Current Addiction Reports (2018) 5:243–250
https://doi.org/10.1007/s40429-018-0180-4

FOOD ADDICTION (A MEULE, SECTION EDITOR)

Bidirectional Associations Between Eating and Alcohol Use During Restricted Intake

Jenna R. Cummings[1] · A. Janet Tomiyama[1]

Published online: 26 January 2018

Abstract

Purpose of Review Many individuals seek to improve their health by restricting intake of alcohol or food. This paper critically examines the existing literature on how restricted alcohol use may increase eating and, reciprocally, how restricted eating may increase alcohol use.

Recent Findings Prior non-human animal research suggests that (a) alcohol deprivation causes increased eating behavior and (b) food deprivation causes increased alcohol use. Preliminary observational findings suggest that these effects translate to humans. Future experimental research is needed to identify for whom, for how long, why, and for what specific eating and drinking behaviors these bidirectional effects emerge. We provide a hypothesis-generating tool to guide future research.

Summary Understanding bidirectional associations between eating and alcohol use during restricted intake requires additional research. This research may have important treatment implications for multiple health behavior change interventions as well as treatment for alcohol use and eating disorders including addictions.

Keywords Addiction · Alcohol use · Eating behavior · Multiple health behavior change

Introduction

As many US Americans are aware of, an attempt at nationwide ban of alcohol use failed and the USA repealed Prohibition so that civilians could drink alcohol again. The lesser-told story of Prohibition, however, is one about eating. Indeed, when US Americans began drinking less alcohol they—whether intentionally or not—began eating more candy. The US per capita candy consumption nearly doubled when Prohibition began in 1919, and sugar consumption hit record highs in 1920 [1]. The public's shift to eating sugar was so apparent that The New York Times stated, "The wreckage of the liquor business is being salvaged for the production of candy, ice cream and syrups [1]."

This historical anecdote illustrates the general hypothesis of this paper: when individuals reduce intake of one substance such as alcohol, there may be consequences for intake of other substances such as sweets and vice versa. The purpose of this paper is (a) to critically examine this general hypothesis by reviewing the existing literature on bidirectional associations between eating and alcohol use during restricted intake and (b) to highlight critical future research directions with theoretical and practical value. In this paper, we first review the extant literature on how restricted alcohol use may increase eating. Second, we review the extant literature on how restricted eating may increase alcohol use. Importantly, this review focuses on bidirectional associations between eating and alcohol use *during restricted intake*, that is, when an individual is motivated to eat less food or drink less alcohol.

Understanding bidirectional associations between eating and alcohol use during restricted intake is important because many individuals seek to improve their health by restricting intake of alcohol or food. Managing *both* eating and alcohol use is imperative to overall health. Critically, there would be a health trade-off if an individual reduced intake of either alcohol or certain foods (e.g., processed foods high in sugar and fat) and then replaced that substance with the other. This is because alcohol and certain foods are substances with addictive potential that may elicit addictive-like behavior [2], and the same risk factors that promote addictive-like behavior in response to one of these

This article is part of the Topical Collection on *Food Addiction*

✉ Jenna R. Cummings
jrcummings@ucla.edu

[1] Department of Psychology, University of California, Los Angeles, 1285 Franz Hall, Los Angeles, CA 90095, USA

substances can promote addictive-like behavior in response to the other [3–5]. Moreover, poor diet and alcohol use are associated with lower health-related quality of life [6, 7] and, ultimately, greater mortality in the USA [8]. Given these substantive health impacts, the current paper takes a comprehensive perspective on these health behaviors in relation to achieving overall mental and physical health.

Restricted Alcohol Use May Increase Eating

Non-human Animal Research

There is a small body of non-human animal research that suggests that restricted alcohol use may increase eating. Rats genetically bred for high compared to low alcohol intake prefer the taste of saccharin solutions and will eat more saccharin when ethanol is unavailable [9]. This effect was also observed with bitter, salty, and sour solutions, although the effect was the strongest for saccharin solutions. Furthermore, researchers studying the alcohol deprivation effect showed that rats rapidly increased ethanol consumption following periodic withholding of ethanol; pertaining to this review in particular, the researchers also showed that rats rapidly ate more saccharin following this periodic withholding of ethanol [10]. Taken together, this rodent research suggests that there are shared factors that promote eating and alcohol use and that alcohol deprivation may increase eating behavior.

Non-human animal research provides a controlled model for understanding how restricted alcohol use might increase eating behavior, yet there are a number of limitations. One key limitation is that non-human animal models cannot simulate a person's motivation to reduce drinking; specifically, alcohol is forcibly given to and withheld from non-human animals. Non-human animal models also limit the time frame of alcohol deprivation (i.e., 5–28 days out of a 1–2-year lifespan), and the procedures control the availability of other substances (e.g., food) in the environment. In contrast, people may experience longer time periods where they are motivated to restrict alcohol use, and during these time periods, people may encounter a wide variety of other substances. Another limitation to consider in non-human animal research is that researchers may control the diet of these animals over their whole lifespan whereas people have a longer, more dynamic developmental relationship with food—one that most likely developed far earlier than any relationship with alcohol. When all these experiences are involved, is there support that restricted alcohol use increases eating?

Human Research

There is a small number of observational studies that suggest that when people are motivated to restrict alcohol intake, consumption of different types of food may increase including simple sugars, starches (e.g., potatoes, breads, cereals), sweets (e.g., candy, chocolate), and salty/spicy foods (e.g., chips, pretzels, nachos) [11–13]. For example, in a sample of 64 newly sober outpatients with alcoholism, researchers collected dietary information via 24-h dietary recall early into the outpatient program [11]. Results indicated that those who were sober for greater than 50 days consumed more carbohydrates compared to those who were sober for 50 days or less. In a 4-month daily diary study, participants recovering from alcoholism (i.e., sober for 3–12 months) compared to controls were more likely to eat sweet, starchy, and salty/spicy foods—especially on days that they reported higher levels of stress [13]. In sum, there is preliminary evidence to suggest that when people restrict alcohol intake, they may increase intake of certain foods.

Nonetheless, there are drawbacks to these observational studies: there are few of them and none establish causality. In particular, when comparing participants who remain sober to those who do not, it remains unclear if reducing alcohol intake increases eating or if eating more food helps participants to stay sober. This directionality of effect is especially important to consider as there exist anecdotal claims that eating reduces alcohol intake [14]. Likewise, the samples across all the prior observational studies comprise participants with alcoholism. Multiple studies suggest that those with alcoholism have a general preference for sweet tastes [15]. Thus, it remains unclear if those with an alcohol use disorder generally eat more of certain foods or if the effect specifically emerges after reducing alcohol intake, albeit measuring eating prior to any reductions in alcohol intake may ameliorate this methodological drawback. Overall, there is a clear need for future experimental work to establish causality, to disentangle the direction of effect, and to tease out any confounds.

Future Research Directions

In light of the limitations of the extant literature on how restricted alcohol use may increase eating, there are several future directions for research. Establishing causality with experimental research would be a strong first step in building upon the existing non-human animal and clinical research; however, we have identified other key directions that we consider to be theoretically and practically valuable. In Fig. 1, we highlight these directions with a hypothesis-generating tool to guide future research. Researchers can choose one or more components from each box to generate a critical research question that will move the field forward.

Individual Differences All prior clinical research is limited to samples comprising participants with alcoholism, and it seems that any effect would be limited to those with a drinking history. However, would restricted alcohol use increase eating for

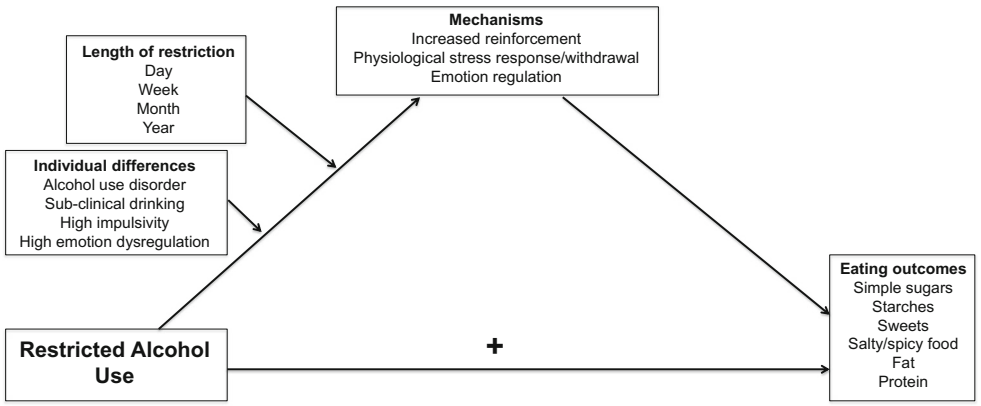

Fig. 1 The effects of restricted alcohol use on eating may depend on different moderators and outcomes investigated and may be mediated by different mechanisms

those who drink less frequently and at a sub-clinical level? For instance, the motivations behind restricting alcohol intake may greatly differ between someone who has an alcohol use disorder versus someone who drinks moderately. Those who have an alcohol use disorder may be trying to overcome the illness whereas those who drink moderately may be trying to improve their lifestyle more generally or to lose weight. The latter motivations may even confound the effect of restricted alcohol use on eating because—in that case—people may be motivated to restrict alcohol and food intake simultaneously.

Likewise, are there specific individual differences that may predispose someone to experience a stronger effect of alcohol restriction on eating? For example, individuals who are more impulsive and who experience greater emotion dysregulation may be especially at risk for the effect of alcohol restriction on eating behavior because these risk factors promote addictive-like behavior [3, 5]; in the absence of drinking, eating may become more addictive-like. Identifying individual differences that may moderate effects is important for targeting any treatment to the individuals most fit for it.

Length of Restriction Prior rodent models have not simulated long-term periods of alcohol restriction, and prior clinical research is varied in the periods of alcohol restriction in relation to when the researchers measured eating behavior (e.g., 3–12 months). In experimental work, researchers can manipulate the length of restriction to be days, weeks, months, or potentially even years and frequently measure eating within the period. This could have direct practical benefits. That is, perhaps the effect of restricted alcohol use on eating is temporary and only occurs within early days of sobriety. This could suggest that any treatment of eating would only need to be implemented during the early transition into alcohol abstinence. On the other hand, if the effect of restricted alcohol use on eating is lasting, it could suggest that any treatment of eating behavior during alcohol abstinence would also need to be long-term.

Mechanisms If restricted alcohol use reliably increases eating behavior, why? Three mechanisms may explain this effect: increased positive reinforcement from food, altered physiological stress responses, and altered psychological mood experiences. First, the restriction of alcohol intake temporarily increases positive reinforcement from substances or aspects about using substances that encourage future use [16]. Although it was previously thought that increased positive reinforcement following restriction of alcohol intake would be limited to alcohol, it is possible that restricted alcohol use might increase positive reinforcement not only most powerfully for the deprived substance (e.g., alcohol) but also more generally for other substances (e.g., food, other drugs).

Second, when someone with a drinking history restricts alcohol intake, they may experience a physiological stress response as a part of withdrawal symptoms. That is, alcohol inhibits excitatory responses of the central nervous system; when a system that is reliant on this no longer receives alcohol, the system may experience excitatory overload leading an individual to experience physiological stress response symptoms such as anxiety, shaking, palpitations, hyperflexia, gastrointestinal upset, and—in severe cases—seizures [17]. During these experiences, an individual may especially crave another substance to ameliorate those withdrawal symptoms. Indeed, preclinical research suggests that high calorie, fatty, and sugary foods may actually dampen physiological stress responses [18•]. Third, many individuals drink alcohol in order to regulate emotional experiences [19], regardless of whether or not alcohol actually helps them succeed in this. When these individuals reduce their alcohol use, they have lost a strategy for coping or avoiding emotional experiences. Comfort eating, or eating high calorie, fatty, and sugary foods in response to negative emotions, may serve as an antidote to this loss [18•]. Indeed, one observational study found that participants recovering from alcoholism were more likely to eat sweet, starchy, and salty/spicy foods on days that they reported higher levels of stress [13].

Eating Outcomes As observed across prior clinical research, alcohol restriction may increase a wide variety of eating behavior ranging from increases in simple sugar intake [11] to

salty/spicy food intake [13]. Measuring all types of food intake in response to alcohol restriction would help clarify which eating behavior is potentially affected the most by alcohol restriction. This has practical importance because each type of food has a unique set of characteristics (e.g., taste, nutritional value, availability) that would need to be considered in any treatment. Additionally, certain foods (e.g., highly processed, with added fats and/or refined carbohydrates) are most strongly associated with addictive-like eating and greater loss of control, pleasure, and craving [20•, 21]. For individuals who had an alcohol use disorder, it may be especially important to know if restriction of alcohol intake increases intake of foods that may trigger addictive-like eating or contribute to the development of so-called food addiction.

Restricted Eating May Increase Alcohol Use

Non-human Animal Research

There is a large body of evidence demonstrating that depriving rats of food increases their motivation to press a lever to obtain a number of drugs including amphetamine, cocaine, heroin, phencyclidine, and—most relevant to the current paper—ethanol [see 22 for a review]. For example, researchers gave rats only enough food to maintain them at 80% of their body weights and then provided them the opportunity to obtain water and ethanol [23]. Results indicated that when food deprived compared to satiated rats drank more ethanol, rats did not drink more water. This showed that the food deprivation selectively enhanced drinking of ethanol rather than generally increased fluid intake. Additionally, researchers replicated the procedure but with varying concentrations of ethanol [24]. Results indicated when food deprived compared to satiated rats drank more ethanol regardless of whether it was a low (4% *W*/*V*) or high (32% *W*/*V*) concentration ethanol solution. In sum, there is substantive non-human animal research that supports the notion that reducing food intake increases alcohol use independent of increases in overall fluid intake.

Although non-human animal models establish a causal pathway from food deprivation to increased alcohol use, limitations prevent translation of these findings to the clinical domain. Again, these models cannot simulate motivations to reduce food intake, they limit deprivation length, and they control all substances in the environment.

Human Research

Although (to our knowledge) no study has been designed to specifically examine the effect of restricted eating on alcohol use, there are other designs that may shed light on how restricted eating increases alcohol use. Randomized controlled trials (RCTs) of dietary restriction interventions, for instance, can provide some preliminary evidence. However, short-term and long-term dietary restriction interventions often include instructions on alcohol use, which precludes understanding of how dietary restriction may independently affect drinking behavior [25–28]. Also, short-term and long-term RCTs of dietary restriction interventions often do not measure alcohol use as an outcome [29–33], and some exclude drinkers from the trials [34, 35]. In sum, this available paradigm for studying food restriction in humans has not yet provided meaningful insight on if food restriction increases alcohol use.

Another paradigm that may allow for the study of the effect of food restriction on alcohol use in humans may be weight loss surgery [36•]. Several cross-sectional and prospective studies have documented that individuals who undergo weight loss surgery—in particular, Roux-en-Y gastric bypass (RYGB)—will successfully eat less food after the surgery, yet may drink more alcohol and develop symptoms of an alcohol use disorder [reviewed in 36•]. For instance, in one study, 19.6% of individuals who underwent RYGB reported symptoms of a substance use disorder and, for 68% of these individuals, these symptoms were occurring for the first time in their life [37]. Research investigating the mechanism behind this RYGB phenomenon has found that RYGB may alter metabolism via decreased eating and weight loss; this alteration in metabolism may contribute to more rapid rises to and higher levels of maximum blood alcohol concentration as well as longer time needed for alcohol elimination [38–40]. Thus, RYGB may be an important paradigm for understanding the effects of restricted eating on alcohol use because biological changes from RYGB force restriction in eating. However, separate biological changes related to RYGB may confound a pure test of the effect of restricted eating on alcohol use.

In observational research, there also have been multiple reports of a strong comorbidity between eating disorders (including anorexia, bulimia, and binge eating disorder) and alcohol use disorders [41]. However, it is difficult to establish the mechanisms behind these broad associations because eating disorders differ in eating patterns (e.g., starvation in anorexia versus binging and purging in bulimia) and both eating and alcohol use disorders incorporate symptoms that are not purely about eating/drinking patterns (e.g., weight concerns, compensatory behaviors, absenteeism, tolerance). Moreover, individuals who chronically fear weight gain may report that they restrict eating (e.g., "Do you deliberately eat less in order not to become heavier?") [42]. This cognitive-behavioral pattern—termed dietary restraint—may uniquely influence alcohol use [see 43 for a review]. For instance, women who scored higher in dietary restraint did not drink more often but did drink larger amounts compared to those who scored lower [44]. It is speculated that the link between dietary restraint and increased alcohol use may be due to ironic processing (i.e., trying to suppress intake backfires) and disinhibition (i.e., continuing to over consume once the diet has been

violated) [43•]. Although these psychological mechanisms may in part explain how restricted food intake increases alcohol use, there are a number of additional plausible mechanisms. As a result, there is still a sizeable research gap for future research to fill.

Future Research Directions

In order to improve upon the existing non-human animal and clinical research, researchers should conduct RCTs of short-term and long-term dietary restriction interventions (without providing instruction on alcohol use) and measure alcohol use as an outcome. In addition, researchers should conduct short-term laboratory studies with paradigms (e.g., fasting, inducing hunger) that can verify causality as well as identify mechanisms. We have again identified key future research directions that we consider to be essential, and these are summarized in Fig. 2.

Food Amounts and Types It is impossible for a person to survive if they abstain from eating food (unlike alcohol). How much food would one have to restrict to observe any potential changes in alcohol use? The answer to this question may have important practical implications; if effects are only observed with severe eating restriction (which is already associated with deleterious health consequences [45]), moderate dietary restriction plans may not need to address alcohol use. Moreover, RCTs of dietary restriction interventions typically target certain food types [25, 31], which begs the question of if restricting intake of different food types may lead to different impacts on alcohol use. For example, perhaps just restricting intake of sweets—or other foods that restricting alcohol use increases—may increase alcohol use but overall calorie restriction would not.

Patterns of Restriction Prior rodent models simulate eating restriction via complete deprivation for 24 h. However, manipulating long-term periods of eating restriction in humans may prove insightful for understanding how substantive changes in eating impact alcohol use. Thus, researchers can manipulate the length of restriction to be days, weeks, months, or years in correspondence with existing dietary plans and frequently measure alcohol use within the period. In addition, researchers can test the effect of other unique dietary restriction patterns. Intermittent fasting—a pattern of dietary restriction wherein individuals repeat periods of 16–48 h of very little eating followed by normal eating—has emerged as a dietary plan that may promote metabolic health and delay aging [46]; might intermittent fasting affect alcohol use differently than more traditional calorie restriction dietary plans? Answering these kinds of questions will provide interventionists with the information to determine if alcohol use treatment needs to be integrated into these varied dietary plans.

Mechanisms It is plausible that the same mechanisms (increased reinforcement, physiological stress responses, and emotion regulation) that explain how restricted alcohol use may increase eating explain how restricted eating may increase alcohol use. Indeed, non-human animal models suggest that food restriction temporarily increases positive reinforcement from other substances [22], certain food deprivation has been shown to induce withdrawal-like symptoms in rodents (e.g., teeth chattering) [47], and many individuals eat to cope with negative emotions [18•, 48]; thus, they may use alcohol as a new way to cope. However, it is also plausible that exclusive mechanisms explain this direction of effect. For instance, research on RYGB has suggested that altered metabolism following restricted eating/weight loss may explain why RYBG could contribute to the development of an alcohol use disorder [38–40]. It is possible that restricted eating may similarly alter metabolism in the short term. Certainly, blood alcohol absorption, concentration, and elimination are altered when individuals do not eat food before drinking alcohol, which all could enhance the effects of alcohol [49, 50].

Drinking Outcomes If restricted eating increases alcohol use, how harmful is this experience to overall mental and physical health? There is a broad range of drinking patterns and not all

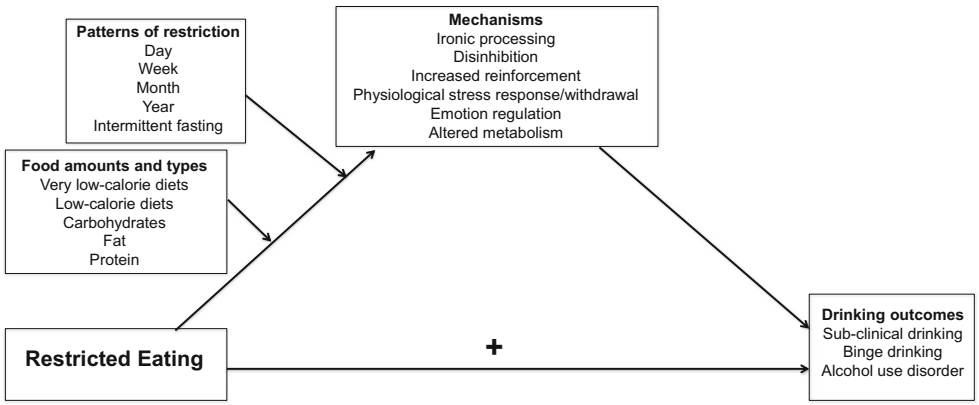

Fig. 2 The effects of restricted eating on alcohol use may depend on different moderators and outcomes investigated and may be mediated by different mechanisms

patterns are associated with marked, negative health consequences. For instance, moderate drinking has been associated with less odds of coronary disease [51], which may be because light to moderate habitual drinking predicts healthier cardiovascular functioning during stress [52]. A systematic review of 34 studies indicates a J-shaped pattern between alcohol use and all-cause mortality such that those who drink moderately (up to two drinks/day in women and up to four drinks/day in men) had maximum protection but those who drank greater amounts had greater odds of mortality [53]. Drinking greater amounts of alcohol each day, however, may be indicative of the development of an alcohol use disorder, which can not only impact physical health but can also burden an individual's mental health via legal (e.g., drunk driving violations), social (e.g., arguments about alcohol with family members), or job (e.g., poor performance, absenteeism) problems [54]. Thus, if someone restricts eating long term, will this increase the likelihood of moderate drinking or will this increase the likelihood of binge drinking and/or the development of an alcohol use disorder?

Information on the severity of the effect is important for treatment. If dietary restriction plans increase the likelihood of binge drinking, dietary restriction plans may need to target risk factors for binge drinking (e.g., alcohol expectancies, social contexts). On the other hand, if dietary restriction plans increase the likelihood of development of an alcohol use disorder, dietary restriction plans may need to also target risk factors for alcohol use disorders (e.g., emotion dysregulation).

Conclusion

The research testing bidirectional associations between eating and alcohol use while restricting intake is in its infancy. However, focusing on the potential clinical implications of understanding these bidirectional associations may inspire basic research that has applied benefits. Indeed, the public and interventionists hold high demand for information regarding successful health behavior change. In our opinion, it is shortsighted to conduct interventions on alcohol use without measuring changes in eating and vice versa. It is generally feasible to incorporate measures of both alcohol use and eating behavior into this research. Simultaneously, there is the potential for tremendous scientific insight given that alcohol use is relevant to the eating literature (i.e., alcohol is a food source) and eating is relevant to the addictions literature (i.e., food may play a role in the maintenance of addictive-like behavior).

Our observations are in accordance with a Multiple Health Behavior Change (MHBC) viewpoint wherein interventionists target clusters of behaviors collectively rather than independently [55, 56, 57•]. MHBC interventions have the potential to increase health benefits, maximize health promotion, and reduce healthcare costs [55], and a meta-analysis suggests that MHBC interventions that focus on two to three behaviors may be the most effective [58•]. If research robustly confirms bidirectional associations between eating and alcohol use during restricted intake, alcohol use and eating may be two prime candidate behaviors for MHBC. Moreover, although MHBC interventions primarily focus on sub-clinical behaviors, if research suggests that bidirectional associations between eating and alcohol use occur during more severe restrictions of intake (e.g., alcohol withdrawal in those with alcohol use disorder, severe restriction of food intake in those with eating disorders), it may be critical to translate this MHBC approach to clinical settings or pharmacotherapy trials. For example, in a case study, clinicians not only administered baclofen to a patient for alcohol use disorder but also observed if baclofen affected eating behavior; although baclofen greatly diminished alcohol craving during abstinence, it had no effects on the patient's high food cravings and bulimia symptoms during the abstinence [59].

Overall, there is preliminary support from non-human animal and clinical research suggesting that restricted alcohol use increases eating and, conversely, restricted eating increases alcohol use. Although this preliminary support provides proof of concept, future clinical research is needed to verify causality. Research is also needed to identify for whom, for how long, why, and for what specific eating and drinking behaviors these effects are contingent upon. Findings from this future research will inform future interventions. Taking a comprehensive approach in treating problematic alcohol use and eating behavior may more efficiently and effectively guide individuals in achieving mental and physical health.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance

1. Taubes G. The case against sugar. First Edit. New York City, New York: Alfred A. Knoff, Penguin Random House LLC; 2016.
2. Schulte EM, Potenza MN, Gearhardt AN. A commentary on the eating addiction versus food addiction perspectives on addictive-like food consumption. Appetite Elsevier Ltd. 2016;115:9–15.
3. Pivarunas B, Conner BT. Impulsivity and emotion dysregulation as predictors of food addiction. Eat Behav. 2015;19:9–14.

4. Gearhardt AN. Neural correlates of food addiction. Arch Gen Psychiatry. 2011;68(8):808–16. https://doi.org/10.1001/archgenpsychiatry.2011.32.
5. Murphy CM, Stojek MK, MacKillop J. Interrelationships among impulsive personality traits, food addiction, and body mass index. Appetite. 2014;73:45–50. https://doi.org/10.1016/j.appet.2013.10.008.
6. Imtiaz S, Loheswaran G, Le Foll B, Rehm J. Longitudinal alcohol consumption patterns and health-related quality of life: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Drug Alcohol Rev. 2017; https://doi.org/10.1111/dar.12503.
7. Hassan MK, Joshi AV, Madhavan SS, Amonkar MM. Obesity and health-related quality of life: a cross-sectional analysis of the US population. Int J Obes. 2003;27(10):1227–32. https://doi.org/10.1038/sj.ijo.0802396.
8. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. JAMA. 2004;291(10):1238–45.
9. Sinclair JD, Kampov-Polevoy A, Stewart R, Li TK. Taste preferences in rat lines selected for low and high alcohol consumption. Alcohol. 1992;9(2):155–60. https://doi.org/10.1016/0741-8329(92)90027-8.
10. Wayner MJ, Greenberg I, Tartaglione R, Nolley D, Fraley S, Cott A. A new factor affecting the consumption of ethyl alcohol and other sapid fluids. Physiol Behav. 1972;8(2):345–62. https://doi.org/10.1016/0031-9384(72)90383-6.
11. Yung L, Gordis E, Holt J. Dietary choices and likelihood of abstinence among alcoholic patients in an outpatient clinic. Drug Alcohol Depend. 1983;12(4):355–62. https://doi.org/10.1016/0376-8716(83)90007-8.
12. Johnson MW, De Vries J, Houghton MI. The female alcoholic. Nurs Res. 1966;15(4):343–7.
13. Rosenfield SN, Stevenson JS. Perception of daily stress and oral coping behaviors in normal, overweight, and recovering alcoholic women. Res Nurs Health. 1988;11(3):165–74. https://doi.org/10.1002/nur.4770110305.
14. Alcoholics Anonymous. Living sober. Alcoholics Anonymous World Services; 1975.
15. Kampov-Polevoy AB, Garbutt JC, Janowsky DS. Association between preference for sweets and excessive alcohol intake: a review of animal and human studies. Alcohol Alcohol. 1999;34(3):386–95. https://doi.org/10.1093/alcalc/34.3.386.
16. Heyser CJ, Schulteis G, Koob GF. Increased ethanol self-administration after a period of imposed ethanol deprivation in rats trained in a limited access paradigm. Alcohol Clin Exp Res. 1997;21(5):784–91.
17. Stobart Gallagher MA, Gomaz AE. Alcohol withdrawal. In: StatPearls. Treasure Island: StatPearls Publishing LLC; 2017.
18.• Tomiyama AJ, Finch LE, Cummings JR. Did that brownie do its job? Stress, eating, and the biobehavioral effects of comfort food. In: Scott RA, Kosslyn SM, editors. Emerging Trends in the Social and Behavioral Sciences. Hoboken, NJ, USA: John Wiley & Sons, Inc.; 2015. **This paper reviews non-human animal and clinical research on comfort eating (i.e., eating in response to negative emotion) and if comfort eating actually dampens a physiological stress response. This paper also draws parallels between comfort eating and alcohol stress-response dampening, which may be important for undertanding mechanisms behind the bidirectional associations between eating and alcohol use during restricted intake.**
19. Cooper ML, Frone MR, Russell M, Mudar P. Drinking to regulate positive and negative emotions: a motivational model of alcohol use. J Pers Soc Psychol. 1995;69(5):990–1005. https://doi.org/10.1037/0022-3514.69.5.990.
20.• Schulte EM, Avena NM, Gearhardt AN. Which foods may be addictive? The roles of processing, fat content, and glycemic load. PLoS One. 2015;10(2):e0117959. **This paper systematically tests which foods were associated with reports of addictive-like eating. Results indicated that highly processed foods, with added fat and/or refined carbohydrates, were most strongly associated with addictive-like eating. If restricted alcohol use promotes eating of these specific foods, it may trigger addictive-like eating.**
21. Schulte EM, Smeal JK, Gearhardt AN, Eissenberg T, Gustafson J, Yanovski J. Foods are differentially associated with subjective effect report questions of abuse liability. PLoS One. 2017;12(8):e0184220. https://doi.org/10.1371/journal.pone.0184220.
22. Carroll ME, Meisch RA. Increased drug-reinforced behavior due to food deprivation. In: Thompson T, Dews PB, Barrett JE, editors. Advances in Behavioral Pharmacology 1984. p. 47–88.
23. Meisch RA, Thompson T. Ethanol as a reinforcer: effects of fixed-ratio size and food deprivation. Psychopharmacologia. 1973;28(2):171–83. https://doi.org/10.1007/BF00421402.
24. Meisch RA, Thompson T. Ethanol intake as a function of concentration during food deprivation and satiation. Pharmacol Biochem Behav. 1974;2(5):589–96. https://doi.org/10.1016/0091-3057(74)90025-2.
25. Stevens V, Obarzanek E, Cook N, Lee I-M, Md S, Appel L, et al. Long-term weight loss and changes in blood pressure: results of the trials of hypertension prevention, phase II. Ann Intern Med. 2001;134(1):1–11. https://doi.org/10.7326/0003-4819-134-1-200101020-00007.
26. Sone H, Tanaka S, Iimuro S, Tanaka S, Oida K, Yamasaki Y, et al. Long-term lifestyle intervention lowers the incidence of stroke in Japanese patients with type 2 diabetes: a nationwide multicentre randomised controlled trial (the Japan diabetes complications study). Diabetologia. 2010;53(3):419–28. https://doi.org/10.1007/s00125-009-1622-2.
27. Jakobsen LH, Kondrup J, Zellner M, Tetens I, Roth E. Effect of a high protein meat diet on muscle and cognitive functions: a randomized controlled dietary intervention trial in healthy men. Clin Nutr. 2011;30(3):303–11. https://doi.org/10.1016/j.clnu.2010.12.010.
28. Zou P, Dennis C, Lee R, Parry M. Dietary approach to stop hypertension with sodium reduction for Chinese Canadians (DASHNa-CC): a pilot randomized controlled trial. J Nutr Heal Aging. 2016:1–8.
29. Heshka S, Anderson JW, Atkinson RL, Greenway FL, Hill JO, Phinney SD, et al. Weight loss with self-help compared with a structured commercial program: a randomized trial. 2014;289(14).
30. Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. English J. 2002;346(6):393–403.
31. Kuller LH, Simkin-Silverman LR, Wing RR, Meilahn EN, Ives DG. Women's healthy lifestyle project: a randomized clinical trial: results at 54 months. Circulation. 2001;103(1):32–7. https://doi.org/10.1161/01.CIR.103.1.32.
32. Howard BV, Manson JE, Stefanick ML, Beresford SA, Frank G, Jones B, et al. Low-fat dietary pattern and weight change over 7 years: the Women's Health Initiative Dietary Modification Trial. JAMA. 2006;295(1):39–49. https://doi.org/10.1001/jama.295.1.39.
33. Kalarchian MA, Marcus MD, Courcoulas AP, Lutz C, Cheng Y, Sweeny G. Structured dietary intervention to facilitate weight loss after bariatric surgery: a randomized, controlled pilot study. Obesity. 2016;24(9):1906–12. https://doi.org/10.1002/oby.21591.
34. Browning JD, Baker JA, Rogers T, Davis J, Satapati S, Burgess SC. Short-term weight loss and hepatic triglyceride reduction: evidence of a metabolic advantage with dietary carbohydrate restriction. Am J Clin Nutr. 2011;93(5):1048–52. https://doi.org/10.3945/ajcn.110.007674.
35. Calvo-Malvar M, Leis R, Benitez-Estevez AJ, Sanchez-Castro J, Gude F. A randomized, family-focused dietary intervention to

evaluate the Atlantic diet: the GALIAT study protocol. BMC Public Health. 2016;16(1):1–9.

36.• Ivezaj V, Stoeckel LE, Avena NM, Benoit SC, Conason A, Davis JF, et al. Obesity and addiction: Can a complication of surgery help us understand the connection? Obes Rev. 2017;18(7, 75):–765. **This paper reviews several studies that report that after weight loss, surgery individuals increase alcohol use. This paper suggests potential mechanisms behind this phenomenon, which may in part explain why restricted eating increases alcohol use.**

37. Ivezaj V, Saules KK, Schuh LM. New-onset substance use disorder after gastric bypass surgery: rates and associated characteristics. Obes Surg. 2014;24(11):1975–80. https://doi.org/10.1007/s11695-014-1317-8.

38. Woodard GA, Downey J, Hernandez-Boussard T, Morton JM. Impaired alcohol metabolism after gastric bypass surgery: a case-crossover trial. J Am Coll Surg Elsevier Inc. 2011;212(2):209–14. https://doi.org/10.1016/j.jamcollsurg.2010.09.020.

39. Pepino MY, Okunade AL, Eagon JC, Bartholow BD, Bucholz K, Klein S, et al. Effect of Roux-en-Y gastric bypass surgery: converting 2 alcoholic drinks to 4. JAMA Surg. 2015;150(11):1096–8. https://doi.org/10.1001/jamasurg.2015.1884.

40. Steffen KJ, D. P, Engel SG, Pollert GA, Cao L, Mitchell JE. Blood alcohol concentrations rise rapidly and dramatically following Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2013;9(3):470–3. https://doi.org/10.1016/j.soard.2013.02.002.

41. Sinha R, O'Malley SS. Alcohol and eating disorders: implications for alcohol treatment and health services research. Alcohol Clin Exp Res. 2000;24(8):1312–9. https://doi.org/10.1111/j.1530-0277.2000.tb02097.x.

42. van Strien T, Frijters J, Bergers G, Defares P. The Dutch Eating Behaviour Questionnaire (DEBQ) for assessment of restrained, emotional and external eating behaviour. Int J Eat Disord. 1986;5(2):295–315. https://doi.org/10.1002/1098-108X(198602)5:2<295::AID-EAT2260050209>3.0.CO;2-T.

43.• Caton SJ, Nolan LJ, Hetherington MM. Alcohol, appetite and loss of restraint. Curr Obes Rep. 2015;4(1):99–105. **This paper reviews how the cognitive-behavioral pattern of dietary restraint (for fear of weight gain) may increase alcohol use. The authors discuss potential psychological mechanisms that may explain a link between dietary restraint and increased alcohol use.**

44. Stewart SH, Angelopoulos M, Baker JM, Boland FJ. Relations between dietary restraint and patterns of alcohol use in young adult women. Psychol Addict Behav. 2000;14(1):77–82. https://doi.org/10.1037/0893-164X.14.1.77.

45. Keys A, Brozek J, Henschel A, Mickelsen O, Taylor HL. The biology of human starvation. Oxford: University of Minnesota Press; 1950.

46. Mattson MP, Longo VD, Harvie M. Impact of intermittent fasting on health and disease processes. Ageing Res Rev Elsevier BV. 2017;39:46–58. https://doi.org/10.1016/j.arr.2016.10.005.

47. Colantuoni C, Rada P, McCarthy J, Patten C, Avena NM, Chadeayne A, et al. Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. Obes Res. 2002;10(6):478–88. https://doi.org/10.1038/oby.2002.66.

48. Burgess EE, Turan B, Lokken KL, Morse A, Boggiano MM. Profiling motives behind hedonic eating. Preliminary validation of the Palatable Eating Motives Scale. Appetite. Elsevier Ltd; 2014 Jan [cited 2014 May 26];72:66–72.

49. Gentry RT. Effect of food on the pharmacokinetics of alcohol absorption. Alcohol Clin Exp Res. 2000;24(4):403–4. https://doi.org/10.1111/j.1530-0277.2000.tb01996.x.

50. Ramchandani VA, Kwo PY, Li TK. Effect of food and food composition on alcohol elimination rates in healthy men and women. J Clin Pharmacol. 2001;41(12):1345–50. https://doi.org/10.1177/00912700122012814.

51. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA. Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. Bmj. 2011;342(feb22 1):d671–1.

52. Jones A, McMillan MR, Jones RW, Kowalik GT, Steeden JA, Pruessner JC, et al. Habitual alcohol consumption is associated with lower cardiovascular stress responses—a novel explanation for the known cardiovascular benefits of alcohol? Stress. 2013 Jul;16(4):369–76. https://doi.org/10.3109/10253890.2013.777833.

53. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G. Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. Arch Intern Med American Med Assoc. 2006;166(22):2437–45. https://doi.org/10.1001/archinte.166.22.2437.

54. Babor TF, Higgins-biddle JC, Saunders JB, Monteiro MG. The alcohol use disorders identification TEST 2001.

55. Prochaska JJ, Prochaska JO. A review of multiple health behavior change interventions for primary prevention. Am J Lifestyle Med. 2011;5(3):208–21. https://doi.org/10.1177/1559827610391883.

56. Prochaska JO. Multiple health behavior research represents the future of preventive medicine. Prev Med (Baltim). 2008;46(3):281–5. https://doi.org/10.1016/j.ypmed.2008.01.015.

57.• Spring B, King AC, Pagoto SL, Fisher JD, Spring B. Fostering multiple healthy lifestyle behaviors for primary prevention of cancer. Am Psychol. 2015;75–90. **This paper reviews the existing knowledge base on multiple health behavior change combinations. This paper also provides critical insight into the future steps that need to be taken to understand how different behavior combinations affect each other in the context of interventions.**

58.• Wilson K, Senay I, Durantini M, Sánchez F, Hennessy M, Spring B, et al. When it comes to lifestyle recommendations, more is sometimes less: a meta-analysis of theoretical assumptions underlying the effectiveness of interventions promoting multiple behavior domain change. Psychol Bull. 2015;141(2):474–509. **This meta-analysis identifies factors that predict the effectiveness of multiple health behavior change interventions. Results indicated that including two to three behavior targets in an intervention led to the most optimal results; this suggests that interventions pairing eating and alcohol use together as targets may lead to optimal results.**

59. Weibel S, Lalanne L, Riegert M, Bertschy G. Efficacy of high-dose baclofen for alcohol use disorder and comorbid bulimia: a case report. J Dual Diagn. 2015;11(3–4):203–4.