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Food–alcohol competition: As young females eat more food, do they drink less alcohol?

Jenna R Cummings, Lara A Ray and A Janet Tomiyama

Abstract

Seminal health behaviour theories and behaviour modification techniques are applied to health behaviours individually. Limited empirical work investigates how change in one health behaviour may change another. This study proposes a *food–alcohol competition hypothesis*, where individuals tend to consume one rewarding substance to the other's exclusion. In a large sample of adolescent girls assessed yearly from age 15 to 19, Latent Growth Modelling indicated that a tendency to consume processed or sweet high-fat foods 'competed' with a tendency to drink alcohol. In order to best improve overall health, it is important to consider interrelationships between food and alcohol consumption.

Keywords

adolescence, behavioural medicine, drinking behaviour, eating behaviour, theory

Poor diet and alcohol use are key risk factors for chronic illness. Indeed, some estimate up to half of US deaths may be preventable through modification of health behaviours like these (Mokdad et al., 2004). Seminal health behaviour theories and behaviour modification techniques are applied to these behaviours one at a time. However, limited empirical work tests how changes in one type of health behaviour may cause changes in another health behaviour (Spring et al., 2015). Lack of such knowledge is an important problem, because without it, prevention and treatment strategies with the aim of changing one behaviour may be ineffective for (or even detrimental to) improving overall health.

There might be a particular link between diet and alcohol use. Common sense might tell us that food and alcohol are two substances that go hand in hand. Yet, decades ago nutritionists documented the opposite, such that drinkers eat *less food* than non-drinkers (Barboriak et al., 1978; Windham et al., 1983). These records align with an accumulating body of non-human animal and epidemiological research that indicates *inverse* relationships between how frequent an individual drinks alcohol and eating-related factors. The most robust example appears in several studies that report individuals who have higher body mass index (BMI) drink alcohol *less frequently* relative to individuals who have lower BMI (Colditz et al., 1991; Gearhardt and Corbin, 2009; Gearhardt et al., 2012; Kleiner

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et al., 2004; Liu et al., 1994; Pickering et al., 2011; Rohrer et al., 2005; Williamson et al., 1987). Another example is the recurrent observation that rats and humans who have received gastric bypass surgery drink *more alcohol*, while eating *less sugar- and fat-rich food* (Hsu et al., 1998; Thanos et al., 2012). Although these inverse associations are well documented, the mechanisms by which these associations emerge are not clearly identified.

One potential mechanism may regard shared reward processing for food and alcohol. When asked to swish sucrose solutions in their mouths and report how much they like the taste, individuals diagnosed with alcohol use disorders report liking the sweet solutions more than matched controls (see Kampov-Polevoy et al., 1999 for a review). Those with a family history of alcoholism report liking the taste the most, which suggests a physiological overlap in reward preference for food, or at least sweets, and alcohol (Kampov-Polevoy et al., 2001, 2003; Lange et al., 2010; Wronski et al., 2007). Indeed, in the last decade, much evidence has suggested that physiological reinforcement mechanisms implicated in alcohol use, including stimulation of opiate and dopamine neuronal reward pathways, are also implicated in the consumption of certain foods (see Brownell and Gold, 2012 for a comprehensive review).

This shared reward processing led some researchers to hypothesize that one potential way food consumption and alcohol use might interact is through physiological *competition* within individuals. That is, as the shared neuronal reward pathway 'is occupied by one of the behaviours (i.e. food consumption or alcohol consumption), it would block the other (Gearhardt and Corbin, 2009: 217)'. The logic here is similar to the use of naltrexone in alcoholism treatment. Just as naltrexone binds to neuronal reward pathways, blocks them from further stimulation, and decreases alcohol use, so too might food consumption (Volpicelli, 1992). Hereafter, we will label this the *food–alcohol competition hypothesis*.

Nevertheless, the evidence for this hypothesis is limited by three critical methodological concerns. First, almost all studies documenting an inverse relationship between alcohol use and BMI are cross-sectional, with only two prospective studies to our knowledge (Liu et al., 1994; Pickering et al., 2011). If food and alcohol compete within individuals, longitudinal data with multiple time points are necessary to examine *continuous time-related changes* in one behaviour relative to the other. Second, all the prior studies are conducted in adult samples. Although useful in its own right, this poses an issue: individuals have already started drinking alcohol. Using an adolescent sample, alternatively, represents a unique opportunity to capture how these behaviours interact *while individual drinking patterns are developing*. Third, researchers have used BMI and gastric bypass surgery status as proxies of food consumption in these prior studies. Neither is a direct measurement of how frequently or how much food an individual consumes, nor do they capture food characteristics.

The latter point is important because it is not yet established if specific macronutrients (e.g. sugar) or overall characteristics of food (e.g. energy density) activate neuronal reward pathways as strongly as alcohol. Palatability – or how pleasant the food is to taste – appears to be implicated most strongly (Avena et al., 2009). Previous research suggests foods comprised of both sugar and fat, termed *sweet high-fat foods*, are the most palatable (Drewnoski et al., 1992). However, some animal researchers find that sugar alone initiates a rapid reward response (Rada et al., 2005), and others find that fat alone does the same (Biedermann et al., 2012). In human research, it is even argued that palatability can be defined irrespective of any macronutrients, and that pleasant foods are unique to each individual (Rozin and Vollmecke, 1986). Nonetheless, a recent study identified high-processed foods, like those typically served at *fast food* restaurants, as initiating a physiological response most similar to drugs of abuse (Schulte et al., 2015). To understand why an inverse association between diet and alcohol use exists, it will therefore be critical to specify the food that may compete with alcohol.

Thus, there were three main objectives in this study: to examine longitudinal associations during adolescence between alcohol use and three types of food measurement: (a) total kilocalorie intake, (b) specific macronutrient consumption, and (c) processed (fast) food consumption. We hypothesized we would find support for food–alcohol competition. Given the field's mixed findings on the food implicated in reward processing, we hypothesized that food–alcohol competition would manifest across all types of food measurement. That is, we hypothesized there would be inverse longitudinal associations between alcohol use and total kilocalorie intake, specific macronutrient, and fast food consumption.

Method

Participants

The National Heart, Lung, and Blood Institute Growth and Health Study (NGHS) was a longitudinal study that followed 2379 Black ($n=1213$) and White girls ($n=1166$) annually from age 10 to age 19. There was 89 per cent retention in the final year. Participants who did not complete an assessment at age 19 ($n=297$) did not differ from those who did complete an assessment at age 19 in baseline alcohol use ($p=.24$).

Procedure

See Morrison et al. (1999) for a detailed description of the NGHS study recruitment, design, interviewing techniques, and physical examination. In brief, baseline and annual assessments included demographic information, dietary intake, and other health behaviours. Prior to age 15, the researchers did not ask the girls about alcohol use, and consequently only alcohol and food data collected from ages 15 through 19 were used. For this study, the University Office of the Human Research Protection Program approved all research activities.

Measures

Alcohol use. Participants responded to the following question regarding the frequency of alcohol use: *During the past 30 days, on how many days did you have at least one drink of alcohol?* Participants responded on a Likert scale ranging from 1 – *I have never had a drink of alcohol* to 8 – *All 30 days*. This operationalization of alcohol use is similar to that of the cross-sectional studies depicting an inverse relationship between alcohol use and BMI (Kleiner et al., 2004; Rohrer et al., 2005).

Food consumption. Researchers collected a 3-day food record from participants; these data were collected when the participants were 16, 17, and 19 years old. Trained dietitians instructed the girls on recording all food and drink, excluding alcohol, consumed during two consecutive weekdays and one weekend day within 30 days of the assessment visit. The researchers calculated the average total kilocalorie intake per day and grams consumed specific to the macronutrients of sugar and fat. Since previous research suggests *sweet high-fat foods* are the most palatable (Drewnoski et al., 1992), a sugar × fat interaction term was created by taking the cross product of the untransformed sugar and fat variables. This value was centred for a more stable model fit. Finally, to gauge processed food consumption, we examined participants' responses to the following question regarding frequency of eating fast food in the 30 days prior to assessment visit: *How often did you eat fast food from a place like McDonald's, Kentucky Fried Chicken, Pizza Hut, or any other place where you can buy fast food? (This means food eaten there or carried out).* Participants responded on a Likert scale ranging from 1 – *Never* to 7 – *More than 7 times a week*.

Covariates. Participants self-reported Black or White race. Parents or caregivers of the participants reported household income as one of four categories (less than US$5000, US$5000–US$20,000, US$20,000–US$40,000, or US$ 40,000 or more) and highest educational attainment (less than high school, 1–3 years post-high school, or 4-year college degree or more) at the baseline assessment. Finally, participants responded to a question at each assessment

Table 1. Growth factor mean, variance, covariance estimates, and residual scores.

	Intercept		Slope		Intercept × slope covariance	Residual variances[a]					Model fit	
	Mean	Variance	Mean	Variance		Age 15	Age 16	Age 17	Age 18	Age 19	CFI	RMSEA
Alcohol use	1.07***	.02***	.11***	.003***	−.003***	.77***	.49***	.50***	.46***	.43***	.89	.11
Food consumption												
Kilocalories[b]	7.47***	.08***	.02*	.01**	−.02*	–	.38***	.53***	–	.52***	.98	.08
Sugar[b]	4.74***	.16***	.02*	.01*	−.02**	–	.49***	.56***	–	.63***	1.00	<.001
Fat[b]	4.19***	.16***	−.01	.01*	−.02**	–	.42***	.51***	–	.46***	1.00	.05
Sugar x fat[b]	8.91***	.16***	.02*	.01*	−.02**	–	.47***	.49***	–	.46***	1.00	<.001
Fast food	3.10***	.77***	.07***	.05***	−.08***	.57***	.67***	.82***	.80***	.77***	.96	.07

CFI: Comparative Fit Index; RMSEA: Root Mean Square Error of Approximation.
All analyses performed on log-transformed scores.
[a]Standardized values reported.
[b]Model estimation conducted with data at ages 16, 17, and 19; time factor loadings set at 1, 2, and 4.
*$p < .05$; **$p < .01$; ***$p < .001$.

asking if they had started their period. Age of menarche was derived from this question across all assessments.

Data analysis

Initial descriptive examination of all variables revealed skew and kurtosis, which were corrected via natural log transformations. Unconditional growth models were fit to alcohol and food consumption variables separately to select the form of the individual growth curve trajectories; all variables, except for fat intake, individually, evidenced positive slopes such that individuals were increasing in consumption over time (Table 1). To evaluate longitudinal change in alcohol use and food consumption relative to one another, multivariate growth models estimated the covariance between the intercept and slopes of these variables. Covariance pathways within these variables' intercepts and slopes were included in the model to provide the most conservative tests of significance. In Step 1, multivariate growth model analyses were conducted without covariates. Step 2 included significant covariates identified by univariate growth model analyses. All model estimations were conducted in MPlus 6.12 using the conventional Full Information Maximum Likelihood with missing-at-random assumptions. Since total kilocalorie and macronutrient data were only available for ages 16, 17, and 19, only alcohol use data for those ages were used in those models.

Results

Descriptive statistics appear in Table 2. In Step 1, no prospective relationships between intercept and slope estimates, or associations between slope estimates, emerged between alcohol use and total kilocalorie intake, alcohol use and sugar intake, and alcohol use and fat intake. No prospective relationships between intercept and slope estimates emerged between alcohol use and sugar × fat intake. In contrast, slope estimates for alcohol use and sugar × fat were significantly negatively associated. Given that consumption increased over 5 years, this indicates that there were individual-level attenuations in the group-level increases. Adolescent girls increasing in intake of foods high in sugar and fat from age 15 to 19 were not increasing in their alcohol use frequency from age 15 to 19. Reciprocally, adolescent girls increasing in their alcohol use frequency were not increasing in intake of foods high in sugar and fat.

There were significant prospective associations between intercept and slope estimates for alcohol use and fast food consumption. Adolescent girls who ate fast food less frequently at age 15 increased in frequency of alcohol use from age 15 to 19. Adolescent girls who used alcohol less frequently at age 15 increased in frequency of fast food consumption from age 15 to 19. No significant

Table 2. Descriptive statistics, and covariate relationships with estimates of intercept and slope.

	Mean (SD)[a]					Covariance with Intercept/Slope			
	Age 15	Age 16	Age 17	Age 18	Age 19	Race[b]	Household income	Parental education	Age of menarche
Alcohol consumption	1.92 (1.05)	2.41 (.81)	2.51 (.88)	2.68 (1.05)	2.93 (1.23)	−.17***/−.17***	−.14***/.19***	−.10**/.15***	−.13**/.15***
Food consumption									
Total[c]	–	1883.25 (657.42)	1844.52 (652.24)	–	1904.78 (665.59)	.17***/−.04	−.02/−.07	−.02/.02	.09*/−.02
Sugar[d]	–	127.64 (56.00)	130.51 (58.13)	–	130.51 (59.37)	.09**/−.02	.02/−.01	−.004/−.001	.02/−.002
Fat[d]	–	71.97 (32.25)	69.70 (32.14)	–	69.77 (33.11)	.26***/.02	−.06/−.09	−.09*/−.04	.05/.03
Fast food	3.04 (1.13)	3.14 (1.17)	3.36 (1.24)	3.36 (1.25)	3.29 (1.27)	.23***/.01	.02/.02	−.18***/−.06	.02/−.001

SD: Standard Deviation.
[a]Non-transformed scores used.
[b]Race was dummy coded as 0 = White and 1 = Black.
[c]Total food consumption was measured in kilocalories.
[d]Sugar and fat consumption were measured in grams.
*p < .05; **p < .01; ***p < .001.

association emerged between slope estimates. In Step 2, including the covariates, this trend of results remained but the association between alcohol use and sugar × fat intake slopes attenuated to marginal significance, and the relationship between fast food consumption intercept and alcohol use slope became non-significant. Figure 1 illustrates the relationships between alcohol use and total kilocalorie intake, sugar × fat intake, and fast food consumption. Multivariate growth model estimates are presented in full in Table 3.

Discussion

This study suggests that the tendency to consume *processed* or *sweet high-fat* foods may compete with a tendency to consume alcohol. This competition arose over 5 years during adolescence, a period where one might expect food–alcohol competition to emerge in response to drinking behaviour development. In contrast, no competition (nor any significant relationships) emerged between alcohol use and total kilocalorie intake, and between alcohol use and sugar intake and fat intake, separately.

These results add support for the food–alcohol competition hypothesis, but only between alcohol and processed or sweet high-fat foods. This finding is critical as it, first, supports the hypothesis that shared reward processing is a mechanism involved in the relationship between alcohol consumption and eating behaviour. Second, it suggests that only these types of foods can initiate a reward response comparable to alcohol, and therefore compete with alcohol. This corroborates prior animal research suggesting that foods containing both sugar and fat activate neuronal reward pathways most strongly (Avena et al., 2009), and human research suggesting that processed foods initiate the strongest physiological reward response (Schulte et al., 2015). It challenges prior work that contends only one macronutrient is needed to create a reward response similar to alcohol (Biedermann et al., 2012; Rada et al., 2005). Here, we tested the interaction of sugar and fat intake and fast food consumption as measures of palatable food; yet, we believe future research can develop

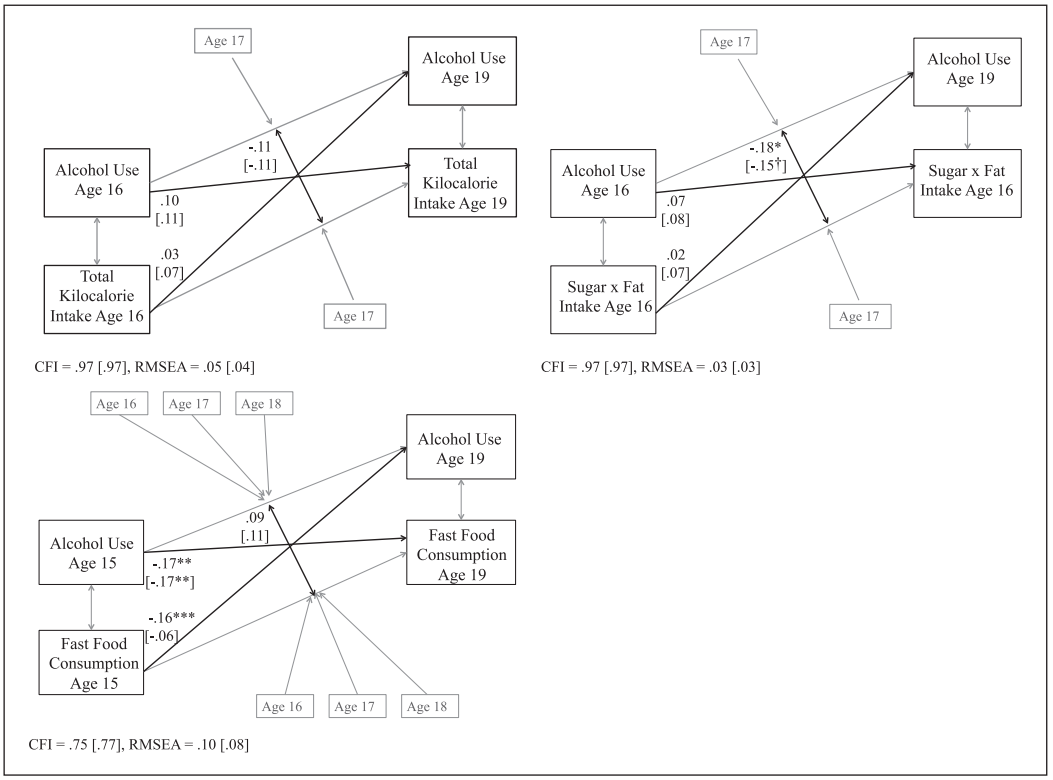

Figure 1. Multivariate growth models between alcohol use and total kilocalorie intake, sugar × fat intake, and fast food consumption. Grey lines represent covariance pathways within variables' intercepts and slopes for the most conservative test of significance. Black lines represent tested longitudinal relationships. Step 2 model estimates adjusting for the covariates of race, household income, parental education, and age of menarche are bracketed. Standardized values reported.
CFI: Comparative Fit Index; RMSEA: Root Mean Square Error of Approximation.
†$p = .051$; *$p < .05$; **$p < .01$; ***$p < .001$.

from this definition. Randomized experiments manipulating consumption/restriction of different palatable foods/alcohol and observing changes in alcohol/food consumption could elucidate causal relationships. For instance, might unprocessed foods high in sugar and fat (e.g. avocados, coconuts) compete with alcohol?

This study's results suggest novel strategies for health behaviour change. For example, if the goal is to reduce adolescent drinking, it may be beneficial to pose consumption of processed/sweet high-fat foods as an attractive alternative, particularly at occasions when youth have the opportunity to drink alcohol. For other populations where drinking is of major concern, like individuals with alcoholism, perhaps eating processed/sweet high-fat foods could serve as a harm reduction approach to regulate and reduce alcohol cravings. This is a practice that exists anecdotally in the community, as illustrated by a passage in Alcoholics Anonymous' (1975) book *Living Sober*:

> This booklet is based on our own personal experience, rather than on scientific reports. So we cannot explain precisely, in technical terms, why this should be so. We can only pass on the word that thousands of us – even many who said they had never liked sweets – have found that eating or drinking something sweet allays the urge to drink. (p. 22)

Table 3. Longitudinal relationships between alcohol use and food consumption.

	Alcohol use Intercept with	Alcohol use Slope with		Model fit	
	Slope	Intercept	Slope	CFI	RMSEA
Step 1					
Food consumption					
Kilocalories[a]	.10	.03	−.11	.97	.05
Sugar[a]	.03	.09	−.14	.96	.05
Fat[a]	.05	−.07	−.11	.98	.04
Sugar × fat[a]	.07	.02	−.18*	.97	.03
Fast food	−.17**	−.16***	.09	.75	.10
Step 2					
Food consumption					
Kilocalories[a]	.11	.07	−.11	.97	.04
Sugar[a]	.03	.10	−.15	.97	.04
Fat[a]	.05	−.07	−.11	.98	.04
Sugar × fat[a]	.08	.07	−.15†	.97	.03
Fast food	−.17**	−.06	.11	.77	.08

CFI: Comparative Fit Index; RMSEA: Root Mean Square Error of Approximation.
Step 1: multivariate growth analyses without covariates. Step 2: multivariate growth analyses adjusted for the covariates of race, household income, parental education, and age of menarche. All analyses performed on log-transformed scores. Standardized values reported.
[a]Model estimation conducted with data at ages 16, 17, and 19; time factor loadings set at 1, 2, and 4.
†$p = .051$; *$p < .05$; **$p < .01$; ***$p < .001$.

These strategies might raise concerns of increased obesity rates; yet those who drink alcohol more frequently already tend to have lower BMIs, and harm reduction approaches focus on the individual to find a strategy that mitigates the most salient harm in his or her life (Marlatt, 1996). Nevertheless, we believe it is too early to make clinical recommendations. It will be critical for future work to weigh benefits against risks and devise solutions that harness the power of food–alcohol competition, while minimizing negative health consequences.

The results should be interpreted in light of study limitations. Although from a large sample, the adolescent participants had lower frequencies of alcohol use compared to studies of heavy adult drinkers, which might have restricted the range. Additionally, alcohol use was measured by a single-item once per year. A more comprehensive measure of alcohol use in a sample of heavier drinkers would provide the opportunity to specify how food–alcohol competition functions. For example, future work that measures drinking quantity along with drinking frequency can test for dose–response effects, or up to what alcohol content sweet high-fat/processed foods can compete with. Studies that include measurement of binge drinking occasions – a type of drinking pattern that may be especially relevant to this age group – can test pivotal clinical questions such as: does increasing palatable food consumption in adolescence not only attenuate frequency of drinking, but also decrease the likelihood of a binge-level dose?

Like most in this work (see Gearhardt and Corbin, 2009 for exceptions), this sample also included only females. Thus, future replicative work could use longitudinal samples inclusive of both genders and heavier drinking frequencies; this type of replication could also address how gender may moderate food–alcohol competition. Moreover, this study did not include measurement of individual difference factors

typically assessed with drinking behaviour, such as family history of alcoholism. Inclusion of genetic moderators, in particular, could strengthen future research; if genes intensify the inverse relationship between sweet high-fat/processed foods and alcohol, it lends support to the contention that physiological pathways are involved in the competition between these rewards. This kind of logic was used in one prior study that demonstrated the inverse relationship between alcohol consumption and BMI was stronger for those with a family history of alcoholism (Gearhardt and Corbin, 2009).

As a final point of discussion, although prior research and the pattern of our results lend support to physiological competition between these two rewards, the self-report nature of these data does not directly test this mechanism. Alternative mechanisms might then also explain the competition. Indeed, in some cases, the statistical significance of the food–alcohol competition models weakened to marginal or no significance when adding covariates. This suggests that these covariates may play a role in food–alcohol competition, and that food–alcohol competition might arise for reasons beyond overlapping physiological reward pathways. For example, income may limit how much money can be spent on food versus alcohol, adding a potential economic competition between the two rewards. Also, culture (for which race served as a proxy in our study) might shape which rewards are preferred. For instance, it is a norm among African Americans that alcohol need not to be integrated into aspects of social life, such as mealtimes, religious activities, and celebrations (Zapolski et al., 2014). Might this cultural practice decrease alcohol consumption and instead promote consumption of sweet high-fat foods? We recommend future work not only continue to measure these critical covariates, but perhaps with tailored questions (e.g. *In times of financial struggle, do you buy alcohol as frequently as food? Do you feel that your culture encourages you to drink less alcohol?*) to begin to test alternative mechanisms.

Overall, this study is methodologically and theoretically unique in its examination of how change in one health behaviour longitudinally relates to change in another. The results provide insight on the food–alcohol competition hypothesis, specifically regarding the nature of food (only processed, sweet high-fat foods) potentially implicated in these processes. Future work will contribute to theory on food–alcohol competition, enabling us to determine precisely how these health behaviours interact at the individual level. This type of research echoes calls to implement multiple health behaviour research and intervention approaches in prevention of chronic illness (Prochaska, 2008). There are unresolved questions about which behaviours can be intervened upon simultaneously in order to maximize positive behavioural synergies and minimize negative ones (Spring et al., 2015). Palatable food consumption and alcohol use might be two particular behaviours that require co-action. In order to best improve overall health, it is therefore essential to carefully consider interrelationships between these behaviours in current health behaviour theory and behaviour modification.

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