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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Twenty-third Meeting of the Nutrition Society (One Hundred and Sixty-ninth of the Scottish Group) was held in the Kelvin Conference Centre, Wolfson Hall, West of Scotland Science Park, Glasgow on Monday and Tuesday, 17/18 *March* 1986, *when the following papers were read:*

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Large neutral amino acids in the diet and neurotransmitter concentrations in the chick brain. By LYDIA HARRISON and J. P. F. D'MELLO, *Department of Agricultural Biochemistry, Edinburgh School of Agriculture, West Mains Road, Edinburgh EH9 3JG*

Tyrosine and phenylalanine are the essential precursors of the neurotransmitters norepinephrine (NE) and dopamine (DA), as tryptophan is of 5-hydroxytryptamine (5HT). In the rat, uptake of these amino acid precursors by the brain is influenced by dietary concentrations of the other large neutral amino acids leucine, isoleucine and valine (Fernstrom & Wurtman, 1972; Gibson & Wurtman, 1978).

The present study was designed to investigate whether the concentrations of NE, DA, 5HT and selected metabolites of these neurotransmitters in the chick brain are affected by excess dietary concentrations of leucine, isoleucine and valine. The results are presented in the Table.

Concentrations of neurotransmitters and their metabolites (ng/g wet weight brain tissue)

Amino acid supplement (g/kg)†	NE	DA	DOPAC	HVA	5HT	5HIAA
Nil	296	246	68	90	572	169
Leucine (60) + isoleucine (34·7) + valine (40)	186**	150**	101	57*	357**	73**
Leucine + isoleucine + valine mixture + phenylalanine (8) + tryptophan (4)	269	221	88	71	470	123*
SEM	13·08	15·60	41·01	6·36	40·08	13·04

Mean values significantly different from control levels: *$P<0.05$; **$P<0.01$.
†Feeding period 7–19 d of age; chicks killed at 19 d of age.

Terminal brain concentrations of NE, DA and their metabolite homovanillic acid (HVA) were significantly ($P<0.05$ for HVA, $P<0.01$ for NE and DA) reduced by feeding excess leucine, isoleucine and valine although dihydroxyphenylacetic acid (DOPAC) concentrations remained unchanged. 5HT and its metabolite 5-hydroxyindoleacetic acid (5HIAA) also fell significantly ($P<0.01$) on feeding excess branched-chain amino acids. Supplementation of the diet containing excess leucine, isoleucine and valine with phenylalanine and tryptophan restored neurotransmitter and metabolite concentrations to those of control values except for 5HIAA, whose concentration nevertheless was increased.

The reduction in neurotransmitter levels caused by the leucine + isoleucine + valine supplements is consistent with an increased competition for brain uptake of phenylalanine and tryptophan and thus decreased synthesis of NE, DA and 5HT. Increasing dietary phenylalanine and tryptophan increases their transport into the brain and hence raises the levels of the corresponding neurotransmitters. The results thus support the existence of a functional blood–brain barrier in the young chick, with competition occurring among the large neutral amino acids for uptake, as in the rat.

L.H. acknowledges receipt of an AFRC research assistantship.

Fernstrom, J. D. & Wurtman, R. J. (1972). *Science* **178**, 414–416.
Gibson, C. J. & Wurtman, R. J. (1978). *Life Sciences* **22**, 1399–1406.

Effect of litter size on parametrial white fat cell size and lipolysis in lactating rats. By M. A. RADCLIFFE and S. C. HAY, *Department of Physiology, University of Aberdeen, Marischal College, Aberdeen AB9 1AS* and D. M. CAMPBELL, *Department of Obstetrics & Gynaecology, University Medical Buildings, Foresterhill, Aberdeen AB9 2ZD*

Part of the decrease in white fat cell size that occurs in lactating rats has been attributed to decreased triacylglycerol (TAG) deposition, but the role of TAG mobilization has not been defined clearly (Vernon & Flint, 1984). Steingrimsdottir *et al.* (1980*a*,*b*) have shown that maternal fat depot weight, mean fat cell size and fat tissue lipoprotein lipase activity decrease in proportion to increased lactational demand as determined by litter size. We have now investigated the effect of litter size on lipolysis.

Hooded Lister dams maintained on stock diet (Labsure CRM) were mated at 9–10 weeks of age. Dam body-weight, food intake and litter weight were measured in two groups nursing litters of four or twelve pups until they were killed at 15–17 d post-partum. Basal lipolysis and isoprenaline-stimulated lipolysis (expressed as E_{max} and EC_{50}, see Table) were then estimated by glycerol release from fat cells incubated at 37° in HEPES-buffered Krebs saline containing bovine serum albumin (40 g/l), pH 7·4. Cell diameters were measured by microscopy (*n* 120).

Litter size (no. of pups) . . .	4		12		Statistical significance (Student's *t* test): *P*
No. of observations . . .	12		12		
	Mean	SD	Mean	SD	
Dam live body-wt (g)	266·7	9·52	261·3	10·20	NS
Pup mean live body-wt (g)	27·7	5·26	18·8	1·65	<0·001
Food intake during lactation (g/d)	29·0	2·77	39·8	4·47	<0·001
Fat depot wet wt (g)	2·55	0·560	2·18	0·248	=0·05
Fat cell mean diameter (μm)	105·4	3·78	96·3	4·36	<0·001
Lipolysis:					
Basal*	65·3	45·27	101·1	61·22	NS
Isoprenaline-stimulated					
E_{max}*	360·9	99·95	330·8	75·95	NS
EC_{50} (nM)	6·75	4·365	3·43	2·024	<0·05

E_{max}, maximal increment above basal; EC_{50}, concentration required for half maximal stimulation; NS, not significant.

*nmol glycerol liberated/10^4 cells in 90 min.

Mean parametrial depot wet weight and cell diameter were lower and mean food intake was greater in the dams nursing large litters compared with those nursing small litters. Mean values for basal lipolysis and E_{max} were not different in the two groups, but EC_{50} was lower in fat cells from the large-litter group. The results confirm and extend those of Steingrimsdottir *et al.* (1980*b*). We conclude that TAG mobilization may be more readily stimulated by β-receptor activation in white fat cells of dams nursing large litters than of those nursing small litters.

Steingrimsdottir, L., Brasel, J. A. & Greenwood, M. R. C. (1980*a*). *Metabolism* **29**, 837–841.

Steingrimsdottir, L., Greenwood, M. R. C. & Brasel, J. A. (1980*b*). *Journal of Nutrition* **110**, 600–609.

Vernon, R. G. & Flint, D. J. (1984). In *Physiological Strategies in Lactation*, pp. 119–145 [M. Peaker, R. G. Vernon and C. H. Knight, editors]. London: Academic Press.

A comparison of continuous and intermittent postoperative nasogastric nutrition after major head and neck surgery. By A. WEIR[1], R. A. RICHARDSON[2], K. CARR[2], A. SHENKIN[3], O. J. GARDEN[4] and G. G. BROWNING[1], *University Departments of* [1]*Otolaryngology,* [2]*Dietetics,* [3]*Biochemistry and* [4]*Surgery, Royal Infirmary, Glasgow G31 2ER*

It has been suggested that continuous nasogastric feeding may be metabolically less efficient, giving a poorer nitrogen balance than intermittent feeding in the postoperative period (Campbell *et al.* 1983). The aim of the present study was to compare the effects of continuous and intermittent feeding on N balance and other biochemical indices.

Fourteen patients were randomly allocated to continuous or intermittent feeding. Each group consisted of six patients who had undergone laryngectomy and one, a major oral procedure. Both groups were well matched with respect to age, sex distribution and weight. Patients were fed comparable amounts of Clinifeed-Iso (Roussel Laboratories Ltd) via a nasogastric tube to a maximum of 7530 kJ (1800 kcal) and 8·1 g N/d diluted to 3 litres with water for the 5-d study period. Daily urine collections were made and analysed for urinary total N. Blood was taken preoperatively and at the end of the study period for estimation of serum albumin, transferrin, alanine aminotransferase (*EC* 2.6.1.2), γ-glutamyltransferase (*EC* 2.3.2.2) and phosphate. Unpaired *t* and Mann-Whitney tests were used to determine the significance of differences.

The mean daily energy intake (continuous: 5824 (SD 2142) kJ *v.* intermittent: 6640 (SD 2200) kJ (continuous: 1392 (SD 512) kcal *v.* intermittent: 1587 (SD 525) kcal)) were not significantly different. There was no significant difference between the two groups with respect to estimated N balance (continuous: −3·5 (SD 3·1) g *v.* intermittent: −2·2 (SD 4·1) g). Serum biochemical changes are shown in the Table.

	Continuous				Intermittent			
Feeding period . . .	Start		End		Start		End	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Albumin (g/l)	39·3	4·8	36·0	5·1	36·3	4·5	31·3	4·9
Transferrin (g/l)	2·6	0·5	2·0	0·4	2·3	0·4	1·7	0·4
Alanine aminotransferase (unit/l)	21·2	5·3	23·5	6·9	14·2	7·1	15·3	5·5
γ-Glutamyltransferase (unit/l)	40·2	40·2	26·0	12·9	30·8	16·5	38·1	25·2
Phosphate (mmol/l)	1·1	0·2	1·3*	0·2	1·2	0·2	1·2	0·4

Mean value was significantly different from start value: *$P<0.05$.

No significant difference was seen in patient tolerance. This study has failed to demonstrate improvement in N balance when enteral nutrition is administered intermittently.

Campbell, I. T., Morton, R. P., Cole, J. A., Raine, C. H., Shapiro, L. M. & Stell, P. M. (1983). *American Journal of Clinical Nutrition* **38**, 870–878.

Heat production and physical activity in laying hens on an intermittent-lighting regime. By M. G. MacLeod and T. R. Jewitt, *AFRC Poultry Research Centre, Roslin, Midlothian EH25 9PS*

Observations on diurnal variation in the heat production rate (H) and physical activity (A) of domestic fowl (MacLeod *et al.* 1982) suggested that reducing the duration of lighting provision might reduce daily energy expenditure. This hypothesis was tested in the particular case of laying hens kept on an intermittent daily lighting pattern (15 × (13 min light (L)–47 min dark (D)) + 9 h D) and compared with a standard pattern of 14 h L + 10 h D. The intermittent pattern gave 3 h 15 min of light/d but was probably interpreted by the birds to give a 'subjective day' of 14 h 13 min, the time between the first and last periods of light.

Sixteen 50-week-old Rhode-Island-Red hens, caged in pairs, were randomly divided between treatments; they were observed in respiration calorimeters fitted with Doppler-radar activity meters, for 24 h periods at 4-d intervals. H and A were measured continuously in 13-min sample periods. Ambient temperature was 20°. A standard layer diet and water were available *ad lib.* The results (see Table) are the means for four bird-pairs (n 4).

Lighting pattern . . .	Standard		Intermittent	
	Mean	SEM	Mean	SEM
Total A (units/d)	33057	2200	24090**	1635
Total 24 h H (kJ/kg $W^{0.75}$)	477	8·8	493NS	7·9
Night H (J/kg $W^{0.75}$ per min)	263	3·8	277*	6·1
Subjective-day dark H (J/kg $W^{0.75}$ per min)	—	—	375	6·6
Subjective-day zero-A H (J/kg $W^{0.75}$ per min)	349	13·5	323NS	10·3
H cost of 1 A unit during subjective day (J/kg $W^{0.75}$)	0·83	0·120	2·42***	0·292

W, body-weight; NS, not significant.
*P<0·05, **P<0·01, ***P<0·001.

In terms of H, the intermittent group responded differently (P<0·001) to night-time and day-time darkness. Total H did not differ between lighting treatments, despite significantly lower total A in the intermittent group. Zero-A H during the subjective day (calculated by regressing H against A) was also the same in both regimes. H therefore remained at the same level in both groups because of a large increase in the H cost per unit A in the intermittent group. The H cost per unit A in the standard group was similar to that recorded with similar birds in previous experiments. The large increase in unit A cost in the intermittent group may be due to a large postural-change component but needs further experimental elucidation.

MacLeod, M. G., Jewitt, T. R., White, J., Verbrugge, M. & Mitchell, M. A. (1982). In *Proceedings of the 9th EAAP Symposium on the Energy Metabolism of Farm Animals* [A. Ekern and F. Sundstol, editors], pp. 297–300. Aas: Agricultural University of Norway.

Activation of pyruvate dehydrogenase in rat white adipose tissue by both insulin and noradrenaline is muted during lactation. By E. KILGOUR and R. G. VERNON, *Hannah Research Institute, Ayr KA6 5HL*

Increased sympathetic nervous activity induces lipolysis and glycogenolysis in white adipose tissue; we now show that it also increases pyruvate dehydrogenase (PDH) activity and hence glucose oxidation.

PDH controls the irreversible conversion of carbohydrate into acetyl CoA for lipogenesis and for oxidation and so has a key role in the control of glucose metabolism. Insulin increases the proportion of PDH in the active state in rat white adipose tissue (Saggerson, 1985). The role of catecholamines in the control of white adipose tissue PDH activity is less clear: studies in vitro show that concentrations of adrenaline above 0·1 μM decrease the proportion of PDH in the active form while lower concentrations of adrenaline increase the proportion of PDH in the active form (Saggerson, 1985). We have therefore investigated the effect of noradrenaline on rat white adipose tissue PDH activity in vivo.

Four to six female rats were under pentobarbital anaesthesia (60 mg/kg body-weight) throughout the experimental period. Parametrial adipose tissue was removed 20 min after intraperitoneal injection of either noradrenaline (1·5 mg/kg live weight), glucose (1 g/kg live weight) or saline (9 g sodium chloride/l) and was immediately frozen in liquid nitrogen for subsequent determination of PDH activity by the method of Stansbie *et al.* (1976). When propranolol, prazosin or yohimbine were administered they were injected intraperitoneally 10 min before the noradrenaline.

Noradrenaline had no effect on total PDH activity but increased the proportion of PDH in the active state. This effect was completely blocked by the α_1-antagonist prazosin and by the β-antagonist propranolol and was partially blocked by the α_2-antagonist yohimbine. Stimulation of white adipose tissue PDH activity by noradrenaline probably accounts for the elevated PDH activity in white adipose tissue from rats killed by cervical dislocation and the higher PDH activity in white adipose tissue in rats at 10.00 hours than at 12.00 hours.

During lactation glucose metabolism in peripheral tissues is decreased to accommodate the demands of the mammary gland (Williamson, 1980; Vernon & Flint, 1983). We have found that the ability of insulin to activate PDH in white adipose tissue is lost during lactation. Preliminary studies indicate that the stimulation of white adipose tissue PDH activity by noradrenaline is also muted during lactation both in vivo and in vitro.

E. Kilgour was supported by the AFRC.

Saggerson, E. D. (1985). In *New Perspectives in Adipose Tissue*, pp. 87–120 [A. Cryer and R. L. R. Van, editors]. London: Butterworths.

Stansbie, D., Brownsey, R. W., Crettaz, M. & Denton, R. M. (1976). *Biochemical Journal* **160**, 413–416.

Vernon, R. G. & Flint, D. J. (1983). *Proceedings of the Nutrition Society* **42**, 315–331.

Williamson, D. H. (1980). *FEBS Letters* **117**, Suppl., K93–K97.

Control of liver enzyme activity by the autonomic nerves in sheep. By M. H. ANIL[1], N. JESSOP[2] and J. M. FORBES[1],[1]*Department of Animal Physiology and Nutrition, University of Leeds, Leeds LS2 9JT* and [2]*Edinburgh School of Agriculture, West Mains Road, Edinburgh EH9 3JG*

In addition to carrying afferent information from the hepatic receptors, the efferent divisions of the sympathetic (Edwards, 1972) and parasympathetic nerves also seem to exert some influence on the metabolic activity in the liver (Lautt, 1983). The present experiment was designed to extend our knowledge of the role of efferent nerves in the control of liver function in sheep.

Four adult sheep were adrenalectomized and pancreatectomized under chloralose anaesthesia, and stimulating electrodes were placed on the peripheral ends of the cut left splanchnic nerve and the cervical vagus. Specimens of liver were taken before and after 5 min of stimulation (0·5 ms duration, 100/s frequency, 20–50 V amplitude) and immediately freeze-clamped and cooled in liquid nitrogen. The activities of phosphorylase A and of total phosphorylase (*EC* 2.4.1.1) were measured using the method of Shimazu & Amakawa (1968). A period of 20 min was allowed between periods of stimulation. It was found that enzyme activity returned to control levels within this time. The results are shown in the Table.

Table. *Phosphorylase A activity as percentage of total phosphorylase*

Control			Splanchnic stimulation			Vagal stimulation			Vagal + splanchnic stimulation	
Mean	SEM	*n*	Mean	SEM	*n*	Mean	SEM	*n*	Mean	*n*
33·7	6·15	4	50·5	4·54	4	32·8	5·8	4	41·1	2

Statistical analyses using paired *t* tests showed that splanchnic-nerve stimulation increased enzyme activity (P=0·003, n 4), vagal stimulation had no effect, whereas simultaneous stimulation of both nerves seemed to counteract the splanchnic-nerve effect.

The sympathetic nerves can have a direct effect on the liver parenchyma of the sheep to stimulate glycogenolysis and parasympathetic innervation may inhibit this effect.

Edwards, A. V. (1972). *Journal of Physiology* **220**, 315–334.
Lautt, W. W. (1983). *Progress in Neurobiology* **21**, 323–348.
Shimazu, T. & Amakawa, A. (1968). *Biochimica et Biophysica Acta* **165**, 335–348.

Absorption of water and electrolytes from hypotonic, isotonic and hypertonic solutions. By J. B. LEIPER (Introduced by M. GLEESON), *Department of Environmental and Occupational Medicine, University Medical School, Foresterhill, Aberdeen AB9 2ZD*

Net absorption of water from the small intestine is considered to be due to a bulk-flow effect promoted by the active transport of solute from the lumen into the mucosa (Sladen, 1972). However, reports of enhanced water uptake from hypotonic solutions (Wapnir & Lifshitz, 1985) have suggested that the osmolality of the luminal contents may play a major role in water absorption. We have examined, using a steady-state perfusion technique, net water and electrolyte transport from potable spring water (W); Quosh®, a still-orange squash drink (S); Lucozade®, a carbonated glucose drink (L); and Dioralyte®, a commercially available glucose–electrolyte solution (D), in the normal human jejunum (n 8). A multilumen tube was positioned in the jejunum with the perfusion port just distal to the ligament of Treitz. The tube used incorporated a 150 mm 'mixing' segment and a 300 mm 'test' segment. Transit through the 'mixing' segment altered the composition of all solutions. W increased its mean (SD) osmolality from 10 (5) mosmol/kg to 98 (49) mosmol/kg; S (256 (5) mosmol/kg) remained moderately hypotonic (249 (19) mosmol/kg) with respect to plasma; the osmolality of D dropped from 299 (5) to 282 (9) mosmol/kg but remained essentially isotonic with plasma; hypertonic L dropped from 631 (5) to 488 (53) mosmol/kg.

In the 'test' segment, mean net water absorption from D (7·1 (2·3) ml/10 mm per h) was higher than that from hypotonic solutions W (1·8 (2·8) ml/10 mm per h, $P<0.001$) or S (5·4 (3·3) ml/10 mm per h, $P<0.05$). Hypertonic solution L produced net secretion of water into the lumen (−3·6 (6·4) ml/10 mm per h, $P<0.001$). W, L and S produced net secretion of sodium and potassium into the jejunum, but these ions were absorbed from D (Na 67 (287), K 155 (59) mmol/10 mm per h). During perfusion the luminal contents tended to become isotonic and isoionic with respect to plasma. The isotonic solution D promoted the greatest absorption of water, Na, K and glucose.

Active co-transport of glucose and Na from the lumen to the mucosa is influenced by the relative proportion of these solutes (Sladen & Dawson, 1969). Net water absorption from the jejunum appears to be determined mainly by the rate of active transport of solute, rather than the overall osmotic gradient across the mucosa.

This study was approved by the local Ethical Committee.

Sladen, G. E. G. (1972). In *Transport Across the Intestine*, pp. 14–34. [W. L. Burland and P. D. Samuel, editors]. Edinburgh: Churchill-Livingstone.
Sladen, G. E. G. & Dawson, A. M. (1969). *Clinical Science* **36**, 119–131.
Wapnir, R. A. & Lifshitz, F. (1985). *Pediatric Research* **19**, 894–898.

The effect of intravenous infusion of noradrenaline on lipolysis in adult sheep. By M. E. SYMONDS, M. J. HANNAH, RACHEL J. KIRKMAN and M. A. LOMAX, *Department of Physiology & Biochemistry, University of Reading, Whiteknights, Reading RG6 2AJ*

Lipolytic rate in vivo may be estimated by measuring the entry rates of either glycerol or non-esterified fatty acids (NEFA). It has been proposed that NEFA entry rate will underestimate total lipolytic activity because fatty acids may be recycled within adipocytes (Vernon, 1981) and therefore glycerol entry rate represents a better estimate of the rate of lipolysis in vivo. The present study examines the effects of the lipolytic agent noradrenaline (NA) on lipolysis by simultaneously measuring glycerol and NEFA entry rates.

Four adult wethers (weight 80–85 kg) were given a diet of barley concentrate and ammonia-treated straw twice daily at 09.00 and 18.00 hours. The sheep received continuous infusions via a jugular vein catheter of either sterile saline (9 g sodium chloride/l) or NA (0·2 μg/kg live weight per min) plus ascorbic acid (1 g/l) for a period of 40 h. The infusions commenced at 18.00 hours and blood samples were taken after 15 and 39 h. During the final 12 h, [1-^{14}C]palmitic acid and [2-^{3}H]glycerol were also infused in order to measure the metabolism of NEFA and glycerol.

Infusion	Glycerol concentration (mM)		Glycerol entry rate (mmol/min)		NEFA concentration (mM)		NEFA entry rate (mmol/min)		CO_2 from NEFA (%)	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Saline	0·101	0·011	0·142	0·030	0·19	0·03	0·42	0·09	5	1
NA	0·140	0·022	0·192	0·032	1·30†	0·45	2·78*	0·81	41*	11

Significantly different from saline (Student's *t* test): †P=0·06, *P<0·05.

NA infusion resulted in a significant increase in the entry rate of NEFA but had little effect on glycerol entry rate. Bergman (1968) observed that NA has a greater effect on NEFA than on glycerol concentrations. These results suggest that NA promotes partial hydrolysis of triglycerides and therefore has a greater effect on the release of NEFA than on the release of glycerol from adipose tissue. NA also stimulated the contribution of NEFA carbon to the whole body carbon dioxide pool indicating a possible increase in the rate of total fat oxidation. It is concluded that neither glycerol nor NEFA entry rates necessarily reflect the true lipolytic rate in vivo.

M.E.S. acknowledges the support of a MAFF studentship.

Bergman, E. N. (1968). *American Journal of Physiology* **215**, 865–873.
Vernon, R. G. (1981). *Progress in Lipid Research* **19**, 23–106.

Effect of *Escherichia coli* enterotoxin (STa) on mucosal surface pH in vivo in rat proximal jejunum. By G. T. A. MCEWAN[1], M. N. BURGESS[2] and M. L. LUCAS[1], [1]*Institute of Physiology, University of Glasgow, Glasgow G12 8QQ* and [2]*Beecham Pharmaceuticals, Walton Oaks, Surrey*

The mucosal surface pH of rat proximal jejunum is significantly more acid than neutral luminal fluid or incubation media both in vivo and in vitro (Lucas, 1984). Intestinal derangement may cause the mucosal surface to become neutral. To test this, the effect of *Escherichia coli* enterotoxin (STa) on the surface pH of rat proximal jejunum in vivo was investigated.

Male Wistar rats (290–310 g) were anaesthetized (80 mg/kg body-weight) with pentobarbitone (May and Baker) and maintained at 37°. A 50 mm loop of proximal jejunum was mounted in a perfusion chamber (Lucas & Cannon, 1983) which allowed access to mucosa with intact mesenteric vasculature. Glucose-free Krebs–phosphate buffer was perfused through the chamber at 1 ml/min. In experiments investigating the effects of *E. coli* enterotoxin, 50 μg of purified toxin from *E. coli* strain P16 were added per ml of perfusate. Surface pH was monitored by placing a miniaturized glass pH electrode (Microelectrodes Inc, NH, USA), connected to a Pye Unicam 9409 digital voltmeter, onto the mucosal surface using a Prior micromanipulator.

Period of experiment (min)	Surface pH: Control (*n* 10) Mean	SEM	*E. coli* toxin (*n* 10) Mean	SEM
0	6·34	0·05	6·30NS	0·04
30	6·31	0·04	6·91•••	0·08
60	6·24	0·03	6·91•••	0·06

NS, not significant.
Mean value significantly different from control value (Student's *t* test): •••$P<0.001$.

Surface pH was significantly ($P<0.001$) increased by *E. coli* enterotoxin compared with control experiments where no significant change was recorded over the experimental period.

Abolition of the jejunal acid microclimate should result in malabsorption of weak acids and enhanced weak base absorption. This has been demonstrated from studies on the effect of bacterial toxin challenge on drug absorption from the small intestine (Lynch & Lucas, 1983).

Lucas, M. L. (1984). In *Intestinal Absorption and Secretion*, pp. 39–54 [E. Skadhauge and K. Heintze, editors]. Lancaster: MTP.
Lucas, M. L. & Cannon, M. J. (1983). *Biochimica et Biophysica Acta* **730**, 41–48.
Lynch, J. & Lucas, M. L. (1983). *Gut* **24**, 989.

Changes to the pars oesophagea in pigs before and after weaning onto a dry diet. By P. D. CRANWELL, K. D. CHANDLER, C. J. CAIN, *School of Agriculture, La Trobe University, Victoria* 3083, *Australia*, D. J. MCGILLIVERY, *Regional Veterinary Laboratory, Hamilton, Victoria* 3300, *Australia* and D. P. HENNESSY, *'Attwood' Veterinary Research Laboratory, Westmeadows, Victoria* 3047, *Australia*

The pars oesophagea, the smallest region of the gastric mucosa in the pig, surrounds the cardia and is covered with stratified, squamous, non-glandular epithelium. Although this region is the major site of gastric ulcers in pigs of 2 months and older, little information is available about its earlier development.

In the present study the pars oesophagea was examined in 105 Large White × Landrace pigs from seventeen litters which were either reared by the sow until 49 d, with no access to solid food, or reared by the sow for 21–35 d, given access to solid food at 14 d and entirely dependent on solid food after weaning. The solid food used throughout the experiment was Pig Creep Starter Crumbles + Mecadox (Barastoc Products, Victoria, Australia); it contained 220 g crude protein (nitrogen × 6·25)/kg and 40 g crude fibre/kg. Grains used in the diet were hammer-milled (screen size 4 mm).

					Growth rate (g/d)				Stomach wt: body-wt (g/kg)	
					Before weaning		After weaning			
	n	Age (d)	Body-wt (kg)	Period from weaning to slaughter (d)	Mean	SE	Mean	SE	Mean	SE
Sucking pigs (no solid food)	33	1–49	0·8–14·3	—	247[a]	7	—	—	4·5[a]	0·1
Sucking pigs (solid food)	11	20–34	6·3–13·6	—	310[b]	15	—	—	4·4[a]	0·1
Weaned pigs	17	30–45	4·6–16·5	2–10	253[a]†	10	306[a]	29	5·7[b]	0·2
	21	34–52	6·8–25·0	11–20			349[a]	27	6·3[c]	0·1
	13	51–59	18·4–27·7	21–30			503[b]	17	5·6[b]	0·2
	10	61–72	25·7–37·9	31–44			643[c]	11	5·7[b]	0·2

[a, b, c]Within a column, mean values with different superscript letters were significantly different: $P<0·05$.
†Growth rate before weaning for all weaned pigs (n 61).

In the sucking pig (<14 d) the surface of the pars oesophagea was normally white, flat to undulating, smooth and glistening; however, in the older sucking pigs (>14 d) it was sometimes bile stained and the surface undulations more pronounced. In sucking pigs given access to solid food the pars oesophagea was thicker and in some cases the surface was corrugated. In pigs 2–44 d after weaning, the pars oesophagea could be described in the following categories: (1) normal, flat or undulating; (2) thickened and corrugated; (3) bile stained and peeling; (4) irregular, corrugated, elevated and roughened; (5) wart-like, bile stained parakeratotic plaques; (6) fissures or erosions, or both. Normal to near-normal pars oesophageas were found in 47% of pigs 2–10 d after weaning but in only 9% of pigs 11–44 d after weaning. Pre-ulcerative changes ((3)–(4)) were found in 47% of pigs 2–10 d after weaning and in 34% of pigs 11–44 d after weaning. More severe damage ((5)–(6)) was found in 6% of pigs 2–10 d after weaning and in 57% of pigs 11–44 d after weaning.