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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Fifty-eighth Meeting of the Nutrition Society was held at the Bristol Polytechnic, Coldharbour Lane, Bristol, on Tuesday, 7 April, 1981, *when the following papers were read:*

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Protein turnover in man measured by the excretion of ^{15}N in urea and ammonia.

By E. B. FERN, P. J. GARLICK, M. A. MCNURLAN and J. POWELL-TUCK, *Clinical Nutrition and Metabolism Unit, London School of Hygiene and Tropical Medicine, 4 St. Pancras Way, London NW1 2PE*

In man, most methods for measuring the rate of whole-body protein turnover with the stable isotope of nitrogen are based on the transfer of label from an amino acid, usually glycine, to an end product in the urine, commonly urea (Picou & Taylor-Roberts, 1969) or ammonia (Waterlow *et al.* 1978). However, it is known that, even for the same experimental subject, rates of turnover calculated from urea and ammonia do not correspond (Nicholson, 1970; Golden & Waterlow, 1977; Waterlow *et al.* 1978). This discrepancy has not been investigated.

We have modified the single dose method of Waterlow *et al.* (1978) to obtain, over a short period of 9 h, rates of turnover both from the excretion of isotope in ammonia and from the excretion in urea. The estimates from urea were made possible by monitoring the size of the body pool over the experimental period and by allowing for isotope retained in this compartment at the end.

With the modified method comparisons of the rates of protein synthesis were made in four situations, namely, in the absorptive and postabsorptive condition when the isotope was given either orally or intravenously.

Whole-body protein synthesis (g protein/9 h) after dosing with 200 mg [^{15}N]glycine

(Values are means with their standard errors; no. of observations in parentheses)

	Postabsorptive							Absorptive						
	Ammonia		Urea		EPA			Ammonia		Urea		EPA		
Dosing method	Mean	SE	Mean	SE	Mean	SE		Mean	SE	Mean	SE	Mean	SE	
Oral	60·3	3·3	63·9	8·5	62·2	4·4	(4)	82·1	6·4**	114·8	4·9	98·4	3·3	(8)
Intravenous	52·7	4·6*	81·6	6·3	67·2	2·9	(4)	56·0	8·8**	151·5	9·0	103·8	3·8	(4)

Statistical significance of differences between ammonia and urea; $^{*}P=0.05$; $^{**}P<0.01$ (paired t test).

The Table shows that only when [^{15}N]glycine was given orally in a postabsorptive period did the rate of synthesis from ammonia equal the rate from urea. In the other three situations the rate based on ammonia was always significantly lower than the rate from urea. The results suggest that estimates of synthesis from ammonia are inversely related to those from urea and that the mean value of the two rates, the end-product average (EPA), is a useful parameter for judging changes in protein turnover. The end product average gives rates of whole-body synthesis comparable with those calculated by independent methods and does not appear to be affected by the route of isotope administration.

Golden, M. H. N. & Waterlow, J. C. (1977). *Clin. Sci. Mol. Med.* **53**, 277.
Nicholson, J. F. (1970). *Pediat. Res.* **4**, 389.
Picou, D. & Taylor-Roberts, T. (1969). *Clin. Sci.* **36**, 283.
Waterlow, J. C., Golden, M. H. N. & Garlick, P. J. (1978). *Am. J. Physiol.* **235**, E165.

0029–6651/81/4003–358A $00.35

Further examination of the source of urinary N^{τ}-methyl histidine in the rat. By P. C. BATES and D. J. MILLWARD, *Clinical Nutrition and Metabolism Unit, Department of Human Nutrition, London School of Hygiene and Tropical Medicine, 4 St. Pancras Way NW1 2PE*

We have reported that skeletal muscle may contribute a lower proportion of urinary N^{τ}-methyl histidine (MH) than is commonly supposed (Millward *et al.* 1980). The evidence for this is a lower turnover rate of protein-bound MH in muscle than that necessary to account for the observed excretion rate and the presence of rapidly turning over pools of MH in the rat indicated by the decay rate of urinary MH labelled by [^{14}C]methyl methionine injection. We have performed two further experiments. To confirm the low turnover rate of muscle MH we have measured the relative steady state synthesis rates of protein-bound MH and the average rate for mixed muscle proteins. Groups of fully grown female rats (380 g) were injected with [^{14}C]histidine either fed or after an overnight fast and the relative labelling of MH and histidine measured in mixed muscle proteins after 60 min. The ratio of MH to histidine labelling was $0{\cdot}407 \pm 0{\cdot}092$ and $0{\cdot}415 \pm 0{\cdot}12$ in the fed and fasted rats respectively. Since the over-all rate of synthesis measured by [^{14}C]tyrosine infusion was 4·2%/d this indicates that the steady state turnover rate of MH in muscle was 1·71 and 1·74%/d.

The observed rate of MH excretion in these rats was 4·37 μmol/d and assuming their whole body MH content to be 105 μmol (Nishizawa *et al.* 1977) the fractional turnover rate of whole body MH was 4·16%/d. Thus skeletal muscle must account for less than 41% of excreted MH.

In a second experiment we measured the decay of labelling in urinary and muscle MH in similar rats fed MH-free diets for 60 d after the injection of [^{14}C]methyl methionine. As we have previously shown there was a rapid fall in labelling initially indicating rapidly turning over pools of MH in the body. However, there was no decay of labelling in muscle protein-bound MH during the first 30 d, after which time a slow decay rate of 1·4%/d was observed. However, after 25 d the labelling of urinary MH was lower than that of muscle indicating either the contribution of non-muscle sources to urinary MH or non-random degradation of muscle proteins.

These experiments further support the possibility that non-muscle sources of MH make a major contribution to urinary MH excretion in the rat.

Millward, D. J., Bates, P. C., Grimble, G. K., Brown, J. G., Nathan, M. & Rennie, M. J. (1980). *Biochem J* **190**, 225.
Nishizawa, N., Shimbo, M., Hareyama, S. & Funabiki, R. (1977). *Br. J. Nutr.* **37**, 345.

0029–6651/81/4003–358A $00.35

Nitrogen turnover measurement by ^{15}N glycine priming prior to continuous infusion in healthy man. By A. J. W. SIM, *Department of Surgery, Royal Infirmary, Glasgow,* B. SUGDEN, V. R. YOUNG and F. D. MOORE, *Department of Surgery of Harvard Medical School, Peter Bent Brigham Hospital* and *Department of Nutrition and Food Science, Massachusetts Institute of Technology, Boston, Massachusetts, USA*

Nitrogen turnover is a third independent variable which allows interpretation of N balance data in terms of whole-body protein synthesis and breakdown. N turnover has been measured by both single (San Pietro & Rittenberg, 1953) and continuous (Picou & Taylor-Roberts, 1969) administration of ^{15}N-labelled amino acids. The latter method requires administration of isotope for up to 60 h (Sim *et al.* 1979) before a plateau of excretion of ^{15}N-labelled urinary urea is achieved.

Four healthy male subjects were studied for 6 d periods on two separate occasions with a nutritional intake of 1 g protein/kg per d and 126 kJ (30 kcal)/kg per d. N balance was calculated daily and N turnover measurements commenced on the fourth study day. In the first study the N turnover was measured by the continuous intravenous infusion of 0·4 mg ^{15}N/kg per d for 60 h. In the second study a priming injection of 0·4 mg ^{15}N/kg was administered prior to the same continuous infusion.

The mean N balances were $-0·1 \pm 1·3$ and $0·5 \pm 2·3$ g N/d respectively. In the priming dose study a plateau of enrichment was achieved between 12 and 18 h. Similar rates of N turnover, whole body protein synthesis and breakdown were achieved.

	Nitrogen turnover mgN/kg per d		Protein synthesis g protein/kg per d		Protein breakdown g protein/kg per d	
	Mean	SD	Mean	SD	Mean	SD
Continuous infusion	570	66	2·57	0·36	2·56	0·39
Continuous infusion + priming injection	605	66	2·93	0·37	2·79	0·40

We conclude that the addition of a priming injection of ^{15}N glycine produces an earlier plateau of urinary urea enrichment with ^{15}N and allows N turnover to be calculated after a shorter constant infusion.

Picou, D. & Taylor-Roberts, T. (1969). *Clin. Sci.* **36,** 283.
San Pietro, N. & Rittenberg, D. (1953). *J. biol. Chem.* **201,** 457.
Sim, A. J. W., Wolfe, B. M. Young, V. R., Clarke, D. & Moore, F. D. (1979). *Lancet* i, 68.

0029–6651/81/4003–358A $00.35

Effect of environmental temperature on protein metabolism following burn injury in the rat. By G. GEDEON,[1] A. SHENKIN,[1] G. S. FELL,[1] G. AL-SHAMMA,[1] C. GOLL[2] and J. R. RICHARDS,[3] [1]*Departments of Biochemistry and* [2]*Surgery, Glasgow Royal Infirmary and* [3]*Department of Surgery, Hairmyres Hospital, East Kilbride*

The increase in net protein catabolism which follows a severe injury in man or animals can be partly attenuated by an elevation in the environmental temperature (Cuthbertson, 1979). We have used a standard burn injury in the rat as a model to investigate this effect.

Male Wistar rats (210–250 g body-weight) were subjected to a full thickness dorsal skin burn of 25% of their body surface area and were subsequently maintained for 50–60 d either at 20° or 30° on 15 g/d 20% lactalbumin diet. Control animals were pair fed to the test group. Nitrogen balance was measured throughout and body composition analysed chemically at intervals on sacrificed animals. 3-methylhistidine (3MH) was measured on acid hydrolysed urine (Tomas *et al.* 1979) and catecholamines estimated by an automated fluorometric procedure (Wood & Mainwaring-Burton, 1974).

The animals kept at 20° developed; (1) progressive loss of body-weight, (2) an increase in urinary N and negative N balance throughout, (3) a sustained increase in urine 3MH (at day 0, (mean ± SD) 0·7±0·15; at day 10, 1·85±0·25 μmol/d per 100 g body-weight, with similar levels beyond), (4) loss of protein from both carcass and pelt, (5) an increase in urine noradrenaline (from 1·1±0·2 μg/d at day 0 to 4·3±1·1 μg/d at day 10) and in adrenaline (0·4±0·16 μg/d at day 0 to 0·9±0·2 μg/d at day 10).

In animals at 30° it was noted that (1) body-weight fell for the first 6 d, and subsequently increased but weight gain was not as rapid as in control animals, (2) urine N increased for 6 d post burn and then decreased but remained higher than in control animals, (3) urine 3MH increased from 0·83±0·05 μmol/d per 100 g body-weight at day 0 to 1·36±0·69 μmol/d per 100 g body-weight at day 6, but thereafter was not significantly higher than controls, (4) body-weight gain was predominantly water; protein and fat content increased in the carcass but decreased in the pelt, (5) urine catecholamines (noradrenaline 1·1±0·5 μg at day 10; adrenaline 0·2±0·1 μg at day 10) were lower than in the 20° group ($P<0·001$) but remained higher than the control group throughout the study period.

Urine noradrenaline correlated significantly with both urine N (20° rats r 0·90; 30° rats r 0·49) and urine 3MH (20° rats r 0·92; 30° rats r 0·71).

It is concluded that burn injury in the rat causes an increase in net protein catabolism from both skeletal muscle and pelt protein and that these changes can be reduced by an elevation in environmental temperature. Our findings also support the hypothesis that these effects may, in part, be mediated by catecholamines.

Cuthbertson, D. P. (1979). *J. Par. Ent. Nutr.* **3**, 108.
Tomas, F. M., Munro, H. N. & Young, V. R. (1979). *Biochem. J.* **178**, 139.
Wood, W. G. & Mainwaring-Burton, R. W. (1974). *Clin. Chim. Acta* **61**, 297.

0029–6651/81/4003–358A $00.35

The effect of different energy sources in parenteral nutrition on protein metabolism postoperatively. By E. ROGALY, *University of Witwatersrand, Johannesburg,* M. B. CLAGUE, M. J. KEIR, P. D. WRIGHT and I. D. A. JOHNSTON, *Department of Surgery, University of Newcastle upon Tyne*

A number of different solutions are available as energy sources in parenteral nutrition but the commonly employed regimes involve the use of glucose alone or in combination with a fat emulsion as the energy supply. In contrast to glucose, fat emulsions alone induce no nitrogen sparing (Craig *et al.* 1977), whereas, when administered with amino acids, a combination of glucose and fat emulsion was more efficacious at increasing whole body nitrogen than an isoenergetic amount of glucose alone (Macfie *et al.* 1981).

To evaluate this further ten female patients undergoing elective cholecystectomy were intravenously fed from the day of their surgery. Each patient was given an isoenergetic isonitrogenous solution with respect to body-weight (200 mg N/kg per d with 34·3 kcal/kg per d) at a constant rate, but in half the patients (group 1) energy was supplied as glucose alone whereas in the remaining patients (group 2) energy was provided as glucose (17·1 kcal/kg per d) and intralipid (17·1 kcal/kg per d). Rates of protein synthesis and breakdown for the whole body were determined in each patient preoperatively on an oral diet (123 mg N/kg per d with 18 kcal/kg per d) as a baseline, and again on the second postoperative day whilst receiving their parenteral nutrition, employing a constant rate infusion of 1-[1-^{14}C]leucine (James *et al.* 1974).

Both groups were well matched for age, body-weight, activity of disease preoperatively and their degree of trauma at the time of their postoperative study (Clague *et al.* 1980). The results are shown in the Table.

Protein metabolism (g/kg per d)

	Preoperative (oral nutrition)						Change postoperatively (IV feeding)					
	Synthesis		Breakdown		Balance		Synthesis		Breakdown		Balance	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Group 1	1·91	0·38	1·50	0·47	+0·41	0·05	−0·42	0·25	−0·66	0·25	+0·24	0·04
Group 2	2·16	0·20	1·57	0·23	+0·45	0·08	−0·67	0·30	−0·94	0·34	+0·27	0·04

Protein metabolism was similar in both groups before surgery and changed in an identical manner postoperatively. It is concluded that both regimes are equally effective in fuelling protein metabolism following surgery.

Clague, M. B., Carmichael, M. J., Keir, M. J., Wright, P. D. & Johnston, I. D. A. (1980). *Br. J. Surg.* **67,** 366.
Craig, R. P., Tweedle, D. E. F., Davidson, H. A. & Johnson, I. D. A. (1977). *Lancet* ii, 8.
James, W. P. T., Garlick, P. J., Sender, P. M. & Waterlow, J. C. (1974). *Dynamic studies with radio-isotopes in medicine.* IAEA report no. 185. p. 461. Vienna: International Atomic Energy Authority.
Macfie, J., Smith, R. C. & Hill, G. L. (1981). *Gastroenterology* **80,** 103.

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The effect of supplemental parenteral nutrition on protein synthesis and breakdown in patients with colorectal neoplasia. By M. J. CARMICHAEL, *University of Texas Medical Centre, Houston, USA,* M. B. CLAGUE, M. J. KEIR, P. D. WRIGHT and I. D. A. JOHNSTON, *Department of Surgery, University of Newcastle upon Tyne*

Parenteral nutrition can be of value in improving the nutritional status of some patients with gastrointestinal malignancies and may improve their tolerance to therapy and reduce the incidence of postoperative complications (Copeland & Dudrick, 1976).

To evaluate the mechanism underlying the improved nitrogen retention a study was planned in four patients with colorectal neoplasia requiring parenteral nutrition. Rates of protein synthesis and breakdown for the tumour–host conglomerate were determined before and one week after instituting a course of preoperative supplemental parenteral nutrition (144 mg N/kg per d with 30 kcal/kg per d; energy:nitrogen 200:1), employing a constant rate infusion of 1-[1-^{14}C]leucine (James *et al.* 1974). Oral nutrition was provided *ad lib.* between the studies, but maintained at the same level during both studies (120 mg N/kg per d with 15 kcal/kg per d; energy:nitrogen 125:1). Approval for the studies was granted by the Local Ethical Committee and the Isotope Advisory Panel in London. The results are shown in the Table.

Protein metabolism (g/kg per d)

	Synthesis				Breakdown			
Patient . . .	1	2	3	4	1	2	3	4
Oral feeding	2·09	1·66	2·89	1·16	1·78	1·26	2·49	0·54
Oral + IV feeding	2·52	1·96	3·08	1·76	1·53	0·56	1·76	0·32
Mean change		+0·38				−0·53		
Paired '*t*' test		$P<0·03$				$P<0·04$		

The improved N retention from supplemental parenteral nutrition appears to be derived both from stimulation of protein synthesis and reduction in breakdown. Increased synthesis probably occurs in response to a higher N intake as has been demonstrated in normal man (Garlick *et al.* 1978). The reduced breakdown could be attributed to simply overestimating the specific radioactivity of the labelled amino acid at the site of protein synthesis but, if real, could represent reduced gluconeogenesis consequent on the improved energy:N value.

Copeland, E. M. & Dudrick, S. J. (1976). *Current problems in cancer. Year book.* Chicago: Medical Publishers.

Garlick, P. J., Clugston, G. A., Swick, R. W., Meinertzhagen, I. H. & Waterlow, J. C. (1978). *Proc. Nutr. Soc.* **37**, 33A.

James, W. P. T., Garlick, P. J., Sender, P. M. & Waterlow, J. C. (1974). *Dynamic studies with radio-isotopes in medicine.* IAEA report no. 185. p. 461. Vienna: International Atomic Energy Agency.

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Rate of protein turnover in obese women on energy restriction and its relationship to extrathyroidal T_4 metabolism. By K. S. NAIR, D. HALLIDAY, M. LALLOZ and J. S. GARROW, *Nutrition Section, Clinical Research Centre, Harrow, Middlesex*

There are conflicting reports concerning the effect of energy restriction on the rate of protein turnover in obese women (Garrow, 1980). Recent results (Nair *et al.* 1980) indicated that, if a group of obese patients is maintained on a diet supplying 3·4 MJ (800 kcal) and 100 g protein/d, both the rate of protein turnover and metabolic rate decrease. It was observed that these effects could be reversed by the administration of tri-iodothyronine (T_3).

To extend this investigation, ten obese women were subjected to the above dietary regime for 3 weeks. Of these women, three received 120 μg T_3 daily, three received 25 mg tetra-iodothyronine (T_4) whilst the remainder received no hormone supplement. Protein turnover was estimated using the [^{15}N]glycine–ammonia end product method (Waterlow *et al.* 1978) at weekly intervals. [^{15}N]glycine was administered orally under standard conditions and calculations were based on the ensuing mean urinary ammonia [^{15}N]enrichment. Serum T_3, rT_3 and T_4 levels were also measured at weekly intervals. The procedure was approved by the Northwick Park Hospital Ethical Committee.

Week of study	T_3:rT_3 Mean	T_3:rT_3 SD	Rate of protein turnover (g/d) Mean	Rate of protein turnover (g/d) SD	Conditions of study
1	7·1	1·9	467·6	75·0	
2	6·1	2·0	459·8	64·0	Energy restriction only
3	5·6	2·0	450·8	54·4	
1	5·5	1·27	509·7	119·2	Energy restriction
2	2·9	0·47	446·7	34·0	+
3	2·98	1·12	435·7	5·5	0·25 mg T_4/d
1	5·3	0·93	558·3	61·08	Energy restriction
2	23·3	5·65	652·3	75·8	+
3	27·2	10·53	715·7	64·61	120 μg T_3/d

A highly significant correlation was obtained between T_3:rT_3 and protein turnover rate (r 0·8, $P<0·001$). This finding suggests the possible importance of extrathyroidal T_4 metabolism in the regulation of protein turnover under the conditions of this study.

Garrow, J. S. (1980). *Nitrogen Metabolism in Man.* [J. C. Waterlow and J. M. L. Stephen, editors]. Applied Science Publishers.

Nair, K. S., Halliday, D. & Garrow, J. S. (1980). *Recent Advances in Obesity Research, III.* Rome: Newman Publishing Ltd.

Waterlow, J. C., Golden, M. H. N. & Garlick, P. J. (1978). *Am. J. Physiol.* **235,** E165.

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Manipulation of thyroid status in the rat by means of the osmotic minipump. By J. G. BROWN and D. J. MILLWARD, *Clinical Nutrition and Metabolism Unit, Department of Human Nutrition, London School of Hygiene and Tropical Medicine, 4 St. Pancras Way, London NW1 2PE*

The manipulation of thyroid hormone levels during investigations of the hormones' action is usually achieved through single or repeated injections of T_4 or T_3. Such administration does not enable physiological levels of the hormone to be maintained throughout the day. We have examined the use of an osmotic minipump implanted into the peritoneal cavity in maintaining T_3 concentrations within the physiological range. The effects of daily injections of T_3 (2 μg/100 g body-weight per d) has been compared with delivery from a minipump (Alzet 2001) at rates of 2 and 0·75 μgT_3/100 g body-weight per d. Measurements were made of serum T_3 concentrations and growth rates of treated and untreated thyroidectomized and euthyroid rats.

Thyroidectomy induced a fall in growth rate from 4·5%/d to less than 2%/d after 7 d. Treatment with T_3 at 0·75 μg/d maintained growth at the euthyroid rate, but treatment at 2 μg/d by a minipump or injection depressed growth. The difficulty of maintaining fixed levels of T_3 by injection is illustrated in the Table. Serum T_3 levels measured 24 h after injection and therefore representing minimum levels during each 24 h period were high initially but decreased to low levels after 6 d. This was presumably as a result of increased T_3 degradation or clearance induced by the high transient levels after the injections or both. In contrast the minipumps maintained more constant T_3 levels with 0·75 μg/d giving values close to the euthyroid levels and 2 μg/d inducing moderate hyperthyroidism. We are using this method of T_3 administration to examine the dose response of muscle protein synthesis and degradation to changes in T_3 concentrations within the physiological range.

Period after implantation (d)...	Serum T_3 (ng/ml)											
	0		1		2		4		6		7	
Groups	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Thyroidectomized untreated	1·02	0·11	0·50	0·11	0·22	0·09	<0·1		<0·1		<0·1	
Sham thyroidectomy	1·16	0·19	1·04	0·10	1·00	0·09	1·13	0·13	1·00	0·10	0·95	0·14
Thyroidectomy + 0·75 μg/100 g body-wt. per d by minipump	1·17	0·10	1·47	0·19	1·46	0·26	1·18	0·23	1·06	0·28	0·76	0·11
Thyroidectomy + 2 μg/100 g body-wt. per d by minipump	0·91	0·15	3·73	0·67	3·71	0·48	2·59	0·40	2·32	0·47	2·10	0·39
Thyroidectomy + 2 μg/100 g body-wt. per d by injection	1·12	0·15	3·45	0·50	1·80	0·31	0·87	0·16	0·36	0·07	0·24	0·05

This work is generously supported by the Muscular Dystrophy Group of Great Britain and the MRC.

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Diurnal pattern of protein turnover in obese subjects given a protein-free diet. By G. A. CLUGSTON and P. J. GARLICK, *Clinical Nutrition and Metabolism Unit, London School of Hygiene and Tropical Medicine, 4 St. Pancras Way, London NW1 2PE*

We have previously shown that in subjects given the total day's intake of a normal diet over 12 h, followed by a 12 h fast, there was a diurnal pattern of rates of protein turnover related to the intake of food (Garlick *et al.* 1980*a*). We have also shown that after 3 weeks on a low-energy protein-free diet rates of protein turnover are decreased (Garlick *et al.* 1980*b*), but these measurements were only made in the fed state (day). The present study was therefore performed to discover whether the diurnal rhythm persisted on a protein-free diet.

Four obese female subjects were given a normal diet (approximately 8·4 MJ and 70 g protein) for 3 d. On the third day rates of whole-body protein synthesis, breakdown and oxidation were measured by constant infusion of [1^{14}C]leucine for periods in excess of 24 h (Garlick *et al.* 1980*b*). For the first 12 h (day) the day's intake was given as twelve equal hourly feeds, after which the subjects fasted for 12 h (night). During the fasting period rates of protein synthesis and oxidation fell, as described previously, but the rise in breakdown was much larger than previously reported (Garlick *et al.* 1980*a*). The subjects were then given a low-energy, protein-free diet (2·1 MJ glucose syrup) for 3 weeks, after which the measurement of protein turnover was repeated. Rates of protein synthesis and oxidation during the feeding period were not only lower than those during the corresponding period on the normal diet, but were lower than those observed during fasting after the normal diet. However, during the period of fasting after the protein-free diet rates of protein synthesis, breakdown and oxidation were the same as during feeding. We conclude that the diurnal variation in rates of protein metabolism that occurs in response to intake of a normal diet is eliminated after adaptation to a low-energy protein-free diet.

Rates of whole-body protein synthesis, breakdown, oxidation and intake (g protein/12 h)

	Synthesis	Breakdown	Oxidation	Intake
Normal diet:				
Feeding	121	77	41	70
Fasting	92	112	20	0
Protein-free diet:				
Feeding	72	79	7	0
Fasting	70	77	6	0

Garlick, P. J., Clugston, G. A., Swick, R. W. & Waterlow, J. C. (1980*a*). *Am. J. Clin. Nutr.* **33**, 1983.
Garlick, P. J., Clugston, G. A. & Waterlow, J. C. (1980*b*). *Am. J. Physiol.* **238**, E235.

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