not crossover designs; for example, the control condition always came first) were not included (e.g., Van Strien et al., 2013). Studies with a known failed manipulation (indicating that its manipulation was more appropriately described as negative affect than stress) (*n* = 5), (Klatzkin et al., 2019; Laessle & Schulz, 2009; Schmalbach et al., 2021; Schulz & Laessle, 2012; Vitt et al., 2021) studies that met review inclusion criteria but did not report whether stress as a condition influenced any of the food choice outcomes or other forms of eating behavior (*n* = 2), (Appelhans et al., 2010; Klatzkin et al., 2018) and studies that did not report sufficient information to compute effect sizes (*n* = 2), (Chang et al., 2022; Leow et al., 2021) are reported in Table 2.

Across all studies that experimentally manipulated stress using a validated stress-inducing paradigm and subsequently assessed choices for food that was consumed, stress generally increases the extent to which individuals choose unhealthy food options (see Table 1). Notably, larger studies were more likely to report significant, unmoderated increases in food consumption under stress (see Table 1), which is

<sup>1</sup> String: ("acute stress" OR "stress induction" OR "Trier Social Stress Test" OR "cold pressor" OR "stress manipulation") AND (("food choice" OR "food choices" OR "eating behavior" OR "food consumption" OR "eating task" OR "food was administered" OR "snacks") OR ("macronutrient" OR "macronutrients") AND ("carbohydrates" OR "fats" OR "saturated fat" OR "protein"))



**Fig. 1.** Simplistic model of how stress can lead to changes in food choices and consumption, which, in the case of large-scale stressors, would presumably further influence broader food economies. Although this figure emphasizes the economic consequences of stress-induced dietary changes, we view these as second-order effects that stem from more direct and well-established public health outcomes such as increased obesity, diabetes, and other chronic diseases.

consistent with a recent meta-analysis of studies related to stress and eating (Hill et al., 2022). This meta-analysis, which had broader inclusion criteria than our review (e.g., pre-post noncounterbalanced designs), found that stress decreased consumption of healthy foods and increased consumption of unhealthy foods (Hill et al., 2022).

A closer look at Table 1 highlights some interesting trends. First, an effect of stress on food choices or eating was enormously more likely to occur when participants were not required to eat any of the target foods: Every study (i.e., all seven) that did not require participants to eat nor tell participants that the food was offered as a reward found a significant effect of stress on food choices or eating behavior (Chaput et al., 2008; Clauss & Byrd-Craven, 2019; Hitze et al., 2010; Lyu & Jackson, 2016; Rutters et al., 2009; Siervo et al., 2018; Wingenfeld et al., 2017). Notably, such designs that use deception and avoid presenting the food as a reward or thanks help to reduce demand characteristics for eating, which may be necessary to observe the effects of stress on eating behavior. Six other studies (Chang et al., 2022; Kistenmacher et al., 2018; Leow et al., 2021; Masih et al., 2019; Van Strien et al., 2013; Zellner et al., 2006; Zellner et al., 2007) told participants that they earned the food or that it was otherwise compensation (e.g., in place of lunch during lunchtime) or a token of appreciation. Of these studies, two found that stress increased consumption of high-fat and/or high-fat, high-sugar foods (Kistenmacher et al., 2018; Zellner et al., 2006); three found no significant differences but numeric increases in unhealthy food consumption under stress (Chang et al., 2022; Leow et al., 2021; Masih et al., 2019); and one found less unhealthy food consumption under stress (Zellner et al., 2007). Of the 13 studies that required participants to eat at least some of the target food(s), none found a significant main effect of stress (Born et al., 2010; Geliebter et al., 2012; Hamm et al., 2021; Herhaus & Petrowski, 2021; Herhaus et al., 2020; Herhaus et al., 2018; Klatzkin et al., 2024; Lattimore & Caswell, 2004; Lemmens et al., 2011; Lemmens et al., 2011; Lusterms et al., 2024; Petrowski et al., 2014; Raspopow et al., 2010). Requiring participants to eat some of the food, or telling participants that the food is a reward, earned, or in some way compensation, may thus diminish what may otherwise be a robust effect of stress on eating behavior and food consumption.

Second, studies with stressors that stressed participants more through cognitive demand than social evaluation tended to be more likely to produce an increase in eating (stressor primarily cognitive: 6/10 found stress increased unhealthy food consumption; stressor

primarily social or pain: 4/18 found stress increased unhealthy food consumption; some studies included each stressor type separately), though it should be noted that the overwhelming majority of those studies used the Trier Social Stress Test, which requires participants to change contexts (i.e., enter a different room and give the speech to evaluators seen only during the stressor—thus producing a spatial and social context unique to the stressor) between stress and the other parts of the study (Kirschbaum et al., 1993). Such a context shift appears to matter more than stressor type in the effects of stress on some cognitive processes (Riddell et al., 2023; Sazma et al., 2019; Shields et al., 2017), so future research should aim to clarify whether either a context shift or the stressor type is more influential in contributing to increases in unhealthy food consumption post-stress-manipulation.

Third, interestingly, no study found that stress increased consumption of healthy foods—all studies found that stress decreased consumption of healthy foods relative to the control condition, at least to a very small extent. It is thus possible that the effect of stress on food choices is more consistent in decreasing consumption of healthy foods. We note, however, that fewer studies reported the effects of stress on fruit or vegetable consumption (i.e., healthy foods) than those reporting the effects of stress on unhealthy foods (i.e., high-sugar and/or high-fat foods) or on all foods.

In terms of macronutrients, the majority of studies that found macronutrient differences found that stress increased consumption of carbohydrates and/or fats (Chaput et al., 2008; Clauss & Byrd-Craven, 2019; Hitze et al., 2010; Kistenmacher et al., 2018; Lyu & Jackson, 2016; Rutters et al., 2009; Siervo et al., 2018; Zellner et al., 2006), stress typically had no effect on protein consumption (though see Lemmens et al., 2011). These findings are consistent with findings on stress eating and stress-related hypothalamic-pituitary-adrenal (HPA) axis activity (Chiu & Tomiyama, 2023; Finch & Tomiyama, 2014; Tomiyama, 2019).

Before leaving this section, we believe that it is important to note two major limitations. The first is that publication bias may have skewed or altered our inferences. Null results are less likely to be published than significant results (Shields et al., 2016; Shields et al., 2017), entailing that positive or significant negative findings are likely overrepresented in our review. Although many of the studies represented in our review did report null effects (e.g., Herhaus & Petrowski, 2021; Herhaus et al., 2018), the specific conditions and categories of studies we highlighted as particularly influential may have been disproportionately likely—for some unknown reason—to have unpublished null results. We thus

caution against strong inferences related to the most important conditions and highlight general, albeit weak, effects of stress on eating behavior within these studies.

The second limitation of this review section relates to its generalizability. The experimental literature we reviewed within this section focused on acute stress, because this is the only form of stress we can ethically experimentally manipulate—and thus infer cause and effect—within humans. However, the literature we reviewed within economics focuses on chronic stressors and their associations with food consumption on a broader scale. The consistency between these literatures in findings relating stress to food consumption suggests to us that the stressors we reviewed within the economics section may, perhaps, exert similar effects to those exerted by the acute stress manipulations. We also note that a robust nonhuman animal literature exists that has manipulated chronic stress and found that it contributes to increased food consumption and obesity (e.g., Bartolomucci et al., 2004; Bartolomucci et al., 2009; Kuo et al., 2007; Patterson et al., 2013; Rowland & Antelman, 1976), similar to the acute stress literature in humans.

#### 4. An integrative model: from stress to population-level changes in food choices

Taken together, the literature supports a framework whereby stress, via multiple pathways (e.g., self-regulation, taste sensitivity and perception, valuation processing, hormonal effects, and a direct reduction of resources, among potential other pathways), influences food choices, collectively decreasing healthy food choices and increasing hyperpalatable (i.e., unhealthy; high-fat and high-sugar and/or high-salt) food choices.

These dynamics are illustrated in Fig. 1, which illustrates the connections between stressors, changes in food choices, and broader economic consequences. Stress, triggered by exogenous factors like economic downturns, job loss, pandemics, and global events such as war or natural disasters, can influence food consumption through several psychological and physiological mechanisms. Stress-induced alterations to valuation processing, taste perception, and self-regulation impair decision-making and contribute to increased consumption of hyperpalatable, unhealthy foods. Hormonal effects and a reduction of available resources further exacerbate these tendencies, leading to fewer healthy food choices and more consumption of fast food, carbohydrates, and fats.

From an economic standpoint, these consumption pattern changes can have significant, policy-relevant consequences for national and global agri-food systems. As consumer preferences shift toward unhealthy and processed foods during times of stress, demand for these products rises, altering market dynamics. This may result in a reshaping of demand curves, driving up prices for certain food categories while reducing demand for healthier options. Additionally, large-scale stressors, such as pandemics or natural disasters, could lead to disruptions in food supply chains, further amplifying these economic shifts. These factors affect food producers and retailers, creating critical implications for public health policy and labor productivity, as increased consumption of unhealthy foods could contribute to rising healthcare costs and economic inefficiency.

##### 4.1. Testable predictions

The relationships between stress and food choice lead to at least four testable predictions. First, although large-scale stressful events (e.g., economic recessions or pandemics) may affect food choices through a variety of pathways unrelated to stress itself (e.g., closures of nearby grocery stores, changes in income, etc.), if our model is correct, then during periods of large-scale stress (e.g., economic recessions or pandemics), we would anticipate a measurable increase in consumer demand for hyperpalatable and fast foods, coupled with a decrease in the consumption of healthier food options like fruits and vegetables. That is,

our model suggests that shifts in grocery purchases and/or consumer interest should have occurred during prior nationwide stressors, and that they should occur following future nationwide stressors. Some of these shifts have been documented (e.g., in response to COVID; Smith et al., 2021), but others (e.g., following the September 11 attacks, the Cuban missile crisis, or the challenger explosion) have not been examined to our knowledge. Our model suggests that a study examining such stressful national events would likely observe corresponding shifts in grocery purchases. It should be noted, however, that these events were more than just stressors, and many nonstress mechanisms (e.g., changes to financial resources, food availability, etc.) may explain these findings.

Second, the stress-induced demand increase for fast foods and processed snacks should lead to a price increase relative to healthier options. This prediction can be tested by analyzing price trends in food categories over time in response to major stress events (Nekmahmud, 2024).

Third, our model further suggests that nationwide stressors should increase the rate of overweight and obesity. Although outside the context of our model, this prediction has been examined in response to the COVID-19 pandemic and borne out in the expected direction, such that the pandemic was associated with a rapid increase in overweight and obesity in both children and adults (Anderson et al., 2023; Restrepo, 2022; Wuerdeman et al., 2023), despite increases in intense exercise (Restrepo, 2022; Robertson et al., 2022). It should be noted, however, that—just as in the examples above—the COVID-19 pandemic was more than a stressor alone, and many other mechanisms (e.g., closure of food outlets) may explain this finding.

Finally, and related to the above, we anticipate that increased consumption of unhealthy foods during periods of stress will lead to higher healthcare costs and reduced workforce productivity due to a rise in diet-related diseases, such as heart disease and diabetes. To the extent that findings related to alcohol consumption can be used to make an analogical argument about unhealthy food consumption, we note that the same pattern is observed with alcohol consumption: Increased alcohol consumption can lead to reduced macroeconomic growth (Cesur & Kelly, 2014). This prediction can be tested by comparing healthcare expenditure and productivity metrics before and after large-scale stress events. We note that this prediction being borne out by data would not show strong evidence for our model—stress can influence health and productivity through numerous other health behaviors. Instead, we note that this is simply a prediction of our model, and one which we would expect to find support in such data.

##### 4.2. Contextual considerations

In addition to influencing consumption patterns and price responsiveness, stress likely alters more foundational aspects of economic decision-making, such as time preferences and risk perceptions. For example, heightened stress may lead individuals to discount future health consequences more steeply, thereby increasing the appeal of immediate gratification through hyperpalatable food consumption. This dynamic validates the economic notion of stress in that a stressful event shifts what people consume because of how they evaluate tradeoffs, particularly between short-term rewards and long-term health.

Moreover, the behavioral consequences of stress are unlikely to operate in a vacuum. Firms in sectors such as food retail may recognize and respond to aggregate shifts in consumer stress. For instance, food manufacturers may adjust product formulations, packaging, or marketing strategies to align with increased demand for convenience or indulgence during periods of widespread stress. They might also simply reduce the number of choice offerings to reduce the implied stress within the consumer decision-making process (Malone & Lusk, 2017). Retailers might alter pricing or promotional marketing channel strategies to target stressed consumers, either intentionally or inadvertently (Harris-Lagoudakis, 2022). These potential firm-level responses create feedback loops that may reinforce or amplify stress-related dietary

patterns. Although a full treatment of these structural dynamics is beyond the scope of this review, acknowledging them helps position stress as a behavioral variable with implications for supply-side behavior and regulatory policy.

## 5. Future directions

Although economics has become increasingly experimental, we separate future directions for psychoneuroendocrinology and economics to highlight discipline-specific gaps and opportunities. Psychologists and neuroscientists have developed detailed models of stress and self-regulation, while economists have built robust models of consumption but less frequently employ biologically validated stress paradigms. This structure is intended to clarify areas for integration and encourage future research that combines the strengths of both disciplines.

### 5.1. Future directions for psychoneuroendocrinology

Although much is known about how stress influences food choices and consumption, much is still unclear. At the level of specificity of food choices, experimental studies ranged from one food offered to participants (Lyu & Jackson, 2016) to a large buffet (Hitze et al., 2010). Although many studies have attempted some level of granularity in understanding how stress influences food choices (e.g., Hitze et al., 2010), the type of granularity differing across studies makes current synthesis difficult. For example, one study found that stress increased choices for crispy foods (Born et al., 2010). Many studies have found that stress increased consumption of high-fat foods (e.g., Chaput et al., 2008; Clauss & Byrd-Craven, 2019; Kistenmacher et al., 2018; Zellner et al., 2006); however, many of them used crispy high-fat items, such as potato chips, as their high-fat food, which couples fat with crispiness, thus making it difficult to determine if stress influenced preference for high-fat foods or crispy foods. This example serves to illustrate that future work might benefit from considering greater granularity when studying these effects.

In terms of confounds, the overwhelming majority of experimental studies of stress and food choices have used within-subjects, crossover designs, and they usually used some form of deception about why the food was presented (e.g., to decrease experimenter demands or the Hawthorne effect). Such a form of deception appears to be important to observing stress effects on food choices and consumption (e.g., see above). However, using a within-subjects, crossover design makes deception related to the food consumption period—which is stable, unlike stress, and which follows stress—much less believable, and almost no within-subjects, crossover design reported testing whether stress condition interacted with session order (though two did, and both observed order effects; see Chang et al., 2022; Hamm et al., 2021). Such tests are crucial when using within-subjects, crossover designs, because strong order effects can mask or alter manipulation effects (Charness et al., 2012; Greenwald, 1976; Poulton, 1973). Future work on the topic should thus report this test whenever a within-subjects, crossover design is used. It is likely that any effect of stress on food choices that relies at least in part on deception—critical for avoiding demand characteristics related to eating—will be observable only during the first lab visit.

From the perspective of mechanism, very little work to date has examined mediating mechanisms that may underpin the effects of stress on food consumption (e.g., self-regulation, reward, or more direct taste perception or hormonal effects) within studies of stress and food consumption. Future work on the topic may benefit from examining mediating mechanisms to identify points of intervention that could work at the level of public policy.

From the perspective of moderators, a few studies have suggested that there may be different effects of stress on food choice depending upon stressor type. For example, an achievement-related stressor had a greater effect on increasing consumption of hyperpalatable foods in men than did a rejection-related stressor, whereas the opposite effect was

true for women (Clauss & Byrd-Craven, 2019). Similarly, another study suggested that a cognitively demanding task may be more likely to increase hyperpalatable food consumption than pain (Lattimore and Caswell (2004). Across studies, those that used the Trier Social Stress Test were generally less likely to observe an effect of stress on food choices or consumption. Taken together, these findings suggest that the type of stress on experiences may differentially contribute to changes in food choices or consumption, which highlights the importance of more research on this topic. Some forms of stress, such as heat stress (Goodin et al., 2012; Veilleux et al., 2018)—which will presumably become increasingly more relevant as time goes on—have not been studied in relation to food choices at all. Future research that aims to determine whether other, less-studied forms of stress or stressor inductions do or do not influence food choices thus seems timely and useful.

### 5.2. Future directions for economics

This review emphasizes the importance of integrating neuropsychological insights into economic decision-making models, particularly in the context of stress and food choices. While traditional economic models often focus on rational decision-making and market dynamics, the highlighted findings indicate that stress and its neuropsychological effects significantly influence consumer behavior, particularly food consumption patterns. Specifically, stress alters self-regulation, taste perception, and hormonal responses, leading to a preference for hyperpalatable, unhealthy food options over healthier alternatives. Future research should develop more biologically consistent economic models that account for the neuropsychological mechanisms influencing food choices. By incorporating variables such as stress-induced changes in cognitive function and decision-making into these models, economists might gain a more robust understanding of consumer behavior under stress. This approach can improve the accuracy of predictions related to food demand, pricing, and public health outcomes, particularly during large-scale stress events such as economic downturns or global pandemics. In turn, these models might inform more effective policy interventions aimed at improving public health and food security, ensuring that both psychological and economic factors are considered when addressing issues such as obesity, food insecurity, and diet-related diseases.

As another future direction, much of the empirical evidence on this topic to date comes from studies that rely on laboratory acute stress manipulations (e.g., the Trier Social Stress Test). These studies have generally, though not entirely, been conducted by groups labs that lack formal or extensive training in economics. Although these studies offer strong internal validity and allow for controlled measurement of stress responses, they may not fully capture the prolonged, diffuse stressors experienced in many real-world economic contexts, such as persistent financial hardship or food insecurity. The behavioral and biological effects of chronic economic stressors may differ meaningfully from those induced by laboratory acute stress manipulations, potentially producing longer-term or otherwise different shifts in dietary patterns, regulatory function, and risk perception. Future research should aim to clarify how chronic economic stressors influences food choice behavior and explore experimental designs that better mimic economic stress environments.

In addition to informing consumer-level behavioral models in the academic literature, the insights reviewed here also suggest that some food-related policy design and implementation approaches are likely to be more effective. Interventions such as nudges may benefit from incorporating behavioral design tools that reframe food choices in high-stress environments like schools, clinics, or workplaces. These environments represent key points of intervention where the choice architecture could be adjusted to promote healthier decisions when self-regulation is compromised. At the same time, policymakers should remain cautious about unintended consequences; for example, restricting access to hyperpalatable foods without addressing the underlying stressors may inadvertently trigger compensatory behaviors that

**Table 3**

Knowns and unknowns related to stress and food choices in the literatures surveyed.

Change in Food Choice or Consumption Related to Stress?	Evidence From PNE	Evidence From Economics
Fruit	↓/-	↓
Vegetable	↓	↓
Meat	-	?
Nuts	?	?
Dairy	?	?
Simple carbohydrates	↑	↑
Complex carbohydrates	↑?	↑
Hyperpalatable foods	↑	↑
“Fast food”	?	↑↓
Different Food Choice Effects by Stressor Type?	Suggestive	Suggestive

Note: PNE = psychoneuroendocrinology.

undermine the intended outcomes. Expanding the policy lens to consider both behavioral constraints and environmental complexity will be crucial for crafting effective, realistic, and ethically sound interventions.

### 5.3. Future directions for both disciplines

Both psychoneuroendocrinology and economics should be able to fruitfully extend work in this domain by clarifying effects and associations. In the table below (Table 3), we summarize what is known, and what is unknown, in the field of psychoneuroendocrinology and economics related to stress and food choices.

Importantly, the effects reviewed within this manuscript presumably do not apply to all populations equally. A number of demographic or other differences that exist across populations are known to moderate at least some effects of stress. Those factors include sex (Shields et al., 2025), age (Knauff et al., 2025), social support (Moseley et al., 2021), socioeconomic status (Deer et al., 2021; Hunter & Shields, 2022), and more. The existence of these moderating factors thus implies a non-negligible amount of heterogeneity within the effects of stress, and presumably within the effects of stress on food choices and consumption. Developing an understanding of the moderators of the effects of stress on food choices could thus be a fruitful avenue for future research aimed at developing a comprehensive model of the effects of stress on food choices food consumption.

## 6. Summary and conclusion

As both stress and excess weight continue to increase within many Western nations, understanding the dynamics among them continues to grow in importance. In this review, we have highlighted the effects of stress on food choices and consumption, surveying mechanisms at the psychological level and describing expected and observed population-level dynamics. Despite the advances made in this area, much is still unknown, and future research should presumably aim to take a more granular approach to these effects—both in terms of stressor type and in terms of foods examined. Nonetheless, some general, albeit tentative, conclusions can be made. In particular, stress, perhaps via its effects on taste sensitivity, hormones, learning, self-regulation and related processes, and resources, among other potential factors, appears—at a meta-analytic level, despite heterogeneity in the literature and with weak effects—to influence food choices and consumption, appearing to lead to less consumption of healthy foods and an increase in consumption of hyperpalatable foods. At a population level, such findings could have important consequences. These findings thus underscore the importance of stress in public health dynamics related to overweight and

obesity, and they further highlight an important factor that influences food choices and consumption but has largely gone unconsidered in the field of economics.

Although the relationship between stress and unhealthy food choices has been well-documented in the psychological and biomedical sciences, the purpose of this review was to pull those findings out of isolation and into a broader, multidisciplinary research framework. As such, this paper aims to serve as a conceptual bridge between disciplines, particularly highlighting how the neurobiological mechanisms of stress, often overlooked in economic modeling, can inform a more effective and neuroscientifically informed understanding of consumer behavior. By synthesizing evidence from psychoneuroendocrinology and reframing it through the lens of behavioral economics, we hope to equip economists with a clearer framework for incorporating stress into models of food choice, policy design, and demand forecasting. This cross-disciplinary approach can improve the predictive and policy relevance of economic approaches to food behavior under stress.

### CRediT authorship contribution statement

**Grant S. Shields:** Writing – review & editing, Writing – original draft, Data curation, Conceptualization. **Trey Malone:** Writing – review & editing, Writing – original draft, Conceptualization.

### Data availability

Data and syntax are available in the link given in the Author Note.

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