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Short-Term Effects of Alcohol Consumption on Appetite and Energy Intake

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POPPITT, S. D., J. W. ECKHARDT, J. MCGONAGLE, P. R. MURGATROYD AND A. M. PRENTICE. *Short-term effects of alcohol consumption on appetite and energy intake.* PHYSIOL BEHAV **60**(4) 1063–1070, 1996.—The relationship between alcohol intake and obesity remains uncertain. Evidence suggesting that alcohol-derived energy may be unregulated points to an inability to maintain appetite, energy balance and, hence, body weight when alcohol is introduced to the diet. This study investigated the short-term effects of alcohol on hunger and energy intake in 20 lean women. On 4 occasions, subjects were given a randomised preload drink ('alcohol', 'no alcohol', 'carbohydrate', 'water') followed by visual analogue scales (VAS) rating hunger and an *ad lib* test meal. There was no difference in hunger ratings ($p > 0.05$) nor in the amount of energy consumed during the test meal ($F = 1.66$, $p > 0.05$) following any of the 4 preloads. Consumption of the 2 high energy preload drinks ('alcohol', 0.91 MJ; 'CHO', 0.72 MJ) did not result in a compensatory decrease in the amount of energy subsequently eaten (*ad lib* intake: 'alcohol' = 2.62 MJ, 0.32 SEM; 'no alcohol' = 2.98 MJ, 0.28 SEM; 'CHO' = 2.93 MJ, 0.21 SEM; 'water' = 2.82 MJ, 0.25 SEM), suggesting either no physiological recognition or no regulation of energy consumed within a drink in quantities of less than 1 MJ. The addition of either alcoholic or CHO-containing carbonated beverages into the diet will result, in the short-term, to an overall increase in energy intake.

Alcohol CHO Appetite Hunger Satiety Energy intake

RECENT evidence suggests that weight gain and the development of obesity are primarily a disorder of appetite and regulation of energy intake rather than one of metabolism (33,40–42). Although the physiological mechanisms regulating appetite still remain poorly understood, central to the control of energy intake are the individual metabolic fuels, fat, carbohydrate (CHO), and protein plus, in a large proportion of the population, alcohol. On an isoenergetic basis, the individual macronutrients appear to have quite different satiating effects. Although the relative effects of fat, CHO, and protein on subsequent appetite are gradually becoming established (1,5,7,18,23,28,38), very little is known about the effects of alcohol on hunger, satiety, energy intake, and body weight. Alcohol consumption comprises on average between 8–10% of the total daily energy intake in the UK (25), a pattern also seen in many other western countries, leading to concern about the effect of alcohol on body fatness. Many questions remain unanswered, particularly in moderate or 'social' drinkers. Does alcohol consumption increase appetite, total energy intake, and adiposity and, if so, why does the body not recognise alcohol-derived energy and regulate its intake as it does for the other macronutrients?

An alcoholic aperitif just prior to a meal has long been anecdotally suggested to enhance appetite, despite the high energy content of ethanol (29 kJ/g) which, if recognised as a conventional energy source by the body, should suppress both appetite and intake. The suppression of gluconeogenesis by ethanol (13,17), and resultant hypoglycemia (51), has been suggested as a possible mechanism. Emotional disinhibition also commonly follows the consumption of alcohol (45), potentially resulting in an increase in energy intake. Certainly, it is well known that alcohol ingestion is associated with changes in eating behaviour, including an extended meal duration (10) and, in association with the time of day, larger meal size. This may, in fact, be a consequence of alcohol ingestion promoting socialisation, a factor that, in itself, has been shown to have a strong effect on food intake, an increase in the number of people at an eating occasion commonly increasing the amount of food consumed (9).

Community survey studies tend to support the anecdotal evidence, showing that, in moderate alcohol consumers, total energy intake increases when alcohol is introduced into the diet (2,10,14,34,44). This suggests that alcohol-derived energy either isn't recognised or isn't regulated by the body, that appetite remains unchanged or potentially heightened and, therefore, there is no compensatory decrease in the subsequent amount of food eaten.

If alcohol intake is unrecognised as an energy source and there is no compensation by the other macronutrients, the increase in total energy intake in alcohol consumers may lead to a small, yet

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significant, positive energy balance and eventually to gradual weight gain. Drinking even moderate amounts of alcohol over a sustained period should increase body fatness. Epidemiological evidence suggests quite the contrary, however, showing that, certainly in women, moderate alcohol consumers tend to have a lower BMI and so are thinner than individuals who abstain from alcohol (6,8,26). In addition, it has also been suggested from studies conducted in a metabolic ward that the long-term addition of up to 8.5 MJ of alcohol per day results in no additional weight gain (43). This leads to speculation that alcohol is metabolised less efficiently than other substrates, a large amount of energy being lost as heat during futile cycling (35,56). This has since been refuted in a number of metabolic studies showing no greater heat production following alcohol than carbohydrate or fat ingestion (49,53,60). This discrepancy remains unresolved and has lead to a resurgence of interest in the role of alcohol in the control of appetite and energy intake, and subsequent effect on body weight.

Although numerous studies have attempted to determine the relationship between alcohol and food intake in community-based studies, often relying on data from dietary records (2,10,14,34,44), very little information is available from well-controlled metabolic studies as to the direct effect of alcoholic beverages on subsequent appetite and energy intake. Only 2 previous studies (16,57) have measured the effect of ethanol on subsequent food intake within a metabolic facility. One of these studies concluded that alcohol-derived energy appears to be under normal physiological regulation, decreasing food intake in a manner similar to that of an iso-energetic CHO source (16) although, most crucially, addition of both alcohol and CHO into the diet resulted in only partial compensation (37%) for the additional energy. A number of current hypotheses suggest that oxidative signals are important in the regulation of satiety (19,20,36,37). Certainly, both CHO and alcohol have rapid autoregulatory responses, and produce oxidative metabolites (33,51,53,54) that may act as a strong satiety signal, reducing both hunger and energy intake. If correct, it would predict that alcohol is at least as satiating as CHO.

The more recent study by Tremblay and coworkers (57) showed even less regulation for alcohol, concluding that when alcohol is consumed during a meal its energy content is not compensated for by an equivalent decrease in energy intake from other macronutrients and, thus, total energy intake increases when alcohol is consumed. It is certainly difficult to interpret the epidemiological data of an inverse relationship between alcohol consumption and body weight (6,8,26) in light of these well-controlled metabolic studies (16,57).

In this study, we aimed to test, first, the hypothesis that alcohol-derived energy may be recognised and regulated by the body, reducing hunger, increasing satiety, and decreasing food intake immediately following an alcoholic beverage and, second, whether or not any decrease in food intake following alcohol consumption is equivalent to that following a nonalcoholic drink of similar energy content. To test these hypotheses, subjects were given 4 preload drinks of varying energy content, of which only 1 contained alcohol. Cognitive hunger ratings and subsequent energy intake was also assessed.

METHODS

Subjects

Twenty female volunteers took part in the study. All subjects were healthy, lean (body mass index, BMI = 23.0 ± 2.8 kg/m2), nonsmokers, aged between 18 and 57 years (37.0 ± 11.4 years). Subjects completed the Dutch Eating Behaviour Questionnaire (DEBQ; 59) to determine levels of cognitive dietary restraint. The median value was calculated and the data separated into 2 groups of equal size, representing 'unrestrained' (below the median value) and 'restrained' (above the median value) eaters. All subjects provided written consent and were informed that the entire nature of the study could not be revealed. Subjects also were informed that, on one occasion, the preload drink would contain 31 g of alcohol. The study was approved by the Unit's ethics committee.

Study Design

Subjects attended the unit on 4 separate occasions, each of 1-day duration. On each occasion, a randomly assigned preload drink to be fully consumed within 15 min was given, followed at 30 min (t_{30}) by an outcome meal. The short time period of 30 min between presentation of the preload and presentation of the outcome meal was chosen, both to represent the 'real-life' situation of an alcoholic aperatif prior to a meal and as the time period previously suggested to best detect macronutrient-specific effects on subsequent energy intake (48). The meal was provided in excess and subjects allowed to eat as much or as little of both items as they chose. All subjects consumed the preload and outcome meal in isolation to minimise the influence of social interactions, which have been shown to strongly influence the amount of food eaten (9). Before and immediately after the preload drink, 100 mm visual analogue scales (VAS) rating 'hunger', 'fullness', 'desire to eat', 'thoughts of food', 'urge to eat', and 'preoccupation with food' were completed, by subjects placing a mark on each of the 6 scales (27). All measurements of food intake were covert. Subjects were informed that the purpose of the study was to assess the effect of alcohol on blood glucose levels. Small finger prick blood samples were collected prior to and immediately following the preload and analysed for blood glucose using a Glucometer GX™ (Bayer Diagnostics, Evans House, England). This was intended, in part, to provide a distraction from the major purpose of the study and to maintain its covert nature.

Preload Drinks

Four different preload drinks were given in randomised order (Table 1). The 4 treatments were 1. +alcohol ('alcohol'); 2.−alcohol, −energy ('no-alcohol'); 3. −alcohol, +energy ('CHO'); 4. water ('water'). Drink 1 contained 0.89 MJ of alcohol given as 92 g gin (31 g alcohol, Beefeaters™, James Burrough, London) plus 300 g slimline tonic (Coca-Cola™ &

TABLE 1

THE ENERGY AND MACRONUTRIENT CONTENT OF THE 4 PRELOAD DRINKS

	Energy	Alcohol		CHO	
Preload Drink	MJ	g	MJ	g	MJ
1. + Alcohol (alcohol)	0.91	30.6	0.89	1.4	0.02
2. − Alcohol, − energy (no alcohol)	0.03	0	0	1.8	0.03
3. − Alcohol, + energy (CHO)	0.72	0	0	45.4	0.72
4. Water	0.00	0	0	0	0

Drink 1 contained alcohol; drink 2 replaced the alcohol with an alcohol-flavoured placebo; drink 3 replaced the alcohol in part with CHO; drink 4 contained only water.

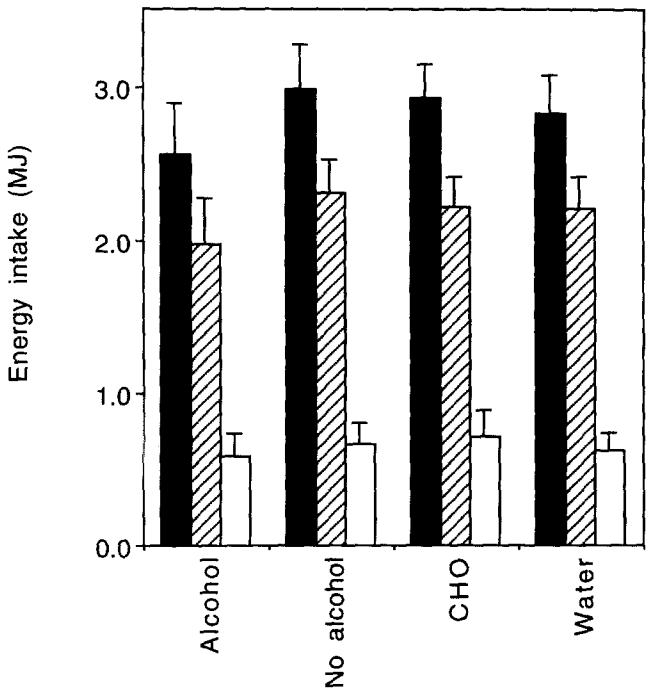

FIG. 1. Mean (SEM) energy intake of an *ad lib* test meal (■), comprising spaghetti bolognese (▨) and fruit loaf dessert (□), given 30 min after preload drinks of alcohol, no alcohol, carbohydrate (CHO), or water. See text for composition of preload drinks.

Schweppes™ Beverages Ltd., Uxbridge, England). Drink 2 was a low energy gin-flavoured placebo containing no alcohol, (gin flavouring from International Flavour & Fragrances Ltd., Haverhill, England), 92 g water and 300 g slimline tonic. Drink 3 was a high-energy gin-flavoured placebo, where the alcohol energy was replaced, in part, by a CHO source, comprising 22.4 g Polycal™ (Cow & Gate Nutricia Ltd., Uxbridge, England) dissolved in 392 g tonic water. The tonic water contained a total of 23 g sucrose. Drink 4 was a control treatment comprising 392 g water alone. Subjects were blinded to the randomisation of treatments. Although the preloads cannot be truely covert, because alcohol has specific pharmacological in addition to sensory properties, the use of gin-flavoured placebo commonly prevented subjects from correctly assigning the presence or absence of alcohol within the drinks.

Protocol

Subjects arrived fasted at the metabolic facility at 0900 h each day and remained sedentary within the confines of the unit until 1900 h each evening, when they were free to return home. While in the metabolic suite, subjects were free to watch TV, read, or take part in similar sedentary activities only. They were prevented from taking any exercise or leaving the unit at any time during the day. Breakfast and lunch, identical on each occasion, were provided at 0900 h and 1300 h, respectively. Breakfast comprised toast and cornflakes (25% fat, 60% CHO, 15% protein) and lunch comprised chicken and rice rissotto (40% fat, 45% CHO, 15% protein). The energy content of these meals was calculated on an individual basis as 1.4 times predicted basal metabolic rate (BMR; 50). Breakfast provided 25% and lunch 33% of the predicted daily intake for each subject. This was intended to maintain the subjects in energy balance and at a constant level of appetite prior to presentation of the preload. No other food was available.

At 1700 h (t_0) VAS scales were completed and the test preload drink, served cold with ice and fresh lemon, was presented to the subjects who were asked to consume the drink within 15 min. VAS scores were repeated at 1715 h (t_{15}). The ad lib outcome meal was given at 1730 h (t_{30}). The meal consisted of 2 courses, both comprising 40% fat, 45% CHO, and 15% protein. The first course was spaghetti bolognese, served as finely chopped spaghetti thoroughly mixed with a meat and tomato sauce and presented in a large casserole serving dish. Subjects were given a constant amount of spaghetti (approximately 1 kg) on each occasion and told to serve themselves freely from this dish. This was followed by a fruit loaf dessert that was cut into 8–10 slices before serving. Both courses were served in excess and were designed to be of homogeneous composition, which ensured that the macronutrient profile was maintained irrespective of the weight of food eaten by the subjects. The meal was covertly weighed before and after consumption to determine the weight and energy content of the food eaten.

Analysis

The effect of the preload drinks on ratings of appetite and energy intake were assessed using 1-way analysis of variance (ANOVA) and Scheffe's post hoc test, using the Data Desk version 4.0 program (Odesta Corporation, Northbrook, Il, USA). Results were analysed including both subject and treatment effects. The retrospective calculation of the limits of detection for between-treatment changes in energy intake were made using the equation:

$$n = 2[(Z_{2\alpha} + Z_{2\beta})\sigma]^2/\delta_o^2 \quad (1)$$

where n, number of subjects; α and β, type I and type II errors ($Z_{2\alpha} = 1.96$, $Z_{2\beta} = 0.842$); σ, average standard deviation of the 2 treatments; δ_o, difference between the treatments to be detected. Results are given as mean ± SEM. Statistical significance was at the 95% confidence level ($p < 0.05$).

RESULTS

Energy Intake

There was no significant difference in the amount of energy consumed during the *ad lib* test meal following any of the 4 treatment preloads ($F = 1.66$, $p > 0.05$, Fig. 1), when analysed for both treatment and subject effects, although intake following the consumption of alcohol tended to be slightly lower than following the consumption of the other 3 preloads (alcohol = 2.62 MJ, 0.32 SEM; no-alcohol = 2.98 MJ, 0.28 SEM; CHO = 2.93

TABLE 2

ENERGY CONTENT, WEIGHT, AND ENERGY DENSITY OF FOOD CONSUMED DURING A TEST MEAL FOLLOWING THE CONSUMPTION OF 4 RANDOMISED PRELOAD DRINKS

	Test Meal		
Preload Drink	Energy (MJ)	Weight (g)	Energy Density (kJ/g)
1. Alcohol	2.62 (0.32)	566.3 (71.3)	4.63
2. No alcohol	2.98 (0.28)	646.0 (62.8)	4.63
3. CHO	2.93 (0.21)	642.2 (46.0)	4.63
4. Water	2.82 (0.25)	612.5 (53.0)	4.63

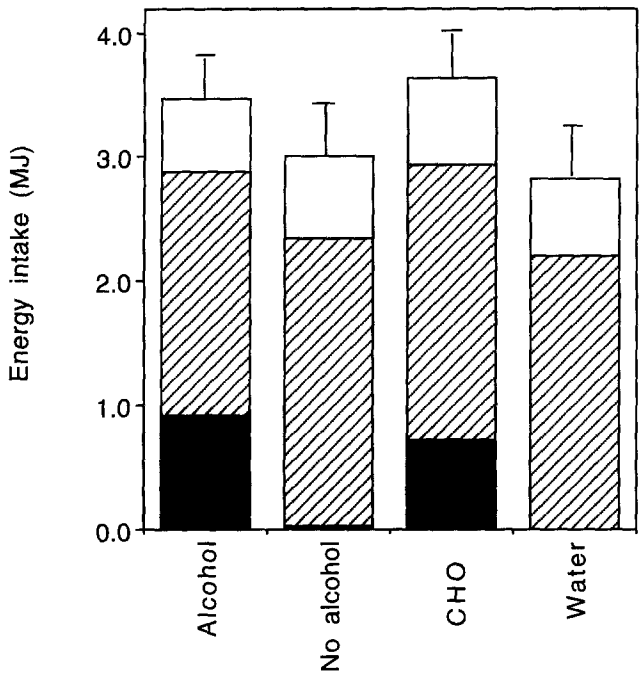

FIG. 2. Mean energy intake (SEM), including the energy content of the preload beverages (■), spaghetti bolognese (▨) and fruit loaf dessert (□), following the 4 preload drinks. CHO, carbohydrate.

MJ, 0.21 SEM; water = 2.82 MJ, 0.25 SEM)(Table 2). Between-subject variability was highly significant for all 4 treatments ($F = 11.8, p < 0.001$). The difference between the alcohol treatment and the water control was only 200 kJ, considerably less than 450 kJ which, estimated from Eqn (1), is required to achieve significance in a study comprising 20 subjects. There was also no difference in the amount of energy consumed in response to the high-energy preloads (alcohol, CHO) vs. the low energy preloads (no-alcohol, water). There was, therefore, no compensation in the amount of food eaten following the addition of less than 1 MJ of either alcohol-derived or CHO-derived energy to the diet, suggesting that there is either no physiological recognition or no response to this amount of energy when consumed in the form of a liquid drink.

There was a significant difference in energy intake for the total meal (calculated as test preload + outcome meal) between the high-energy and the low-energy treatments (High energy: alcohol = 3.53 MJ, 0.32 SEM; CHO = 3.65 MJ, 0.21 SEM; Low energy: no-alcohol = 3.01 MJ, 0.28 SEM; water = 2.82 MJ, 0.25 SEM. $F=7.95, p < 0.05$). This is a consequence of the lack of compensatory decrease in the amount of food eaten following either of the high energy preloads, alcohol, and CHO (Fig. 2). The average total daily energy expenditure (TEE), calculated as 1.2–1.4 × predicted BMR, was 6.8–7.9 MJ/day. To maintain energy balance, because an average of 4.6 MJ was given as breakfast and lunch (which represented 58% of the required daily intake), required an intake of 2.2–3.3 MJ during the evening. The actual intake of the subjects was similar to this on all treatments, confirming that, under the slightly artificial conditions of the metabolic suite, women were neither under- nor overeating substantially.

Appetite Ratings

Only when the alcohol preload was given prior to the test meal was there a decrease in subjective ratings of appetite in response to the drink. This failed to reach significance, however, with the exception of the single question 'How hungry do you feel?' ($F = 5.94, p < 0.05$). Following the no-alcohol, CHO, and water preloads, there was either no effect of the drink on subjective ratings or appetite increased slightly (Fig. 3), particularly surprising following the 0.72 MJ CHO preload.

Figure 4 shows the significant correlation between the subjective hunger ratings and the amount of energy consumed in the test preload when analysed for all treatments ($p < 0.001$), for the questions 'How hungry do you feel?' ($r = 0.37$), 'How strong is your desire to eat?' ($r = 0.45$), 'How much do you think that you could eat now?' ($r = 0.45$), 'Urge to eat?' ($r = 0.41$) and 'Preoccupation with thoughts of food?' ($r = 0.48$). This positive correlation shows that the amount of energy consumed is directly related to cognitive hunger. Only the question 'How full do you feel' was not significantly correlated with intake ($r = 0.10, p > 0.05$). This question is quite different from the other ratings, addressing feelings of satiety rather than hunger. The positive correlation between ratings and intake was maintained for all treatments, with the exception of the high-energy CHO preload (Table 3).

Blood Glucose

Figure 5 shows the change in blood glucose concentrations following both the preload drink and the outcome meal. Immediately following the preloads, there was no significant change in glucose levels in any of the 4 treatments ($F = 1.017, p > 0.05$). However, as would be predicted, consumption of CHO did result in a slight rise [baseline = 4.40 mmol/L, 0.36 SEM; posttreatment = 5.00 mmol/L, 0.51 SEM] and the no-alcohol and water preloads to a slight decrease in blood glucose

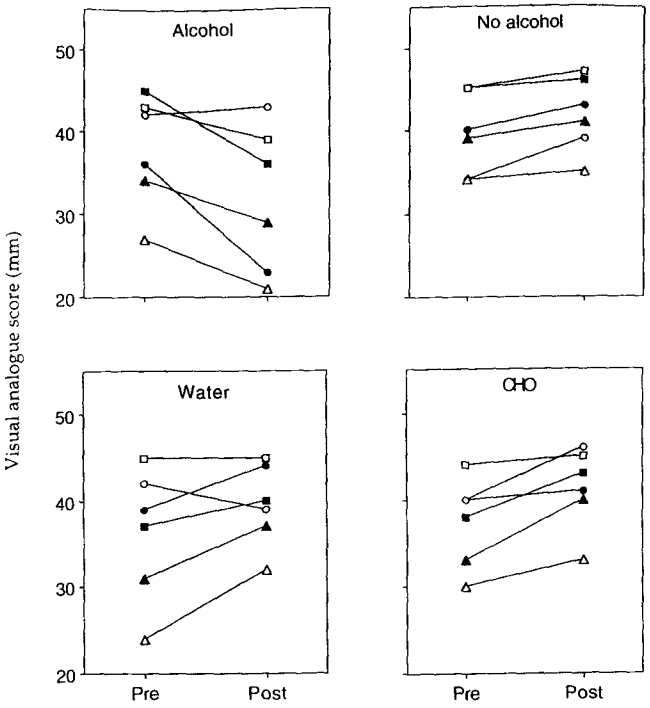

FIG. 3. Changes in visual analogue scale (VAS) rated feelings of hunger pre- and postpreload (How hungry do you feel?, ●; How full do you feel?, ○; How strong is your desire to eat?, ■; How much do think that you could eat now?, □; Urge to eat?, ▲; Preoccupation with thoughts of food?, △). CHO, carbohydrate.

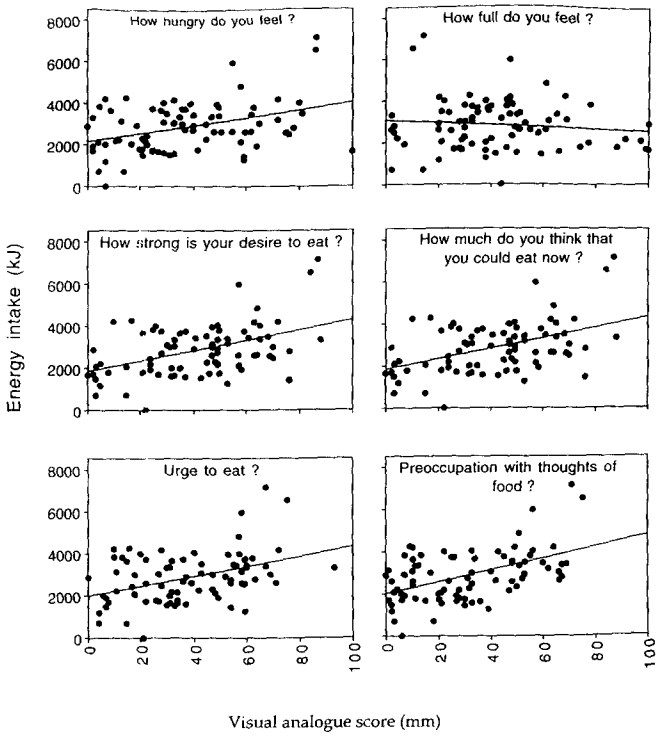

FIG. 4. Correlations between visual analogue scale (VAS) rated feelings of hunger and energy intake following the 4 preload treatments.

concentration [no-alcohol: baseline = 4.43 mmol/L, 0.47 SEM; posttreatment = 4.12 mmol/L, 0.58 SEM; water: baseline = 4.62 mmol/L, 0.51 SEM; posttreatment = 3.62 mmol/L, 0.36 SEM)]. Alcohol also caused a small decrease in glucose concentration [baseline = 4.76 mmol/L, 0.69 SEM; posttreatment = 3.98 mmol/L, 0.36 SEM].

'Restrained' vs. 'non-restrained' eaters

The median restraint score for the entire group was 2.7. Subjects were categorised as 'unrestrained' (< 2.7) or 'restrained' (> 2.7) eaters. Energy intake, when analysed across all treatments, was lower, although not significantly, in the 'restrained' group ('restrained' = 2599.8 kJ, 161 SEM 'non-restrained' = 3113.1 kJ, 192 SEM; $t = 1.922, p > 0.05$). There was no effect of treatment on energy intake in either group, suggesting that the inclusion of a large proportion of self-reported 'restrained' eaters in the study, in whom physiological control may potentially be overridden by cognitive control, did not alter the outcome ('restrained', $F = 0.746, p > 0.05$; 'non-restrained', $F = 0.024, p > 0.05$).

DISCUSSION

To maintain a constant body weight, energy intake and energy expenditure must, necessarily, balance. If the energy consumed increases above expenditure, body energy stores must increase, as will body fatness. In normal-weight individuals, energy balance appears to be maintained, at least in part, by the subconscious control of appetite that ensures that short-term increases in food intake are compensated for by equivalent periods of reduced food intake. This study was designed to test the hypothesis that similar compensation in energy intake occurs following the consumption of both alcohol-derived and CHO-derived high-energy drinks.

The results showed that the consumption of an alcoholic drink 15 min prior to eating had no significant effect on the amount of energy consumed during the subsequent meal. This confirms the results of Tremblay (57), who also showed that, when alcohol was consumed during a meal, its energy content was not compensated for by an equivalent decrease in nonalcohol-derived energy intake. The absence of a significant compensatory decrease in food intake in our study cannot, however, be attributed simply to the inability to detect alcohol-derived energy *per se* because there was no significant difference in intake following any of the 4 preload treatments. Although the inability of the body to detect a difference between the alcohol-derived and the CHO-derived energy is perhaps unsurprising, because isoenegetic manipulations of macronutrients alone may be relatively weak and have been shown, under certain conditions, to elicit comparable changes in intake (11,15,21,55), the absence of any difference in energy intake following both the 2 high-energy and 2 low-energy treatments is more difficult to explain. In this study, neither alcohol-derived nor CHO-derived energy appears to be recognised by the mechanisms controlling food intake, when consumed in quantities of up to 1 MJ.

When analysed in cross-section, the subjective appetite ratings recorded after each of the 4 treatments were reasonably correlated with subsequent energy intake. VAS scores have previously been criticised for failing to demonstrate a relationship between intensity of rated hunger and the amount of food eaten (3,52,58), particularly cognitive impression of fullness (29)

TABLE 3

THE CORRELATION (r) BETWEEN SUBJECTIVE APPETITE RATINGS AND MEASURED ENERGY INTAKE OF AN *AD LIB* OUTCOME MEAL GIVEN SUBSEQUENT TO A RANDOMISED PRELOAD DRINK

Rating Question	All Treatments	Alcohol	No Alcohol	CHO	Water
Number of subjects	80	20	20	20	20
Q1. How hungry do you feel?	0.38*	0.50‡	0.58†	0.10^{ns}	0.15^{ns}
Q2. How full do you feel?	0.10^{ns}	0.05^{ns}	0.27^{ns}	0.06^{ns}	0.00^{ns}
Q3. How strong is your desire to eat?	0.45*	0.51‡	0.60†	0.05^{ns}	0.46‡
Q4. How much could you eat now?	0.45*	0.50‡	0.60†	0.10^{ns}	0.55‡
Q5. Urge to eat?	0.41*	0.44^{ns}	0.52‡	0.07^{ns}	0.52‡
Q6. Preoccupation with thoughts of food?	0.48*	0.56†	0.57†	0.10^{ns}	0.51‡

* $p < 0.001$, † $p < 0.01$, ‡ $p < 0.05$, $^{ns}p > 0.05$.

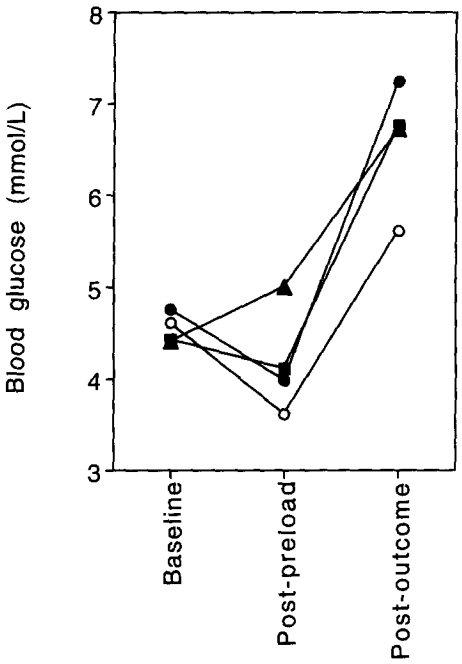

FIG. 5. Mean changes in blood glucose following the 4 preload drinks (alcohol, ●; no alcohol, ■; CHO, ▲; water, ○; SEM has been omitted for clarity, see text).

which, in fact, was also a poor predictor of intake in this study. When each treatment was analysed individually, only following the alcohol preload was there a decrease in ratings of hunger, whereby subjects reported feeling less hungry, had a lower urge to eat, and were less preoccupied with thoughts of food than before the alcoholic drink. This drop in hunger, however, failed to manifest itself as a reduction in food intake at the test meal, suggesting that detectable changes in hunger were of insufficient magnitude to influence the subsequent meal. There was no consistent decrease of hunger or increase in satiety following either the high-energy CHO treatment or the 2 low-energy drinks, suggesting that these preloads went unrecognised. The decrease in hunger following the alcohol treatment cannot, therefore, be attributed simply to the energy content of the preload but, speculatively, may be a consequence of a pharmacological effect prior to the oxidative disposal of ethanol from the blood stream.

There are several reasons that may explain why there was little or no compensatory decrease in the outcome meal following the alcohol and CHO treatments. First, it may be that consuming a single bolus preload of less than 1 MJ is insufficient for detection. Alternately, it is possible that psychological and/or behavioural factors may overide the physiological control of appetite at such low intakes. Studies assessing the short-term effects of alcohol are heavily constrained by the intoxicating effect that accompanies the intake of ethanol. In our current study, 31 g of ethanol given as 92 g of gin (equivalent to approximately 4 standard spirit measures) was assessed in preliminary studies as the maximum single dose that could be given to women without incurring considerable side effects. In the study, subjects reported feelings only of mild intoxication, disinhibition, and loss of cognitive control. Increasing the preload above this level may result in severe dizziness and nausea and, consequently, constrains the maximum alcohol-derived energy that may be given as a single preload to less than 1 MJ.

Several authors have shown that liquid preloads may have a less suppressive effect than isoenergetic solid preloads (4,24,29). In a previous preload study assessing the effect of a sucrose drink given at doses of 0.35 and 0.70 MJ, there was also a lack of recognition of the energy preload and, consequently, subsequent food intake did not fall to compensate the additional energy source (47). In the longer-term metabolic study of Foltin and coworkers (16), however, it was found that, although demonstrating no differential effects between alcohol-derived and CHO-derived energy when given as a liquid preload, there was some compensatory reduction in the amount of food consumed in response to both treatments (16). Subjects were able to both detect and partially respond to these energy sources. This difference might be explained by the fact that the treatments given were considerably higher in energy. Male subjects were given either 2.4 or 4.6 MJ over an entire day, corresponding to 0.6 and 1.15 MJ on each of 4 occasions.

It is also possible that, although the energy source within the drinks was either not recognised or regulated in the short-term, the consumption of a large volume of liquid immediately prior to a meal was detected and resulted in a decrease in food intake in all 4 treatments. Several authors have suggested that gastric distension may have a strong influence on appetite control, hunger decreasing and satiety increasing as the gut fills (22,30–32,39). In this study, 400 ml of liquid was consumed under all treatments 15 min before the outcome meal. Although it requires an additional treatment to be included in the protocol, whereby no preload is given, to fully establish the effect of gut distension on appetite in these women, the fact that there was virtually no decrease in hunger following the drinks suggests that the volume of liquid consumed was actually not a limiting factor concealing any effect of energy content of the preloads. Previous studies have also shown that the addition of a small volume of water to a meal has little effect on energy intake (47) unless, in the absence of fluids, the subjects have become significantly more thirsty, which, interestingly, may reduce rather than increase hunger (12,46).

The timing of the outcome meal following a preload treatment must influence both the hunger ratings and the amount of energy consumed. Although studies using both high-fat and high-CHO yoghurt preloads have shown that a 30-min interval may be optimum in establishing any macronutrient specific effects on intake (48), this may not be the case for liquid preloads where a more rapid absorption from the gut may result in a more rapid, short-term effect on appetite. Satiating effects of a CHO-rich drink have been shown to be strongest when consumed with a meal, gradually declining over the following 30 min (47). If this is correct, the effect of the treatments in our study may have gone undetected.

We can conclude, from our study, that neither alcohol-derived or CHO-derived energy is recognised by any short-term system regulating appetite control when given in doses of less than 1 MJ. The addition of either an alcoholic or high-CHO carbonated drink to a meal will result in the addition of this extra energy into the diet. There is no evidence to suggest that such drinks are regulated via a compensatory decrease in the amount of energy eaten at the subsequent meal. Whether or not the addition of numerous drinks of this kind throughout the day may result in longer-term compensation, perhaps over periods of 24 or 48 h, has yet to be established.

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