

Eating Behaviors of People with Chronic Stress

Subjects: Public, Environmental & Occupational Health

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Psychological stressors frequently occur in modern society, and are associated with general anhedonic traits (inability to experience pleasure) and altered eating behavior. As eating behavior is largely motivated by a desire for pleasure, the Food Pleasure Scale (FPS) was introduced as a new research tool for investigating aspects of pleasure from food-related experiences.

Keywords: food pleasure ; stress ; appetite ; eating behavior

1. Stress

Despite the frequent use of the term 'stress' both in scientific and everyday speech, a standard definition of the condition is absent, and the term has been defined and redefined over decades ^{[1][2][3][4]}. A commonly used definition by Torres and Nowson defines stress as 'the generalised, non-specific response of the body to any factor that overwhelms, or threatens to overwhelm, the body's compensatory abilities to maintain homeostasis' ^[1]. To further understand the development of the scientific notion of stress, the term can be divided into the three sub-terms: 'stressor', 'stress response', and 'allostasis'.

A 'stressor' can be any kind of stimuli which is perceived as stressful by the individual. Thus, it can be anything from a specific working task to a traumatic life event, such as death in the near family. This also implies that all major life events can be deemed as stressful, even the positive events of getting married, having children, and going on vacations. Therefore, many different attempts have been made at defining the stressful events of life for the purpose of being able to measure the magnitude of stress and the adaption needed ^{[5][6][7]}. Nevertheless, there is still little agreement as to what defines a stressful event ^[7].

The 'stress response' is a term in stress research which finds its origin in the work of Lazarus and Folkman ^{[8][9]}, who focused on the appraisal of threats posed during life, and the ability to effectively 'cope' with these threats. From this perspective, stress occurs when an individual perceives an event as threatening or harmful, and simultaneously experiences having inadequate coping resources available ^{[4][5][6]}. If coping with the stressors is successful, the imbalance between the demands of the stressful situation and the resources needed is levelled out, and possible discontent and discomfort are reduced. Oppositely, if coping is not successful, the imbalance and discontentment will proceed and perhaps worsen, and can lead to both physiological and mental symptoms of stress, e.g., headaches, exhaustion, memory loss, or anxiety ^[4].

A central concept in relation to the stress response is that of 'allostasis', first introduced by Bruce McEwen ^{[5][9]}. Allostasis is the physiological process, by which the body responds to a stressor, adapts to that specific stressor, and thus, reaches homeostasis again. Thereby, allostasis is also the physiological and behavioral mechanisms that allow people to cope with challenges, stressors, or high demands, and once the challenge is over, shuts off and allows the body to return to a normal relaxed state. Furthermore, McEwen defines the term 'allostatic load' as 'the wear and tear that results from chronic overactivity or underactivity of allostatic systems' ^[9], which can have pathophysiologic consequences. Allostatic load can be relieved in many ways. Most often, people will turn to behavioral changes, such as the consumption of alcohol and tobacco, increased sleep, less physical activity, and perhaps, an unhealthy diet too ^{[3][7][8][9]}.

In biological stress research, the focus is often on the sympathetic-adrenal-medullary system (SAM) and the hypothalamic-pituitary-adrenal axis (HPA), as well as the cardiovascular, metabolic, and immune systems as allostatic systems, all of which protect the body from stressors ^{[3][6]}. In addition, it is important to understand and discriminate between chronic and acute stress, as the implications of allostasis are quite diverse for the two states of stress. In the event of acute stress, allostasis systems will activate as an appropriate and beneficial reaction, allowing the body to perform fast on a high level. This primarily happens through the SAM system, by the release of stress hormones, which will increase blood pressure and heart rate, expand the heart and muscular blood vessels, as well as accelerate the

metabolism of macronutrients for the release of energy [3][5][6][10]. In the case of chronic stress, a completely different effect of allostatic systems is seen. A prolonged exposure to stress has proven to cause a long and varied list of clinically defined diseases. These health effects are caused by activation of the HPA axis and alterations of health behavior and affective regulation, which can potentially result in diseases such as depression, cardiovascular diseases, diabetes, and infectious and neurodegenerative diseases [3][9][10][11].

2. Stress-Induced Eating and Altered Perception of Reward

The complex relationship between stress and eating behavior has long been acknowledged. Stress can cause irregular eating patterns, altered food behaviors and preferences, and is believed to cause alterations in the perception of pleasure from food [3][12][13]. Research has shown that being stressed can cause non-homeostatic hunger, as food consumption has proved to have calming capacities on the physiological and behavioral stress responses [3][12][13][14]. Oppositely, other studies report a reduced intake due to activation of the SAM system [2][15][16][17]. Thus, approximately 40–70% of people suffering from stress report an increase in food intake, whereas 30–60% report a reduction under stress [2][18]. Yet, the underlying psychobiological mechanisms that shape the direction of change are largely unidentified [17][19]. In the case of chronic stress, it is largely believed that the stress response is governed by an activation of the HPA axis, with a consequent rise in the secretion of glucocorticoids. Glucocorticoids directly and indirectly stimulate food intake, especially highly palatable foods, by activating secretion of the appetite hormones, insulin, leptin, and NPY [2][12]. At the same time, food intake can dampen the physiological stress response by deactivating the HPA axis [12][20]. This deactivation is very often credited to the hedonic effects of the food, as eating will activate neural substrates, such as dopamine, in a similar manner to drug abuse [12][21][22]. Dopamine functions in multiple ways; however, in the context of eating, it is first and foremost a neurotransmitter, which codes for pleasure and enhances the desire for food. Furthermore, dopamine supports the deactivation of the HPA axis [12][22]. Experienced pleasure from food may, therefore, be the main explanation for this comforting effect of food under stressful conditions. In this manner, food intake as a reaction to the physiological and behavioral stress response will concurrently stimulate the reward pathways, and thus, possibly lead to neurobiological adaptations which will promote stress-induced eating in the future too [23][24][25]. Thus, if eating is learned to be an effective coping strategy, then it is likely that highly palatable foods could become addictive, in the same manner as other highly hedonic substances (e.g., alcohol, drugs, or tobacco) [22][26][27].

Hedonia, or, in larger degree, its contradistinction, anhedonia, have been studied primarily as symptoms of mental illnesses such as depression, anxiety, and schizophrenia [22][28][29]. Anhedonia is defined as the lack of ability to perceive pleasure, and thus, the term anhedonic traits is frequently used to describe how anhedonia is expressed in the individual. Prior human studies commonly assume that relatively mild acute stressors, as well as chronic stressors, can lead to impairments in reward function, and thus, result in anhedonic symptoms [30][31]. In fact, animal studies have found that exposure to stressors induce anhedonic and depression-like behavior, as well as dysfunctions in the dopaminergic reward pathways [22][31]. In addition, it is believed that anhedonia is heterogeneous across different mental disorders, depending on which part of the reward pathway is involved [28]. For instance, in depression, anhedonia can be regarded as a transient state, whereas in schizophrenic patients, anhedonia reflects a trait-like characteristic [28]. Many different self-report scales have been constructed for measuring anhedonia, primarily in patients of mental diseases. Examples which have been widely used include the Chapman Physical and Social Anhedonia Scale, the Fawcett–Clark Pleasure Scale, and the Snaith–Hamilton Pleasure Scales [32][33][34]. Though these scales differ, what is common for all these scales is the focus on pleasure from the perspective of anhedonia, thereby only giving attention to the dysfunctional reward systems. Moreover, these scales address anhedonia from a general perspective with respect to food, which means that specific pleasure from food and food experiences is either addressed by a single question or not at all [28][35][36]. Recently, Andersen et al. (2021), at the Department of Food Science, Aarhus University and Department of Psychology, University of Chinese Academy of Sciences, developed a scale for solely evaluating pleasure from food-related experiences [36]. Thereby, it has become possible to investigate the specific relationship between pleasure perception and eating behavior in various consumer groups. Furthermore, this scale lays the foundation for a deeper understanding of why some consumers experience having issues in terms of keeping a healthy diet or making more sustainable food choices, in addition to aiding the development of strategies for alleviating these issues.

3. The Effect of Chronic Stress on Appetite, Meal Patterns, and Food Preferences

In general, the study showed that chronic stress influence meal patterns and specific food preferences, especially in relation to main and snack meals. Increases in sweet and salty snacks and caffeinated drinks, as well as decreases in the intake of fruit and vegetables, were found. More specifically, when investigating the effects of stress severity, it was found

that the 'High stress' group had a lower desire for food than the 'Moderate stress' group. This result was reflected in the changes of meal patterns, as the 'High stress' group had a larger decrease in intake of both breakfast and lunch, as well as snack meals and, particularly, fruits and vegetables. Like the 'High stress' group, the 'Moderate stress' group also showed a decrease of breakfast and lunch meals, but to a lesser degree. Additionally, more respondents suffering from 'Moderate stress' compared to 'High stress' demonstrated an increase in night meals and a tendency towards more post-dinner snacks. These results exemplify, in a discrete way, what the scientific literature has stated for years, namely, that when experiencing chronic stress, approximately 40–70% increase their food intake, whereas 30–60% reduce their intake [2][12][13]. Yet, the psychobiological mechanisms which shape the course of this change are largely unknown (which is why it has not been fully explored), as well as which factors account for these individual differences. Furthermore, several papers have reported that chronic stress is linked to a change in diet towards fewer main meals and higher intake of, especially, highly-palatable snack foods, mainly due to a continued activation of the HPA axis [12][37]. In line with these studies and a previous study on acute stress reporting an increase in the unconscious craving for high-fat sweet snack foods [38], the majority of respondents reported to have increased their intake of sweet snacks after being ill with stress, and a large group likewise reported to have increased their intake of salty snacks.

The food pleasure profile of this sample can be used to expand the understanding of this change in diet. As the most important aspect to food pleasure came from the sensory modalities of the food eaten, it makes sense that foods that have a strong sensory output are chosen more often. Snack foods are generally known to be highly satisfactory both in terms of sensory profile, as well in terms of well-being sensations, giving instant relief from the stress condition [12][37][39]. Conversely, main meals are often chosen and composed upon reasoning around nutrition; the social context, which requires product information; and, potentially, considerations around ethical values. These aspects around a meal require effort to fulfill. Thus, it seems logical that these meals are eaten less, and that these aspects provide pleasure to few people when mental resources (e.g., requirements to cognitive efforts) are low, such as those in the current stress conditions. It can be hypothesized that the same goes for fruit and vegetable intake. Again, this food group represents nutrition and health for many people, and making a healthy choice can seem insuperable under the influence of stress, especially if the condition is at a level which involves the activation of the HPA axis and secretion of glucocorticoids.

One would assume the 'High stress' group would follow the same change in eating pattern of reducing main meals while also increasing intake of snack foods, perhaps with an even more pronounced dietary change. However, as seen from the results, this group only follows the pattern in terms of main meals, and thus, not in the intake of snack meals. The severity of their condition can explain these results, as more severe stress does, to a larger degree, impose anhedonic traits, as it is often seen in people with depression and other mental disorders [22][28][40]. Anhedonic traits were also reflected in the results from the FPS, as the 'High stress' group had a narrower range of aspects providing pleasure, and, in general, rated the aspects lower in terms of importance for pleasure. A loss of appetite as a mere symptom of anhedonia, and thus, may be the cause for their dietary pattern. Therefore, it seems that the dietary changes of the 'High stress' group are not as much of an expression of the HPA axis being activated for a prolonged period of time, but a consequence of a condition that is leaving deeper marks in a person's mental health (ability to perceive pleasure). Further research into the food pleasure profiles and eating behavior patterns of people known to have anhedonic traits is highly recommended to be able to fully understand the impact of mental health on dietary habits and food choice.

Interestingly, the 'High stress' group increased their intake of caffeinated drinks. A positive correlation between occupational stress and coffee consumption has been established years ago [41][42][43], yet why this relationship exists is not quite clear. As the highly stressed group displayed a larger degree of anhedonia, it seems reasonable that they, to some degree, experience an aversion towards food. A simple thing such as a cup of tea or coffee may again be an easier choice for relief of hunger and low energy. Moreover, having a cup of coffee or tea may seem more manageable than cooking up a complete meal. A ten-year longitudinal study including a total of 50,739 US women showed that depression risk decreases with increased caffeinated coffee consumption [41]. Thus, the increase in coffee consumption of the highly stressed group could, in fact, be a positive finding, especially as caffeine has been shown to enhance dopaminergic activity in animal studies, as well as to cause increased well-being, energy, and alertness in human behavioral studies [44][45]. Caffeine may thus be used by the highly stressed group as a way of compensating for anhedonic or depression-like symptoms.

References

1. Torres, S.J.; Nowson, C.A. Relationship between stress, eating behavior, and obesity. *Nutrition* 2007, 23, 887–894.
2. Adam, T.C.; Epel, E.S. Stress, eating and the reward system. *Physiol. Behav.* 2007, 91, 449–458.

3. Epel, E.S.; Crosswell, A.D.; Mayer, S.E.; Prather, A.A.; Slavich, G.M.; Puterman, E.; Mendes, W.B. More than a feeling: A unified view of stress measurement for population science. *Front. Neuroendocr.* 2018, 49, 146–169.
4. Andersen, L.P. Arbejdsrelateret stress—Fra symptomer til et komplekst samspil. *Tidsskr. Arb.* 2007, 9, 11.
5. Rod, N.H. Stress og helbred: Årsager, helbredskonsekvenser og social ulighed, 1st ed.; Munksgaard: Copenhagen, Denmark, 2017; ISBN 978-87-628-1586-5.
6. Cohen, S.; Gianaros, P.J.; Manuck, S.B. A Stage Model of Stress and Disease. *Perspect. Psychol. Sci.* 2016, 11, 456–463.
7. Cohen, S.; Murphy, M.L.; Prather, A.A. Ten Surprising Facts about Stressful Life Events and Disease Risk. *Annu. Rev. Psychol.* 2019, 70, 577–597.
8. Brinkmann, S. Stress i Arbejdslivet: Konstitueringen Af En Epidemi. In *Nye Perspektiver på Stress*; Andersen, M.F., Brinkmann, S., Eds.; Klim: Aarhus, Denmark, 2013; pp. 75–92. ISBN 9788771291674.
9. McEwen, B.S. Protective and Damaging Effects of Stress Mediators. *N. Engl. J. Med.* 1998, 338, 171–179.
10. Lupien, S.J.; McEwen, B.S.; Gunnar, M.R.; Heim, C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat. Rev. Neurosci.* 2009, 10, 434–445.
11. Cohen, S.; Doyle, W.J.; Alper, C.M.; Janicki-Deverts, D.; Turner, R.B. Sleep Habits and Susceptibility to the Common Cold. *Arch. Intern. Med.* 2009, 169, 62–67.
12. Sinha, R.; Jastreboff, A.M. Stress as a Common Risk Factor for Obesity and Addiction. *Biol. Psychiatry* 2013, 73, 827–835.
13. Tomiyama, A.J. Stress and Obesity. *Annu. Rev. Psychol.* 2019, 70, 703–718.
14. O'Loughlin, I.; Newton-John, T.R. 'Dis-comfort eating': An investigation into the use of food as a coping strategy for the management of chronic pain. *Appetite* 2019, 140, 288–297.
15. Gibson, E.L. Emotional influences on food choice: Sensory, physiological and psychological pathways. *Physiol. Behav.* 2006, 89, 53–61.
16. Wallis, D.; Hetherington, M. Emotions and eating. Self-reported and experimentally induced changes in food intake under stress. *Appetite* 2009, 52, 355–362.
17. Ans, A.H.; Anjum, I.; Satija, V.; Inayat, A.; Asghar, Z.; Akram, I.; Shrestha, B. Neurohormonal Regulation of Appetite and its Relationship with Stress: A Mini Literature Review. *Cureus* 2018, 10, e3032.
18. Epel, E.; Lapidus, R.; McEwen, B.; Brownell, K. Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology* 2000, 26, 37–49.
19. Oliver, G.; Wardle, J. Perceived Effects of Stress on Food Choice. *Physiol. Behav.* 1999, 66, 511–515.
20. Finch, L.E.; Tiongo-Hofschneider, L.; Tomiyama, A.J. Stress-Induced Eating Dampens Physiological and Behavioral Stress Responses. In *Nutrition in the Prevention and Treatment of Abdominal Obesity*; Elsevier: Amsterdam, The Netherlands, 2019; pp. 175–187. ISBN 9780128160930.
21. Finlayson, G.; King, N.; Blundell, J.E. Liking vs. wanting food: Importance for human appetite control and weight regulation. *Neurosci. Biobehav. Rev.* 2007, 31, 987–1002.
22. Berridge, K.C.; Kringelbach, M.L. Pleasure Systems in the Brain. *Neuron* 2015, 86, 646–664.
23. Havermans, R.C. "You Say it's Liking, I Say it's Wanting ...". On the difficulty of disentangling food reward in man. *Appetite* 2011, 57, 286–294.
24. Berridge, K.C. 'Liking' and 'wanting' food rewards: Brain substrates and roles in eating disorders. *Physiol. Behav.* 2009, 97, 537–550.
25. Berridge, K.C.; Ho, C.-Y.; Richard, J.M.; DiFeliceantonio, A.G. The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Res.* 2010, 1350, 43–64.
26. Morris, M.J.; Beilharz, J.E.; Maniam, J.; Reichelt, A.C.; Westbrook, R.F. Why is obesity such a problem in the 21st century? The intersection of palatable food, cues and reward pathways, stress, and cognition. *Neurosci. Biobehav. Rev.* 2015, 58, 36–45.
27. Ruddock, H.K.; Hardman, C.A. Guilty pleasures: The effect of perceived overeating on food addiction attributions and snack choice. *Appetite* 2018, 121, 9–17.
28. Thomsen, K.R.; Whybrow, P.C.; Kringelbach, M.L. Reconceptualizing anhedonia: Novel perspectives on balancing the pleasure networks in the human brain. *Front. Behav. Neurosci.* 2015, 9, 49.

29. Gard, D.E.; Gard, M.G.; Kring, A.M.; John, O.P. Anticipatory and consummatory components of the experience of pleasure: A scale development study. *J. Res. Pers.* 2006, 40, 1086–1102.
30. Stanton, C.H.; Holmes, A.J.; Chang, S.W.; Joormann, J. From Stress to Anhedonia: Molecular Processes through Functional Circuits. *Trends Neurosci.* 2018, 42, 23–42.
31. Pizzagalli, D.A.; Bogdan, R.; Ratner, K.G.; Jahn, A.L. Increased perceived stress is associated with blunted hedonic capacity: Potential implications for depression research. *Behav. Res. Ther.* 2007, 45, 2742–2753.
32. Fawcett, J.; Clark, D.C.; Scheftner, W.A.; Gibbons, R.D. Assessing Anhedonia in Psychiatric Patients. *Arch. Gen. Psychiatry* 1983, 40, 79–84.
33. Chapman, L.J.; Chapman, J.P.; Raulin, M.L. Scales for physical and social anhedonia. *J. Abnorm. Psychol.* 1976, 85, 374–382.
34. Snaith, R.P.; Hamilton, M.; Morley, S.; Humayan, A.; Hargreaves, D.; Trigwell, P. A Scale for the Assessment of Hedonic Tone the Snaith–Hamilton Pleasure Scale. *Br. J. Psychiatry* 1995, 167, 99–103.
35. Hyldelund, N.B.; Worck, S.; Olsen, A. Convenience may increase vegetable intake among young consumers. *Food Qual. Prefer.* 2020, 83, 103925.
36. Andersen, B.V.; Chan, R.C.K.; Byrne, D.V. A Conceptual Framework for Multi-Dimensional Measurements of Food Related Pleasure—The Food Pleasure Scale. *Foods* 2021, 10, 2044.
37. Tomiyama, A.J.; Finch, L.E.; Cummings, J.R. Did That Brownie Do Its Job? Stress, Eating, and the Biobehavioral Effects of Comfort Food. *Emerg. Trends Soc. Behav. Sci.* 2015, 1–15.
38. Hyldelund, N.B.; Dalgaard, V.L.; Byrne, D.V.; Andersen, B.V. Why Being ‘Stressed’ Is ‘Desserts’ in Reverse—The Effect of Acute Psychosocial Stress on Food Pleasure and Food Choice. *Foods* 2022, 11, 1756.
39. Finch, L.E.; Cummings, J.R.; Tomiyama, A.J. Cookie or clementine? Psychophysiological stress reactivity and recovery after eating healthy and unhealthy comfort foods. *Psychoneuroendocrinology* 2019, 107, 26–36.
40. Pelizza, L.; Ferrari, A. Anhedonia in schizophrenia and major depression: State or trait? *Ann. Gen. Psychiatry* 2009, 8, 22.
41. Lucas, M. Coffee, Caffeine, and Risk of Depression among Women. *Arch. Intern. Med.* 2011, 171, 1571–1578.
42. Conway, T.L.; Vickers, R.R.; Ward, H.W.; Rahe, R.H. Occupational Stress and Variation in Cigarette, Coffee, and Alcohol Consumption. *J. Health Soc. Behav.* 1981, 22, 155.
43. Schreiber, G.B.; Robins, M.; Maffeo, C.E.; Masters, M.N.; Bond, A.P.; Morganstein, D. Confounders contributing to the reported associations of coffee or caffeine with disease. *Prev. Med.* 1988, 17, 295–309.
44. Alasmari, F. Caffeine induces neurobehavioral effects through modulating neurotransmitters. *Saudi Pharm. J.* 2020, 28, 445–451.
45. Garrett, B.E.; Griffiths, R.R. The Role of Dopamine in the Behavioral Effects of Caffeine in Animals and Humans. *Pharmacol. Biochem. Behav.* 1997, 57, 533–541.

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