Australian radiation oncology facility were eligible for inclusion. Sixty patients (51M;9F; mean age 61.9±14.0yr) were enrolled and randomised to receive either usual care (UC)(n=31) provided by the nurses or nutrition intervention (NI)(n=29) in the form of regular and intensive nutrition counselling by a dietitian. Baseline characteristics did not differ between groups. Nutritional status was measured by the scored Patient-Generated-Subjective Global Assessment (PG-SGA). Body weight, BMI and percentage weight loss in the previous six months were also recorded. QoL was measured by the European Organisation for the Research and Treatment of Cancer (EORTC) QLQ-C30 version 3. Outcomes were assessed at the commencement of radiotherapy and after four weeks. Three subjects were lost to followup. Results: The UC group had a significantly greater deterioration in PG-SGA score with a mean change of 6.6 compared with 1.9 in the NI group (p=0.001). The UC group experienced significant deteriorations in weight and QoL compared with the NI group (Table 1). Nutrition measures used in practice to provide broad assessments, such as BMI and SGA do not appear to reflect subtle changes in nutritional status over this period. Conclusion: Early and intensive nutrition intervention provides beneficial outcomes in terms of minimising weight loss and deterioration in nutritional status and QoL in oncology outpatients receiving radiotherapy to the head, neck or GIT area. Tools which are composed of multiple constructs appear to be more useful in determining outcomes compared with tools designed for broad assessment * A positive value for change in PG-SGA score or change in percentage weight loss in the past six months reflects a deterioration in nutritional status ** A negative value for change in QoL reflects a deterioration in quality of life.

Table 1. Change in nutritional status and QoL after four weeks of radiotherapy

Variable N	Usual Care (UC) (30)	Nutrition Intervention (NI) (27)	P
Change in PG-SGA score*	6.6 ± 5.0	1.9 ± 5.6	0.001
Change in SGA	No change (<1)	No change (<1)	
Change in weight (kg)	-2.1 ± 2.9	0.18 ± 2.0	0.001
Change in BMI (kg/m2)	-0.7 ± 1.0	0.2 ± 0.9	
Change in percentage weight loss in past 6 months (kg)*	2.6 ± 3.4	-0.22 ± 3.3	0.003
Change Quality of life**	-15.0 ± 15.1	-2.5 ± 19.6	0.009

008 Cysteine Supplementation Normalizes Plasma Taurine Concentrations in Low Birth Weight Premature Infants Requiring Parenteral Nutrition Support. Michael C. Storm, Ph.D., The University of Tennessee Health Science Center, Memphis, TN; Richard A. Helms, Pharm.D., The University of Tennessee Health Science Center, Memphis, TN **Background**: Cysteine (CysH) is a conditionally essential amino acid (AA) in the infant and young child. Our previous work has shown that CysH supplementation in infants who require parenteral nutrition (PN) results in a more normal plasma sulfhydral amino acid profile. However, the relation between CysH dose during PN and resulting plasma amino acid levels has not been systematically examined. We recently demonstrated that CysH supplementation at 20,30, and 40 mg/g AA in PN resulted in sequentially increasing concentrations of plasma taurine (Tau) in children receiving home PN due to short bowel syndrome. Plasma Tau concentrations were within our normal range for children at CysH doses of 30 and 40 mg/g AA in PN. **Methods**: In the current study, we evaluated CysH doses of 0, 10, 20, and 40 mg/g AA in LBW premature infants who required PN. This was a double blinded, randomized trial to evaluate the resulting plasma concentrations of Tau, methionine (Met), and cystine (Cys). Eighteen infants with a gestational age 30-37 weeks and a post-natal age less than 4 weeks who required PN for at least two weeks were enrolled. Infants were excluded if they had an inborn genetic error or significant organ failure. Target PN dosage was 2.5 gAA/kg/d with a caloric goal (carbohydrate and fat) of 125 kcal/kg/d. All infants initially received PN with no supplemented CysH for three days. Infants then received one of six randomly assigned CysH dosing schedules. Each CysH dose was administered for three days. On the morning of the last day in each dosing interval (study days 3, 6, 9, and 12) blood was drawn between 8 and 10 AM and immediately processed for amino acid analysis. Samples were deproteinized with 5'-sulfosalicylic acid (40 mg/mL) and stored at -70° C until assay. All samples were analyzed upon completion of the last study period for that subject. Samples were analyzed on a Beckman 6300 AA Analyzer using a four buffer (lithium citrate) expanded physiological program. **Results**: The resulting plasma AA concentrations(nmoles/mL, mean / SEM) are:

CysH Dose (mg/g AA)	**0**	**10**	**20**	**40**
Plasma AA (mean of normal)				
Tau (83.9)	**41.2 / 5.47**	**54.8 / 5.22**	**64.8 / 4.27**	**76.1 / 9.60**
Cys (51.9)	**38.9 / 3.71**	**45.8 / 4.89**	**46.7 / 3.31**	**54.3 / 7.90**
Met (35.8)	**74.1 / 7.59**	**59.5 / 4.08**	**60.6 / 3.44**	**65.3 / 4.55**

Plasma Tau and Cys concentrations generally increased with CysH dose. Plasma Met was initially above our normal range and tended to fall with CysH dosing. All other AA were within our normal range. Plasma Tau was positively correlated to CysH dosing (r2=0.982). The increases in plasma Tau with CysH dose were similar to those we previously observed in our study of SBS patients on home PN. **Conclusion**: We conclude that CysH supplementation to PN in LBW infants results in normal plasma Tau

concentrations and is necessary in this population to produce a more normal plasma sulfhydral AA profile.

009 Fatty acid metabolism in malabsorption treated with TPN. Karen C. McCowen, MD, Beth Israel Deaconess Medical Center, Boston, MA; Pei-Ra Ling, MD, Beth Israel Deaconess Medical Center, Boston, MA; Justin A. Maykel, MD, Beth Israel Deaconess Medical Center, Boston, MA ; Mario Ollero, PhD, Beth Israel Deaconess Medical Center, Boston, MA; Bruce R. Bistrian, MD, Beth Israel Deaconess Medical Center, Boston, MA

Despite being well-nourished without essential fatty acid deficiency (EFAD), a marked increase in arachidonic acid (AA) to linoleic acid (LA) ratio was present in plasma phospholipid of home TPN patients. Substantial increases in soluble TNF receptor and IL6 were found in blood and urine Vs healthy controls. Our hypothesis is that AA is produced in excess from LA due to its presentation to the liver by parenteral rather than enteral route. Arachidonic acid excess may lead to a pro-inflammatory milieu. We therefore investigated fatty acid metabolism in a rodent model of malabsorption.

Sprague Dawley rats, ~300g, underwent laparotomy and 80% small bowel resection, (or sham surgery) and placement of jugular vein catheters. Rats (n=32, 16 sham, 16 short gut) were randomly assigned to TPN with lipid, or fat-free TPN, continued for 5 d. Only tap water was allowed orally. TPN contained amino acids, dextrose, and essential micronutrients at 200 kcal/kg/day. Rats randomized to lipid (n=8 per group) got 30% non-protein energy as fat, using 20% Intralipid. After 5 d, blood was drawn for plasma phospholipid fatty acid analysis.

After 5 d, weight loss was 36 ±18g in short gut, 28 ±9g in sham. Analysis of phospholipids demonstrated that while EFAD had not developed, 20:3n-9 (Mead acid) was relatively increased in fat-free TPN groups, as were other distal very long chain fatty acids (Table below). Both nutrition (TPN/lipid Vs fat-free TPN) and surgery type (sham Vs short gut) were significant in determining AA levels. Relatively elevated AA occurred in both groups of fat-free rats, suggesting increased Δ6 desaturase activity, as expected. In contrast, AA was lower (suggesting appropriately down-regulated Δ6 desaturase) in sham animals given TPN/fat, but NOT in short gut animals fed TPN/lipid. The ratio of LA/ AA was suggestive of lower turnover of LA in sham rats given lipid compared with the other groups.

These results suggest that IV lipid was not appropriately sensed in the short gut rat, analogous to our human patients. The short gut rat may have a heightened desaturase activity inappropriate with adequate delivery of essential fatty acid provided parenterally. Therefore, the short gut rat is an appropriate model to study further this vexing problem.

Serum phospholipid fatty acid profiles, mol%, mean ± SEM

Fatty acids	Short gut TPN/ lipid	Short gut Fat-free	Sham TPN/ lipid	Sham Fat free
C18:2w6 (LA)	14.8 + 1	14.6 + 0.9	17.9 + 1	14.8 + 1
C18:3w3*	0.08 + 0.01	0.03 + 0.01	0.1 + 0.01	0.01 + 0.01
C20:3w6*	0.4 + 0.06	0.7 + 0.06	0.6 + 0.06	0.7 + 0.06
C20:3w9*	0.09 + 0.02	0.2 + 0.02	0.1 + 0.02	0.2 + 0.02
C20:4w6 (AA)	20.1 + 1	19.6 + 0.9	17.8 + 1^	22 + 1
C20:5w3#	0.3 + 0.09	0.5 + 0.08	0.2 + 0.09	0.5 + 0.09
LA/ AA	0.8 + 0.1	0.8 + 0.09	1 + 0.1^^	0.7 + 0.1

*p<0.01, #p<0.05, fat free Vs lipid; ^p<0.05, ^^p = 0.057 Vs other 3 groups;
All statistics by 2 way ANOVA

010 Accuracy of methods to estimate ionized and "corrected" serum calcium concentrations in critically ill multiple trauma patients receiving specialized nutrition support. Roland N. Dickerson, Pharm.D., University of Tennessee, Memphis, TN; Kathryn L. Holliday, MS, RD, Regional Medical Center, Memphis, TN; Angelina C. Tidwell, PharmD, University of Tennessee, Memphis, TN; Shanna K. Chennault, PharmD, Regional Medical Center, Memphis, TN; Martin A. Croce, MD, University of Tennessee, Memphis, TN; Gayle Minard, MD, University of Tennessee, Memphis, TN; Rex O. Brown, PharmD, University of Tennessee, Memphis, TN

The accuracy of twenty-three published methods to estimate serum ionized calcium (iCa) and "corrected" total serum calcium (totCa) concentrations in critically-ill, multiple trauma patients was evaluated. Eight of these formulas estimated iCa and fifteen were directed towards predicting a "corrected" totCa. Forty adult patients (29M/11F, 46 ± 18 yrs of age, APACHE II 15.3 ± 6.7, 14 patients with head injury and 35 with mechanical ventilation) admitted to the trauma intensive care unit who received specialized nutrition support were recruited for study. Patients who received blood products, intravenous calcium or therapeutic doses of heparin within 24 hours prior to the laboratory measurements, had a history of cancer, bone disease, parathyroid disease, hyperphosphatemia (≥ 6 mg/dl), or renal failure requiring dialysis were excluded from study entry. Serum chemistries, arterial blood gas measurements, and ionized calcium concentrations were simultaneously collected from each patient.

Patients were studied 5.8 ± 4.3 days post-injury and were receiving enteral nutrition (n=35), parenteral nutrition (n=4) or both (n=1). None of the patients had hypomagnesemia (serum Mg ≤ 1.5 mEq/L). Eight patients (20%) were hypocalcemic (iCa ≤ 1.12 mmol/L) and 3 (7.5%) were hypercalcemic (iCa ≥ 1.32 mmol/L). Serum totCa correlated modestly with iCa (r^2 = 0.207, p<0.01.). No significant differences were found for iCa for patients with sepsis (n=23) versus without sepsis (n=17; 1.18 ± 0.07 versus 1.21 ± 0.09 mmol/L, p=N.S., respectively), those who received intravenous lipid emulsion/propofol (n=15) versus no lipid/propofol (n=25; 1.19 ± 0.08 versus 1.19 ± 0.07 mmol/L, p=N.S., respectively) or between those with a low (< 7.35), normal, or increased (> 7.45) arterial pH (1.20 ± 0.06 versus 1.20 ± 0.08 versus 1.18 ± 0.08 mmol/L, p=N.S., respectively). However, those with a serum albumin ≤ 2 g/dL (n=18) had a lower ionized calcium concentration than those (n=12) with an albumin of > 2 g/dL (1.16 ± 0.04 versus 1.22 ± 0.08 mmol/L, p<0.01, respectively).