AMYLOPECTIN STARCH INDUCES INSULIN RESISTANCE WHICH IS NON-REVERSIBLE IN RATS

C.E. WISEMAN, J.A. HIGGINS, G.S. DENYER and J.C. BRAND MILLER

Starches which have a high amylopectin content are digested and absorbed more quickly and thus produce larger post-prandial glycaemic and insulinaemic responses than starches with a high amylose content. Rats fed a high-amylopectin diet have been shown to develop insulin resistance compared with rats fed a high-amylose diet. The aim of this study was to determine whether amylopectin-induced insulin resistance could be prevented or reversed by a period of high-amylose feeding.

Six groups of six Wistar rats were used. The two test groups were fed for a total of 16 weeks: one group on a high-amylose diet for eight weeks then a high-amylopectin diet for a further eight weeks, the other group received the diets in the reverse order (eight weeks' high-amylopectin diet then eight weeks' high-amylose diet). Four control groups were fed either the high-amylopectin or high-amylose diet only for eight or 16 weeks. The high-carbohydrate diet (67% energy from carbohydrate) was presented as two, 10 g meals per day (300 kJ/day). The diets were identical except for the starch content. The starch in the high-amylose diet was 61% amylose whereas the starch in the high-amylopectin diet was 0% amylose. The degree of insulin sensitivity was determined at the end of each dietary period using an intravenous glucose tolerance test (IVGTT).

Glucose tolerance did not significantly differ at any time point between any group. However insulin responses to the IVGTT varied greatly (Figure). Rats fed the high-amylose diet remained relatively insulin sensitive whereas rats maintained on the high-amylopectin diet became progressively insulin resistant, shown by the increasingly large insulin response to the glucose bolus. The level of insulin resistance in rats fed both diets over 16 weeks was similar to that observed in rats fed on the high-amylopectin diet for 16 weeks, despite having an eight week period of high-amylose feeding.

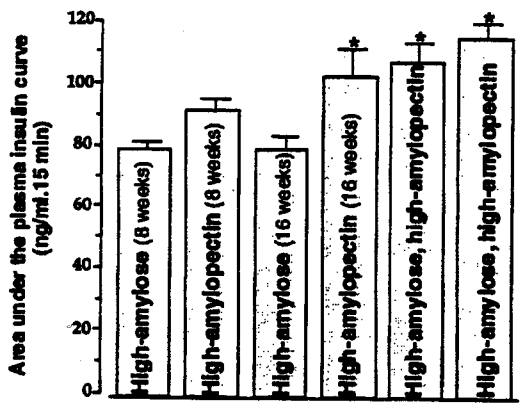

Figure. Area under the plasma insulin curve was calculatede from the insulin response 0 to 15 minutes following infusion of a 1 g/kg bolus of glucose. Results are expressed as mean ± SEM (n=4-6).
*P<0.01 for a difference with rats fed high-amylose diet for eight weeks.

These findings suggest that amylopectin-induced insulin resistance cannot be reversed or prevented by either a subsequent or previous period of amylose-feeding. There may be important ramifications for the primary prevention of non-insulin dependent diabetes mellitus (NIDDM) if these results can be extrapolated to humans.

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IN SEARCH OF MORE LOW GLYCAEMIC INDEX FOODS

J. BRAND MILLER, L. BELL, K. DENNING and D. BROWNE

The glycaemic index (GI) of foods is a ranking of foods based on their postprandial glycaemic response. Blood glucose changes after different foods containing equivalent amounts of carbohydrate are compared with the response to a reference food (usually white bread or glucose) and expressed on a scale where the GI of the reference food = 100. The GI approach has been shown to be useful in the dietary treatment of diabetes and obesity, research on appetite and in sports performance (Brand Miller et al. 1994). To date over 500 foods have been tested to determine their GI (Foster-Powell and Brand Miller 1995) There is still a need, however, to expand the list of foods with a known GI value. The aim of the present study was to determine the GI values of some commonly consumed 'brand-name' Australian foods, some of which we believed would have a low GI.

Eight healthy volunteers with normal glucose tolerance consumed 50 g carbohydrate portions of 16 foods. White bread was the reference food but the final result was multiplied by 0.7 in order to use a GI scale where glucose = 100. Capillary blood was sampled at 0, 15, 30, 45, 60, 90 and 120 minutes, centrifuged and the plasma glucose concentration analysed by the glucose hexokinase method (Brand Miller et al. 1995). The incremental area under the curve was calculated using Simpson's rule with the fasting level as baseline.

On the basis of our findings, the following foods were classified as low GI foods, based on values less than or equal to 50 (mean ± SE): Burgen Oat Bran and Honey Loaf (31 ± 3), Maggi 2-Minute Noodles (46 ± 5), Fielder's Ploughman's loaf (47 ± 3). Seven foods were classed as intermediate GI foods (ie GI between 51 and 69 inclusive): Kelloggs Special K (54 ± 4), Kelloggs Miniwheats (58 ± 8), Vogel's Honey and Oats Bread (55 ± 5), Vogel's Roggenbrot (59 ± 5), Riga Sunflower and Barley Bread (57 ± 6), Latina Gnocchi (68 ± 9), Green's Microwave Popcorn (55 ± 7). The following foods were considered high GI foods (GI > 70): Parker's Kavli (Norwegian crispbread) (71 ± 7), Parker's Pretzels (83 + 9), Uncle Toby's Breakfast Bars Fibreplus (78 ± 9), Uncle Toby's Wheatbites (72 ± 11), Buttercup Wonderwhite™ Bread (80 ± 8).

The findings indicate that very few modern starchy foods have a low glycaemic index. Products with a low GI have achieved this by incorporation of high levels of whole kernels or providing a consistency similar to that of pasta. The food industry can play an important role in providing more low GI starchy foods by modifications to product formulation. Lowering the GI of the overall diet may reduce a population's risk of developing non-insulin-dependent diabetes and coronary heart disease.

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OXIDATION OF LOW DENSITY LIPOPROTEINS IN RENAL TRANSPLANT PATIENTS: EFFECT OF DIETARY ALTERATION

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Cardiovascular disease (CVD) is the major cause of death in renal patients whose risk is substantially increased compared to age-matched individuals from the general population (USRDS 1991). The risk of CVD remains high after renal transplant (Kasiske 1988). The factors responsible are not well delineated, but many patients have lipid abnormalities. Oxidation modification of low density lipoprotein (LDL) is also postulated as a possible important factor in the development of atherosclerosis (Richard et al. 1991). In healthy subjects and hyperlipidaemic subjects, dietary alteration has been shown to alter the susceptibility of LDL to oxidative modification (Corboy et al. 1993; Abbey et al. 1993). This study investigates the effect of alteration in dietary fat on lipids and LDL oxidation in a group of renal transplant patients.

Seven patients who had undergone a renal transplant at least six months previously and who had normal or mildly impaired renal function (plasma creatinine 80-180 μmol/l) were recruited into a dietary study to assess the effect of increasing the amount of monounsaturated fatty acids (MUFA) in their diet. After baseline blood sampling, the patients entered a control period or a MUFA diet period for one month each. During the control period they consumed their usual diet. During the MUFA diet they consumed a cereal containing MUFA and used rapeseed margarine and oil as a replacement for their usual spread and cooking fat. The subjects completed a seven-day diet record between weeks three and four. Venous blood specimens were taken at the end of each period for the measurement of plasma cholesterol, LDL and HDL cholesterol, triglycerides, TBARS, conjugated dienes (lag time, rate and maximum production).

Dietary analysis showed a significant increase in fat content on the MUFA diet — the mean (SD) MUFA intake being 25(6)g and 39(8)g on the control and MUFA diet respectively, and a decrease in carbohydrate intake (particularly of sugars). Polyunsaturated fatty acid intake increased slightly and saturated fatty acids remained constant. The other components of the diet were not significantly different. Body weight remained stable. Plasma LDL cholesterol was significantly lower on the MUFA diet ($P<0.01$), and the % free cholesterol was significantly increased and the % cholesterol esters significantly less. TBARS were not significantly different. The lag time of conjugated diene formation was significantly increased on the MUFA diet: mean (SD) 87 (12) mins vs. 72 (8) mins ($P<0.05$) on the usual diet and the rate of diene formation was much less ($P<0.001$).

Given the high CHD risk of renal transplant patients, a reduction in susceptibility of their LDL to oxidation, resulting from an increase in MUFA in the diet, may be beneficial and should be further explored.

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ACUTE EFFECTS OF DIETARY CLENBUTEROL ON BLOOD FLOW IN HEPATIC PORTAL VEIN, HEPATIC ARTERY AND COMMON ILIAC ARTERY LAMBS

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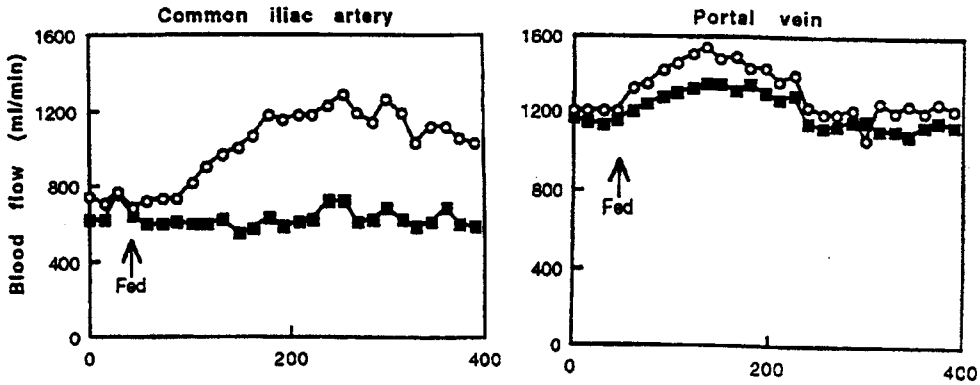
Potent effects of the β adrenergic agonist clenbuterol on partitioning of nutrients towards lean tissue growth are well known (Moloney et al. 1991), but the mechanism by which this occurs is not clear. Previous studies have indicated a direct action on skeletal muscle as well as an increase in blood flow to skeletal muscle tissues (Eisemann et al. 1988). In the latter study the indicator dilution technique was used to measure blood flow in steers. The effects of clenbuterol on real time changes in blood flow to skeletal muscle tissues and portal drained viscera are not known. Therefore, in this study we have made simultaneous measurements of real time blood flow in the hepatic portal vein, hepatic artery and in the common iliac artery in lambs fed clenbuterol.

Six cross-bred ewe lambs (25-30 kg liveweight) were surgically prepared by placement of ultrasonic perivascular flow probes around the portal vein, hepatic artery and common iliac artery. The lambs were fed a mixture of good quality lucerne and rolled barley in the ratio 60:40 (w:w). After a minimum of a seven-day recovery period portal blood flow was monitored continuously for one hour pre-feeding and for seven hours post-feeding for three consecutive days. On day two, clenbuterol was mixed with the feed at a dose of 0.3 mg/kg feed. The mean blood flows are presented in the Figure (day one: solid squares, day two: open circles).



Feeding clenbuterol resulted in a prompt increase in blood flow in the common iliac artery ($P < 0.05$) and the flow remained elevated during the measurement period on day two. No significant changes in the blood flow were observed in either hepatic portal vein or in the hepatic artery. The hepatic arterial blood flow (60-90 ml/min) was only about 5% of the portal blood flow. The results demonstrate a marked increase in blood flow to hindquarters but no change in blood flow to portal drained viscera in response to clenbuterol. This lack of response in the hepatic portal and arterial blood flows are likely due to lack of β adrenergic receptors in these vascular beds.

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IMMUNISATION OF CHICKS WITH SOMATOSTATIN IMPROVES GROWTH

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In our laboratory immunisation of hatchling chickens with somatostatin (SRIF) has promoted growth in some but not all experiments (Sutton and Westbrook, unpublished data). The present study was conducted to assess whether the variability in responses following immunisation might be related to nutrition and/or genotype.

Eighty chicks of mixed sexes and of each of two genotypes (Strain IM98 Ingham Pty Ltd, Packenham, Victoria; and Strain Cobb - Biadia Poultry, Tullamarine, Victoria) were allocated randomly to groups of 20 birds/genotype and maintained on deep litter. They were fed crumbles (Barastock Stockfeed Pty Ltd, Packenham, Victoria); for 21 days starter crumbles were fed, between days 22-24 a mixture (50:50) of starter: finisher crumbles were fed and thereafter finisher crumbles. Water was available ad libitum. Two groups of each genotype were offered feed ad libitum (AL) and the others were offered 90% (R) of the corresponding intakes for the genotype. All birds received intra-peritoneal injections on day zero of either placebo (NI birds) or SRIF [I birds; SRIF-14 (Novobiochem, Switzerland) in a proprietary delivery system (Inovax Pty Ltd, Nedlands, Western Australia); 8 μg SRIF/bird] followed by an oral dose of the same preparation on day 21. One group of birds from each nutritional level x genotype combination was immunised. All birds were sacrificed on day 43. Results are summarised in the Table.

Strain/treatment	AGR (g/day)	Fat pad (% BW)	Glucose (mM)	a-NH2-N (mM)	Insulin (ng/ml)	GH (ng/ml)
Cobb-NI-AL	56.3	2.2	10.9[a]	6.6	0.2[a]	1.0[a]
Cobb-I-AL	69.9	2.1	10.2[b]	5.7	0.4[b]	2.5[b]
Cobb-NI-R	64.9	2.0	11.6	7.8	0.2	2.6[c]
Cobb-I-R	58.1	1.8	11.5	5.7[b]	0.3	0.9[d]
IM98-NI-AL	68.8	2.1[a]	9.7	7.3	0.4	1.6
IM98-I-AL	70.9	1.7[b]	9.7	5.8	0.3	1.5
IM98-NI-R	62.1	1.5	12.6	7.3[a]	0.2	1.2
IM98-I-R	56.3	1.5	10.6	5.5[b]	0.2	0.8

a,b,c,d within genotype values with different superscripts differ significantly

Between about 14-30 days all birds developed bronchitis which was treated by adding antibiotic to drinking water. In spite of this there were indications that, by comparison with NI birds, the AL-I birds had greater absolute growth rates (AGR), and all I birds generally had lower proportions of body fat, and lower plasma concentrations of glucose; α-amino-nitrogen (α-NH_2-N), insulin and growth hormone (GH). Although differences were not always statistically significant the consistent trends lead us to conclude that in chicks of both strains, immunisation against SRIF increased growth, particularly in the AL-birds.

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EFFECT OF EXERCISE ON PORTAL AND HEPATIC BLOOD FLOW IN LAMBS

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Quantitative assessment of the absorption and uptake of nutrients by the gut and liver requires absolute measures of portal (PVf) and hepatic arterial (HAf) blood flows. In many metabolic studies these flows have been determined by the indicator dilution technique, which requires steady state conditions and cannot measure hepatic arterial blood flow directly. The present study was conducted to examine the effects of exercise on HAf and PVf measured directly in real time.

Five ewe lambs weighing 28-33 kg were housed in metabolism cages and fed to maintenance. Ultrasonic blood flow probes (Transonics Inc. Ithaca, NY) were fitted around the portal vein and hepatic artery. The sheep were exercised on a moving belt treadmill at a speed of 0.7 m/s and inclination of 9° for 1 h. HAf and PVf were measured prior, during and post exercise at 1 second intervals and averaged over five minutes. Measurements were recorded on a datalogger and are presented as a percentage of resting values .

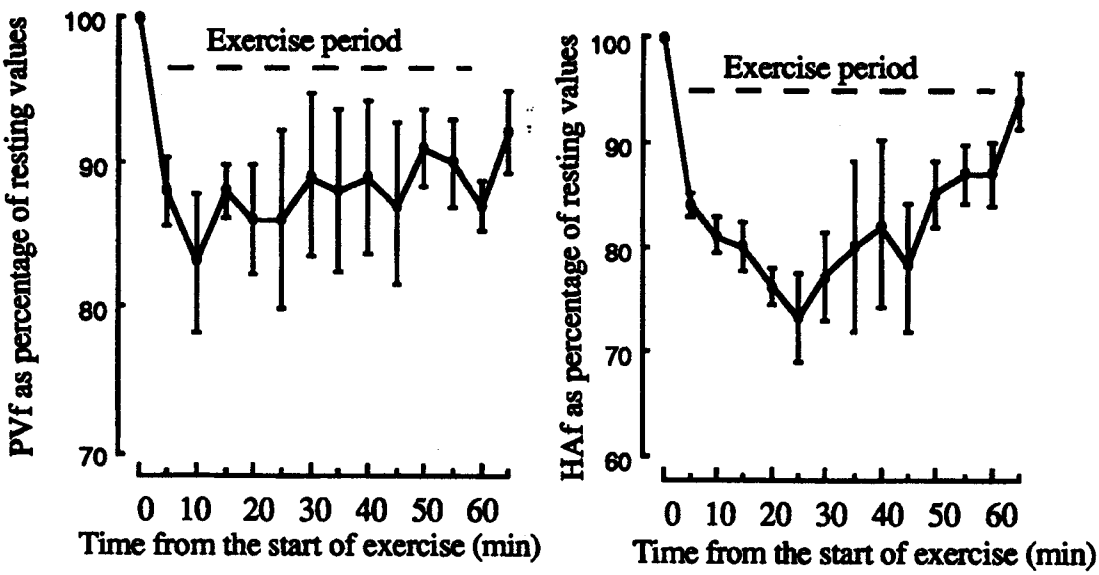

Figure 1. Percentage changes in PVf and HAf during exercise and 5 min post exercise

Mean (± s.e.m.) values for HAf and PVf during rest were 61±2.6 and 1255±120.6 mls/min respectively. These values decreased by a mean of 20% and 13% respectively during exercise and tended to stabilise to pre exercise levels five min post exercise (Figure 1). The decrease in HAf contrasts with the results of Brockman (1987) who measured a three-fold increase in HAf in response to exercise when using the indicator dilution technique. The decrease in PVf and HAf measured in response to exercise in this study would be mediated by changes in the vascular resistances in the mesenteric, splenic and hepatic vasculature. The acute decline in flows at the onset of exercise could be associated with sympathetic stimulation of α-adrenergic receptors in these vessels, while the rebound of HAf towards control values with time may be due to metabolic factors.

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ARACHIDONIC ACID TO EICOSAPENTAENOIC ACID RATIO IN BLOOD CORRELATES POSITIVELY WITH CLINICAL SYMPTOMS OF DEPRESSION

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Major depression is occurring at a younger age and at a higher incidence than prior to 1913 (Klerman 1988; Klerman and Weissman 1989). In epidemiological studies reviewed by Klerman (1988) these findings, primarily for Westernised societies, were not explained by changing diagnostic criteria, changing attitudes of health professionals and societies, reporting bias differential mortality, institutional or other artefacts. Smith (1991) and Hibbeln and Salem (1995) have hypothesised that the sharp rises in depression and other neurological disorders this century have been fuelled by increased consumption of LA-rich vegetable oils. Consistent with this hypothesis are the substantially elevated levels of prostaglandin E_2 and thromboxane B_2 (both derived from AA, a metabolite of LA) reported in patients with both unipolar and bipolar depression (Lieb et al. 1983).

In this study of 20 moderate to severely depressed patients, diagnosed using current research diagnostic criteria (DSM IVR 1995) and excluding known bipolar affective disorder and reactive depression, we investigated the relationships between severity of depression and levels and ratios of n-3 and n-6 long-chain polyunsaturated fatty acids (PUFA) in plasma and erythrocyte phospholipids (PL). Severity of depression was measured using the 21 item Hamilton Depression Rating Scale (1960) (HRS) and a second linear rating scale (LRS) of severity of depressive symptoms which omitted anxiety symptoms. There was a significant correlation between the ratio of erythrocyte PL arachidonic acid (AA) to eicosapentaenoic acid (EPA) and the severity of depression as rated by the HRS ($p<0.05$) and the LRS for depression ($p<0.01$). There was also a significant negative correlation between erythrocyte EPA and the LRS ($p<0.05$). The AA/EPA ratio in the plasma PL and the ratio of erythrocyte long-chain (C20 and C22 carbon) n-6 to longchain n-3 PUFA were also significantly correlated with the LRS ($p<0.05$). These findings do not appear to be simply explained by differences in dietary intake of EPA. We cannot determine whether the high ratios of AA/EPA in both plasma and erythrocyte PL are the result of depression or whether the tissue PUFA changes predate the depressive symptoms. However, we suggest that our findings provide a basis for studying the effect of the nutritional supplementation of subjects with depression which is aimed at reducing the AA/EPA ratio in tissues and the severity of depression.

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COMPARISON OF LOW SATURATED FAT DIETS WITH DIFFERENT α-LINOLENIC:LINOLEIC ACID RATIOS ON BLOOD LIPIDS

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It is recommended that Australians reduce dietary fat to 30% of energy intake (en) with polyunsaturated fat to contribute about 6% en (NHMRC 1992). The intake of the ω6 fatty acid, linoleic acid (LA), has been encouraged because of its LDL cholesterol-lowering properties. The aim of the current study was to determine if substitution of LA, with the ω3 fatty acid, α-linolenic acid (ALA), would adversely affect blood lipid profiles. Increasing the dietary ratio of ALA:LA to increase tissue accumulation of very long chain ω3 fatty acids, may have beneficial effects on the tendency to thrombosis (Allman et al. 1995).

Eighteen healthy men, aged 18 to 35 years, consumed a typical Australian diet for two weeks. At this time they were randomly allocated to one of two low fat diets:- saturated fat (10% en), monounsaturated (13% en) and polyunsaturated (7% en). Both were identical in macro- and micronutrient content with the only difference being the ALA:LA such that calculated ALA:LA was 1.4:1 (ALA-rich diet) or 1:34 (ALA-poor diet). Diets were consumed for six weeks. Blood was sampled at the beginning, mid-point and end-point of the test diets to measure total and HDL cholesterol and triglycerides. Erythrocyte membrane fatty acids were analysed and used to monitor dietary compliance.

The table shows the effects of the diets on blood lipids and erythrocyte fatty acids. No differences were detected between the two diets with respect to plasma cholesterol but the triglycerides increased on the ALA-poor diet compared to the ALA-rich ($P<0.05$). The changes in the fatty acids were indicative of compliance. The very long chain polyunsaturated ω3 fatty acids, eicosapentaenoic acid (C20:5) and docosapentaenoic acid (C22:5) increased on the ALA-rich diet ($P<0.05$).

		Baseline[1] 0 weeks	Mid-point[1] 3 weeks	End-point[1] 6 weeks
Total cholesterol	ALA-rich	3.7 ± 0.5	3.7 ± 0.2	3.6 ± 0.2
(mmol/L)	ALA-poor	3.5 ± 0.3	3.7 ± 0.2	3.4 ± 0.2
LDL cholesterol	ALA-rich	2.3 ± 0.2	2.3 ± 0.2	2.2 ± 0.2
(mmol/L)	ALA-poor	2.1 ± 0.2	2.1 ± 0.2	1.8 ± 0.2
HDL cholesterol	ALA-rich	1.1 ± 0.1	1.0 ± 0.1	1.1 ± 0.1
(mmol/L)	ALA-poor	1.2 ± 0.5	1.2 ± 0.1	1.1 ± 0.1
Triglycerides	ALA-rich	0.8 ± 0.1	0.9 ± 0.1	0.8 ± 0.1
mmol/L	ALA-poor	0.6 ± 0.1	0.8 ± 0.1	0.9 ± 0.1

[1]means ± sem

In conclusion, substitution of LA with ALA had no adverse effects on plasma lipid concentrations. However, the study was conducted in a group of healthy normolipidaemic young men and the results cannot necessarily be extrapolated to older people with dyslipidaemia.

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FRYING FATS AND PLASMA LIPIDS

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An estimated one-third of fat energy in the Australian diet is derived from commercially prepared foods (Baghurst et al. 1987) with fried foods probably contributing a significant proportion of this. Current commercial frying fats are high in saturated fatty acids (COFA 1989) and comprise primarily palm oil and tallow. The purpose of this study was to compare the effects on plasma cholesterol levels of three types of cooking fats suitable for frying: (1) A partially hydrogenated (20% trans fatty acids) semi-solid frying fat (Test Blend), (2) Sunola™, a high oleic genetic variant of sunflower oil, and (3) Palm oil, one of the most commonly used commercial frying oils.

Twenty three free-living hypercholesterolemic men and women participated in a double blind, randomised cross-over trial comprising a two week baseline period (<30% fat energy) followed by three by three-week intervention periods which comprised a background diet (15% fat energy) that was identical throughout the study plus the test foods that contained the oils (20% fat energy) under investigation. Plasma lipids (Mean ± SD) at the end of the baseline and each of the dietary periods are summarised in the Table below.

Plasma lipids mmol/L	Baseline	Palm	Sunola	Test blend
Total cholesterol	5.93 ± 0.84	6.23 ± 0.96b	5.72 ± 0.82a	5.93 ± 0.91
LDL cholesterol	4.07 ± 0.71	4.18 ± 0.81b	3.77 ± 0.58	3.88 ± 0.70
Triglycerides	1.60 ± 0.59	1.83 ± 0.70	1.66 ± 0.70a	1.86 ± 0.84
HDL cholesterol	1.14 ± 0.32	1.25 ± 0.33b	1.22 ± 0.32a	1.18 ± 0.30
LDL/HDL ratio	3.88 ± 1.26	3.60 ± 1.33c	3.29 ± 0.98a	3.50 ± 1.18

[a] P<0.05 Sunola™ vs test [b] P<0.001 palm vs mean Sunola™ and test [c] P<0.05 palm vs mean Sunola™ and test

We conclude that monounsaturated oils such as Sunola™ are clearly preferably to palm oil in terms of cardiovascular risk. Further, monounsaturated blends containing moderate TFAs (<20%) such as the test blend in this study may also lower LDL without altering the LDL/HDL ratio relative to palm oil. The high LDL/HDL ratio noted on the baseline low fat diet suggests that changes in this ratio during dietary intervention may not necessarily reflect changes in cardiovascular risk.

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ARACHIDONIC ACID SUPPLEMENTATION CAUSES AN INCREASED THROMBOXANE TO PROSTACYCLIN RATIO EVEN IN THE PRESENCE OF n-3 FATTY ACIDS

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There is significant interest in the interrelationship between long chain n-3 and n-6 fatty acids due to their ability to modulate eicosanoid production and therefore thrombosis tendency. In general, intake of arachidonic acid (AA) results in enhanced eicosanoid production, whereas n-3 fatty acids (FA) appear to modulate eicosanoid production by decreasing the prostacyclin to thromboxane ratio (Whelan et al. 1993).

This study was designed to investigate the consequences of ingestion of both AA and n-3 long chain FA on the production of thromboxane and prostacyclin in rats. In addition, we wanted to determine whether the inclusion of high levels of long chain n-3 FA would have any beneficial effect on eicosanoid production in the presence of increasing levels of dietary AA. Four groups of male Sprague-Dawley rats were fed a control diet enriched with eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (approximately 100 mg/day) for 24 days. During the last 10 days the four groups were orally supplemented with either 0, 30, 60 and 90 mg/day of ethyl arachidonate. A further group of rats was fed the control diet for 24 days. In vitro aortic prostacyclin (6-keto-PGF1α) production, serum thromboxane (TXB_2) production and plasma platelet and aortic phospholipid (PL) FA were measured.

Enriching the diet with n-3 FA resulted in significant reductions in tissue AA levels and an increase in the n-3 FA, particularly EPA. Despite these changes in FA, no changes were observed for in vitro eicosanoid synthesis compared with the control animals. The inclusion of AA in the diet resulted in dose-dependent increases in tissue AA levels with corresponding decreases in EPA (plasma, platelet and aortic FA PL) compared with the n-3 enriched-fed rats. These changes resulted in significant step-wise increases for both aortic prostacyclin and serum thromboxane production. The dietary AA caused a differential (two-fold) increase in thromboxane relative to prostacyclin for all three levels of AA supplementation. The major effect of AA supplementation on FA PL content was to displace EPA from the membrane PL. This was observed even with as little as 30 mg/day of AA. The increases in eicosanoid production are most likely due to a combination of increased AA content and a reduction in EPA (a cyclooxygenase inhibitor). The results indicated that in the combined presence of dietary AA and n-3 FA, the n-3 FA do not appear to exert a beneficial effect on the production of eicosanoids.

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CONTRIBUTION OF THE BREEDING HERD TO METHANE EMISSIONS DURING THE PRODUCTION OF BEEF FROM PASTURE

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The inventory of greenhouse gas emissions in Australia for 1988 and 1990 was published recently by the National Greenhouse Gas Inventory Committee (NGGIC 1994a). The inventory includes estimates of methane emissions by livestock which were calculated using algorithms set out in the Workbook for Livestock (NGGIC 1994b). For beef cattle, the calculations required both population statistics and estimates of feed intake and digestibility for the nominated years. Calculated methane emissions were summed by season and class (bulls, cows etc.) for each State to obtain the value for Australia. This approach emphasises the contribution of feed quality and animal numbers to overall methane emissions but not that of other components of production, eg, the breeding herd. For beef production, the contribution to methane emissions per beef carcass of the breeding herd required to produce a slaughter beast can be assessed from estimates of its reproductive performance.

Data for each State were obtained from the Australian Bureau of Statistics and the Australian Bureau of Agricultural and Resource Economics to allow calculation for 1990 of branding rate (calves branded per breeding cow, which allows for fertility, fecundity and embryo and perinatal mortality), weaner mortality, cow and bull replacement rates (which include culling and mortality) and bull ratio (bulls per breeding cow). Then, assuming one breeding cycle per year, the number of cows/slaughter beast = {1/[(branding rate) x (1 - weaner mortality) x (1 - cow replacement)]} and of bulls/slaughter beast = {[(cows/slaughter beast) x (bulls/cow)]/(1 - bull replacement)}. Cows/beast values for NSW/ACT, VIC, QLD, SA, WA, TAS, NT and Australia were, respectively, 1.675, 1.614, 2.072, 1.611, 1.749, 1.851, 2.449 and 1.814; bulls/beast values were 0.083, 0.114, 0.118, 0.107, 0.119, 0.120, 0.143 and 0. 108.

Thus, for Australia in 1990, methane emissions from 1.81 breeding cows and 0.11 bulls must be added to the lifetime emissions of a beast slaughtered from pasture to obtain the emissions per carcass. If it is assumed that a beast was slaughtered at 2.5 years' of age, that emissions during its first year were 50% of adult steer emissions and that breeding cows and bulls emitted, respectively, 5 and 35% more than steers, then methane emitted per carcass would be the annual emissions of [(0.5 + 1.5) + (1.81 x 1.05) + (0.11 x 1.35)] = 4.05 steers, of which 51% were associated with the breeding herd. It can be seen that reductions in emissions per carcass can be achieved not only by improving feed quality, which reduces both methane produced per unit of feed and age at slaughter for a given carcass weight, but also by improvements in the components of reproductive performance.

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ENERGY AND AMINO ACID UTILIZATION OF WHEAT-BASED DIETS BY POULTRY: INFLUENCE OF GENOTYPE AND ENZYME SUPPLEMENTATION

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Several studies have demonstrated that the anti-nutritive effects of wheat can be overcome by supplementation with exogenous enzyme preparations, which lower the viscosity of intestinal contents and improve nutrient digestibility and absorption. Although the efficacy of enzyme supplementation in improving growth performance and apparent metabolizable energy (AME) has been well established, there is limited information on the influence of added enzymes on the amino acid utilization of wheat-based diets. In a previous study conducted in our laboratory, addition of two commercial enzyme preparations to diets containing 918 g/kg wheat was shown to improve the apparent amino acid digestibility (AAAD) by chickens (Hew et al. 1995). The present study was undertaken to investigate further the effect of enzyme supplementation (Avizyme 1300®) on the AME and AAAD of practical diets containing 408 g/kg wheat in broiler chickens. An additional objective of the experiment was to obtain information on the influence of genotype on energy and amino acid utilization.

Male broiler chicks from three commercial hatcheries, designated as strain A, B and C, were obtained at one day of age and reared under similar management conditions. On day 35, 40 birds from each strain were selected and groups of four were randomly assigned to each of 30 pens. Enzyme treatment (unsupplemented or supplemented) was then assigned within strain to five pens. The unsupplemented basal diet contained 408 g/kg wheat and celite (20 g/kg) was included as an analytical marker. The birds were fed the experimental diets (pelleted form) from days 35 to 42, and total collection of excreta was carried out during the last three days to determine the AME values. At the end of the trial, ileal contents were obtained and processed, and the apparent ileal digestibilities were calculated as described previously (Siriwan et al. 1993).

Strain x diet type interaction was not significant for any of the parameters evaluated. Enzyme supplementation resulted in 2.0% improvement ($P < 0.05$) in the AME of wheat-based diets. The AME contents of basal and supplemented diets were 13.34 and 13.61 MJ/kg, respectively. Although Strain A (13.50 MJ/kg) and Strain C (13.61 MJ/kg) had numerically higher energy utilization values than Strain B (13.25 MJ/kg), the differences were not statistically significant. The digestibility values of all amino acids were one to two percentage units higher in enzyme supplemented diets, but the differences were significant ($P < 0.05$ to 0.001) only for Asp, Ser, Gly, Val, Ile, His and Arg. Significant ($P < 0.001$) strain effects were observed for AAAD values, with Strain A recording the highest (80.2 - 94.5%) and Strain B the lowest (70.9 - 91.1%).

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CARCASS COMPOSITION AND ENERGY DEPOSITION IN LAMBS FED BARLEY OR FISH MEAL DIETS

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Fish meal contains proteins resistant to degradation in the rumen and when used as a supplement in diets for young sheep fish meal can promote rapid liveweight gains with reduced carcass fatness (Ponnampalam and Hosking 1994). However, fish meal fed as a sole supplement is expensive and poorly accepted by sheep. This study was undertaken to test the efficacy of a pelleted fish meal mixture, formulated to improve palatability and ease of feeding.

Cryptorchid Romney Marsh x Merino lambs were drenched for the control of helminths and allocated by weight to one of three groups. Two groups (n=6 lambs/group) were then randomly assigned to diets consisting of a chopped oaten hay:lucerne hay mixture (3:1) fed ad libitum and supplemented daily with either whole barley grain or fish meal pellets (FMP) at the rate of 1% liveweight. The FMP were prepared by extruding fish meal and milled lucerne hay (1:3 w/w) through a 10mm die. Intake and liveweight gain were monitored over a 10 week period. The lambs were then humanely killed and dissected. Carcass gain and composition were determined by reference to the third group of animals killed at the commencement of the study (Sainz et al. 1994). The supplements were approximately isoenergetic and provided 50 and 164 g/d rumen undegraded protein (UDP) for barley and FMP supplements, respectively.

	Barley	FMP	sem	Significance (P)
Initial liveweight (kg)	26.8	26.8	1.73	
Final liveweight (kg)	34.7	43.3	1.57	<0.01
Carcass weight (kg)	15.6	18.5	0.86	<0.05
Fat content	4.1	4.1	0.39	ns
Protein content	2.4	3.1	0.13	<0.01
Carcass energy content (MJ)				
Protein	58	73	3.1	<0.001
Total energy	220	234	17.2	ns

FMP increased liveweight and carcass weight by 25% and 19%, respectively. Fat content (kg) in the carcass was similar for each diet while protein content was increased 30% by FMP. Overall energy retention in the carcass was similar for both diets although FMP resulted in significantly more (26%) energy retained as protein. Variations in energy deposition in the carcass parallelled differences (P<0.001) in ration intake. DM intakes were 1.12 and 1.44 kgDM/day (sem 0.034) for barley and FMP diets, respectively. The results confirm the effectiveness of FMP for delivery of high UDP mixtures and the role for such supplements as a potent means for the manipulation of body growth and composition through the diet.

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