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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Thirty-sixth Meeting of the Nutrition Society was held in Dudley Road Hospital, Birmingham on Tuesday and Wednesday, 7/8 *April* 1987, *when the following papers were read:*

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Moderate alcohol consumption affects lipid metabolism and blood haemostasis. By TH. OCKHUIZEN, H. VAN DE POL, J. VEENSTRA, W. VAN DOKKUM and M. WEDEL (introduced by R. F. GRIMBLE), *TNO-CIVO Toxicology and Nutrition Institute, PO Box* 360, 3700 *AJ Zeist, The Netherlands*

The present study was designed to test the hypotheses that the decreased relative risk for cardiovascular disease in moderate alcohol consumers (as compared with abstainers) relates to alterations in lipid metabolism as well as to decreased blood clotting or thrombosis tendency, or both.

In Expt 1, short-term effects of moderate alcohol consumption on blood constituents were studied in twelve male volunteers who drank standardized amounts of wine. Four regimens, namely 0, 2 or 4 glasses/d or 14 glasses as 'binge drinking' during the weekend, were followed for 5 weeks each in a randomized order. The results showed a clear dose-related response to drinking for several blood constituents. Most marked was a decrease in the tissue-type plasminogen activator activity and, to a lesser degree, an increase in plasminogen levels. Collagen-induced platelet aggregation was reduced, affecting all indices measured. Levels of high-density-lipoprotein$_3$ (HDL_3)-cholesterol, γ-glutamyltransferase and urate showed a small but significant increase.

In Expt 2, long-term effects of moderate alcohol drinking on indices of lipid metabolism and blood haemostasis were studied. Twelve healthy men consumed 4 glasses wine/d for 20 weeks and the effects were compared with those in six men who refrained from drinking (controls). Remarkable were the seasonal differences in the concentrations of HDL_3-cholesterol and γ-glutamyltransferase in the experimental and control groups. At the beginning of the experiment there were no significant differences in the concentrations of these substances; however, after 5 weeks the concentrations of both were significantly higher in the drinking group than in the controls, confirming the results in Expt 1. For the remaining 15 weeks the levels of both substances remained significantly higher in the experimental group and the differences remained in the same order of magnitude.

It is concluded that drinking 4 glasses wine/d affected the liver enzyme γ-glutamyltransferase only in the first few weeks. Serum concentrations became somewhat higher compared with the control period but they stayed well within the normal range, suggesting no harmful effect. Furthermore, the results are only partially in accordance with the above mentioned hypotheses and show that there was a stronger response by coagulation and fibrinolyses factors than blood lipids to the consumption of alcohol.

The relation of alcohol intake and diet to the changing pattern of acute pancreatitis. By M. LARVIN and M. J. MCMAHON, *University Department of Surgery, General Infirmary, Leeds LS1 3EX*

Acute pancreatitis is becoming more common in the UK (Office of Population Censuses and Surveys, 1974–86) but the relation of the increasing prevalence to diet and alcohol consumption remains unclear. The present study is based upon an analysis of 621 attacks studied prospectively in West Yorkshire between 1975 and 1986. At its conclusion, or after further investigation, each attack was attributed to gallstones (detected radiologically, at laparotomy or autopsy), alcohol (mean daily consumption >50 g for at least 3 months, no gallstones), or other (alternative or unexplained causes). Alcohol consumption was quantified by interrogation of patients, corroborating the details with relatives whenever possible.

Significant changes (two-sample rank test) were observed in the age and sex distribution of patients during the periods of study (1975–77, median age 67 years, male:female ratio 0·6; 1984–86, median age 59 years ($P<0·05$), male:female ratio 1 ($P<0·05$)). There was a progressive rise in the incidence of alcohol as the cause of acute pancreatitis from 7% in 1975–77 to 26% in 1984–86, but gallstones have continued to cause about 50% of attacks (47–53%). Alternative or unexplained causes have fallen from 41% in 1975–77 to 27% in 1984–86. The median age in the alcohol group was 38 years, with a male:female ratio of 7·9, whilst the median age of patients in the gallstone group was 66 years, male:female ratio 0·5.

Alcohol-related acute pancreatitis showed an annual peak incidence in the summer months, 27% of these attacks occurring during July and August. An autumn peak for attacks due to gallstones was noted, with 25% of attacks presenting in September or October.

The study shows that alcohol-related acute pancreatitis is becoming more common, and the peak annual incidence is related to high alcohol consumption during holiday periods. The reason for the peak incidence of gallstone-related acute pancreatitis in the autumn is unclear, but seasonal dietary changes constitute a possible explanation.

Office of Population Censuses and Surveys (1974–86). *OPCS Reports*. London: H.M. Stationery Office.

Recent alcohol intake and blood pressure. By RAVI MAHESWARAN, JASWINDER S. GILL and D. GARETH BEEVERS, *University Department of Medicine, Dudley Road Hospital, Birmingham B18 7QH*

The relation between alcohol consumption and blood pressure (BP) has been demonstrated in several epidemiological studies. Clinical studies suggest that regular alcohol consumption over a few days may raise BP, which settles soon after stopping alcohol intake. It is, therefore, possible that alcohol exerts a fairly rapid and reversible effect on BP.

We have examined 258 men in an occupational screening survey to investigate whether BP related better with 'recent' alcohol intake (i.e. intake on days 1 to 3 before examination) or with 'previous' alcohol intake (i.e. intake on days 5 to 7 before examination). Alcohol intake was assessed by a 7 d retrospective diary. Subjects were examined on every day of the week (except for weekends).

A positive relation between alcohol intake for the whole week and diastolic BP was observed ($F = 2{\cdot}79$, $P<0{\cdot}05$) using analysis of covariance. Recent intake significantly influenced both systolic ($F = 5{\cdot}80$, $P<0{\cdot}01$) and diastolic ($F = 6{\cdot}79$, $P<0{\cdot}01$) BP. Previous alcohol intake did not have any effect on BP. Subjects who drank 40 g alcohol/d previously, but none recently, had BP readings similar to those of non-drinkers (see Table).

(Mean values, no. of subjects in parentheses)

		Systolic and diastolic BP		
Recent alcohol intake (g/d) . . .		0	1–40	40
Previous alcohol intake (g/d):	0	122/74 (50)	125/72 (40)	132/80 (10)
	1–40	125/74 (28)	123/73 (58)	128/77 (21)
	>40	120/71 (8)	124/74 (16)	134/78 (27)

We conclude that the effect of alcohol on BP in epidemiological studies is predominantly related to alcohol consumption in the few days immediately preceding BP measurement.

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Which foods contain dietary fibre? The beliefs of 7351 respondents in the Health and Lifestyle Survey 1984–85. By MARGARET J. WHICHELOW, *Cambridge University School of Clinical Medicine, Addenbrooke's Hospital, Cambridge CB2 2QQ*

In recent years emphasis has been put on encouraging the nation to increase its dietary fibre intake. For this to be implemented the public needs to know which foods contain fibre. The Health and Lifestyle Survey 1984–85, which was a nationwide study of a randomly selected representative sample of 9003 adults in Great Britain, provided the opportunity to find out whether the public could discriminate between fibre-rich and fibre-free foods. 7351 of the respondents were asked if they thought each of ten common foods (five fibre-rich and five fibre-free) contained dietary fibre. The proportions of respondents correctly identifying the fibre contents of the foods were: Weetabix® 93.4%, digestive biscuits 80·0%, eggs 78·7%, cheese 74·5%, orange juice 66·2%, grilled fish 57·2%, roast meat 50·7% apples 49·8%, potatoes 46·4% and white bread 41·7%. The respondents were best informed about Weetabix and digestive biscuits which are commercially advertised as being high-fibre foods. That more than 40% of respondents thought that grilled fish and roast meat contained fibre may be due to their 'fibrous' appearance, in the case of white bread the fact that nearly 60% believed that it was fibre-free is probably associated with the emphasis put on the high-fibre content of wholemeal bread.

The distribution of the correct answers given by the respondents was: none 1·4%, one 0·6%, two 1·4%, three 4·3%, four 8·3%, five 12·8%, six 18·1%, seven 23·4%, eight 17·6%, nine 9·6% and ten 2·6%. There was a clear relation with education (see Table).

Relation between the mean number of correct answers about the dietary fibre content of ten foods and educational qualifications

Educational qualification . . .	None	Work related	'O' level	'A' level	Degree/ diploma
♂ No. of correct answers:					
Mean	5·85	6·38	6·46	6·77	7·19
SEM	0·06	0·15	0·07	0·10	0·08
♀ No. of correct answers:					
Mean	5·96	6·69	6·74	7·10	7·51
SEM	0·04	0·18	0·05	0·11	0·07

There was also an association of knowledge about dietary fibre with age. The smallest proportion of respondents giving eight or more correct answers (15%) were among those over 70 years old, whereas the largest percentage (35%) were amongst the 30- and 40-year-olds.

The results indicate considerable misunderstanding about dietary fibre, particularly among the elderly and the less well educated.

This survey was supported by The Health Promotion Research Trust.

Glycine: limiting amino acid for rapid growth. By C. PERSAUD[1], T. FORRESTER[2] and A. A. JACKSON[1], [1]*Department of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO9 3TU* and [2]*Tropical Metabolism Research Unit, University of the West Indies, Mona, Kingston 7, Jamaica*

The evidence is accumulating to support the suggestion that glycine is a conditionally indispensable amino acid. We have shown in normal adults that the urinary excretion of 5-oxoproline (5OP) increases when benzoic acid is used to deplete the body pool of glycine. Hence, 5-oxoprolinuria can be used as an indirect index of glycine status. The metabolic demand for glycine is particularly high during periods of rapid growth and we have measured high rates of urinary 5OP during recovery from malnutrition. In the present study we have looked at the effect of dietary supplementation with glycine during catch-up growth.

Nine males, aged between 10 and 24 months, were given a dietary supplement of glycine, 1·7 mmol/kg per d, for 48 h during rapid catch-up weight gain. Before and at the end of each period of supplementation urine was collected for 12 h for the measurement of 5OP, and the concentration of glutathione (GSH) was measured in blood.

In seven of the nine children there was a fall of up to 75% in 5OP excretion (Δ5OP, −10 μmol/kg per d, range −0·02 to −22) with a concomitant rise of up to 40% in GSH (ΔGSH +5·5 mmol/g haemoglobin (Hb), range +0·7 to +10) over the period of supplementation. In two children increased 5OP excretion (Δ5OP +18 μmol/kg per d) was associated with a fall in GSH (ΔGSH −4·4 mmol/g Hb). These two children showed a higher rate of weight gain (+18 g/kg per d) than any of the other seven (+10 g/kg per d, range 0–18).

The usual association with 5-oxoprolinuria is a congenital deficiency of the enzyme glutathione synthetase (*EC* 6.3.2.3), which forms glutathione from glutamylcysteine and glycine. The results of the present study suggest that limited availability of glycine may produce an increase in 5OP excretion which in some children can be improved with dietary supplementation. Glycine is required to satisfy a number of metabolic reactions, of which protein synthesis is quantitatively the most important. During rapid growth the available glycine has to satisfy a number of conflicting demands. We found that in two children who were growing at a very rapid rate there was an increase in 5OP excretion and a decrease in GSH concentration with the supplemental glycine. Although the effect of glycine supplementation may be complex, we would interpret this as indicating that the demands on glycine for new tissue synthesis were sufficiently great to divert available glycine from GSH production.

We consider that these results support the suggestion that glycine can act as a specific limiting nutrient during periods of rapid weight gain.

The effect of splenectomy on whole body protein turnover in homozygous sickle cell disease. By V. BADALOO[1], A. EMOND[1], S. VENUGOPAL[1], G. SERJEANT[1] and A. A. JACKSON[1,2], [1]*Faculty of Medical Sciences, University of the West Indies, Mona, Kingston 7, Jamaica* and [2]*Department of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO9 3TU*

In patients with homozygous sickle cell disease the continued haemolysis creates a demand for increased erythrocyte production, reflected in an increase in whole body protein turnover. With splenomegaly the rate of erythrocyte destruction increases, and removal of the spleen improves well being. We have measured whole body protein turnover in eight children being considered for splenectomy, and in four of them after removal of the spleen.

The first study was carried out at a time when the children were being considered for splenectomy before preoperative transfusion. The second study was conducted at least 6 weeks after the operation, by which time the acute influence of the operative experience was unlikely to influence the results. Protein turnover was measured with an oral prime/intermittent infusion of [^{15}N]glycine over 12–16 h on a standard hospital diet. Isotopic excretion in urinary urea and ammonia were used to calculate protein turnover.

The ages of the children ranged from 10 months to 11 years. Before splenectomy, mean nitrogen flux was 1·15 (SD 0·19) g N/kg per d with ammonia as the end-product, and 1·50 (SD 0·40) g N/kg per d using urea. In the four children who had the spleen removed, N flux decreased to 0·75 (SD 0·32) g N/kg per d with ammonia and 1·31 (SD 0·42) g N/kg per d with urea. Each individual showed a decrease in N flux of between 23 and 47% following splenectomy.

The basis of the decrease in N flux and the associated fall in protein breakdown and synthesis with splenectomy is presumably related to a fall in the excessive haemolysis, associated with a large spleen. The four children in whom a decision was made not to remove the spleen tended to have lower values for protein turnover, suggestive of less aggressively active disease.

Protein turnover and energy expenditure tend to change in the same direction in various physiological and pathological states: protein turnover being an energy-consuming process. We have found previously that in adult sicklers in a stable state, about 50% of basal energy expenditure could be accounted for by protein turnover. The reduction in protein turnover after splenectomy could result in a theoretical saving of about 22 kJ/kg per d, an amount sufficient to allow for the deposition of balanced new tissue of 1 g/kg per d, the normal rate of growth of a 1-year-old child. In a similar group of children we have found an accelerated rate of gain in height after splenectomy.

The energy cost of compensating for the accelerated haemolysis seen in splenomegaly seems to be considerable. Splenectomy may occasion a saving of energy which allows for the growth spurt that follows the operation.

Fat metabolism during prolonged exercise: influence of carnitine supplementation. By C. WILLIAMS[1], M. P. WALKER[1], M. G. NUTE[1], J. JACKSON[2] and S. BROOKS[1], [1]*Department of Physical Education and Sports Science, University of Technology, Loughborough, Leics. LE11 3TU* and [2]*Department of Renal and Metabolic Medicine, St Mary's Hospital, Portsmouth PO3 6AD*

Fatigue during prolonged exercise occurs as a result of a reduction in muscle glycogen concentration in spite of the availability of an abundance of fatty acids. There is, however, some evidence that the concentration of muscle carnitine decreases during prolonged exercise (Lennon *et al.* 1983) and this may contribute to the onset of fatigue by reducing the rate of fat metabolism. The purpose of the present study was to examine the influence of carnitine supplementation of the endurance capacity of a group of fourteen experienced male runners. Each runner completed two treadmill runs to exhaustion, at a speed equivalent to 70% maximum oxygen consumption (V_{O_2max}). After the first run (control) the subjects were randomly assigned to either the carnitine group, which received a daily supplement of carnitine (50 mg/kg), or the placebo group, for a period of 3 weeks before the second run. Blood and expired air samples were taken before, during and immediately after exercise for the analyses of plasma carnitine, free fatty acids (FFA), glycerol, the catecholamines, blood glucose, lactate concentrations and the determination of the respiratory exchange ratio (R). The performance times, the concentrations of plasma FFA and glycerol, along with the values for blood glucose and R at exhaustion are shown in the Table.

Group . . .	Control		Placebo		Carnitine	
	Mean	SD	Mean	SD	Mean	SD
Performance time (min)	107·0	29·4	107·4	30·5	112·0	46·7
FFA (mM)	0·73	0·34	0·87	0·24	0·91	0·44
Glycerol (mM)	0·58	0·19	0·56	0·22	0·57	0·23
Glucose (mM)	5·23	1·47	4·47	0·96	4·58*	1·39
R	0·93	0·04	0·92	0·03	0·90*	0·03

Significantly lower than the control values: *$P<0.05$.

Carnitine supplementation was effective in increasing plasma carnitine concentrations before ($P<0.05$) and during ($P<0.01$) the second run but there was no significant improvement in endurance capacity. However, while there were no differences in the O_2 cost of the two runs, only the carnitine group had significantly lower R values and blood glucose concentrations at exhaustion, suggesting an increased fat metabolism.

The financial support of Powell & Scholefield Ltd is gratefully acknowledged.

Lennon, D. L. F., Stratman, F. W., Shrago, E., Nagle, F. J., Madden, M., Hanson, P. & Carter, A. L. (1983). *Journal of Applied Physiology* **55**, 489–495.

Influence of acute food restriction on rabbit skeletal muscle sarcoplasmic reticulum Ca^{2+}/Mg^{2+} ATPase (*EC* 3.6.1.3). By S. A. WOOTTON and J. MCWHIRTER, *Departments of Human Nutrition and Biochemistry, Southampton University, Southampton SO9 3TU*

Recent studies have suggested that alterations in the ability to regulate intracellular calcium within the skeletal muscle fibre may contribute to the altered contractile characteristics observed in malnourishment (Russell *et al.* 1984).

As the principal mechanism for the regulation of intracellular Ca resides within the sarcoplasmic reticulum (SR), we have determined the influence of acute food restriction on the Ca^{2+}/Mg^{2+}-dependent ATPase of SR in the rabbit.

Five female New Zealand White rabbits were maintained on their normal stock diet (Labsure CRB) and water *ad lib.* for a period of 7 d, whilst five food-restricted rabbits (FR) received water *ad lib.* and a quantity of food equal to 25% of the intake of their weight-matched littermates (controls) on the previous day. On the morning of the 8th day, the rabbits were killed under anaesthesia (Interval; May & Baker, Dagenham, Essex; intravenously) and the hind-limb muscles rapidly removed.

ATPase activity of vesicles prepared from homogenates of white and red muscles was determined using a linked enzyme assay in the absence (coupled activity) and presence (uncoupled activity) of the Ca ionophore A23187 according to the method of McWhirter *et al.* (1987). Protein concentration was determined by the absorption at 280 nm, calibrated against Biuret.

The body-weights of the FR group fell from 3·354 (SD 0·545) kg to 3·116 (SD 0·591) kg ($P<0.05$) whilst that of the controls increased slightly from 3·324 (SD 0·767) kg to 3·382 (SD 0·687) kg (not significant).

		Coupled activity (μmol P_i/mg protein)		Uncoupled activity (μmol P_i/mg protein)		Ratio of uncoupled: coupled activity	
Muscle		Mean	SD	Mean	SD	Mean	SD
Control	White	0·93	0·24	11·10	1·69	12·88	5·27
	Red	0·94	0·23	9·98	1·86	11·06	3·19
FR	White	1·29*	0·43	9·60	2·93	8·72*	4·57
	Red	1·11	0·46	7·88	4·00	7·72*	4·08

P_i, inorganic phosphate.
Significantly different from control value (Wilcoxon Ranked Pairs): *$P<0.05$.

As the ratio of ATPase:total protein remained unchanged, the lowered uncoupled:coupled ratios would suggest a decreased efficiency of Ca pumping following food restriction.

McWhirter, J., Gould, G. W., East, J. M. & Lee, A. G. (1987). *Biochemical Journal* (In the Press).

Russell, D. McR., Atwood, H. L., Whittaker, J. S., Hakura, T., Walker, P. M., Mickle, D. A. G. & Jeejeebhoy, K. N. (1984). *Clinical Science* **67**, 185–194.

Responses of lean and obese Zucker rats to two types of *Escherichia coli* endotoxin. By R. F. GRIMBLE and DENISE HOLDEN, *Department of Human Nutrition, Southampton University Medical School, Southampton SO9 5NH*

An impaired immune system and reduced capacity for thermogenesis are characteristics of genetically obese rodents (Meade & Sheera, 1979; Trayhurn *et al.* 1979). Many of the metabolic effects of endotoxins, such as fever, have been attributed to interleukin 1 (IL1), a product of the immune system. Some forms of endotoxin produce a rapid depression of rectal temperature for several hours (Wan & Grimble, 1986). The present study examined the responses of lean and obese Zucker rats to hyperthermic (ES, strain 0127:B8, butanol extract) and hypothermic (ED, strain 0127:B8, TCA extract) varieties of *Escherichia coli* endotoxin.

Male lean and obese Zucker rats (body-weight 148 (SE 2) and 148 (SE 3) g respectively) were given an intraperitoneal injection of endotoxins ES or ED (400 μg/kg body-weight) or sterile, non-pyrogenic saline (9 g sodium chloride/l). Rectal temperatures were measured, before injection and at 2-h intervals thereafter, for 6 h. Food was removed from all animals. Rats were decapitated 24 h after injection, blood collected, and liver, spleen and tibialis muscle rapidly removed, weighed and frozen in liquid nitrogen. Tissues were stored at −20° until analysed for protein by the Lowry method. Serum albumin was measured by the bromocresol green method and serum zinc by atomic absorption spectroscopy.

	Lean			Obese		
Rectal temperature (°) after treatment (period of treatment, h):	Saline (*n* 5) Mean	ES (*n* 5) Mean	ED (*n* 5) Mean	Saline (*n* 5) Mean	ES (*n* 5) Mean	ED (*n* 5) Mean
0	36·7	36·9	36·9	36·4	36·4	36·2
2	36·9	37·4	35·8	36·4	36·3	34·8
4	37·1	36·8	36·0*	36·5	36·4	33·9*†
6	36·4	37·4*	36·3	36·7	36·4	33·9**†
Spleen wt (mg)	442	492	502	336†	340††	422*
Serum Zn (μg/ml)	2·04	1·84	1·72	2·68	2·46	1·60**
Serum albumin (mg/ml)	39·1	35·9**	34·5**	39·2	36·3**	33·9**
Tibialis protein (mg)	39·1	39·6	28·9*	27·8†	26·6†	26·6
Liver protein (g)	0·97	1·06	1·10	1·22	1·31†	1·38**††

Significantly different (ANOVA) from saline group: *$P<0.05$, **$P<0.01$; from lean group: †$P<0.05$, ††$P<0.01$.

Impaired responsiveness of some, but not all, target tissues for endotoxin action occurred in obese rats. Obese rats were unable to mount a fever in response to ES and suffered from a severe fall in rectal temperature in response to ED. Muscle protein loss was not apparent in obese rats; however, other metabolic responses to endotoxins were normal.

Meade, C. J. & Sheera, J. (1979). In *Animal Models of Obesity*, pp. 205–220 [M. F. V. Festing, editor]. London: Macmillan Press.

Trayhurn, P., Thurlby, P. L., Woodward, C. J. H. & James, W. P. T. (1979). In *Animal Models of Obesity*, pp. 191–203 [M. F. V. Festing, editor]. London: Macmillan Press.

Wan, J. & Grimble, R. (1986). *Proceedings of the Nutrition Society* **45**, 83A.

The effects of alcohol on blood pressure in rabbits. By PETER G. BURSZTYN and ZOE GILLBE, *School of Biochemical & Physiological Sciences, University of Southampton, Southampton SO9 3TU*

In epidemiological studies, blood pressure (BP) has been related to alcohol intake (e.g. Klatsky *et al.* 1977). However, experiments have given equivocal results. This is partly due to the difficulty of performing such studies blind, and to the psychological consequences of drinking alcohol. Animal experiments should avoid these non-pharmacological problems, but also give equivocal results. Moreover, animal studies tend to use very high doses.

Rabbits were caged individually from weaning for 7 weeks (mean body-weight 3 kg). Drinking water was replaced with ethanol (30 g/l) for 4 weeks, when water was given again. One group of eight animals served as a control for 4 weeks alongside a group of eight receiving alcohol. The first group was then given alcohol. A third group of eight rabbits received ethanol (40 g/l) for 5 weeks. BP was measured daily using a Grant–Rothschild ear capsule. Food and water intakes and animal weights were measured daily. Results for all 24 animals are given in the Table.

	Control period			Alcohol ingestion					Control period		
Week ...	−2	−1	0	1	2	3	4	5	1	2	3
BP (mmHg)											
Mean	61·0	61·0	60·5	60·9	60·8	60·3	59·9	60·4	59·7	59·6	59·5
SE	0·25	0·29	0·25	0·23	0·27	0·27	0·31	0·21	0·41	0·34	0·28
Ethanol intake (g/d)											
Mean	0	0	0	2·7	4·1	4·6	4·7	5·4	0	0	0
SE	–	–	–	0·18	0·17	0·22	0·20	0·11	–	–	–
Fluid intake (g/d)											
Mean	190	186	195	155**	138**	140**	142**	135**	206	190	192
SE	7·6	6·7	8·2	9·5	5·7	6·9	5·8	4·5	7·9	6·4	8·6
Energy intake (kJ/d)											
Mean	987	1013	1046	929**	971**	954**	987	510**	971*	1046	1100
SE	15·1	27·2	23·8	21·3	21·3	21·8	20·5	28·5	18·4	20·9	28·0
Wt gain (g/d)											
Mean	13·2	16·5	11·3	7·0**	6·7**	10·0	10·4*	6·8**	8·1*	10·7	9·9
SE	2·1	1·5	0·9	1·5	1·7	2·3	1·5	1·0	1·3	1·7	1·8

Significantly different from the mean of the first three control weeks (paired *t* test): $^{*}P \leqslant 0.05$, $^{**}P \leqslant 0.01$.

The animals were not fond of alcohol. Fluid and energy intakes and weight gain all decreased when ethanol was given. Alcohol ingestion failed to increase their energy intake as it does in man (Bursztyn, 1986).

Alcohol constituted 17% of energy intake during the experimental period (equivalent to moderate drinking), and this dose was not pressor. Indeed, two recent experiments were only able to demonstrate a small increase in BP in men drinking moderately (Puddey *et al.* 1985; Howes & Reid, 1986).

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Howes, L. G. & Reid, J. L. (1986). *Journal of Hypertension* **4**, 421–425.
Klatsky, A. L., Friedman, G. D., Sieglaub, A. B. & Gerard, M. J. (1977). *New England Journal of Medicine* **296**, 1194–1200.
Puddey, I. B., Beilin, L. J., Vandongen, R., Rouse, I. L. & Rogers, P. (1985). *Hypertension* **7**, 707–713.