Appetite xxx (2010) xxx–xxx

Contents lists available at ScienceDirect

Appetite

journal homepage: www.elsevier.com/locate/appet

Research report

Energy compensation in enterally fed children

Laura Kane [a], Charlotte Wright [a,*], Wan Fathin Fariza [a], Marion Hetherington [b]

[a] Paediatric Epidemiology and Community Health (PEACH), Faculty of Medicine, University of Glasgow, QMH Tower, Yorkhill Hospitals, Glasgow G3 8SJ, UK
[b] Institute of Psychological Sciences, University of Leeds, Leeds LS2 9JT, UK

ARTICLE INFO

Article history:
Received 9 June 2010
Received in revised form 1 November 2010
Accepted 3 November 2010

Keywords:
Energy compensation
Energy intake
Enteral feeding
Preload
Satiety

ABSTRACT

Limited exposure to solid food in early childhood may affect the development of appetite regulation. We used formal satiation studies to assess energy compensation in children who have been artificially fed. Subjects were 11 children, median age 4.5 years (range 1–10) who were formerly ($n = 4$) or currently ($n = 5$) mainly tube fed or supplement fed ($n = 2$), with a range of surgical or neurodevelopmental problems. On 2 separate days a high-energy preload (HEP) and low-energy preload (LEP) drink were given followed by a multi-item test lunch. A compensation index (COMPX) score was derived as follows: COMPX (%) = $[(\text{Meal}_{\text{lep}} - \text{Meal}_{\text{hep}})/(\text{Preload}_{\text{hep}} - \text{Preload}_{\text{lep}})] \times 100$. The median (range) COMPX of the participants was 70% (−73% to 178%). The 8 boys tended to compensate more (median 99%) than the 3 girls (30%; P Mann–Whitney = 0.1), but there was no clear association of compensation with age. Although a small preliminary study, this suggests that children who have been artificially fed demonstrate energy compensation comparable to that of normally fed children.

Introduction

Enteral feeding, via gastrostomy or nasogastic tube provides vital nutritional support for neonates and infants with major medical or surgical problems who are unable to meet their dietary requirements through normal oral intake alone. However, when the acute medical issues preventing feeding have resolved, some infants seem unable to make the transition to oral feeding and in extreme circumstances may remain dependent on enteral feeding for years, despite being apparently capable of eating (Mason, Harris, & Blissett, 2005). Children in these circumstances often appear to have no interest in food and may lose weight rapidly when their feeds are reduced (Wright, Smith, & Morrison, 2010). This leads to the suggestion that such children may have disordered appetite and energy regulation, either as a result of their underlying condition or because of the disruptive effect of long-term tube feeding. All individuals need to be intrinsically capable of regulating their nutrient intake to maintain energy balance. Healthy children have been shown to demonstrate energy self-regulation both in the short term, adjusting energy intake in response to food or drink consumed before a meal (Birch & Deysher, 1985, 1986; Birch, McPhee, Bryant, & Johnson, 1993; Birch, McPhee, & Sullivan, 1989; Hetherington, Wood, & Lyburn, 2000; Shea, Stein, Basch, Contento, & Zybert, 1992) and the longer term, over 24 h (Birch, Johnson, Andresen, Peters, & Schulte, 1991).

Satiety is a complex process governed by negative feedback resulting from gastric and duodenal distension, release of peripheral appetite suppressing (anorexigenic) signals, such as leptin, and stimulation of the central suppressive leptin–melanocortin pathway, which communicates with the paraventricular nucleus or "satiety centre" (Druce & Bloom, 2006). Satiety is also dependent upon inhibition of positive feedback from the appetite-stimulating (orexigenic) pathway, including orosensory stimulation (Druce & Bloom, 2006). Meal termination ensues when opposing positive and negative afferent feedback signals are of equal force (Norton, Anderson, & Hetherington, 2006). However, psychological food motivation and environmental stimuli may override physiological regulation of food intake (Bellisle & Dalix, 2001; Blass et al., 2006; De Castro, 1994; Hetherington, Anderson, Norton, & Newson, 2006; Lumeng & Hillman, 2007; Mrdjenovic & Levitsky, 2005; Norton et al., 2006; Shide & Rolls, 1991).

Artificial feeding could affect both physiological appetite regulation and eating behaviour in many ways. Tube-feeding bypasses part of the upper gastrointestinal tract, so that long-term enterally fed children may lack innate somatic satiety cues. The child may also have missed a critical period of associative learning (Birch, 1998; Hetherington, 2002; Rolls, Engell, & Birch, 2000) and have disordered daily eating patterns (Poustie et al., 2006). Other studies have suggested that both the age at which tube feeding started (Mason et al., 2005) and when reduction in feeds began (Wright et al., 2010) may critically affect the ease of transition.

* Corresponding author.
E-mail address: cmw7a@clinmed.gla.ac.uk (C. Wright).

0195-6663/$ – see front matter © 2010 Elsevier Ltd. All rights reserved.
doi:10.1016/j.appet.2010.11.002

The effectiveness of appetite regulation can be assessed objectively, by measuring short term *energy compensation*. A preloading experimental model was first used in adults by Booth, Campbell, and Chase (1970), who demonstrated that consuming a glucose "preload" prompted a reduction in energy intake during a subsequent test meal, with the reduction approximately equal to that of the preload energy content. This model has since been developed and used extensively in children (Birch & Deysher, 1985, 1986; Birch et al., 1989). The Birch model compares how children adjust their energy consumption, during two standardised *ad libitum* test meals, eaten 30 min after a solid or liquid snack ("*preload*"), which varies only in energy content: high energy in one condition, low in the other. This model has been widely tested in healthy children, particularly in relation to obesity risk (Cecil, Palmer, & Wrieden, 2005; Johnson & Birch, 1994; Johnson & Taylor-Holloway, 2006; Mrdjenovic & Levitsky, 2005). On average, after a high energy preload, infants and preschool children reduce their meal intake by between 50% and 100% (Birch & Deysher, 1986; Johnson & Birch, 1994) of the energy content of that preload, but older children compensate much less (Cecil et al., 2005; Johnson & Taylor-Holloway, 2006). Only one previous study has examined energy regulation in a clinical group; this study (Kasese-Hara, Wright, & Drewett, 2002) found that weight-faltering children seemed not to compensate; they ate the same, smaller amount after both high and low energy preload. No studies have yet formally assessed energy regulation in enterally fed children.

The aim of this study was to establish if children who are currently or formerly enterally fed demonstrate energy compensation in laboratory-based satiation studies. We predicted that artificially fed children would demonstrate poor energy compensation, because of limited exposure to solid food in early life.

Methodology

Participants

Children were recruited from the feeding clinic at the Royal Hospital for Sick Children, Glasgow, set up specifically to assist the withdrawal of children from artificial feeding (Wright et al., 2010). The hospital provides tertiary care for the West of Scotland and manages a range of severe medical and surgical conditions. Most of the children referred have survived major neonatal health problems and many remain chronically ill or impaired. Children attending the clinic at the time of the study (June 2006–March 2007) were invited to take part if they were aged between 1 and 10 years, were being or had been enterally fed and were able to take at least some food orally. Ethics approval for this study was granted by Yorkhill Local Research Ethics Committee. Written informed consent was gained from the parents before commencing the first satiation study.

Procedure

The studies were conducted by 2 research students (LK and WF) between July 2006 and March 2007. Parents of eligible children were sent a letter of invitation and an information sheet, followed up by a telephone call. Parents who agreed to take part were given a menu from which they selected a number of foods that their child was likely to eat at the test meals. Each participant then attended two lunchtime sessions roughly one week apart, at the Laboratory for Human Ingestive Behaviour, Glasgow Caledonian University. Parents were asked to fast their children for at least 2 h prior to each study, and researchers assessed compliance by informally asking participants what they had eaten that day.

Before each meal, the participant was presented with a high-energy preload (HEP) drink or low-energy preload (LEP) drink, which they were encouraged to finish. Children who were unable to drink the whole preload and who were tube fed, received the drink via their nasogastric or gastrostomy tube. Participants received the preloads in a random order and the child and family were not told which was which. The preloads of choice were based on a blackcurrant-flavoured soft drink ('*Ribena*'®) and prepared as follows:

HEP (200 ml = 506 kJ) 50 ml blackcurrant '*Ribena*'® mixed with 130 ml bottled still water and 20 g of maltodextrin ('*Maxijul*'®).
LEP (200 ml = 1.25 kJ): 50 ml blackcurrant '*Really Light Ribena*'® mixed with 150 ml bottled still water.

Where participants would not take the standard '*Ribena*'® preloads, alternatives were offered (Table 2). Where possible the energy content of the alternative preloads was then matched to that of the standard '*Ribena*'® preloads (506 kJ and 1.25 kJ), so that if necessary the preload volume varied.

Thirty minutes after the children consumed the preload, they were presented with their pre-selected multi-item lunch, served on a tray. Participants ate the meal *ad libitum*, with normal parental feeding assistance or encouragement (such as spoon feeding, cutting up or handing food, depending on developmental stage) and the meal ended when children demonstrated signs of satiation as determined by the parents, such as stopping eating, refusing to be fed further or saying that they were full. Again this was dependent on developmental stage. Participants were offered the same selection of foods on both occasions.

The preload volume consumed was recorded. Each item was weighed before and after the meal using digital scales (SECA). Energy intake for all items was calculated using energy values supplied by the food manufacturer. Efforts were made to take spillage into account.

Analysis

The primary outcome measure was the compensation index (COMPX) score, derived from the various energy intakes as follows:

$$\text{COMPX}\,(\%) = \left[\frac{(\text{Meal}_{\text{lep}} - \text{Meal}_{\text{hep}})}{(\text{Preload}_{\text{hep}} - \text{Preload}_{\text{lep}})}\right] \times 100$$

The COMPX (Birch & Deysher, 1985) reflects the precision of energy compensation. 100% represents perfect calorie for calorie compensation.

Because of the small numbers it was often unclear whether the data were normally distributed, so the Mann–Whitney *U* tests were used to compare participant subgroups. The differences in total meal energy intake within individuals were normally distributed, so a one sample *t*-test was used for these.

Results

Twenty-four children were eligible and invited to take part in the study. Of these, eight families declined consent and two children (twins) were out of the country. Three families wished to take part, but were unable to complete one or both stages due to illness or other competing commitments. Thus 11 children (3 girls, 8 boys) completed both sessions. All had been enterally fed for between 6 months and 6.6 years because of a range of medical and surgical conditions (see Table 1) but 5 had already stopped, 1–23 months previously.

The individual and median energy intakes are shown in Table 2. Alternative preloads were needed for 4 participants, two of which were of lower volume (80 and 120 mls) in each condition, and two of lower volume (100 and 110 mls) for the LEP only. Only about half

Please cite this article in press as: Kane, L., et al. Energy compensation in enterally fed children. *Appetite* (2010), doi:10.1016/j.appet.2010.11.002

Table 1
Clinical, demographic and functional characteristics of participants.

Subject	Sex	Age (yrs)	Height *Z* score	Weight *Z* score	BMI *Z* score	Enteral feeding method and duration	Clinical history	Functional capacity
1[FM] JD	M	2.5	−0.48	−0.78	−1.56	Gastrostomy/NG fed since age 2 m	Diaphragmatic hernia	Normal development for age
2[LK] EH	F	3.6	−2.79	−2.47	−0.56	Gastrostomy /NG fed since birth	Goldenhar syndrome, visual impairment (right eye), gastrostomy and fundoplication	Normal cognition and development for age
3[LK] AC	M	4.5	−4.06	−3.69	−0.47	Gastrostomy fed since age 3.5 yrs	Complex cardiac disease, pulmonary hypertension (home oxygen and nitric-oxide)	Normal cognition and development for age
4[LK] LF	M	4.9	−1.15	1.02	2.43	Gastrostomy/NG fed since birth	Preterm (27 wks), IUGR, gastrostomy and fundoplication, chronic lung disease, sensory integration disorder	Normal development for age, speech delay
5[FM] KF	F	1.9	−4.04	−3.78	−1.01	NG fed since birth	Downs Syndrome: duodenal atresia, Hirschsprung's disease, congenital cardiac anomaly	Global developmental delay, sitting unsupported, but unable to walk, finger feeds, unable to use cutlery, limited ability to chew
6[FM] DC	M	7.2	−2.36	−5.45	−5.6	Gastrostomy/NG fed from 3.5 m; stopped 2 months earlier	Preterm (31 wks), IUGR, Russell–Silver Syndrome	Normal development and cognition for age
7[LK] MS	F	9.8	*	−2.64	*	Gastrostomy fed from age 5.5–7.5 yrs; still dependant on sip feeds[a]	Cerebral palsy, quadriplegic	Global developmental delay, unable to sit unsupported or feed self, mum feeds with spoon
8[FM] BC	M	10.3	−1.36	−1.21	−0.54	Gastrostomy fed from age 2.5 to 8.5 yrs	Congenital adypsia (absence of thirst),	Mild learning difficulty
9[FM] RP	M	1.2	+0.56	−1.98	−3.42	NG fed from birth −8 m; ceased 4 months before study	Oesophageal atresia, trachea-oesophageal fistula, gastro-oesophageal reflux	Normal development for age
10[LK] DG	M	3.7	−2.27	0.09	2.30	NG fed from 1 m to 3.3 yrs; ceased 4 months before study	Complex congenital cardiac disease	Mild developmental delay
11[LK] CC	M	3.8	0.16	−0.84	−1.52	NG fed from birth to 6 m; still dependant on sip feeds[a]	Preterm (25 wks), chronic lung disease, visual deficit, gastro-oesophageal reflux	Global developmental delay, sits without support, unsteady on feet, no speech, unable to chew, mum feeds from spoon

[FM]: Study conducted by researcher F.M.
[LK]: Study conducted by researcher L.K.
* Unable to stand.
[a] High energy sip feeds.

Table 2
Details of (a) energy intake (kJ) during test meals and (b) preload volume.

Subject	LE pre-load	HE pre-load	LE meal intake	HE meal intake	Total intake at LE study	Total intake at HE study	Δ Meal intake (LE–HE)	COMPX (%)
(a) Energy intake during test meals after low energy (LE) and high energy (HE) preload								
1 = JD	1.25	506	178	0.00	179	506	178	35.2
2 = EH*	0.00	508	188	33.5	188	542	155	30.4
3 = AC*	0.00	628	432	8.37	432	636	424	67.5
4 = LF	1.26	425	872	138	873	563	734	173
5 = KF	1.26	506	684	1053	685	1559	-369	-73.1
6 = DC	1.26	506	975	563	976	1069	412	81.6
7 = MS*	0.42	497	1267	918	1267	1415	349	70.3
8 = BC	1.26	506	2128	2315	2129	2821	−187	−37.0
9 = RP	1.26	506	1163	343	1164	849	820	162
10 = DG	1.26	506	1472	887	1473	1393	585	116
11 = CC*	90.0	490	1123	413	1213	903	710	178
Median	1.26	506	975	413	976	903	412	70.3

	LEP (volume offered)	HEP (volume offered)
(b) Standard and non standard preloads and volumes		
Standard preload	High energy ribena (200 mls)	Low energy ribena (200 mls)
2 = EH	Still water (81 mls)	Nutrini (81 mls)
3 = AC	Sparkling water (200 mls)	Nutrini (100 mls)
7 = MS	Ribena (200 mls)	Chocolate Milk (110 mls)
11 = CC	Diluted infatrini (121 mls)	Infatrini (121 mls)

* Non-standard preload composition or volume.

the children drank the entire preload volume on both occasions, but all took 80% or more. Energy intake at meals was also extremely variable, but the median energy intake during the meals following the LEP was 412 kJ higher than those following the HEP (Table 2, Fig. 1) and the mean difference (346 kJ) was significantly different from zero (one sample *t*-test $p = 0.013$).

The median (mean) COMPX score of the 11 children was 70% (73%) but there was substantial variability within the group

Please cite this article in press as: Kane, L., et al. Energy compensation in enterally fed children. *Appetite* (2010), doi:10.1016/j.appet.2010.11.002

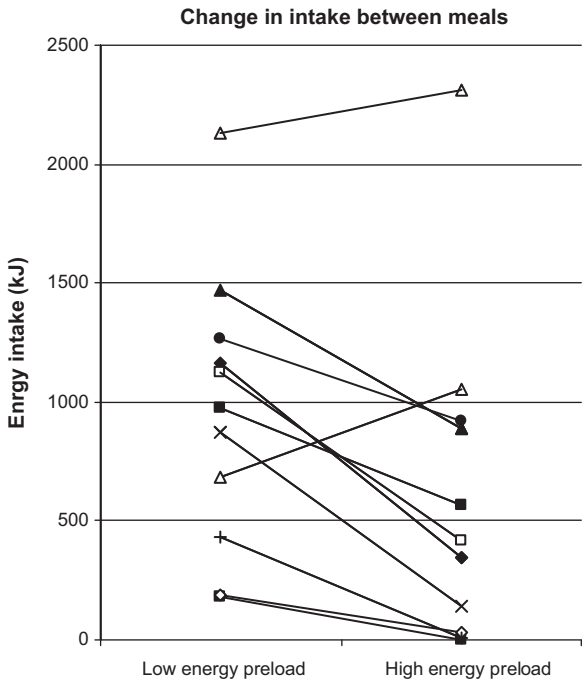

Fig. 1. Meal intake per child after different preloads.

(SD = 82%) (Table 2). Five children showed partial compensation of between 30% and 82%, and another slightly overcompensated (116%), but 3 others substantially over compensated (162–178%) while 2 others showed paradoxical compensation (−73% and −37%) and ate more after the *high* energy preload. There was a trend for boys to compensate rather more accurately (median 99%) than girls (median 30%; *P* Mann–Whitney = 0.1) (Fig. 2) but no clear trend to reduce compensation with age or with whether currently tube-fed (Fig. 2). There was also a trend towards worse compensation in those with the longest duration of tube feeding (Spearman's rho = −0.44, $p = 0.17$) an effect that seemed to be independent of age (Fig. 3).

Discussion

This was a small study in a highly heterogeneous sample, but it nonetheless produced results quite similar to those seen in healthy children (Birch & Fisher, 2000; Cecil et al., 2005; Hetherington et al., 2000). Despite the impression that members of this clinical group do not experience hunger and satiation, all but 2 of the children reduced their subsequent meal intake after a high energy preload. The range of responses was wide, but similar wide ranges have been described in healthy children (Johnson, 2000; Johnson & Birch, 1994).

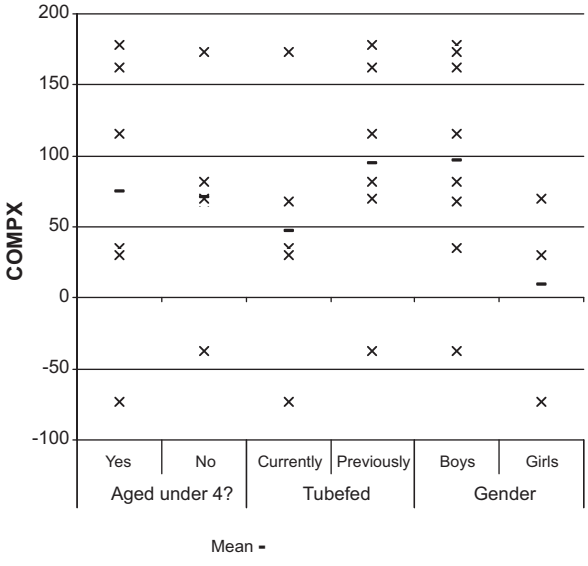

Fig. 2. COMPX scores by gender, age and whether tube fed.

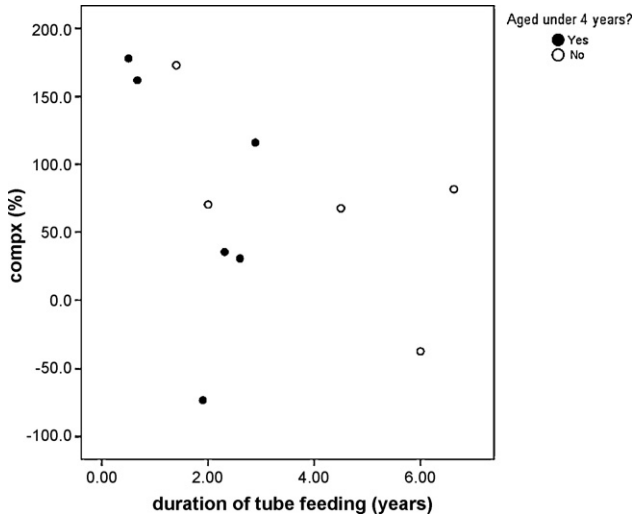

Fig. 3. COMPX scores by length of tube feeding.

The trend for boys to compensate more accurately than girls has also been described previously (Davy, Van Walleghen, & Orr, 2007; Johnson & Birch, 1994). In healthy children the precision of energy intake regulation usually declines as age increases (Birch & Deysher, 1986; Cecil et al., 2005; Hetherington et al., 2000; Johnson, 2000; Rolls, Dimeo, & Shide, 1995). This trend was not seen in this small sample, but many of the younger children were the ones who were still tube fed, which may also explain why current tube feeding did not seem to be associated with degree of compensation.

Practical difficulties

Recruitment and attendance were extremely difficult in this group due to conflicting medical appointments and illness during the study period. For example, one participant failed to complete the study as he was admitted to intensive care before completing the second feeding session. The children, as a group, tended to be highly selective eaters and the researchers had to modify the standardized menu to accommodate each child's preference. The neophobic tendencies of the children, combined with their limited repertoire of foods, resulted in a marked between-participant variation between the preload drinks and the foods presented as test meals, but all children received the same meal on both test occasions.

Even when foods the child liked were offered, intake was relatively low, with an average of only 975 kJ taken at the meal after the low energy preload. This is much lower than 1700 kJ per meal on average which participants of this age would be expected to eat at a meal, but some of this difference reflects the fact that they were also, on average, very small children, with lower requirements.

This fussiness also extended to the preloads. For example, one child would not drink anything except his high energy formula milk. Using certain preloads prevented blinding, as the researchers were unable to mask drinks that varied in colour, viscosity, taste and smell. However, blinding was achieved in 8 of the 11 children studied. The lack of preload uniformity also imposed inter-child

Please cite this article in press as: Kane, L., et al. Energy compensation in enterally fed children. *Appetite* (2010), doi:10.1016/j.appet.2010.11.002

volume and energy-density inconsistencies, but these were necessary in order to ensure a consistent energy content. This meant the volume or energy density offered for some children was more than twice that offered to others, but in all but 2 cases, the volume offered on each meal occasion to a given child was the same.

Strengths and limitations

A great strength of this study is that it tests the compensation model in a clinical group, rather than in healthy children. Only one previous study has examined compensation in a clinical group: comparing children with idiopathic weight faltering to healthy community controls. In that study the weight faltering children ate far less than did the controls at both meals and showed no evidence of compensation. The children in our study were selected because of prior illness and tube dependency rather than under eating and the very different result probably reflects their different clinical characteristics.

Although small, this study was conducted as rigorously as possible, but due to the difficulties of working with sick children, numbers were limited and not all aspects of the protocol could be completely observed. Thus these results should be viewed as only preliminary findings. Future work is needed to study a larger group of such children over a longer period, though the logistic difficulties and comparative rarity of this clinical problem will make this a challenging task.

It must be recognised that satiation studies evaluate only short-term energy adjustment, which may not reflect longer-term energy compensation (Birch et al., 1991). For example in our study one child showed little reduction in meal intake after the HEP, but the parents later described him as eating far less throughout the following day. The compensation model captures only 2 meals in a child's life, and these may not be typical of the child's normal eating practices. Other studies confirm considerable within-child variation in compensation when studies are repeated (Mitchell, 2009). This group did show highly variable individual responses, as has been shown in healthy children (Johnson, 2000; Johnson and Birch 1994) but this variability may merely reflect the highly variable eating behaviour of all young children (Birch et al., 1991).

Implications for practice

Parents and clinicians often describe enterally fed children as having no sense of hunger or appetite and are reluctant to reduce enteral feeds, assuming that the children will not increase their oral intake to match the amount of feed lost (Wright et al., 2010). These results suggest, however, that the children's lack of hunger and low intake while on enteral feeds reflects effective compensation, rather than the reverse. These results are also clinically relevant to the use of high-energy supplement drinks, which are commonly used in this sort of clinical group. There has been almost no formal evaluation of high-energy supplements in this age range, but one study found no evidence of long-term efficacy (Poustie et al., 2006). The results of our study, as well as all the prior work in healthy children, would suggest that on average using high-energy supplements in addition to solid food will increase net energy intake only slightly and at the expense of reducing oral intake.

Conclusion

This small study suggests that children who have been enterally fed from an early age may have the same capacity to regulate their energy intake as healthy children.

References

Bellisle, F., & Dalix, A. (2001). Cognitive restraint can be offset by distraction, leading to increased meal intake in women. *The American Journal of Clinical Nutrition, 74*(2), 197–200.
Birch, L. L., & Deysher, M. (1985). Conditioned and unconditioned caloric compensation. Evidence for self-regulation of food intake in young children. *Learning and Motivation, 16*, 341–355.
Birch, L. L., & Deysher, M. (1986). Caloric compensation and sensory specific satiety. Evidence for self regulation of food intake by young children. *Appetite, 7*(4), 323–331.
Birch, L. L., McPhee, L., & Sullivan, S. (1989). Children's food intake following drinks sweetened with sucrose or aspartame. Time course effects. *Physiology & Behavior, 45*(2), 387–395.
Birch, L. L., Johnson, S. L., Andresen, G., Peters, J. C., & Schulte, M. C. (1991). The Variability of Young Children's Energy Intake. *New England Journal of Medicine, 324*(4), 232–235.
Birch, L. L., McPhee, L. S., Bryant, J. L., & Johnson, S. L. (1993). Children's Lunch Intake. Effects of Midmorning Snacks Varying in Energy Density and Fat Content. *Appetite, 20*(2), 83–94.
Birch, L. L. (1998). Development of food acceptance patterns in the first years of life. *The Proceedings of the Nutrition Society, 57*, 617–624.
Birch, L. L., & Fisher, J. P. (2000). Mothers' child-feeding practices influence daughters' eating and weight. *The American Journal of Clinical Nutrition, 71*(5), 1054–1061.
Blass, E. M., Anderson, D. R., Kirkorian, H. L., Pempek, T. A., Price, I., & Koleini, M. F. (2006). On the road to obesity. Television viewing increases intake of high-density foods. *Physiology & Behavior, 88*(4–5), 597–604.
Booth, D. A., Campbell, A. T., & Chase, A. (1970). Temporal bounds of post-ingestive glucose induced satiety in man. *Nature, 228*(5276), 1104–1105.
Cecil, J. E., Palmer, C. N., Wrieden, W., et al. (2005). Energy intake of children after preloads. Adjustment, not compensation. *The American Journal of Clinical Nutrition, 82*(2), 302–308.
Davy, B. M., Van Walleghen, E. L., & Orr, J. S. (2007). Sex differences in acute energy intake regulation. *Appetite, 49*(1), 141–147.
De Castro, J. M. (1994). Family and friends produce greater social facilitation of food intake than other companions. *Physiology & Behavior, 56*(3), 445–455.
Druce, M., & Bloom, S. R. (2006). The regulation of appetite. *Archives of Disease in Childhood, 91*(2), 183–187.
Hetherington, M. M., Wood, C., & Lyburn, S. C. (2000). Response to energy dilution in the short term. Evidence of nutritional wisdom in young children? *Nutritional Neuroscience, 3*, 321–329.
Hetherington, M. M. (2002). The physiological-psychological dichotomy in the study of food intake. *The Proceedings of the Nutrition Society, 61*(4), 497–507.
Hetherington, M. M., Anderson, A. S., Norton, G. N., & Newson, L. (2006). Situational effects on meal intake. A comparison of eating alone and eating with others. *Physiology & Behavior, 88*, 498–505.
Johnson, S. L., & Birch, L. L. (1994). Parents' and children's adiposity and eating style. *Pediatrics, 94*(5), 653–661.
Johnson, S. L. (2000). Improving Preschoolers' Self-Regulation of Energy Intake. *Pediatrics, 106*(6), 1429–1435.
Johnson, S. L., & Taylor-Holloway, L. A. (2006). Non-Hispanic white and Hispanic elementary school children's self-regulation of energy intake. *The American Journal of Clinical Nutrition, 83*(6), 1276–1282.
Kasese-Hara, M., Wright, C., & Drewett, R. (2002). Energy compensation in young children who fail to thrive. *Journal of Child Psychology and Psychiatry, 43*(4), 449–456.
Lumeng, J. C., & Hillman, K. H. (2007). Eating in larger group increases food consumption. *Archives of Disease in Childhood, 92*(5), 384–387.
Mason, S. J., Harris, G., & Blissett, J. (2005). Tube feeding in infancy. Implications for the development of normal eating and drinking skills. *Dysphagia, 20*(1), 46–61.
Mitchell, V. (2009) PhD thesis, Glasgow Caledonian University
Mrdjenovic, G., & Levitsky, D. A. (2005). Children eat what they are served. The imprecise regulation of energy intake. *Appetite, 44*(3), 273–282.
Norton, G. N., Anderson, A. S., & Hetherington, M. M. (2006). Volume and variety. Relative effects on food intake. *Physiology & Behavior, 87*(4), 714–722.
Poustie, V. J., Russell, J. E., Watling, R. M., Ashby, D., Smyth, R. L., & CALICO Trial Collaborative Group, (2006). Oral protein energy supplements for children with cystic fibrosis: CALICO multicentre randomised controlled trial. *British Medical Journal, 18*(332(7542)), 632–636.
Rolls, B. J., Dimeo, K. A., & Shide, D. J. (1995). Age-related impairments in the regulation of food intake. *The American Journal of Clinical Nutrition, 62*(5), 923–931.
Rolls, B. J., Engell, D., & Birch, L. L. (2000). Serving portion size influences 5-year-old but not 3-year-old children's food intakes. *Journal of the American Dietetic Association, 100*(2), 232–234.
Shea, S., Stein, A. D., Basch, C. E., Contento, I. R., & Zybert, P. (1992). Variability and self-regulation of energy intake in young children in their everyday environment. *Paediatrics, 90*(4), 542–546.
Shide, D. J., & Rolls, B. J. (1991). Social facilitation of caloric intake in humans by friends but not strangers. *International Journal of Obesity, 15*, 8.
Wright, C. M., Smith, K., & Morrison, J. (2010). Withdrawing feeds from children on long term enteral feeding. Factors associated with success and failure. *Archives of Disease in Childhood* http://adc.bmj.com/content/early/2010/07/22/adc.2009.179861.full.pdf.

Please cite this article in press as: Kane, L., et al. Energy compensation in enterally fed children. *Appetite* (2010), doi:10.1016/j.appet.2010.11.002