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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Twenty-first Meeting of the Nutrition Society was held in Lecture Theatre E, Main Lecture Theatre Block, Brunel University, Uxbridge, Middlesex on Tuesday and Wednesday, 17/18 *December* 1985, *when the following papers were read:*

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Effect of inhibition of corticosterone production by Trilostane on the catabolic response of the rat to the *Escherichia coli* endotoxin. By M. M. JEPSON J. M. PELL and D. J. MILLWARD, *Nutrition Research Unit, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

The response to infection involves growth inhibition, muscle wasting and increased acute-phase protein synthesis in the liver and these changes can be induced acutely in fed and fasted rats with the *Escherichia coli* lipopolysaccharide endotoxin, LPS (Jepson *et al.* 1986). Plasma corticosterone is increased in infection and may be involved in some of these changes, since increased corticosterone levels are known to reduce mortality in endotoxaemia.

We have assessed the response of the rat to LPS after inhibiting the increase in corticosterone with Trilostane, a drug which is known to block corticosterone synthesis. Measurements were made in fed rats 24 h after treatment with LPS (3 mg/kg). Three doses of Trilostane (50 mg/kg) were given in all: the first 18 h before the LPS, the second with the LPS and the third 6 h later. Measurements were made of protein content and synthesis in muscle (gastrocnemius and plantaris) and liver, plasma insulin and corticosterone, and muscle glutamine.

	Muscle protein				Muscle glutamine (μmol/g)		Liver protein				Insulin (μ units/ml)	
	Synthesis (%/d)		Content (mg)				Synthesis (%/d)		Content (mg)			
Treatment	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Saline*	15·6	1·6	89·8	5·4	7·7	0·8	95	16	778	31	27·8	14·7
+ LPS	11·0	1·3	83·6	2·3	5·9	1·4	131	19	833	59	46·1	13·2
Trilostane	16·8	0·7	88·0	4·3	7·7	0·6	111	9	732	54	34·1	7·7
+ LPS	10·1	1·6	80·0	7·5	5·3	0·7	118	11	735	83	30·0	8·1

*9 g sodium chloride/l

The effects of Trilostane on plasma corticosterone concentrations could not be measured because it interferes with the steroid assay. Treatment with the drug alone had no effect on growth or muscle and liver protein turnover. The protective effect of corticosterone was demonstrated in that treatment with both LPS and Trilostane was lethal for half of that group. This may have been due to the absence of the increased hepatic protein synthesis. The reduced muscle protein synthesis was independent of the different insulin concentrations and was not affected by the drug, thus it appears that the inhibition of muscle growth is likely to be a direct effect of the LPS. The fall in protein synthesis was correlated with the fall in the glutamine concentration gradient across muscle (a sensitive index of the sodium gradient) so that the mechanism of the response to LPS may involve interference with ion gradients.

The authors are grateful to Stirling Winthrop for the gift of Trilostane. This work was supported by the British Diabetic Association, and the MRC.

Jepson, M. M., Pell, J. M. & Millward, D. J. (1986). *Proceedings of the Nutrition Society* **45**. 36A.

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Inhibitory effects of indomethacin on some features of the metabolic response to *Escherichia coli* endotoxin in rats. By JENNIFER WAN and R. F. GRIMBLE, *Nutrition Department, Southampton University Medical School, Southampton SO9 3TU*

Escherichia coli endotoxin produces a depression of serum zinc, decreased rates of protein synthesis and loss of protein from skeletal muscle, skin and bone. Chronic feeding of coconut oil, which is poor in linoleate, suppresses these changes. The suppressive effects could be due to decreased generation of cyclooxygenase and lipoxygenase products from arachidonate. Only the depression of serum Zn is mimicked by lipoxygenase inhibitor AA861 (Wan & Grimble, 1986), thus other suppressive effects could be via a reduction of products of the cyclooxygenase pathway. This possibility was investigated by pretreating rats with indomethacin before treatment with *E. coli* endotoxin.

Female Wistar rats (234±3 g) were assigned to five groups. Two received indomethacin (Indo. 5; 5 mg/kg body-weight) orally in a vehicle or carboxymethylcellulose (CMC; 50 g/l), one group a higher dose (Indo. 10; 10 mg/kg body-weight) in vehicle and two groups CMC alone. After 1 h, half the rats given Indo. 5, half those given CMC alone and all rats given Indo. 10 received *E. coli* endotoxin (Difco; 400 μg/kg body-weight) intraperitoneally (i.p.). The remaining rats received sterile saline (9 g sodium chloride/l) i.p. All rats were pair-fed on a standard laboratory chow to the amount eaten by the Indo. 10 group (6±1 g). Rats were killed by decapitation 24 h after injection, the blood collected, pelts removed, various tissues rapidly dissected and frozen in liquid nitrogen.

Pretreatment (*n*) . . .	CMC (5)		CMC (5)		Indo. 10 (4)		Indo. 5 (5)		Indo. 5 (5)	
E. coli endotoxin . . .	−		+		+		+		−	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Pelt wt (g/kg body-wt)	175	4	158*	5	158*	9	164	5	170	6
Liver total protein (g)	1·62	0·03	1·99*	0·11	2·19*	0·08	1·81*	0·04	1·73*	0·02
Protein concentration (g/kg)										
Muscle (thigh)	196	3	175*	3	191†	3	188†	1	197	5
Skin (abdomen)	130	1	118*	1	124†	2	125	4	129	2
Femur	97	2	81*	3	101†	2	88*	2	101	2
Serum Zn (μg/ml)	1·55	0·09	0·62*	0·04	0·57*	0·05	0·61*	0·04	1·10*	0·05
Corticosterone (ng/ml)	332	22	490*	20	297†	24	382†	30	316	34

Significantly different from the group receiving CMC alone: *$P<0.05$, or CMC and endotoxin: †$P<0.05$

Protein loss from muscle, skin and bone was inhibited by indomethacin but loss of Zn from serum was not. Changes in Zn and protein metabolism during the acute-phase response are thus under the control of separate branches of arachidonate metabolism. Inhibition of both pathways is involved in the suppressive effect of coconut oil on the response.

Wan, J. & Grimble, R. F. (1986). *Proceedings of the Nutrition Society* **45**, 38A.

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Energy balance of corticosterone-treated rats in relation to body composition. By C. J. H. WOODWARD and P. W. EMERY, *Department of Nutrition, King's College (KQC), Campden Hill Road, London W8 7AH*

Treatment of rats with corticosteroids leads to substantial weight loss without reducing food consumption (Bellamy, 1964). The alterations of energy balance which accompany these changes are, however, unclear. Both increased and decreased energy expenditure have been recorded at different doses (Coyer *et al.* 1985). In the present study we have attempted to clarify this question.

Two groups of male Sprague-Dawley rats, weighing approximately 140 g, were used. One group received corticosterone by daily subcutaneous injection (50 mg/kg per d). After 11 d the animals were killed for carcass analysis. Data on initial body composition were obtained from a third group killed at the start of the study. Metabolizable energy intake (MEI) was calculated from gross energy intake and excreted energy. Energy expenditure was calculated from MEI and energy retention.

	Tissue deposition				Energy expenditure	
	Fat (g/d)	CP (g/d)	Water (g/d)	Energy (kJ/d)	(kJ/d)	(kJ/d per kg body-wt$^{0.75}$)
Steroid-treated	0·96	0·18	−0·23	41	249	1033
Control	1·05	1·08	4·45	65	263	942
$P<$	NS	0·001	0·001	0·05	NS	0·01

NS, not significant (Student's *t* test).

Weight gain was significantly reduced by corticosterone treatment (1·1 *v* 7·0 g/d; $P<0·001$). This difference resulted from reduced deposition of crude protein (nitrogen × 6·25, CP) and water, but not of fat (see Table). MEI was reduced in rats receiving corticosterone (291 *v* ·327 kJ/d; $P<0·01$) and this accounted for the deficit of energy deposition in the treated rats. Whole-body energy expenditure did not differ significantly between the groups.

It is concluded that, because of altered body composition, the energy deficit of corticosterone-treated rats is less pronounced than their weight deficit. Energy expenditure may be viewed as normal or increased, depending on the method of expression.

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Bellamy, D. (1964). *Journal of Endocrinology* **31**, 83–84.
Coyer, P., Cox, M., Rivers, J. P. W. & Millward, D. J. (1985). *British Journal of Nutrition* **53**, 491–499.

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Relation between increased heat production and 3,5,3′-triiodothyronine concentration in rats treated with catabolic doses of corticosterone.

By PENNY COYER, M. COX, J. P. W. RIVERS and D. J. MILLWARD, *Nutrition Research Unit, Department of Human Nutrition, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

Although low doses of corticosteroid hormones suppress heat production (Galpin *et al.* 1983), we have shown that catabolic doses increase it (Coyer *et al.* 1985). We have now investigated whether the increased heat production could reflect thyroidal changes and report here measurements of plasma free and total 3,5,3′-triiodothyronine (T_3) concentrations.

Ten weight-matched groups of male Lister Hooded rats, 80 g initial weight, individually caged and fed *ad lib.* (200 g casein/kg diet), received a daily subcutaneous injection of either 100 mg corticosterone/kg body-weight or vehicle only. On days 1, 3, 4, 5 and 8, oxygen consumption ($\dot{V}_{O_2}$) was measured for 2 h during the day in treated (T) and control (C) rats, after which they were immediately killed and serum collected for measurement of free and total T_3 (Metachem Diagnostics, Northampton).

			Body-wt (g)		$\dot{V}_{O_2}$ (l/2 h per kg body-wt$^{0.75}$)		Total T_3 (ng/ml)		Free T_3 (pg/ml)	
Day	Group	*n*	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	T	4	81·3	2·3	4·0*	0·5	1·7*	0·3	8·65	1·25
	C	4	84·9	2·4	3·5	0·1	1·36	0·0	8·55	0·99
3	T	5	75·5	4·8	4·9*	0·1	1·43	0·22	8·48	1·17
	C	4	97·3	5·4	3·4	0·2	1·37	0·10	8·90	0·81
4	T	4	74·4	1·9	3·4	0·4	1·35	0·36	8·39	2·20
	C	4	105·9	6·0	3·1	0·2	1·35	0·01	8·72	0·74
5	T	4	74·5	7·8	3·4	0·7	0·94*	0·18	6·60*	0·74
	C	5	111·2	6·8	3·4	0·4	1·38	0·23	8·22	0·81
8	T	4	74·6	9·5	3·4	0·4	1·37	0·32	7·75	0·59
	C	5	132·1	6·6	3·1	0·4	1·30	0·21	7·92	0·54

*Significantly different from control (two sample Wilcoxon Rank test): *$P<0.05$.

The treatment completely inhibited growth but increased $\dot{V}_{O_2}$, which peaked on day 3. Although total T_3 increased on day 1, subsequent levels were either unchanged or decreased, while free T_3 was never elevated and was depressed on day 5. The peak increase in $\dot{V}_{O_2}$ thus occurred without increases in either free or total T_3, and $\dot{V}_{O_2}$ was maintained despite a fall in these hormones. Unless the increased $\dot{V}_{O_2}$ occurred in tissues where T_3 status is independent of serum levels of the free hormone, thyroid hormones cannot mediate the increased heat production caused by these doses of corticosterone.

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Coyer, P., Cox, M., Rivers, J. P. W. & Millward, D. J. (1985). *British Journal of Nutrition* **53**, 491–499.

Galpin, K. S., Henderson, R. G., James, W. P. T. & Trayhurn, P. (1983). *Biochemical Journal* **214**, 265–270.

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Gut transit time measured by three markers. By MARGARET LAWSON, *Department of Food and Biological Sciences, Polytechnic of North London, Holloway Road, London N7* and VALDA BUNKER and BARBARA CLAYTON, *Chemical Pathology and Human Metabolism, Medical Faculty of the University of Southampton, Southampton General Hospital, Southampton*

Measurement of gut transit time by carmine red (CR) has been criticized as it is thought to give a falsely low result (Allen *et al.* 1979). Other substances such as polyethylene glycol (PEG) and radio-opaque pellets (ROP) have been described as giving a truer picture. No difference was found in the time of appearance of PEG, ROP and a third faecal marker, chromic oxide, when they were given simultaneously (Simpson *et al.* 1979).

In the present study a single dose of CR, PEG and ROP was given to ten healthy adults eating their normal diet. Stools were collected for 120 h and were X-rayed to detect the presence of ROP. After homogenization, each stool was graded 0–4 for concentration of CR by two observers, using a stool collected before the administration of marker for comparison of colour. PEG was analysed using a modified turbidometric method.

Comparison of gut transit times using three markers in ten adults

	Carmine red			Radio-opaque pellets			Polyethylene glycol		
Subject no.	A	B	C	A	B	C	A	B	C
1	24	30	72	24	44	72	30	48	72
2	37	87–97	>120	37	85	>120	37	97	>120
3	24	48	75	48	48	120	24	48	75
4	15	15	48	15	15	72	15	15	48
5	28	48	72	28	48	72	28	48	72
6	24	48	73	24	24	73	24	48	73
7	40	81–88	>120	12	81–88	>120	20	20–104	>120
8	12	24	24	12	24	68	12	24	24
9	28	48	74	28	48	74	23	48	74
10	21	21	85	21	21	62	10	21	85

A, Period before the first appearance of the marker in the stools (h), B, period before the maximum concentration of marker in stools (h), C, period before the markers were cleared from the stools (h).

The first appearance of all three markers occurred in the same sample in five subjects; maximum marker concentration occurred in the same sample in seven subjects. Four subjects cleared the three markers at the same time.

These results agree with the observation that the initial appearance of a faecal marker is not the best indicator of gut transit time (Fisher *et al.* 1978), and suggest that for subjects eating their normal diet there is little difference in gut transit time when measured by carmine red, polyethylene glycol or radio-opaque pellets.

Allen, L. H., Raynolds, W. L. & Margen, S. (1979). *American Journal of Clinical Nutrition* **32**, 427–440.
Fisher, M. T., Atkins, P. R. & Joplin, C. F. (1978). *Clinica Chimica Acta* **41**, 109–119.
Simpson, F. G., Hall, G. P., Kelleher, J. & Losowsky, M. (1979). *Gut* **20**, 581–584.

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Influence of wheat bran, cellulose, pectin and low or high viscosity guar gum on glucose and water absorption from pig jejunum. By A. G. LOW[1], TERESA ZEBROWSKA[2], L. M. J. HEPPELL[1] and H. A. SMITH[1], [1]*The Animal and Grassland Research Institute, Shinfield, Reading RG2 9AQ* and [2]*Institute of Animal Physiology and Nutrition,* 05–110 *Jablonna, Warsaw, Poland*

The property of soluble types of dietary fibre to decrease the rate of glucose absorption and peripheral blood glucose levels has been associated with their ability to increase the viscosity of liquid meals (Jenkins *et al.* 1978). The aim of the present study was to examine the relation between soluble and insoluble types of dietary fibre, viscosity and glucose absorption from the jejunum of pigs.

Five 35 kg pigs were prepared with two re-entrant cannulas 2·0 and 3·0 m from the pylorus. The jejunum was perfused (8 ml/min) for 6 h/d (3 d/treatment) with a modified Ringer solution containing glucose (20 g/l) and 6·7 g/l of one of the following: wheat bran, cellulose powder (Brown & Co, Berlin, USA), high methoxy citrus pectin (H. P. Bulmer, Hereford) and low or high viscosity guar gum (Meyprogat 60 and 150; Meyhall Chemical (UK) Co., New Ferry Merseyside). The low viscosity guar gum was only given to three pigs. Full details of the perfusion system are given by Rainbird *et al.* (1984).

Perfusate viscosity and % absorption of glucose and water

	Control	Bran	Cellulose	Pectin	Guar gum 60	Guar gum 150
Viscosity (Ns/m^2)†	0·001	0·006	0·003	0·013	0·011	0·179
Glucose	$70·4^a$	$59·6^b$	$57·1^b$	$53·9^b$	$53·7^b$	$40·5^c$
Water	$49·3^a$	$31·8^b$	$29·6^b$	$34·6^b$	$30·0^b$	$18·6^c$

[a, b, c]Mean values followed by a different superscript letter were significantly different: $P<0·01$.

†Measured at 20° and a shear rate of 100/s.

It is evident that even those types of dietary fibre which are largely insoluble and which do not increase viscosity can decrease the rate of glucose and water absorption. At the same time, the influence of soluble types of dietary fibre does not appear to be related in a direct manner to the viscosity which they induce. Further studies are merited to examine the relations between the physico-chemical properties of dietary fibre and their effects on absorption.

Jenkins, D. J. A., Wolever, T. M. S., Leeds, A. R., Gussell, M. A., Haisman, P., Dilawari, J., Goff, D. V., Metz, G. L. & Alberti, K. G. M. M. (1978). *British Medical Journal* i, 1392–1394.

Rainbird, A. L., Low, A. G. & Zebrowska, T. (1984). *British Journal of Nutrition* 52, 489–498.

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Comparative changes in the lipid compositions of liver and bile of the female broiler bird. By R. C. NOBLE and R. MCCARTNEY, *Hannah Research Institute, Ayr KA6 5HL* and D. BROWN, *West of Scotland Agricultural College, Ayr* and P. F. DODDS, *Wye College (University of London), Ashford, Kent TN25 5AH*

It has already been shown that development of the chick embryo with its heavy emphasis on lipid assimilation and unusual lipid accumulation in the liver is associated with a unique biliary lipid pattern (Noble & Connor, 1984). In the broiler bird the intensity of modern poultry meat production results in a continuing lipid stress being placed upon the liver during the early stage of rapid growth. A comparative study has therefore been made of biliary and liver lipid compositions during this and the subsequent egg-laying periods of the broiler bird.

Lipid compositions of the bile and liver from broiler birds during the period of intense growth, i.e. 7 weeks of age (see Table), showed the bile to have a high concentration of triglyceride and reduced concentration of phospholipid. In spite of a substantially increased level of liver triglyceride at the onset of lay, i.e. 24 weeks, the concentration of triglyceride in the bile had reduced considerably and the composition approached that commonly displayed by mammalian species. However, with the accumulation of extremely high levels of triglyceride in the liver during the later stages of egg laying, i.e. 46 weeks, an elevated biliary concentration of triglyceride again appeared.

The lipid composition (weight % of total lipid) of gall bladder bile and liver of the broiler bird

Age (weeks) . . .	7		20		24		42	
	Bile	Liver	Bile	Liver	Bile	Liver	Bile	Liver
Triglyceride	31·6	16·5	1·1	16·5	trace	57·0	4·0	74·7
Free cholesterol	6·8	11·2	9·3	13·0	21·4	5·8	5·8	2·3
Phospholipid	56·5	64·4	88·4	63·3	77·6	32·2	82·0	17·7
Lipid (g/l)	21·1		17·0		5·0		3·4	
Lipid (mg/g wet wt)	21		20		40		72	

At 7 weeks of age the fatty acid composition of the bile triglyceride differed from that of the liver which in turn was different from that of the liver at 24 weeks. In particular, the bile triglyceride displayed lower levels of oleic but higher levels of arachidonic and docosahexaenoic acids.

It is suggested that during the rapid period of growth involving intense liver lipid metabolism, the bile plays a specific role in liver lipid homeostasis. With the onset of egg laying and the consequential daily excretion of some 6 g of liver lipid into the egg, the role is reduced.

Noble, R. C. & Connor, K. (1984). *Proceedings of the Nutrition Society* **43**, 52A.

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Effects of diet on triacylglycerol levels in the liver, bile and serum and on the activities of enzymes concerned with lipid synthesis in the liver of the laying hen. By P. F. DODDS[1], K. E. CROSS[1], R. C. NOBLE[2] and K. CONNOR[2], [1]*Wye College* (*University of London*), *Ashford, Kent TN*25 5*AH* and [2]*Hannah Research Institute, Ayr KA*6 5*HL*

At the onset of the lay, the livers of laying strains of hens are stimulated by oestrogen to synthesize massive amounts of triacylglycerol (TG) for egg production. An excess of TG synthesis over secretion can result in pathological fatty liver syndromes such as the fatty liver haemorrhagic syndrome. Such a condition could provide a convenient model with which to study the control of lipid synthesis in laying hens.

White Hi-Sex birds (17-weeks-old) were paired on a weight and age basis and given one of two diets that were isoenergetic and isonitrogenous. Diet A contained (g/kg) 660 ground maize and 222 soya-bean meal. These were replaced by barley and fish meal respectively in diet B. The diets were given to the birds for a minimum of 23 weeks before liver, blood and bile samples were taken.

The livers from all the birds had a 'fatty' appearance and a few showed haemorrhages. The livers of the birds given diet A contained significantly more TG than those given diet B, whether expressed in absolute or percentage terms (see Table). Serum TG concentrations and the proportions of TG in total bile lipid were also significantly higher in the birds given diet A. The TG content of the bile was similar to that seen in broiler birds of a similar age (Noble *et al.* 1986). As the birds became older (>75 weeks) there was a decrease in biliary TG which coincided with a fall in egg production.

	Diet A			Diet B		
	Mean	SD	*n*	Mean	SD	*n*
Bird wt (g)	1862	146	14	1866	180	14
Age (weeks)	70·0	12·8	14	69·4	15·0	15
Liver TG (g/liver)	3·86	1·56	14	2·50	1·16	13
Liver TG (% liver wet wt)	15·7	6·0	14	14·1	5·14	13
Serum TG (mg/ml)	4·51	0·96	9	3·86	0·72	7
Bile TG (% bile lipid)	7·19	6·22	14	3·46	3·20	13
GPAT (nmol/min per mg protein)	5·67	2·71	12	5·29	2·81	10
mic-PAP (nmol/min per mg protein)	18·5	9·7	11	16·9	8·9	8

There were large variations in hepatic enzyme activities and consequently there were no significant differences between treatments. However, regression analyses revealed an unexpected negative correlation (r −0·57, $P<0·01$) between liver TG concentration and glycerol 3-phosphate acyltransferase (GPAT; *EC* 2.3.1.15) activity in the combined groups, whereas microsomal phosphatidate phosphatase (mic-PAP; *EC* 3.1.3.4) was positively correlated (r 0·72, $P<0·02$) with liver TG level in the birds given diet A. This suggests that mic-PAP, but not GPAT, may be rate limiting in the increased production of TG seen in these birds.

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Noble, R. C., McCartney, R., Brown, D. & Dodds P. F. (1986). *Proceedings of the Nutrition Society* **45**, 56A.

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Normal anthropometric values for a large group of London subjects: comparison with surgical patients. By N. CARVER[1], ELS VAN PERSIJN VAN MEERTEN[2], A. W. JOHNSON[3] and A. J. W. SIM[1], [1]*Surgical Unit, St Mary's Hospital, London W2*, [2]*Leiden University, Leiden, The Netherlands* and [3]*Department of Medicine, St James's University Hospital, Leeds*

Clinical studies using anthropometric measurements to determine body composition have tended to use published reference values or values obtained from out-patients departments. These values may not be appropriate for the particular clinical population, therefore this study has used a large number of normal subjects from a population which is the source of the clinical input for a London teaching hospital. These values were compared with those obtained from successive surgical patients attending for elective operation.

Normal subjects (209 female, 189 male) were assessed for height, weight, skinfold thickness (four sites, both sides), mid-arm circumference and hand-grip dynamometry. Bone-free arm muscle area (Heymsfield *et al.* 1982), Benn index (Frisancho & Flegel, 1982) and body fat mass were calculated and the results stratified into age-bands. Similar data manipulations were conducted on the results from seventy-eight surgical patients.

The normal population showed age and sex changes in height and weight as found by others (Durnin & Womersley, 1974); however, there were considerable differences in the mean values for several of the measured variables when compared with those in the literature (Frisancho, 1984). Dynamometry in particular failed to correlate with any of the anthropometric measurements when age and sex were taken into account. The Benn index correlated well with body fat.

The patient sample showed unusual results in that those male patients attending for minor surgery had significantly lower values for dynamometry, arm muscle area, body fat mass and Benn index ($P<0{\cdot}005$) when compared with the normal group; however, there were no differences detected between the female minor-operation patient group and the control group. Dynamometry did not appear to be related to any anthropometric variable.

These results suggest that reference values for clinical studies should be established using the indigenous population.

Durnin, J. V. G. A. & Womersley, J. (1974). *British Journal of Nutrition* **32**, 77–97.
Frisancho, A. R. (1984). *American Journal of Clinical Nutrition* **40**, 808–819.
Frisancho, A. R. & Flegel, P. N. (1982). *American Journal of Clinical Nutrition* **36**, 697–699.
Heymsfield, S. B., McManus, C., Smith, J., Stevens, V. & Nixon, D. W. (1982). *American Journal of Clinical Nutrition* **36**, 680–690.

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Aspartate aminotransferase (*EC* 2.6.1.1) activation by pyridoxal phosphate as an index of vitamin B_6 nutritional status: validity in the presence of oestrogen conjugates. By ABDELAZIZ DENG BIOR, *Department of Biochemistry, Faculty of Medicine, University of Khartoum, PO Box* 102, *Khartoum, Sudan* and DAVID A. BENDER, *Courtauld Institute of Biochemistry, The Middlesex Hospital Medical School, London W1P 7PN*

Previous studies have shown that the tryptophan load test is not valid as an index of vitamin B_6 nutritional status in women receiving oestrogens. This is because oestrogen conjugates inhibit kynureninase (*EC* 3.7.1.3) independently of vitamin B_6 (Bender & Wynick, 1981; Bender, 1983). The present study was undertaken in order to determine whether the activation of aspartate aminotransferase (*EC* 2.6.1.1.) by pyridoxal phosphate is similarly affected by oestrogen conjugates, and hence whether this test for vitamin B_6 nutritional status is likely to be reliable in women receiving oestrogens.

A partially purified preparation of aspartate aminotransferase from pig heart (Sigma Ltd, Poole, Dorset) was used; activity was determined by the release of ^3H_2O from [2,3-^{3}H]aspartate (Schuster *et al.* 1978) and the partially resolved apo-enzyme was prepared as described by Scardi *et al.* (1962). The results in the Table show that oestrone sulphate was not an inhibitor of aspartate animotransferase, and indeed at the unphysiologically high concentration used (1 mM) was a slight activator.

The effect of adding 1 mM-oestrone sulphate to aspartate aminotransferase

(Mean values and standard deviations for six replicate determinations at five concentrations of substrate)

	Control		+1 mM-oestrone sulphate	
	Mean	SD	Mean	SD
K_m aspartate (mM)	48	5·6	45 NS	4·4
K_m α-oxoglutarate (mM)	1·11	0·099	0·96•	0·093
V_{max} (μmol/min)	2·1	0·42	2·3 NS	0·41
Activity of partially resolved apoenzyme (μmol/min) at saturated substrate concentration:				
Without pyridoxal phosphate	1·24	0·063	1·45•	0·054
+1 mM pyridoxal phosphate	2·24	0·085	2·3 NS	0·41

NS, not significant. •$0·01 > P > 0·05$.

It seems likely that the activation of aspartate aminotransferase by pyridoxal phosphate is a reliable index of vitamin B_6 nutritional status in the presence of oestrogen conjugates, although the activation of the apo-enzyme may mask a marginal deficiency.

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Birth weight and feeding practices of infants in Southall, Middlesex. By B. QURESHI[1], FARAH SHEIKH[2], D DONALDSON[3], JANE B. MORGAN[2] and J. W. T. DICKERSON[2], [1]*Featherstone Road Health Clinic, Hartingdon Road, Southall, Middlesex UB2 5BQ* [2]*Department of Biochemistry, University of Surrey, Guildford, Surrey GU2 5XH* and [3]*Department of Pathology, New East Surrey Hospital, Three Arch Road, Redhill, Surrey RH1 5RH*

Asians represent a major group among ethnic minorities in the British community, and number approximately 800 000. In Southall, Middlesex, where 47% of the population are Asians, longitudinal data were collected over a 5 year period by one observer (BQ) on birth weights and feeding practices in a randomly selected sample of infants from four groups, Punjabi Indians, Pakistanis, East African Asians and English.

Female birth weights were similar to those for males in the three Asian groups. Birth weights for the male English babies tended to be higher than those of females but in both sexes were lower than national values (50th percentile 3500 g and 3400 g for males and females respectively; Tanner *et al.* 1966). Pakistani infants of both sexes were heavier at birth than Punjabi and East African Asians (Table).

Birth weights (g) for infants by sex and ethnic group

	English			Punjabi			Pakistani			East African Asians		
Sex	*n*	Mean	SEM	*n*	Mean	SEM	*n*	Mean	SEM	*n*	Mean	SEM
Male	42	3445*†	70	75	3210	42	15	3300	82	8	3230	163
Female	44	3245	52	70	3175	43	16	3210	106	10	3075	96

Values exclude twins and infants with birth weights under 2500 g.
*Significantly greater compared with Punjabi male ($P<0.005$).
†Significantly greater compared with English female ($P<0.01$).

The incidence of breast feeding for the whole population on which longitudinal data were available (124 infants) was 40·1%, and mean duration (9·5 weeks) was shorter than the national average (Wharton, 1982). By 26 weeks less than 2% of the population were breast feeding, the reason most commonly stated (32%) by mothers for cessation of breast feeding was insufficient milk. Overweight mothers breast fed for a longer period (13·5 weeks) than either normal weight (7·7 weeks) or underweight mothers (10·7 weeks). By 12 weeks, 50% of all infants had received solid food, the most popular choice (80·0%) being commercial baby foods.

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The inhibition of peroxidation in liver microsomes by glutathione but not by metallothionein. By J. R. ARTHUR, I. BREMNER, P. C. MORRICE and C. F. MILLS, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

Degradation of lipids, mainly polyunsaturated fatty acids, by free-radical-mediated mechanisms may occur in several disease states. This lipid peroxidation can be initiated by free radicals generated during metabolism involving oxygen (Halliwell & Gutteridge, 1984). However, many systems exist in the cell to scavenge O_2-derived radicals and their precursors. Metallothionein (Mt) reacts efficiently with $OH^{\cdot}$ radicals in vitro (Thornally & Vasak, 1985) and, as its biosynthesis is induced under conditions of stress, it has been suggested that it functions as an antioxidant in vivo.

We have studied the inhibitory effects of rat liver zinc-metallothionein (ZnMt) and of pig liver copper–Zn metallothionein (CuMt) on lipid peroxidation induced by Fe/ADP or xanthine/xanthine oxidase (*EC* 1.1.3.22) (X/Xo) in incubations of liver microsomes. Reduced glutathione (GSH) and the spin trap phenylbutylnitrone (PBN), both of which will react with free radicals, were tested for inhibitory action in the same system.

Formation of thiobarbituric acid (TBA) reactive substance (nmol malondialdehyde/mg protein) after incubations of microsomes for 10 *min (Fe/ADP) or* 15 *min (X/Xo)*

(Mean values with their standard errors for four determinations)

	X/Xo		Fe/ADP	
	Mean	SEM	Mean	SEM
No additions	4·07	0·14	2·38	0·13
ZnMt, 0·1 mM	4·74	0·69	2·29	0·08
CuMt, 0·1 mM	8·76***	0·67	2·77	0·40
GSH, 0·1 mM	1·61***	0·11	0·77***	0·19
GSH, 1·0 mM	0·83***	0·07	0·57***	0·05
PBN, 0·1 mM	1·19***	0·14	1·05***	0·13

Significantly different from values for no additions: ***$P<0.001$.

Addition of ZnMt or CuMt did not cause any inhibition of TBA-reactive substance formation when lipid peroxidation was initiated by either Fe/ADP or X/Xo, whereas both GSH and PBN effectively inhibited lipid peroxidation (see Table). GSH, PBN and ZnMt did not affect the rate of the xanthine oxidase reaction monitored by the formation of uric acid.

Concentrations of GSH in liver cells can range up to 10 mM whereas those of Mt will rarely exceed the 0·1 mM used in these incubations. It therefore seems unlikely that Mt will act as an important direct radical scavenger in the liver cell. Sequestration of free Cu by Mt may, however, be important in the prevention of radical generation.

Halliwell, B. & Gutteridge, J. M. C. (1984). *Biochemical Journal* **219**, 1–14.
Thornally, P. J. & Vasak, M. (1985). *Biochimica et Biophysica Acta* **827**, 36–44.

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Nutritional status of vitamins E, C and B_2 in alcoholics in relation to the plasma phospholipid-2-esterified 9,11-linoleic acid: 9,12-linoleic acid value. By D. I. THURNHAM[1], B. J. CRUMP[2], J. A. DAVIES[1], R. D. SITUNAYAKE[2] and M. DAVIS[2], [1]*Wolfson Research Laboratory, Queen Elizabeth Hospital, Birmingham B15 2TH* and [2]*Department of Medicine Dudley Road Hospital, Birmingham B18 7QH*

Evidence from experimental and human studies suggests that oxygen free-radicals may be involved in the aetiology of alcoholic liver disease. Recent work has shown that the % molar ratio (R; μM 9,11-linoleic acid (LA):9,12-LA × 100) of the 9,11-isomer of LA to 18:2Δ9,12-LA is increased in the plasma of alcoholics who have recently consumed alcohol (Fink *et al.* 1985). This isomer may be formed by hydrogen abstraction of LA with the subsequent production of a diene conjugated free-radical. The presence of antioxidants may block further peroxidation, producing the stable isomer found in the blood. We have measured the nutritional status of vitamins E, C and B_2 in sixty-eight alcoholics (>80 g alcohol/d for >5 years), twenty-seven ex-alcoholics (no alcohol for >3 months) and sixty-nine people randomly selected in a local factory.

	Alcoholics		Ex-alcoholics		Control	
	Mean	SEM	Mean	SEM	Mean	SEM
Plasma vitamin E (mg/g lipid)	0·93[a]	0·04	1·15[b]	0·05	1·53[b]	0·05
<0·8	(19/60, 30%)		(3/25, 12%)		(0/40, 0%)	
Plasma vitamin C (μM)	34·8	2·24	38·3	3·36	32·6	2·82
<11·4	(1/65, 2%)		(0/27, 0%)		(6/65, 9%)	
Vitamin B_2 (EGRAC)	1·33	0·04	1·29	0·03	1·27	0·02
>1·30	(31/63, 49%)		(11/27, 41%)		(23/65, 35%)	
% molar R	3·02[A]	2·09	2·02[B]	0·85	2·07[B]	0·66
>3·39	(18/65, 28%)		(2/27, 7%)		(1/50, 2%)	

EGRAC, erythrocyte glutathione reductase activation coefficient.

[a,b]Within rows, values with different superscript letters were significantly different (Duncan's multiple range test): $P<0·05$.

[A,B]Within rows, values with different superscript letters were significantly different (Wilcoxon and Rank test): $P<0·05$.

The Table shows that vitamin E and B_2 status was poor in the alcoholics and vitamin B_2 status was poor in both other groups. There was no correlation between R and the vitamins studied in the ex-alcoholics or controls but in the alcoholics a tendancy to an inverse relation with vitamin E status was found (r −0·18, n 55, not significant). The lack of significance may be concealed as the amount of and time since alcohol was consumed were variable and both are major determinants of R. Also, in the alcoholics, vitamin E status was correlated with vitamin C (r 0·37, n 55, $P<0·01$). It is possible that the poor vitamin E status may result from its increased utilization as a free-radical scavenger and in this function it may be supported by vitamin C.

Fink, R., Clemens, M. R., Marjot, D. H., Patsalos, P., Cawood, P., Norden, A. G., Iversen, S. A. & Dormandy, T. L. (1985). *Lancet* **ii**, 291–294.

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Selenium and neutrophil function in mice. By J. R. ARTHUR, R. BOYNE, P. C. MORRICE and F. NICOL, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB* (Introduced by I. BREMNER)

Severe selenium deficiency has been demonstrated to impair the ability of neutrophils from rats, cattle and mice to kill the yeast *Candida albicans* (Boyne & Arthur, 1985). This defect has been attributed to loss of neutrophil glutathione peroxidase (GSHpx; *EC* 1.11.1.9) (Arthur & Boyne, 1985).

We have now examined in more detail the relations between neutrophil GSHpx and candidacidal activities. Consumption by mice from weaning, of low-Se diets (<0·01 mg Se/kg diet) caused a steady decrease in vitro in candidacidal activity of peritoneal-derived neutrophils. Sixty-three days after weaning, mean candidacidal activity was 9·85 (SEM 0·59)% (nine animals, expressed as % of cells containing dead *C. albicans*). In Se-supplemented mice (0·1 mg Se/kg diet) mean neutrophil candidacidal activity was 55·0 (SEM 2·2)% (four animals). Intra-peritoneal injection of Na_2SeO_3 into Se-deficient mice, 58 d after weaning, restored neutrophil candidacidal activity (measured in mice killed 5 d post-injection) in two distinct stages (see Table). Partial restoration of activity to 13·60 (SEM 1·2)% occurred with doses of <20 μg Se/kg body-weight, whilst further increases in activity did not occur until higher Se doses were used (100–1000 μg Se/kg body-weight). The 20 μg dose of Se caused small but significant rises in neutrophil but not liver GSHpx activity, whereas the higher doses of Se increased GSHpx in both tissues.

Neutrophil candidacidal activity and liver and neutrophil GSHpx activities in mice dosed with differing amounts of Se

Se (μg/kg body-wt) . . .	0		20		100		1000	
No. of animals . . .	9		5		5		5	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Candidacidal activity (%)	9·85	0·59	13·60*	1·2	13·25*	1·25	42·00*	0·94
GSHpx activity (units/mg protein):								
Neutrophil	0·041	0·005	0·082*	0·007	0·134*	0·030	0·383*	0·023
Liver	0·027	0·004	0·028	0·004	0·159*	0·035	0·868*	0·060

Significantly different from 0 Se: *P<0·05.

The neutrophil may therefore have at least two pools of Se-containing GSHpx, both of which are necessary for maintenance of normal microbicidal activity.

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Lipid peroxidation during muscular exercise in man: inferences from the pulmonary excretion of n-pentane, isopentane and nitrogen. By M. T. SNIDER, P.-O. BALKE, K. E. OERTER, N. A. FRANCALANCIA, A. P. BULL, K. A. PASKO and M. E. ROBBINS, *Department of Anesthesia and Physiology, Pennsylvania State University, Hershey, Pennsylvania* 17033, *USA* (Introduced by R. F. GRIMBLE)

Free radicals generated during exercise may peroxidize linoleic and arachidonic acids in cellular membranes. These lipid peroxides can decompose to n-pentane (n-P) which appears in the exhaled breath (Dillard *et al.* 1978). Since n-P is a common air pollutant, endogenous production by lipid peroxidation may be masked by the mobilization of exogenous n-pentane stored in body tissues. To separate these factors we measured the kinetics of n-P and isopentane (i-P) excretion by the lung in volunteers after breathing hydrocarbon-free oxygen–helium for 2 h and compared these with computer simulations using multicompartmental models.

Thirteen volunteers breathed hydrocarbon-free O_2–He (20:80 v/v) for 2 h before leg cycling for 20 min at 50% $\dot{V}_{O_2max}$. An on-line, gas-tight collection system carried inspired and expired samples to the gas chromatograph which measured n-P and i-P in the pl/l range. Mean n-P excretions after 20 min of exercise (2320 fmol/kg per min) were significantly greater than pre- and post-exercise rest values (1020 fmol/kg per min, $P<0{\cdot}01$). This could be taken as evidence of increased n-P excretion from lipid peroxidation during exercise, but the excretion of i-P, an air pollutant having no known biological origin, was similarly increased ($P<0.01$) as was nitrogen ($P<0{\cdot}01$).

We measured the solubilities of n-P and i-P in beef heart, skeletal muscle, liver, kidney, brain and olive oil. Computer simulations of the uptake and distribution of n-P and i-P imply that hydrocarbon-free gas must be breathed for 3 d to wash out exogenous pollutant pentanes. If exercise is simulated during this 3 d wash out period, n-P and i-P excretion increases because exercise induces an increase in blood flow to muscle and fat which washes out exogenously stored pentane. We conclude that lipid peroxidation cannot be detected during moderate exercise using n-P excretion as a marker.

The present work was partially supported by HL-7477.

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Vitamin C restriction and physical performance. By E. J. VAN DER BEEK, W. VAN DOKKUM, J. SCHRIJVER and M. WEDEL, *TNO CIVO-Toxicology and Nutrition Institute, PO Box* 360, 3700 *AJ Zeist, The Netherlands*

The functional consequences of a combined marginal biochemical status of vitamins B_1, B_2, B_6 and C has recently been described (van der Beek *et al.* 1984). In order to elucidate the effect of individual vitamin restriction on physical performance, a 13-week double-blind study with a vitamin C-deficient diet was carried out.

An experimental group of six healthy male volunteers (mean age 24·0 years) was given a basal diet composed of normal foodstuffs for an experimental period of 7 weeks. This diet provided 10 mg vitamin C/d (20% of the Dutch recommended daily allowance (RDA)). In the first 3 weeks of the experimental period the subjects consumed this diet only, in the last 4 weeks an additional dose of 15 mg vitamin C/d was provided (total intake 25 mg vitamin C/d or 50% of the Dutch RDA). The 7 weeks were preceded and followed by a 3-week control and recovery period in which the basal diet was consumed and vitamin supplementation amounted to twice the RDA of all vitamins. A control group of six males (mean age 23·8 years), matched with the experimental group according to aerobic capacity, received the same diet supplemented with twice the RDA of all vitamins throughout the study. For the duration of the study all subjects were given trace element–mineral supplementation at the level of one times the RDA. Physical performance was determined on a bicycle ergometer and quantified by means of the aerobic power ($\dot{V}_{O_2max}$) and the onset of blood lactate accumulation (OBLA) (4 mM).

Table 1. *Aerobic power and OBLA*

	$\dot{V}_{O_2max}$ (l/min)				OBLA (Watt)			
Group . . .	Experimental		Control		Experimental		Control	
Week	Mean	SD	Mean	SD	Mean	SD	Mean	SD
−3	3·84	0·36	3·71	0·32	246	21	249	12
0	3·78	0·36	3·89	0·28	240	29	240	16
3	3·79	0·45	3·83	0·28	246	23	245	16
7	3·75	0·34	3·78	0·40	245	20	243	11
10	3·81	0·43	3·76	0·23	247	18	234	16

The deficient diet appeared to have caused the vitamin C status to reach borderline to deficient levels (whole blood vitamin C at week 7 (mean and SD): experimental 19·9 (5·2) *v.* control 57·3 (10·5) μmol/l). In the 3-week recovery period the vitamin C status reached values comparable to those measured at the start of the experiment. In the experimental and control groups neither $\dot{V}_{O_2max}$ nor OBLA changed significantly during the study. It is concluded that vitamin C restriction induced a decrease in vitamin C status but did not influence physical performance.

van der Beek, E. J., van Dokkum, W., Schrijver, J., Wesstra, J. A., van de Weerd, H. & Hermus, R. J. J. (1984). *International Journal of Sports Medicine* **5**, Suppl., 28–31.

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Examination of nutritional factors in rheumatoid arthritis: relation with markers of lipid peroxidation. By R. D. SITUNAYAKE[1], D. I. THURNHAM[2], J. A. DAVIES[2], B. J. CRUMP[1], M. DAVIS[1] and B. MCCONKEY[1], [1]*Department of Medicine, Dudley Road Hospital, Birmingham B18 7QH* and [2]*Wolfson Research Laboratory, Queen Elizabeth Hospital, Birmingham B15 2TH.*

Increasing evidence points to a role for lipid peroxidation in rheumatoid arthritis (RA). Diene conjugated 18:2Δ9,11-linoleic acid (9,11-LA) accounts for 90% of the absorbance of 234 nm of human plasma lipid extract and may represent a product formed from 18:2Δ9,12-linoleic acid (9,12-LA) during peroxidation (Cawood *et al.* 1983). Antioxidants may affect this process. We measured vitamins E, C and B_2 in sixty-one patients with active RA, thirty-eight with inactive RA and sixty-nine randomly selected controls. Total plasma diene conjugation (DC, expressed in arbitrary units per g triglyceride + cholesterol) and the % molar ratio, phospholipid-2-esterified 9,11-LA:9,12-LA (R; μM 9,11-LA:9,12-LA × 100) were measured as markers of lipid peroxidation.

	Active RA		Inactive RA		Control	
	Mean	SEM	Mean	SEM	Mean	SEM
Plasma vitamin E (mg/g)*	2·99	0·14	2·85	0·19	2·52	0·10
<1·294	(1/59)		(3/37)		<(0/40)	
Plasma vitamin C (μM)	35·9	3·06	44·6	4·79	32·6	2·82
<11·4	(5/57, 9%)		(0/29, 0%)		<(6/65, 9%)	
Vitamin B_2 (EGRAC)	1·26[b]	0·02	1·16[a]	0·02	1·27[b]	0·02
>1·3	(19/57, 33%)		(3/34, 9%)		<(23/65, 35%)	
DC (mmol/g)*	106[b]	6·5	151[a]	13·2	96†[b]	6·2
>160	(2/50, 4%)		(11/30, 37%)		<(1/23, 4%)	
% molar R	2·28[B]	0·12	2·76[A]	0·18	2·07[B]	0·66
>3·39	(6/61, 10%)		(9/38, 23%)		<(1/59, 2%)	

[a,b]Within rows, values with different superscript letters were significantly different (Duncan's multiple range test): $P<0·01$.

[A,B]Within rows, values with different superscript letters were significantly different (log transformed data-analysis of variance): $P<0·01$.

*Expressed per g triglyceride + cholesterol.

†Based on twenty-three factory controls.

Few patients with RA were deficient in vitamins E or C. Vitamin B_2 (erythrocyte glutathione reductase activation coefficient; EGRAC) status was poorer in the patients with active than with inactive disease but no poorer than that in the controls. The ratio, vitamin E:lipid, correlated with plasma vitamin C (r 0·35, $P<0·02$, n 72), indicating that vitamin C may regenerate α-tocopherol.

DC was inversely related to serum C-reactive protein (CRP) (r −0·25, $P<0·05$, n 80), a marker of the hepatic acute phase response, although R was not. DC and R were weakly correlated (r 0·33, $P<0·02$, n 80) and were inversely related to the vitamin E:lipid value (DC *v.* vitamin E: r −0·29, $P<0·02$, n 80; R *v.* vitamin E: r −0·33, $P<0·02$, n 80).

These results suggest a role for vitamins E and C as antioxidants in RA.

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Factors affecting the integrity of intestinal mucosa in Gambian children.

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The interrelation between diarrhoea and malnutrition in tropical Africa remains ill understood, and little is known of small-bowel function. We have studied seventy-five Gambian infants (from birth to 24 months) living in an area with adequate food supplies. The infants were seen monthly over a period of 8 months and whenever sick. Detailed profiles of growth, development and morbidity were recorded. Small-intestinal integrity was tested at each planned visit by measuring gut permeability. The following were given orally: 5 g lactulose (7·5 ml Duphalac), 0·5 g lactose and 1 g mannitol. Urine was collected for 5 h and analysed for creatinine, lactose, mannitol and lactulose. Intestinal integrity was best expressed as the ratio, lactulose:mannitol (L:M).

Urinary L:M values in infants by diagnosis, and nutritional state as a percentage of the National Centre for Health Statistics (1977) *standard*

Clinical group	L:M Mean	L:M 2SD	No. of tests	No. of patients
Percentage standard weight-for-age				
Well >80%	0·42	0·2–1·4	255	60
Well <80%	0·52*	0·2–2·2	45	15
Malnourished <60%	1·3**	0·2–13	42	27
All diarrhoea	1·3**	0·2–10·4	93	47
Diarrhoea <14 d	1·0**	0·2–10·4	76	32
Diarrhoea >14 d	2·85**	0·2–10·4	17	15

*$P<0·05$, **$P<0·001$.

Tests (296) were performed on apparently well children. Ratios were distributed in a log normal fashion (mean 0·42). For ninety-three infants with diarrhoea, L:M values were increased (mean 1·3). Histology of jejunal mucosa of five sick infants with diarrhoea/malnutrition showed severe enteropathy. Abnormal L:M values (>1·14) occurred occasionally in well-nourished children with superficial skin sepsis (5%) and with respiratory tract infections (12%). Underweight children without apparent disease had near normal L:M values but, with severe malnutrition (<60% standard weight-for-age), values increased markedly (n 42, mean 1·3). Multi-regression analysis of weight gain revealed a correlation between low weight velocity and a raised L:M value ($P<0·01$).

Gambian infants frequently suffer small intestinal pathology associated with increased intestinal permeability which appears to be conditioned by a combination of malnutrition and infection.

National Centre for Health Statistics (1977). *Growth Curves for Children from Birth to* 18 *years*. DHEW publication no. PHF 78–1650.

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Amino acid requirements in acute disease. By A. W. JOHNSON, ADUKE ANIMASHAUN, JANET BERRINGTON, R. V. HEATLEY and M. S. LOSOWSKY, *Department of Medicine, St James's University Hospital, Leeds*

Longenecker & Hause (1959) first suggested that by reference to changes in plasma amino acid concentration following a food challenge, information regarding the ideal amino acid composition of the diet could be obtained. We have modified this approach to investigate the amino acid requirements of patients with Crohn's disease and alcoholic liver disease.

Following an overnight fast, the patients were asked to consume a peptide-containing meal (Vital, Ross Laboratories) and the changes in plasma amino acid concentration followed for 2 h. A decrease in concentration of any individual amino acid following ingestion of a test meal containing that amino acid was taken as indicating that there was an increased demand for the amino acid in total body terms.

Certain amino acids showed consistent changes for the disease status studied. Patients with active Crohn's disease (n 6) showed negative changes for valine and methionine and a large positive change for glutamine (not present in the test meal) when compared with Crohn's patients in remission (n 12).

The response in patients with alcoholic liver disease varied with the grade of encephalopathy. The most clearcut changes were seen in those patients recently admitted (encephalopathy grade 2–3, n 8), in whom serine and glycine showed a decrease in concentration following the challenge, whilst there was a delay in the clearance of the branched-chain amino acids and tryptophan. The response was similar for the branched-chain amino acids in recovered patients (encephalopathy grade 0–1, n 7); however, there were no negative changes observed for either glycine or serine.

These results suggest that in nutritional terms there may be an increased demand for certain amino acids dependent upon the clinical status of the patient, or the normal mechanisms for the clearance of these amino acids is altered.

Longenecker, J. B. & Hause, N. L. (1959). *Archives of Biochemistry and Biophysics* **84**, 46–59.

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Gastric juice 'secretion' of ascorbic acid. By B. J. RATHBONE, A. W. JOHNSON, CHRISTINE JONES, J. KELLEHER and R. V. HEATLEY, *Department of Medicine, St James's University Hospital, Leeds*

Nitrosamines have been implicated in the pathogenesis of both gastric and oesophageal carcinoma. Dietary ascorbic acid (AA) acting as an anti-oxidant is thought to decrease the conversion of nitrites to *N*-nitroso compounds thus protecting the gastric mucosa (Mirvish, 1981). The occurrence in the stomach of locally 'secreted' AA has not been generally recognized.

Fasting plasma AA, white blood cells (wbc) AA and gastric juice AA concentrations were studied in thirty-nine patients attending for upper gastrointestinal endoscopy. Following an overnight fast all patients had gastric juice aspirated and gastric antral and body biopsies taken for histology. Patients were divided into two groups dependent on the presence or absence of histologically proven gastritis, thus when gastric antral and body histology was taken into account the following results were obtained:

	Gastric juice pH		Plasma AA (mg/l)		Gastric juice AA (mg/l)		wbc AA (μg/10⁸ cells)	
n	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Normal 15	2·14	0·27	9·71	1·10	22·41	4·01	23·96	1·43
Gastritis 24	3·52*	0·50	6·75*	0·99	12·66*	2·22	20·82*	2·70

*$P<0.05$

The source of gastric juice AA was also investigated using a short pentagastrin test in seven volunteers. AA concentrations peaked at 10 min post-pentagastrin while acid output peaked at 20–30 min.

These results suggest that there is a considerable 'secretion' of non-dietary AA into the stomach which, taking an average daily gastric secretion of 1500 ml, amounts to approximately 30 mg AA/d. The finding of decreased gastric concentrations of AA in gastritic patients may help explain the increased risk of gastric malignancy found in this condition. The mechanism of gastric AA 'secretion' requires further study, but appears to be uncoupled from that of acid secretion.

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Changes in the endogenous anti-oxidant levels of plasma and erythrocytes during *Plasmodium vinckei* malaria. By R. STOCKER, N. H. HUNT, M. J. WEIDEMANN and I. A. CLARK (Introduced by R. F. GRIMBLE), *Departments of Biochemistry and Zoology, Faculty of Science, and Experimental Pathology, John Curtin School of Medical Research, Australian National University, Canberra, A.C.T. 2601, Australia*

Reactive oxygen species probably play a role in the host immune response to malaria, and may contribute to the tissue damage seen in this disease. The status of endogenous anti-oxidants in erythrocytes (RBC) and plasma has been studied in normal *Plasmodium vinckei*-infected mice.

In heavily infected mice, the RBC levels of vitamins E and C were double that of control values. No α-tocopherol-quinone, an oxidation product of vitamin E, was detectable. The ratio, oxidized:reduced vitamin C decreased from 0·96 in control RBC to 0·57 in RBC from infected animals. The changes in vitamin C status are consistent with a role in protecting vitamin E from irreversible oxidation.

In plasma from infected animals, vitamin C levels were 50% higher, and vitamin E levels 50% lower, than those in controls. There was a small increase, from 0·71 to 0·76 in the ratio, oxidized:reduced forms of vitamin C in plasma from infected mice. Other plasma anti-oxidants changed during *P. vinckei* infection: uric acid levels were double control values and plasma protein concentrations decreased slightly, but significantly. The latter change may have been obscured by the increased haemoglobin level in the plasma. Plasma proteins, particularly albumin, are the major contributors to the anti-oxidant of normal plasma.

These changes in endogenous anti-oxidants could protect the parasitized RBC and its contents against possible oxidative assault from phagocytes but would not provide additional protection to host endothelial cells, which consequently might be damaged.

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