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ORIGINAL PAPER

Caloric intake and weight gain in a neonatal intensive care unit

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Abstract The aim of this paper was to study the weight gain in very-low-birthweight (VLBW) infants by adopting earlier and higher intake of proteins and earlier intake of lipids. We studied 28 VLBW infants admitted to Neonatal Intensive Care Unit during the year 2004 (group 1) and 18 during the first semester of 2006 (group 2). Dietary intakes for group 1 were: 1 g kg^{-1} day^{-1} of proteins started at postnatal day 2 (P2) and 0.5–1 g kg^{-1} day^{-1} of lipids at P3; for group 2, 1–1.5 g kg^{-1} day^{-1} of proteins and 0.5–1 g kg^{-1} day^{-1} of lipids, both started at P1. Caloric intake was significantly higher in group 2 ($p<0.05$), whereas cumulative nutritional deficit was higher in group 1 ($p \leq 0.01$). Weight *z* scores were significantly lower at discharge comparing with *z* scores at birth for each group ($p \leq 0.01$), with no differences between the two groups. Despite a higher protein intake which resulted in a lower nutritional deficit, the weight *z* score did not improve significantly at discharge.

Keywords Caloric intake · Very-low-birthweight infants · Proteins · Lipids

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Introduction

The postnatal development of premature infants is critically dependent on an adequate nutritional intake that mimics a similar gestational stage to which the foetus would be exposed if still in the uterus [5].

After birth, preterm infants lose weight and take variable periods to regain birthweight.

Recommended intakes are commonly interrupted for clinical reasons, and very-low-birthweight (VLBW) infants develop major deficits in caloric intakes during initial hospital stay that are not recovered by the time of hospital discharge [5].

More aggressive total parenteral nutrition with higher energy and protein intakes might reduce the energy and protein deficit. However, early total parenteral nutrition is limited by glucose and lipid intolerance as well as by concerns regarding amino acid metabolism.

Deficient protein or amino acid administration over an extended period may cause significant growth delay or morbidity in VLBW infants [15]. Furthermore, there is evidence that amino acid and lipid solutions can be tolerated by VLBW infants during the first days of postnatal life, although nutritional requirements at this time remain uncertain [15].

More aggressive enteral feeding might also reduce the caloric deficit. Nevertheless, whether earlier introduction and more rapid advancement in enteral volumes is achievable without adverse effects is not clear [10].

We performed a retrospective/prospective study to determine adequate nutritional intakes in very-low-birthweight infants for postnatal catch-up growth assessment and examined the effect of earlier and higher intakes of proteins and of earlier intake of lipids on weight gain.

Materials and methods

Sample

The sample consists of infants with birthweight <1,500 g on admission to a level III Neonatal Intensive Care Unit from Fernando Fonseca's Hospital in Portugal.

Two groups were selected: Group 1 included VLBW infants who were born during the year 2004 (n=35), and group 2 included VLBW infants born during the first semester of 2006 (n=28).

Seventeen VLBW infants were excluded from the study, as six died during hospitalisation, one was transferred to other Hospital, and ten had missing data.

The remaining 46 infants are the sample of this report (group1 with 28 VLBW infants and group 2 with 18 VLBW infants).

Diets

Parenteral feeding

Two different caloric protocols were adopted. Group 1 received 1 g kg^{-1} day^{-1} of proteins started at postnatal day 2 (P2) and 0.5–1 g kg^{-1} day^{-1} of lipids at P3.

Group 2 received 1–1.5 g kg^{-1} day^{-1} of proteins and 0.5–1 g kg^{-1} day^{-1} of lipids started at P1.

Protein and lipid intakes had a daily increase of 0.5 g/kg until 3–3.5 g/kg maximum.

On the first postnatal day, all infants had glucose intake of 3–5 mg kg^{-1} min^{-1} until gradually reaching a maximum of 13 mg kg^{-1} min^{-1}, depending on the blood glucose value (80–150 mg/dl).

Electrolytes were started at P3 and adjusted according to infant needs during parenteral feeding administration. Sodium intake (N) of 4–8 meq kg^{-1} day^{-1} was administered to infants who weighed less than 1,000 g, whilst those weighing 1,000–1,500 g were given a 3–5-meq kg^{-1} day^{-1} dose. Potassium intake (K) of 1–2 meq kg^{-1} day^{-1} and chloride intake of 3–5 meq kg^{-1} day^{-1} were administered to all VLBW infants.

In addition, infants were also provided with 35–60 mg kg^{-1} day^{-1} calcium, 20–35 mg kg^{-1} day^{-1} phosphorus (P) and 0.3–0.4 meq kg^{-1} day^{-1} magnesium intakes.

Enteral feeding

Protocol guidelines determined that the infant should start with mother's milk and/or preterm formula when clinically stable. Term formula was subsequently introduced when the infant reached 38 weeks or more of gestational age or 2 weeks before discharge when the family had low socioeconomical resources. Enteral feeding was stopped temporarily whenever gastric residuals reached more than 50% of the volume of the previous feed; there were bilious residuals or any suspicion of necrotising enterocolitis.

Data collection

Data were collected retrospectively from 28 infant charts for group 1 and prospectively from 18 infant charts for group 2.

Gestational age was calculated from the date of the last menstrual period or foetal ultrasound assessment.

Weight was measured by nurses using an electronic scale, daily during the first week and once a week thereafter.

Daily measurement of caloric intake for the parenteral nutrition valued 1 g of glucose and protein to contain 4 kcal and 1 g of lipid to be equivalent to 9 kcal. Caloric intake from each 100 ml of milk was calculated as amounting to 75 kcal for mother's milk [10], 81 kcal for preterm formula 13% and 69 kcal for term formula 13%.

Caloric deficit was determined by ascertaining the difference between the actual caloric intake and a recommended caloric dietary intake of 120 kcal kg^{-1} day^{-1}. The cumulative deficit was obtained by the sum of the daily caloric deficit.

In order to attend to the heterogeneity that characterises the obstetric population and its socio-demographic, anthropometric and nutritional variables [4], we used the distribution of anthropometric values of our Neonatal Intensive Care Unit (NICU) population published by Cunha et al. [6] to calculate weight z score of preterm infants. Weight z score was obtained by determining the difference between the mean weight for gestational age of our NICU population [6] and each infant's weight (at birth or discharge) divided by standard deviation.

The comorbidities analysed for each group included mechanical ventilation, sepsis, intrauterine growth restriction, necrotising enterocolitis equal or higher than grade IIA, patent ducts arteriosus (PDA), bronchopulmonary dysplasia (BPD), intraventricular haemorrhage (IVH), retinopathy of prematurity (ROP) and gastroesophageal reflux.

Neonatal Therapeutic Intervention Scoring System (NTISS) was analysed during 24 h and later quantified according to a scale that measures therapy points. These include respiratory and cardiovascular as well as metabolic and nutritional parameters, drug therapy, monitoring, transfusion, procedural and vascular access.

Clinical Risk Index for Babies (CRIB) was calculated based on data collected within the first 12 h after birth and is composed of six variables: birthweight, gestational age, maximum and minimum fraction of inspired oxygen (FiO_2), congenital malformations, and base excess, each scored according to severity.

Statistical analysis

Data were analysed using SPSS 13.0 programme (Statistical Package for the Social Sciences, USA), and p values <0.05 were considered statistically significant.

Demographic sample data were presented by mean and standard error and median (minimum–maximum).

Student's t test was used for normally distributed continuous variables and Mann–Whitney test whenever normality failed. Categorical data were analysed using the chi-square test.

Comorbidities between the two groups were compared using the chi-square test. Caloric intake correlations were determined by bivariate Pearson correlation (R=maximum value of correlation).

T test for paired samples was used for comparing weight z score at discharge and at birth.

T test for independent samples was used to compare z scores between the two groups.

Results

Demographic characteristics, comorbidities and feeding aspects of both groups are presented in Table 1.

There were no statistically significant differences in either gender, mean gestational age or mean birthweight between the two groups. In addition, NTISS and CRIB scores, hospitalisation time, duration of parenteral nutrition, start of enteral feeds and time required to achieve full enteral feeds (FEFs) in premature infants were not statistically different between the two groups.

Despite the intention to start protein and lipid intakes earlier in group 2, this was only achieved for protein intake (1.7±0.8 vs 3.1±0.3 days, p=0.00) and not for lipids (p= 0.15; Table 1).

There were no statistically significant differences in comorbidities between the two groups (Table 2).

Protein intake was higher for group 2 during the first 12 postnatal days and similar to group 1 thereafter (Fig. 1).

Group 2 presented a higher caloric intake during the first 22 days of postnatal life (Fig. 1).

Caloric intake was significantly higher in group 2 on P1 and P7 (group 2 vs group 1 mean caloric intake of 30.4 vs 18.6 kcal/kg for P1 and 98.6 vs 76.0 kcal/kg for P2, $p<$ 0,05). Whereas daily caloric deficit was similar in both groups, the cumulative caloric deficit was higher in group 1 and that disparity increased during hospital stay. The difference between groups was statistically significant on P1, P7 and P15 (group 1 vs group 2 mean cumulative deficit of −85.9 vs −79.9 kcal/kg for P1, −428.21 vs −367.8 kcal/kg for P7, −641.1 vs −513.88 kcal/kg for P15, $p\leq$0.01; Fig. 1).

Birthweight z scores did not differ between the two groups, with both presenting significantly lower weight z scores at discharge than at birth ($p\leq$0.01; Fig. 2).

Although group 2 was provided with a higher protein intake which resulted in a lower caloric deficit, neither did weight z score significantly improve at discharge nor were early caloric deficits regained before hospital discharge (Fig. 1).

We also found that factors such as delayed lipid intake and FEF acquisition, longer periods of enteral feeding interruption, full parenteral nutrition and mechanical ventilation all negatively correlated with caloric intake in both groups (p=0.01, n=46; Table 3).

Furthermore, there was no statistically significant correlation between caloric intake and the start of enteral feeds (n=46; Table 3).

However, we detected a significant positive correlation between caloric intake and gestational age (R=0.66, p= 0.01, n=46; Table 3). In fact, when comparing VLBW infants below 29 weeks of gestational age (GA) with those above 30 weeks GA, we noticed that those of GA< 29 weeks not only had longer hospital stay (mean 75.2 days for GA≤29 weeks and 40.3 days for GA≥30 weeks, p= 0.00) but also started total enteric nutrition at a later stage (26.4 vs 16.3 days, p=0,00), had lower caloric intakes at P1 (19.4 vs 27.7, p=0.029), P7 (72.8 vs 99.1, p=0.008), P15 (86.4 vs 146.1, p=0.000) and P22 (98 vs 144.2, p=0.002). Cumulative deficit was higher among those of less GA measured on P22 (−815 vs −627, p=0.019).

During hospital stay, 97.8% of the VLBW infants received predominantly preterm formula (with or without mother's milk), 78% received predominantly mother's milk (exclusively or with preterm/term formula), and 39.1% were fed predominantly term formula (with or without mother's milk). It was observed that only the preterm formula positively correlated with caloric intake (p=0.01) at postnatal days 1, 7, 15 and 22.

Discussion

The effect of early malnutrition on infant growth and development, including long-term consequences to the central nervous system, is well recognised. Indeed, protein and caloric undernutrition results in decreased cell division and myelination in the developing brain with potentially irreversible consequences to cognitive, motor and behavioural development [15].

Difficulties in reaching the recommended intakes are intimately associated with a number of factors, including the need for fluid restriction, intolerance to standard glucose infusions, periods of lipid-free feeding and imma-

Table 1 Demographic, comorbidities and feeding characteristics of group 1 and group 2

VLBW sample data	Group 1 (2004) *N*=28	Group 2 (2006) *N*=28	*p* value
Gender	13 male (46%)	9 male (50%)	0.81[c]
Mean gestational age	29.1 ± 0.6 weeks[a]	29.8 ± 0.7 weeks[a]	0.40[d]
Mean birthweight	1,141.6 ± 50 g[a]	1,245.4 ± 61.7g[a]	0.20[e]
NTISS	14.5 (9–30)[b]	16 (7–22)[b]	0.55[d]
CRIB	1.5 (0–12)[b]	1 (0–10)[b]	0.54[e]
Hospitalisation	62 ± 5 days[a]	56 ± 7 days[a]	0.36[e]
Total parenteral nutrition	25 ± 3 days[a]	22 ± 4 days[a]	0.39[e]
Corticotherapy	21 infants (75%)	17 infants (94%)	0.12[c]
Enteral feeds/Full enteral	3 ± 0.2/23 ± 3 days[a]	3 ± 0.2/19 ± 2 days[a]	0.70/0.32[e]
Beginning of protein intake	3.1 ± 0.3 days[a]	1.7 ± 0.3 days[a]	0.00[e]
Beginning of lipid intake	5.3 ± 0.5 days[a]	4.4 ± 0.8 days[a]	0.15[e]

Group 1 included 28 VLBW infants born during the year 2004 and group 2 18 VLBW infants born during the first semester of 2006

VLBW very-low birthweight, *NTISS* Neonatal Therapeutic Intervention Scoring System, *CRIB* Clinical Risk Index for Babies

[a] Mean and standard error

[b] Median (minimum–maximum)

[c] Chi-square test

[d] *T* test

[e] Mann–Whitney test

turity of intestinal functions that cause frequent interruptions in the enteral feeding [8].

In the second and beginning of the third trimesters of gestation, the estimated placental transfer is 8 to 10 mg kg^{-1} min^{-1} of glucose and 3.6 to 4.8 g kg^{-1} day^{-1} of amino acids [8].

Sauer [20] defends that there can be no comparison between the sterile and temperature-controlled foetal environment in the uterus with that of the preterm infants. Instead, after birth, infants have to regulate their own temperature and defend themselves against infections. Indeed, preterm infants have higher fat accretion in order to improve thermoregulation and increase body energy stores [20]. Therefore, we can speculate that preterm infants might benefit from an even higher score of energy intake when comparing to foetal growth.

Lipids play a fundamental role in providing the necessary dietary energy density to achieve positive energy balance, allowing optimal utilisation of dietary protein for tissue growth and supplying the essential *n*−6 and *n*−3 fatty acids. Failure to provide an adequate lipid intake is likely to result in suboptimal energy intakes, which can significantly contribute to caloric undernutrition and proteolysis [15].

In our study, and despite the lack of a significant difference in lipid introduction between the two groups, we observed a significant positive correlation between earlier lipid introduction and higher caloric intake (n=46, p=0.01 for P7 and p=0.05 for P1, P15, P22; Table 3). Notwithstanding protocol recommendations for group 2 of starting lipids on P1, there was a delay to P4 (mean time). This adjournment might be explained on the basis of clinical debilitations of the infant and neonatologists' cautions regarding adverse clinical outcome. In fact, many authors, such as Dinerstein et al. [8] and Ibrahim et al. [15] argue that an aggressive introduction of earlier proteins and lipids does not increase the incidence of an adverse clinical sequelae.

In group 2, earlier intake of proteins proved insufficient to achieve adequate weight gain. The recommendations regarding the start of protein intake were also not fully accomplished, with a mean time of 1.7 postnatal days, instead of P1. Furthermore, group 2 received a higher protein intake, which translated into higher caloric intake

Table 2 Statistical comparison of comorbidities between group 1 and group 2

Comorbidities	Group 1	Group 2	*p* value[a]
Intrauterine growth retardation	9 (32%)	2 (11%)	0.16
Sepsis	13 (46%)	10 (56%)	0.76
Mechanical ventilation	15 (54%)	11 (61%)	0.76
Bronchopulmonary dysplasia	4 (14%)	2 (11%)	1
Necrotising enterocolitis≥IIA	0 (0%)	1 (5,5%)	0.39
Gastroesophageal reflux	3 (11%)	4 (22%)	0.40

[a] Chi-square test was used. p value<0.05 for statistically significant differences

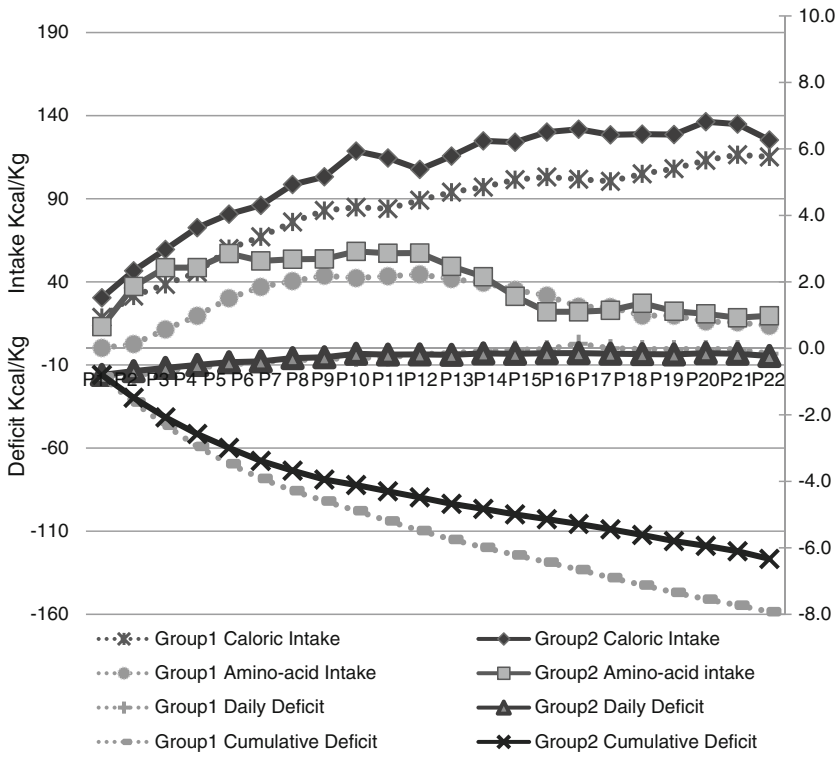

Fig. 1 Daily evaluation of both caloric and amino acid intakes and daily and cumulative caloric deficits. *Marked dotted lines* for group 1 and *continuous lines* for group 2. *X*-axis refers to daily evolution during the first 22 days of postnatal life. *Left*, *y*-axis: positive values refer to calorie intake (kcal/kg), negative values refer to caloric deficit (kcal kg^{-1} day^{-1}) and cumulative deficit (kcal kg^{-1} day^{-1}). *Right*, *y*-axis represents protein intake (g/kg)

(Fig. 1). However, despite a lower caloric deficit, the weight *z* score did not significantly improve at discharge in this group. This finding seems to be associated with a failure to meet minimal nutritional requirements that led to significant early caloric and protein deficits which were not regained before hospital discharge [2, 8, 11]. This conclusion is in agreement with Griffin [14] and Ibrahim et al.

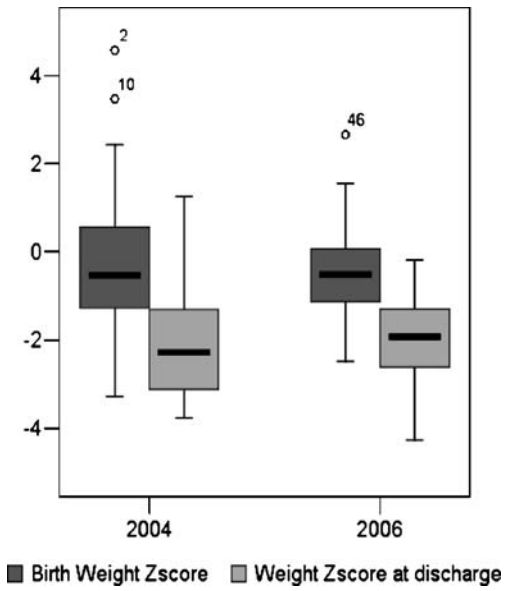

Fig. 2 Changes in *z* score at birth and at discharge for group 1 (2004) and group 2 (2006). Weight *z* score was calculated based on the weight distribution of our NICU population [6]

[15] recommendations for a higher intake of proteins and lipids (3.5 g kg^{-1} day^{-1} of proteins and 3 g kg^{-1} day^{-1} of lipids) to be started immediately after birth (2 h). These authors also endorse higher glucose concentrations, as well as the use of early low-volume trophic feeds and a more aggressive management of hyperglycaemia. In fact, higher protein and lipid intakes cannot only be tolerated without metabolic or respiratory complications but also prevent the development of negative nitrogen balance seen with the conventional management of delivering glucose alone. These advantages not only help reduce the maximum weight loss, thus accelerating the regaining of birthweight, but are also effective in improving growth.

In our study, caloric intake increased during the first 2 weeks in both groups, and the same pattern occurred with the cumulative deficit, against initial expectations. This might be explained by recommended dietary intakes of 120 kcal kg^{-1} day^{-1}, which are, according to Bauer et al. [1], Embleton et al. [10] and Kashyap [16], the optimal nutritional values from the first day of postnatal life.

Initial weight loss occurs in the first week and is followed by a positive weight change until maximum growth velocity is reached approximately 3 weeks later. This observation points to a more critical increase in caloric intake during the first 2 weeks after birth in order to fulfil the requirements for higher growth velocity [2]. We also observed that after the first 2 weeks, the increase of caloric intake stopped and the intake of proteins started to decrease, whereas cumulative deficit continued to increase

Table 3 Statistical correlations of caloric intake with diverse variables concerning both groups (n=46)

Caloric intake correlations (N=46)	R[a]	p value[b]
Gestational age	+0.66	0.01 (P1, P7, P15, P22)
Parenteral nutrition total time	−0.53	0.01 (P1, P7, P22) 0,05 (P15)
First day of lipid intake	−0.42	0.01 (P7) 0,05 (P1, P15, P22)
Total time of enteral feeding interruption	−0.50	0.01 (P1, P7, P15) 0,05 (P22)
First day of full enteral feeds	−0.55	0.01 (P7, P15, P22) 0,05 (P1)
Time of mechanical ventilation	−0.52	0.01 (P7, P15, P22) 0,05 (P1)
First day of enteral feeds	−0.25	>0.05 (P1, P7, P15, P22)

P1 postnatal day 1, *P7* postnatal day 7, *P15* postnatal day 15, *P22* postnatal day 22

[a] R refers to maximum value of bivariate Pearson correlation obtained

[b] p value<0.05 for statistically significant differences

during hospital stay. Since the enteral feeding protocol was the same for both groups, the global worsening of protein/caloric ratio during hospital stay might be closely related to enteral feeding methods. Indeed, a negative correlation was noted, for both groups, between caloric intake and later acquisition of FEF. Longer periods of enteral feeding interruption and of full parenteral nutrition were also found to significantly correlate with lower caloric intakes. In fact, these variables might have been responsible for the lower weight z scores noted in both groups at discharge, recalling the observations of Enrenkranz et al. [9] and Rocha et al. [19]. Different results were obtained in another study, conducted by Gallini et al., in which a group of VLBW infants fed through the parenteral nutrition route during the first 2 weeks of postnatal life had higher caloric intake than the one provided with full enteral feeds. Gallini et al. [12] also observed that full enteral feeding was achieved later in the parenteral nutrition group.

Due to illnesses and immature organ function, initial nutritional support is usually provided via the parenteral route [3]. Enteral feedings are introduced cautiously and often with the sole initial intent of preventing atrophy of the gut (trophic feedings) [3]. Our study reiterates the benefits of enteral over parenteral feeding methods. Diet protocols should be revised to include specific instructions to implement earlier and prolonged enteral feeds, thus preventing long periods of full parenteral nutrition and reducing the caloric deficit in VLBW infants.

We also observed that higher caloric intake was achieved with preterm formula administration instead of human milk or term formula. This finding is congruent with that of Goudoever et al. [13] who demonstrated that infants fed with formula containing lower energy (as human milk) had a markedly lower fat accumulation rate, even lower than intrauterine accretion rates. Nevertheless, more infants were fed simultaneously with both preterm formula and human milk, which could have affected our results.

Kashyap [16] argues that infants fed with fortified human milk gain weight and increase length and head circumference at a rate similar to intrauterine rates. In fact, breast milk confers nutritional, immunological and psychosocial advantages that can overwhelm preterm formulas [17].

As observed in our study, weight loss was more pronounced in the most immature infants [18]. Indeed, the more immature the infant, the more comorbidities and the greater the need for higher protein/energy ratio [16].

Some studies report that therapies employed in the management of VLBW infants may affect nutrient excretion and retention and that dexamethasone exerts the most striking effects on nutrient balance (N, P and K retention affected adversely) [21]. In our study, however, there was no statistically significant difference between the two groups regarding prenatal steroids administration.

As previously reported in other studies, [7, 8, 15], we also did not find any association between a higher incidence of comorbidities such as ROP, IVH, PDA, sepsis or BPD and the administration of higher and earlier lipid and amino acid caloric intakes in group 2. Hence, higher nutrient intake was not responsible for a worst clinical outcome.

In conclusion, our study demonstrated that all VLBW infants accrued a significant cumulative deficit during hospital stay that was not regained before discharge. These low caloric intakes might be explained by a delayed introduction of lipids, a late acquisition of full enteral feeding, as well as by longer periods of enteral feeding interruption and mechanical ventilation. On the other hand, an earlier increase of protein load led to lower caloric deficits, although this still proven insufficient to achieve adequate weight gain at discharge.

Ultimately, more studies are needed in order to better define the caloric needs of VLBW infants and decrease their postnatal undernourishment during earlier developmental stages.

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