PROTEIN TURNOVER IS NOT INFLUENCED BY DIFFERENT ENERGY INTAKES IN PRETERM INFANTS.

J.B. van Goudoever, E.J. Sulkers, D. Halliday' and P.J.J. Sauer. Department of Pediatrics, Sophia Children's Hospital, Rotterdam, The Netherlands; CRC, Harrow, London, England. An energy intake of 120 kcal/kg.d in preterm infants resulted in a

higher fat accretion than in utero. To study if a reduced energy intake would not only reduce fat accretion, but also influence protein(pro) turnover(Q), we measured leucine(leu) kinetics in 4 wk old, orally fed VLBW infants, receiving 3.2 g pro/kg.d. They were fed either 120  $(n = 12, bw = 1.1 \pm 0.2 \text{ kg}, ga = 30 \pm 2 \text{ wk})$  or 100 kcal/kg.d  $(n = 12, bw = 1.1 \pm 0.2 \text{ kg}, ga = 31 \pm 2 \text{ wk})$  <sup>13</sup>C-Leucine was given orally, <sup>13</sup>C-KIC plasma dilution was measured by GC-MS, <sup>13</sup>CO<sub>2</sub> excretion by IRMS. Results: mean  $\pm$  1 sd, Leu kinetics in mmol/kg.hr, Pro kinetics in g/kg.d. C = Catabolism, S = Synthesis, Ox = oxidation, Ret = retention.

	120 K	CAL	100 KCAL			
n	7(AGA)	5(SGA)	7(AGA)	5(SGA)		
Leu Q	339 ± 74	$349 \pm 149$	351 ± 79	349±137		
Leu Ox	71 ±24	63 ± 24	76 ±22	59 ±19		
Pro Q	12.9 ± 2.9	$13.3 \pm 5.7$	$13.4 \pm 3.0$	$13.3 \pm 5.2$		
Pro S	12.0 ± 2.9	12.8 ± 5.8	12.6 ± 3.1	$12.8 \pm 5.2$		
Pro C	9.7 ± 2.8	$10.0 \pm 5.6$	10.2 ± 3.0	$10.2 \pm 3.0$		
Pro Ret	$2.3 \pm 0.3$	2.7 ±0.0	$2.4 \pm 0.3$	$2.5 \pm 0.2$		
°p<0.005 A	GA vs SGA, no eff	ect of feeding (Al	Nalvsis Of VArian	ce).		

Conclusions: 1. Decreasing energy intake from 120 to 100 kcal/kg.d does not increase protein catabolism or alter protein turnover, 2, Protein retention is higher in SGA infants

> OUTCOME AT 3 YEARS FOLLOWING A DOUBLE BLIND NEONATAL STUDY COMPARING TWO PARENTERAL AMINOACID PREPARATIONS (PAA)

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A randomised double blind study of 2 PAA solutions given to 64 neonates showed better survival and more normal plasma aminograms in the group receiving M (McIntosh & Mitchell 1990). 23 on M and 25 on V completed the study. At 3 years (range 2.5-3.5) 32 survivors were identified (19 M, 13 V). 17 M and 10 V were psychometrically tested without knowledge of the original course of illness or aminoacid regimen. (2 M and 2 V could not be obtained for testing and 1 V infant was untestable). 2 of 17 M and 4 of 10 V children had below average cognitive ability. Overall 15 of 21 who received 5 days of preparation M and were tested had average or above cognitive ability compared to 6 of 23 who received preparation V and were tested (OR = 7.08, 95% Cl = 1.59-33.13, P 0.01). Assuming original double blind randomisation of the groups, the outcome following the use of PAA solution M is superior to V. McIntosh N & Mitchell V, Arch Dis Child 1990; 65: 692-699. A randomised double blind study of 2 PAA solutions given to 64

INFLUENCE OF DIFFERENT FAT BLEND ON FAT ABSORPTION IN ORALLY FED PRETERM INFANTS.

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Sophia Children's Hospital, Rotterdam, Clinical Chemi-cal Lab, State Univ. Groningen, The Netherlands. A new preterm formula (PT-F), with a fat composition more resem-bling human milk showed a relative decrease in total fat absorption compared to the former PT-F (78% vs 88%), in 4 weeks old orally fad motors. absorption compared to the former FI-F (103 vs 003), in 4 weeks old orally fed preterm infants. To investigate individual fatty acids (FA) coefficients of absorption(CA), 13 3-d stool collec-tions were analysed for the CC FA spectrum in each PT-F group. Group A received a 40/60 MCT/cornoil mixture, group B received a coco/soy/oleic mixture. CA:8, intake + excretion:mg/kg.d

					Incure .	GVCTC	1011+md/1	4.4	
	A:	intak	excr	(CA)		B:	intake	excr	(CA)
C8:0		1258	0.6	(100)			270	0.3	(100)
C10:0		862	8.5	(99)			235	3.8	(98)
C12:0		70	2.1	(97)	p<0.0005		1109	102	(91)
C14:0		104	3.1	(97)	p<0.0001		521	100	(81)
C16:0		569	142	(75)	p<0.05		689	274	(60)
C18:0		188	47	(75)	p<0.0005		260	136	(48)
C18:1		1345	325	(76)	-		2205	721	(67)
C18:2		2115	478	(77)			1200	356	(70)
C18:3		53	15	(73)	p<0.005		96	15	(88)

Concl: Higher fat excretion on PT-F B is not only due to increased intake of LCT, but also to lower CA of saturated FA. Remarkably, C18:3w3 absorption is lower with MCT/cornoil.

## RESPIRATORY WATER LOSS AND OXYGEN CONSUMPTION IN FULL TERM NEWBORN INFANTS EXPOSED TO COOL AIR

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The response to an ambient air temperature (Tamb, °C) below the thermoneutral zone was studied in nine fullterm newborn infants. Respiratory water loss (RWL,mg/kg min), oxygen consumption (VO<sub>2</sub>,ml/kg min), carbon dioxide production (VCO<sub>2</sub>,ml/kg min), core ( $T_e$ , °C) and peripheral temperature ( $T_p$ , °C), and truncal  $(Q_1, \%)$  and peripheral  $(Q_p, \%)$  skin blood flow were continuously monitored during initial care at a  $T_{anb}$  within the thermoneutral zone (interval A) and after lowering  $T_{anb}$  to below 27.5°C (interval B). Mean values for intervals A and B are given in the table. Values for Q, and Q, depict relative change in blood flow, with the mean value for interval A = 100%.

Interval A	T.mb	RH	T.	T,	<u>RWL</u>	<u>VO</u> ₂	VCO <sub>2</sub>	Q,	Q,
Α	33.0	50%	37.0	35.9	3.7	5.3	3.8	100	100
В	26.6	47%	37.0	31.0	6.1	7.9	5.9	99	79
p <	.001	.05	ns	.001	.01	.001	.001	ns	.01

Newborn infants exposed to cool air react with an increase in respiratory water loss, oxygen consumption, carbon dioxide production and an decrease in peripheral skin blood flow and skin temperature before their core temperature is affected and without increased motor activity. RQ remains unchanged.

> RESPONSE OF ENDOGENOUS GLUCOSE PRODUCTION (EGPR) TO A CHANGE IN BLOOD GLUCOSE (BG) LEVEL IN LOW BIRTH WEIGHT (LBW) INFANTS ON THE FIRST DAY OF LIFE.

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In a previous study we found a large inter-individual variability between the BG level and EGPR. In the present study we have measured the response of EGPR to a change in BG level in individual LBW infants.

Patients: Four infants were studied sofar. Gestational age ranged from 30 to 39 weeks, and birthweight from 1.5 to 2.1 kg. Increase of BG level was obtained by increasing the glucose infusion rate from 3.4 to 6.7 mg/kg/min, prior to the increase a small bolus of glucose was given. Studies were done prior to oral feeding, EGPR was measured with the prime dose, constant rate infusion technique, using 66.[<sup>2</sup>H<sub>2</sub>] glucose as a tracer.

Results: Before the change in BG level, BG was 3.5 mmol/l (mean), glucose turnover was 5.6 ±0.5 mg/kg/min. EGPR was 2.2 ± 0.2 mg/kg/min. After the change of BG, BG level was 5.7 mmol/l (mean), glucose turnover increased to 7.7± 1.0 mg/kg/min. The decrease of EGPR ranged from 13 to 100 %. This decrease was not related to the BG level, but was correlated to the relative increase of BG (r=0.99.p=0.006)

Hypothesis: An increase of BG level induces a decrease of EGPR, the degree of decrease is related to the relative change of BG.

> CHANGES IN BRAIN GLUCOSE UTILIZATION INDUCED BY ACUTE Astrid Nehlig, INSERM U272, Universite de Nancy, 30 rue Lionnois, 54013 NANCY, FRANCE.

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82 Astrid Nehlig, INSERM U2/2, Universite de Nancy, 30 rue Lionnois, 64013 NANCY, FRANCE. Changes in regional glucose utilization might be involved in neuronal damage due to hypoxia. The quanti-tative autoradiographic 2-14C-deoxyglucose technique (20G) was applied to the measurement of the effects of an acute hypoxic exposure on local cerebral glucose utilization (LCGU) in the 10 (P10), 14 (P14), and 21 (P21) day-old rat. The animals were expo-sed to the hypoxic (7% 02/93% N2) or control gas mixture (21% 02/ 79% N2) for 20 min before the initiation and for 45 min of the 20G procedure. At P10, the exposure to the hypoxic gas mixture induced a generalized increased in LCGU which affected 40 struc-tures (including white matter) of the 45 studied. At P14, LCGU increased in 5 areas and decreased in 11 regions, mainly brain-stem and respiratory areas in hypoxic as compared to control rats. But average cerebral glucose utilization was similar in both groups. Finally, at P21, LCGU decreased in 11 structures of hypoxic rats as compared to controls. The increase in LCGU of the hypoxic 10 day-old rat is likely to be the reflection of the stimulation of anaerobic glycolysis in brain structures with a low metabolic rate. Conversely, at P14 and P21, metabolic rate increases and the brain becomes more dependent upon oxygen. These results suggest that immature brain responds to an acute hypoxic aggression in a speci-fic way according to the maturation of oxidative metabolic pathways.