

## Alcohol and its acute effects on resting metabolic rate and diet-induced thermogenesis

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*(Received 26 July 1989 – Accepted 8 May 1990)*

The impact of alcohol (ethanol) on resting energy expenditure of male non-obese volunteers was determined in two studies. In the first study the thermic effect of alcohol on resting metabolic rate (RMR) was assessed in ten male non-obese volunteers. In the second study the impact of alcohol on diet-induced thermogenesis (DIT) was determined in twelve male non-obese volunteers. Energy expenditure was measured with a ventilated-hood system. RMR was measured for 60 min with the subjects in a fasting state. In the first study subjects received in random order 20 g alcohol in concentrations of 75, 180 and 300 ml/l water respectively. After measurement of the RMR the thermic effect of alcohol was measured for 90 min. In the second study volunteers received in random order and in duplicate either a meal of food (2 MJ) plus an alcoholic aperitif (20 g alcohol in a 180 ml/l solution) or an isoenergetic meal of food alone (2.55 MJ) plus a placebo aperitif containing no alcohol. DIT was measured for 240 min. Alcohol induced a significant thermic effect, which varied between 0.22 and 0.30 kJ/min. No systematic difference in DIT was observed among the different concentrations. DIT was not significantly affected by the ingestion of alcohol. Total DIT was 219 (SE 14) kJ for the alcohol treatment and 185 (SE 20) kJ for the control treatment. The results do not support the suggestion that alcohol is less efficiently used as an energy source in comparison with, for example, fats and carbohydrates.

### Alcohol: Resting metabolic rate: Diet-induced thermogenesis

In most Western countries total per capita alcohol consumption has increased dramatically over the last few decades (Sulkunen, 1976; World Health Organization, 1980). A recent nationwide food consumption study in The Netherlands showed that alcohol contributes on average 3% to total daily energy intake in women and 5.4% in men aged 22–49 years (Ministerie van Welzijn, Volksgezondheid en Cultuur & Ministerie van Landbouw en Visserij, 1988). Similar values have been reported for men and women in other Western countries (Spring & Buss, 1977; Gruchow *et al.* 1985). The health effects of excessive alcohol intake are well documented (Klatsky, 1979; Mercy, 1980; Lieber, 1982), but the metabolic consequences and relation to the incidence of disease of moderate alcohol use remain to be definitely assessed. In this respect there is a general interest in the utilization of alcohol as an energy source. The issues that receive most attention are whether alcohol energy is added to or substituted for non-alcoholic energy in the diet, and whether energy intake from alcohol is related to increased levels of adiposity in the same way as energy intake from non-alcoholic sources.

The majority of observational studies show that alcohol usually provides additional energy to the diet, but that its use is not associated with higher levels of adiposity (Bebb

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*et al.* 1971; Jones *et al.* 1982; Fisher & Gordon, 1985; Hillers & Massey, 1985; Camargo *et al.* 1987). In concordance with these findings are the results of various experimental studies (Pirola & Lieber, 1972; McDonald & Margen, 1976; Crouse & Grundy, 1984). In these studies it was found that isoenergetic substitution of non-alcoholic energy by alcohol resulted in weight loss in volunteers and that alcohol added to the diet did not result in weight gain. These findings suggest that alcohol is used less efficiently as an energy source than, for example, fats and carbohydrates, or that alcohol interferes with the efficient utilization of these substrates. However, results from metabolic studies assessing the impact of alcohol ingestion on energy expenditure give only equivocal support for the latter proposition. In the classic report of Atwater & Benedict (1902) it was shown that alcohol use did not increase energy expenditure in man relative to fats or carbohydrates. More recent studies did observe either an increase in, or no effect on, oxygen consumption after alcohol use (Perman, 1962; Barnes *et al.* 1965; Stock & Stuart, 1974; Rosenberg & Durnin, 1978). The reasons for these discrepant findings are unclear, but in general the experimental design, i.e., number and sex of subjects studied, duration of study, amount of alcohol given and way in which alcohol was provided and the technique used to measure energy expenditure differ considerably among the various studies. Thus, in spite of its importance as an energy source in the average adult individual, little is known with certainty about the impact of alcohol on overall energy metabolism. In particular, there is little information on the effect of alcohol on diet-induced thermogenesis (DIT). In the present report we assessed the thermic effect of alcohol in varying concentrations. In addition, we investigated the impact of alcohol on DIT. The results show a significant thermic effect of alcohol on fasting energy expenditure but no systematic potentiation of DIT by alcohol.

#### SUBJECTS AND METHODS

The present study consisted of two parts. In the first study the impact of ingestion of alcohol on post-absorptive resting metabolic rate (RMR) was studied. In the second study we assessed the effect of alcohol on DIT.

##### *Subjects*

Twenty-two male subjects were invited to participate in the studies by an advertisement in the weekly periodical of the University and through friends and relatives. Four subjects participated in both experiments. All subjects except one were students or members of staff of the University. After applying for participation subjects received a letter in which the purpose and nature of the experiment(s) were explained. They also received two questionnaires, one to assess past and present health status and one to obtain information on habitual eating, drinking, smoking and exercise habits. The medical questionnaire was evaluated by a physician (J.G.A.J.H.). After the subjects had given their written consent and when they fulfilled the entry criteria, they were able to participate in the experiments. Subjects had to be apparently healthy (no past or present evidence of hypo- or hyperthyroidism or diabetes mellitus), non-obese (body fat percentage below 25) and to have moderate smoking (less than 10 cigarettes/d) and drinking habits (no more than 5 alcoholic drinks/d). None of the subjects used drugs known to affect energy metabolism, none was on a diet, all subjects were consuming normal balanced meals and all subjects had had weight fluctuations within 2.5 kg for at least 6 months before the start of the study. The protocol of the studies was submitted to and approved by the Medical Ethical Committee of the Wageningen Agricultural University.

### *Experimental design*

*Thermic effect of alcohol.* In this study ten subjects received on three occasions on different days an oral dose of 20 g alcohol dissolved in 335 ml water (75 ml/l solution), 140 ml water (180 ml/l solution) or 85 ml water (300 ml/l solution) after an initial RMR measurement of at least 1 h duration. To these solutions were respectively added 1.2 g of a non-nutritive sweetener (Natrena; Bayer GmbH, Germany) and 0.12 mg aniseed oil (to the 75 ml/l solution) or 0.8 g sweetener and 0.06 mg aniseed oil (to the 180 and 300 ml/l solutions) to improve sensory characteristics (mimicking Anisette). The alcohol solutions were drunk at a temperature of between 10 and 15° within 10 min and with the ventilated hood removed from the subjects' heads. After the alcohol ingestion the hood was replaced and energy expenditure was measured continuously for another 90 min. Energy expenditure was measured with a ventilated-hood system as previously described (Weststrate *et al.* 1989). Subjects received the three alcohol solutions either early in the morning after an overnight fast, or early in the afternoon after a fasting period of at least 4.5 h after a standardized breakfast of 2 MJ. In a separate study in five subjects (values not shown) the thermic effect of drinking 350 ml water plus 1.2 g sweetener was assessed. Water plus sweetener did not induce a systematic thermic effect. Between each session was a time-interval of at least 2 d. Subjects received the three alcohol solutions in randomized order.

*Impact of alcohol on diet-induced thermogenesis.* In this study twelve subjects received on four different days in the afternoon two treatments in duplicate, i.e., either a test treatment consisting of the ingestion of an alcoholic aperitif (containing 20 g ethanol in a 180 ml/l solution) and a yoghurt-based mixed liquid test meal (containing 1.96 MJ, 130 g protein/l, 270 g fat/l, 600 g carbohydrate/l) or a placebo aperitif (containing zero energy) and an isoenergetic yoghurt-based mixed liquid diet (containing 2.55 MJ, 130 g protein/l, 270 g fat/l, 600 g carbohydrate/l). To both aperitifs flavourings were added to mimic Anisette. To the alcoholic aperitif were added 0.8 g of a non-nutritive sweetener (Natrena) and 0.06 mg aniseed oil. To the placebo aperitif were added 0.8 g sweetener, 0.06 mg aniseed oil, 0.03 mg kinin and 0.007 mg maltol. In a separate control experiment in eight subjects (values not shown) the thermic effect of kinin in a much larger dose (22.0 mg in 350 ml water) was tested. Kinin did not induce a significant thermic effect. The impact of maltol on energy expenditure was not studied. The quantity used was extremely low and no effects on energy expenditure were expected. The aperitifs were given at a temperature of between 10 and 15° after RMR had been measured for at least 1 h and with the hood removed from the subjects. After ingestion of the aperitif (within 10 min) the hood was replaced over the subject's head. After 15 min subjects received the liquid test meal through a straw which was passed through the hood. The meals were given at room temperature and were eaten within 5 min. Energy expenditure was then continuously measured for 4 h. Control and test treatments were assigned to the subjects in randomized order. Between treatments there was a time interval of at least 2 d.

### *Energy exchange measurements*

Energy expenditure was measured in subjects resting in a hospital bed in a supine position with a ventilated hood system. Details of the measurement procedure and conditions have been previously published (Den Besten *et al.* 1988; Weststrate *et al.* 1989). Spontaneous movements of the subjects were unobtrusively registered by means of a load cell (TKA-200A; Tokyo Sokki Kenkyujo, Tokyo, Japan) placed under one of the legs of the hospital bed. Once every hour, during calibration of the gas analysers with fresh outside air, subjects were allowed to move briefly for 2–3 min.

Subjects whose energy expenditure was measured in the afternoon were instructed to

have a small breakfast of 2 MJ before 08.00 hours and to avoid coffee drinking, sleeping and moderate or heavy exercise on the morning before the gas-exchange measurements. Under these conditions RMR and DIT measured in the afternoon do not differ systematically from RMR and DIT measured in the morning (Weststrate *et al.* 1989). After the gas-exchange measurements urine was collected for determining urea-nitrogen excretion.

#### *Calculations*

Energy expenditure was calculated as described previously (Jéquier, 1985). The thermic effect of alcohol was calculated as the difference between the average energy expenditure over a period of 90 min and the corresponding baseline RMR. DIT with or without alcohol was calculated as the difference between the average hourly and total (4 h) postprandial energy expenditure and the corresponding baseline RMR.

*Substrate oxidation rates and nutrient balances.* Substrate oxidation rates and nutrient balances were calculated as described previously (Jéquier, 1985; Den Besten *et al.* 1988; Weststrate *et al.* 1989). In addition the contribution of alcohol oxidation to total oxidation was estimated. Alcohol oxidation was assumed to be equal to the average elimination rate of alcohol from the blood, i.e., 100 mg/kg body-weight per h (Bouman & Rand, 1980).

Generally about 93–98% of an ingested dose of alcohol is eliminated from the body by oxidation and negligible amounts leave the body by respiration and in the urine (Bouman & Rand, 1980). We calculated the amount of O<sub>2</sub> needed to oxidize 1 g alcohol (1.4594 litres) and the amount of carbon dioxide thus produced (0.973 litres). Total O<sub>2</sub> consumption and CO<sub>2</sub> production were subsequently corrected for the volumes of O<sub>2</sub> and CO<sub>2</sub> associated with alcohol oxidation. These corrected gas volumes were used in the calculation of the oxidation rates of fats and carbohydrates. It should be noted that there may be considerable inter-individual difference in the elimination rate of alcohol from the blood (Bouman & Rand, 1980). However, since in the present study measurements for each person were made repeatedly under different conditions such variation will not bias conclusions on within-person comparisons of treatments.

*Body composition.* Body fat mass and body fat-free mass were estimated from total body density using Siri's (1956) formula. Total body density was measured in each subject by hydrostatic weighing with simultaneous determination of residual lung volume by a helium dilution technique.

#### *Statistics*

In the first study the thermic effect of alcohol was evaluated using two-sided paired *t* tests. Analysis of variance was used to test differences among the three treatments (75, 180 and 300 ml/l solutions) in thermic effect. In the second study results of the duplicate measurements were pooled and averaged for both treatments. Analysis of differences between treatments were performed using two-sided paired *t* tests. We also performed a two-way ANOVA with repeated measures on the (within-subject) factors meal ± alcohol and time period (duplicates). The results of the ANOVA were similar to the results of the paired *t* tests. Results are expressed as means with their standard errors. *P* < 0.05 was taken as significant.

## RESULTS

### *Subject characteristics*

Table 1 gives some characteristics of the subjects participating in the two experiments. The average age was 27 (SE 1.5) years, and average body fat percentage was 16.5 (SE 1.2). Three

Table 1. *Age, body-weight, body fat percentage and habitual alcohol use of the subjects*  
(Mean values with their standard errors)

	Study 1 Thermic effect of alcohol (n 10)			Study 2 Effect of alcohol on diet- induced thermogenesis (n 12)		
	Mean	SE	Range	Mean	SE	Range
Age (years)	26.5	1.7	22-41	27.3	1.5	21-41
Body-wt (kg)	76.8	3.2	66.2-102.0	78.3	3.0	58.8-101.8
Body fat content (%)	17.0	1.1	13.0-24.7	16.0	1.2	7.9-22.5
Habitual alcohol use*	1.2	0.2	0.0-3	1.4	0.4	0.0-5

\* No. of alcoholic drinks habitually consumed/d.

subjects had a body fat percentage between 20 and 25, but none was obese. Habitual use of alcoholic beverages was on average 1.5 units/d.

#### *Thermic effect of alcohol*

Table 2 shows metabolic rate, O<sub>2</sub> consumption, CO<sub>2</sub> production and respiratory quotients in subjects before and after alcohol ingestion at three different concentrations. None of the variables was significantly different among the three treatments. Alcohol ingestion induced a significant increase in metabolic rate and in O<sub>2</sub> consumption, but not in CO<sub>2</sub> production. As a consequence respiratory quotients decreased significantly after alcohol use. On average, metabolic rate increased by 4.4% (SE 1.40, range 1.1-9.9%) after intake of the 75 ml alcohol/l solution, 4.8% (SE 1.35, range 1.6-13.6%) after ingestion of the 180 ml alcohol/l solution and 6.2% (SE 1.23, range 1.1-11.3%) after use of alcohol in a 300 ml/l solution. These values did not differ significantly. At the end of the 90 min measurement period metabolic rate had not yet returned to baseline. Metabolic rate was elevated on average by 3.4% (75 ml/l), 4.6% (180 ml/l) and 6.2% (300 ml/l). These values did not differ significantly.

#### *Impact of alcohol on DIT*

Table 3 gives pre- and postprandial metabolic rate, O<sub>2</sub> consumption and CO<sub>2</sub> production in subjects who received in duplicate a meal either with or without alcohol as an aperitif. Since we did not observe significant differences in thermic effect among isoenergetic alcohol solutions of varying concentration, the effect of alcohol on DIT was studied in the second study using the moderately concentrated solution of 180 ml/l. Metabolic rate, O<sub>2</sub> consumption and CO<sub>2</sub> production increased significantly after ingestion of the meal either with or without alcohol. No significant difference was found between treatments in the increase in metabolic rate or in O<sub>2</sub> consumption. The postprandial rise in CO<sub>2</sub> production was, however, significantly smaller on the test treatment in comparison with the control treatment. Respiratory quotients remained at their pre-prandial level after ingestion of a meal with alcohol, whereas after ingestion of a meal without alcohol respiratory quotients showed a significant increase. On average DIT was about 16% higher on the test treatment in comparison with the control treatment. The difference in DIT between treatments was, however, not significant.

Fig. 1 shows average hourly DIT values separately for the two treatments. During the second, third and fourth hour DIT was higher on the test treatment compared with the control treatment, but only for the second hour was the difference statistically significant ( $P = 0.03$ ).

Table 2. *Metabolic rate, oxygen consumption, carbon dioxide production and respiratory quotients in subjects before and after ingestion of a dose of 20 g alcohol at three different concentrations*  
(Mean values with their standard errors)

	75						180						300					
	Before		After		Before		After		Before		After		Before		After			
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE		
Alcohol concentration (ml/l)...																		
Metabolic rate (kJ/min)	4.98	0.13	5.20*	0.16	4.95	0.21	5.19*	0.24	4.85	0.22	5.15*	0.24	4.85	0.22	5.15*	0.24	5.15*	0.24
O <sub>2</sub> consumption (ml/min)	247	7.0	261*	8.3	245	11.3	260*	12.5	241	11.0	258*	12.1	241	11.0	258*	12.1	258*	12.1
CO <sub>2</sub> production (ml/min)	206	5.7	204	6.1	206	6.5	203	7.4	201	9.3	200	10.3	201	9.3	200	10.3	200	10.3
Respiratory quotient	0.84	0.02	0.78**	0.01	0.85	0.02	0.78***	0.01	0.84	0.02	0.77***	0.01	0.84	0.02	0.77***	0.01	0.77***	0.01

Differences between values before and after ingestion were significantly different from zero: \*  $P < 0.05$ , \*\*  $P < 0.01$ , \*\*\*  $P < 0.001$ .

Table 3. *Metabolic rate, oxygen consumption, carbon dioxide production, respiratory quotient and diet-induced thermogenesis (DIT) in subjects before and after ingestion of a test meal with or without a dose of 20 g alcohol given as an aperitif*

(Mean values with their standard errors)

	Meal + alcohol†				Control meal‡			
	Before		After		Before		After	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Metabolic rate (KJ/min)	5.34	0.23	6.25	0.25	5.34	0.26	6.12	0.27
O <sub>2</sub> consumption (ml/min)	267	11	312	12	267	13	300	13
CO <sub>2</sub> production (ml/min)	216	9	251	11	216	11	263	11
Respiratory quotient	0.81	0.01	0.80	0.01	0.88	0.01	0.88*	0.01
DIT (kJ)	—	—	219	14	—	—	185	20
DIT — % ME (%)	—	—	8.6	0.6	—	—	7.2	0.8

% ME, percentage of metabolizable energy intake.

Postprandial respiratory quotient was significantly different between treatments: \*  $P < 0.01$ .

† Energy intake 2.543 (SE 0.005) MJ (22.7% derived from alcohol).

‡ Energy intake 2.550 (SE 0.004) MJ.

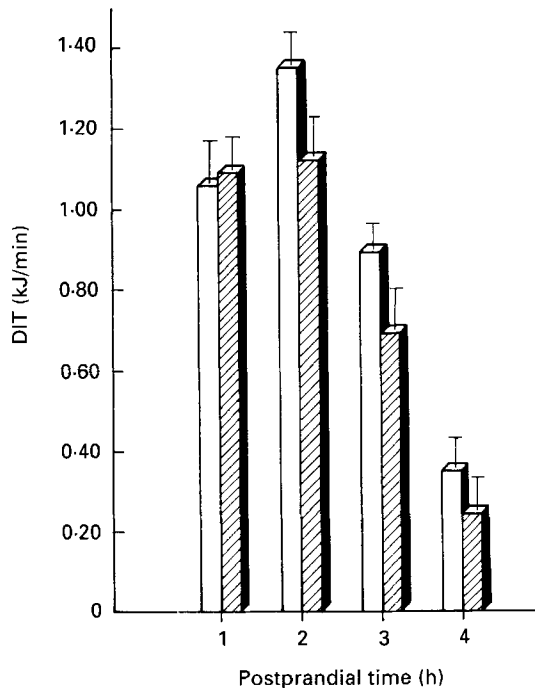


Fig. 1. Diet-induced thermogenesis (DIT) for twelve non-obese male volunteers after ingestion of isocaloric mixed meals of 2.5 MJ with (□) or without (▨) a 20 g alcohol aperitif. Values are means with their standard errors represented by vertical bars.

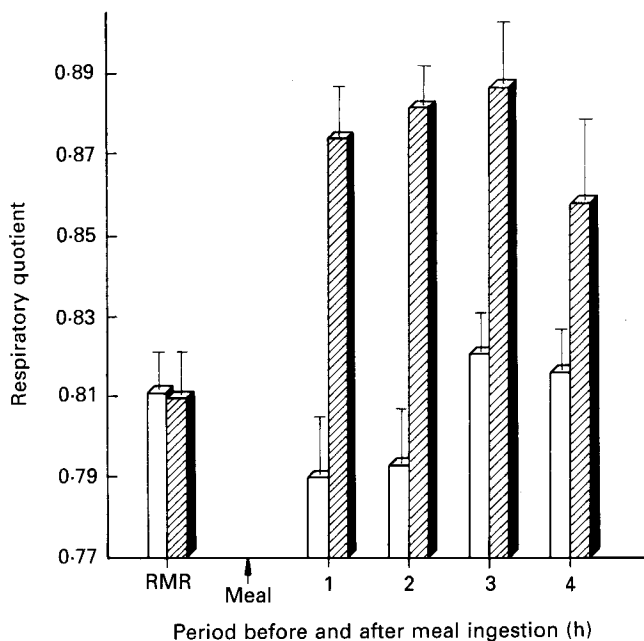


Fig. 2. Respiratory quotients for twelve non-obese male volunteers before and after ingestion of isoenergetic mixed meals of 2.5 MJ with (□) or without (▨) a 20 g alcohol aperitif. Values are means with their standard errors represented by vertical bars.

Fig. 2 gives respiratory quotients for RMR and for the course of the DIT measurement separately for both treatments. Significant differences ( $P < 0.01$ ) were found between treatments in respiratory quotients during the first, second and third hour postprandially.

Table 4 gives energy and nutrient intake and postprandial metabolism of the ingested nutrients for both treatments. Glucose and fat oxidation rates were significantly lower ( $P < 0.001$ ) on the test treatment in comparison with the control treatment. Protein oxidation was not systematically affected by alcohol ingestion. On the control treatment subjects oxidized on average about 50% of the carbohydrate and fat intake. During a 4 h postprandial period, when food was eaten with alcohol about 40% of the glucose intake and about 20% of the fat intake were subsequently oxidized. Energy and carbohydrate and fat storage were not significantly different between treatments.

## DISCUSSION

### *Thermic effect of alcohol*

The results of the present study show that alcohol in a moderate amount of 20 g induces in fasting individuals a significant increase in  $O_2$  consumption and metabolic rate, but not in  $CO_2$  production. The increase in metabolic rate was not significantly related to the concentration of the alcohol solution.

Alcohol induced an increase in metabolic rate of about 4–6% of the ingested energy load (about 0.6 MJ). This is similar to or slightly lower than the thermic effect of fats or carbohydrates (Jéquier, 1984). The results of the present study support the findings of Perman (1962) and Rosenberg & Durnin (1978), but are in contrast with the observations of Stock & Stuart (1974) and Barnes *et al.* (1965). It is, however, difficult to compare these studies, because of differences in experimental design and in the technique used for



assessing alcohol-induced thermogenesis. In contrast to the present study, none of the aforementioned studies employed the ventilated-hood technique to assess energy expenditure. This technique is particularly suitable for measuring energy expenditure accurately over prolonged periods of time, i.e., over several hours, and has been recommended for use in metabolic studies assessing thermogenesis (Kinney, 1980).

In the present study we gave 20 g alcohol dissolved in varying amounts of water with added flavourings. This enabled us to study the impact of alcohol in different concentrations on energy expenditure. Perman (1962) and Rosenberg & Durnin (1978) gave ethanol in a dose of 0.29 g/kg dissolved in 150 ml chilled fruit juice or in a dose of 23 g as 230 ml red wine respectively. Barnes *et al.* (1965) and Stock & Stuart (1974) used whisky in a dose of 1.5 ml/kg and in a quantity of about 80 ml respectively. Besides differences among these studies in the amount of alcohol given, the concentration of the alcohol solutions and the amount of non-alcoholic energy varied. For example, red wine and fruit juice may contain carbohydrates, which can provoke an increase in energy expenditure (Jéquier, 1984).

For the present study we first tested a quantity of 30 g alcohol (180 ml/l) and found that this amount, drunk within 10 min, induced drowsiness and symptoms of mild intoxication in fasting individuals. Since drowsiness may interfere with a correct assessment of metabolic rate, we opted for the smaller dose of 20 g. In fasting subjects such an amount of alcohol should quickly raise the blood alcohol concentrations to values between 5 and 15 mmol/l (DiPadova *et al.* 1987). It was expected that the more concentrated solutions would lead to higher maximal blood alcohol curves (DiPadova *et al.* 1987). At higher blood alcohol levels an increased metabolism of ethanol by the microsomal alcohol-oxidizing system (MEOS) occurs (Pirola & Lieber, 1972). Oxidation of alcohol by MEOS will result in a less efficient coupling (about 20% reduction in net ATP production) of oxidation to ATP synthesis and an increased demand for NADPH compared with oxidation by the alcohol dehydrogenase (*EC* 1.1.1.1; ADH) pathway. The latter pathway operates fully at lower blood alcohol concentrations (Pirola & Lieber, 1972). This may affect thermogenesis in two ways. A relative increase in alcohol oxidation by MEOS may increase the rate of alcohol oxidation to meet the tissues' demand for ATP; second, the eventual NADPH repletion may increase energy, i.e., ATP demand, from other metabolic pathways. Both processes could lead to higher O<sub>2</sub> consumption and, hence, increased thermogenesis. However, we did not find a significant relationship between the thermic effect of alcohol and its concentration. Nonetheless at lower alcohol concentrations (75 and 180 ml/l) the thermic effect of alcohol was on average 4.6 v. 6.2% at the highest alcohol concentration. This is a relative difference of about 30% and this could be partly attributed to an increase in the rate of alcohol oxidation at the highest alcohol concentration to compensate for the less efficient metabolism of alcohol, i.e., a relatively greater part by MEOS than by the ADH pathway. However, the increase in the thermic effect of alcohol at the highest ethanol concentration was not a systematic phenomenon and did not reach statistical significance. It is also possible that the different solutions did not result in clear differences in blood alcohol concentrations and, hence, in alcohol metabolism. Since blood alcohol curves were not measured we cannot test this explanation.

In the present study it was also found that alcohol ingestion and its subsequent oxidation significantly reduced basal respiratory quotients. It is well known that alcohol oxidizes with a respiratory exchange ratio of 0.666, which is much lower than respiratory exchange ratios usually found in individuals fasting overnight (Flatt, 1978).

#### *Impact of alcohol on DIT*

Very few studies have assessed the effect of alcohol on DIT (Stock & Stuart, 1974; Rosenberg & Durnin, 1978). Rosenberg & Durnin (1978) found no significant difference

Table 4. Nutrient and energy intake, utilization and storage in the body over a period of 4 h in subjects after ingestion of a test meal with or without a dose of 20 g alcohol given as an aperitif  
(Mean values with their standard errors)

	Energy (kJ)		Carbohydrate (g)		Fats (g)		Protein (g)		Alcohol (g)									
	M + E	M - E	M + E	M - E	M + E	M - E	M + E	M - E	M + E	M - E								
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE								
Intake	2543	5	2550	4	68.7	0.1	90.8	0.1	14.1	0.0	18.6	0.0	15.0	0.0	19.8	0.0	19.9	0.1
Oxidation	—	—	29.8	3.5	47.5	3.3**	3.1	1.5	9.2	1.6*	17.5	1.5	18.8	1.4	19.8	0.3	—	—
Expenditure	1500	60	1469	65	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Storage	1093	61	1081	66	38.9	3.5	43.3	3.3	11.0	1.5	9.4	1.6	-2.6	1.5	1.0	1.4**	0.1	0.0

M + E, meal with alcohol; M - E, meal without alcohol.

Mean values for M + E and M - E were significantly different: \*  $P < 0.05$ , \*\*  $P < 0.01$ .

between the average increase in metabolic rate over 3 h after an isoenergetic meal of food or of food plus alcohol. However, in the latter study, during the last 30 min of the 3 h postprandial period the increased  $O_2$  consumption was significantly greater after the meal including the alcohol. Stock & Stuart (1974) found that whisky taken with a meal produced a 22% increase in  $O_2$  consumption, which was significantly greater than the 13% increase caused by consuming an isoenergetic non-alcoholic meal. This effect was observed during the entire postprandial period. In both studies, however, at the end of the postprandial period energy expenditure was still elevated by more than 20% above the pre-meal baseline energy expenditure, indicating that the total thermogenic response was not completely measured.

In the present study we compared thermogenesis induced by an isoenergetic meal of food alone and of food plus alcohol. Alcohol was given as an aperitif to minimize effects of food on the absorption of alcohol from the gastrointestinal tract. Control and test treatments were carried out in duplicate to obtain a more accurate estimate of an individual's response in energy metabolism to food. In previous studies it was shown that DIT is prone to considerable within-person variation (Ravussin *et al.* 1986; Weststrate *et al.* 1989). Postprandial measurements were carried out over a period of 4 h, which was sufficiently prolonged to allow an almost complete assessment of the thermogenic response. Thus, energy expenditure averaged over the fourth hour of the postprandial period was on average 4.5% elevated above the corresponding pre-meal baseline energy expenditure on the control treatment and 6.5% on the test treatment. In the present study overall DIT was not potentiated by alcohol ingestion. On average however, and in particular during the last 3 h of the postprandial period, DIT values were slightly higher on the test treatment than on the control treatment. The maximum potentiation of DIT was observed during the second and third hour postprandially.

After alcohol consumption it takes about 30–45 min in fasting subjects to reach the maximal blood alcohol concentrations (DiPadova *et al.* 1987). Depending on the total amount of alcohol ingested, alcohol elimination will take several hours. It can be calculated that in the present study most of the alcohol will have been eliminated within a period of 30–150 min after ingestion of the meal, which was given 15 min after the aperitif. During this period alcohol oxidation contributed on average 3.8 kJ/min to total energy expenditure. Between 55 and 65% of total energy expended was derived from alcohol oxidation. Analysis of the substrate oxidation rates revealed that alcohol was preferentially oxidized in comparison with fats and carbohydrates, but not in comparison with proteins. In the first 180 min of the postprandial period glucose and fat oxidation were markedly reduced, on average by about 22 and 7 g respectively after ingestion of a meal of food plus alcohol in comparison with a meal of food alone. This means that during this period more fats and carbohydrates were stored on the test treatment when compared with the control treatment. Increased storage of nutrients after a meal will increase postprandial energy expenditure (Flatt, 1978). When fats and carbohydrates are oxidized without previous storage, about 2% of the energy content of these substrates has to be expended to provide ATP for handling costs. In contrast, if fats are stored before oxidation total energy dissipated for handling this process is about 4% of the energy content of fat. For glucose stored as glycogen or as fat the costs for processing are about 7 and 24% of the energy content of glucose respectively (Flatt, 1978). The average difference in handling costs of the postprandial processing of the nutrients was calculated at about 0.2–0.3 kJ/min for the first, second and third hour of the postprandial period. Fig. 1 shows that for this period differences were found between treatments in energy expenditure, on average of -0.03, 0.23 and 0.21 kJ/min respectively. In particular the values for the second and third hour are close to what was expected. Probably, over the first hour of the postprandial period

differences in storage of nutrients between the two treatments are small. This could be due to a relatively low alcohol oxidation, especially in the first half of this period. It has also been found (Jéquier, 1984) that nutrient-induced thermogenesis cannot be explained entirely in terms of the biochemistry of energy expenditure associated with the processing of ingested nutrients (Flatt, 1978). Part of nutrient- or meal-induced thermogenesis, i.e., about 30–40%, is thought to be mediated by an increased activity of the sympathetic nervous system after food or nutrient ingestion (Sims & Danforth, 1987). The latter part of the total thermogenic response is usually designated with the term facultative thermogenesis, indicating that this part of thermogenesis might not be operating at the same level in each individual or that it might be susceptible to environmental stimuli. It is possible that nutrient-induced facultative thermogenesis is effectively operative in the period immediately after food ingestion (0–60 min) and that ethanol reduces the amount of energy expended by the facultative mechanism.

The results of the present study on the thermic effect of alcohol and the impact of alcohol on DIT do not indicate that moderate amounts of alcohol are less efficiently used as an energy source in comparison with fats or carbohydrates. We therefore tentatively conclude that the observation that alcohol provides additional energy to the diet, but that its use is not associated with increased levels of adiposity, is due to methodological problems of accurately quantifying energy and alcohol intake in free-living humans or of the accurate quantification of factors other than diet and alcohol that affect energy balance or body-weight in man, for example, the degree of physical activity.

In conclusion we can state that alcohol consumed in a moderate amount has a significant thermic effect, probably similar to fats or carbohydrates. No significant potentiation by alcohol ingestion of DIT was found. Alcohol given in combination with a mixed liquid meal had a substantial impact on substrate oxidation and storage rates, in particular during the period at which alcohol oxidation was at its maximum. During this period alcohol ingestion slightly increased DIT. Overall postprandial energy retention was, however, similar for an isoenergetic meal of food plus alcohol and a meal of food alone. Substituting moderate amounts of energy from fats and carbohydrates by alcohol in non-alcoholics is not expected to decrease energy retention significantly or to be useful as an adjuvant in weight-reducing regimens.

The authors wish to thank the subjects for their enthusiastic participation. They are also indebted to Quest International (Naarden, The Netherlands) for advice on flavouring the ethanol solutions and for providing the necessary flavouring agents.

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