Published in final edited form as:
JPEN J Parenter Enteral Nutr. 2010 ; 34(1): 13–20. doi:10.1177/0148607109354088.

A.S.P.E.N. Clinical Guidelines: Nutrition Support of Hospitalized Pediatric Patients With Obesity

Cheryl Jesuit, MS, RD, LDN[1], **Cristin Dillon, MS, RD, LDN**[2], **Charlene Compher, PhD, RD, FADA, CNSD, LDN**[3], **American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Board of Directors**, and **Carine M. Lenders, MD, MS, ScD**[1,2]

[1]Nutrition and Fitness for Life Program, Boston Medical Center, Boston, Massachusetts

[2]Pediatric Nutrition Support Service, Boston Medical Center, Philadelphia, Pennsylvania

[3]University of Pennsylvania School of Nursing, Philadelphia, Pennsylvania

Pediatric obesity has reached epidemic proportions in the United States,[1] and there are reports of greater discharge diagnosis of obesity-related complications such as diabetes, sleep apnea, and gallbladder disease and longer length of stay.[2] The origin of pediatric obesity is multifactorial and leads to numerous complications[3,4] affecting inflammatory processes[5] as well as nutrient metabolism.[6–9] As a result, current estimations of nutrition status[10–12] and requirements among obese patients remain unclear.[13–15] Recognizing that body mass index (BMI) may predict obesity-related complications even in adulthood, the Institute of Medicine (IOM)[16] and, more recently, the American Academy of Pediatrics (AAP)[4] recommend that the term *obesity* be used in children aged 2–20 years (BMI ≥95th percentile). Once obesity has been identified, the role of nutrition support is to prevent complications associated with the provision of enteral or parenteral feedings. Undernutrition may result in energy and protein deprivation,[17,18] whereas overzealous nutrition support may result in hypophosphatemia, typically observed in refeeding syndrome, and hyperglycemia; all of these complications may affect morbidity and mortality risk.[19] Thus, neither undernutrition nor overnutrition can be recommended during hospitalization of the obese child.

Methods

The American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) consists of healthcare professionals representing the disciplines of medicine, nursing, pharmacy, dietetics, and nutrition science. The mission of A.S.P.E.N. is to improve patient care by advancing the science and practice of nutrition support therapy. A.S.P.E.N. vigorously works to support quality patient care, education, and research in the fields of nutrition and metabolic support in all healthcare settings. These clinical guidelines were developed under the guidance of the A.S.P.E.N. Board of Directors. Promotion of safe and effective patient care by nutrition support practitioners is a critical role of the A.S.P.E.N. organization. The

Address correspondence to: Charlene Compher, PhD, RD, FADA, CNSD, LDN, University of Pennsylvania School of Nursing, Claire M. Fagin Hall, 418 Curie Boulevard, Philadelphia, PA 19104-4217; compherc@nursing.upenn.edu..

Financial disclosure: Dr. Lenders and the Nutrition and Fitness for Life clinic received foundation support from Loomis, Sayles & Company, LP; the New Balance Foundation; the Physician Nutrition Specialist Award from the American Society for Nutrition; and the Boston Red Sox.

A.S.P.E.N. Board of Directors has published clinical guidelines since 1986.[20–22] Starting in 2007, A.S.P.E.N. has revised these clinical guidelines on an ongoing basis by reviewing about 20% of the chapters each year in order to keep them as current as possible.

These A.S.P.E.N. clinical guidelines are general. They are based upon general conclusions of health professionals who, in developing such guidelines, have balanced potential benefits to be derived from a particular mode of medical therapy against certain risks inherent with such therapy. However, the professional judgment of the attending health professional is the primary component of quality medical care. Because guidelines cannot account for every variation in circumstances, the practitioner must always exercise professional judgment in their application. These clinical guidelines are intended to supplement, but not replace, professional training and judgment.

These clinical guidelines were created in accordance with the IOM recommendations as "systematically developed statements to assist practitioner and patient decisions about appropriate healthcare for specific clinical circumstances."[23] These clinical guidelines are for use by healthcare professionals who provide nutrition support services and offer clinical advice for managing adult and pediatric patients in inpatient and outpatient (ambulatory, home, and specialized care) settings. The utility of the clinical guidelines is attested to by the frequent citation of this document in peer-reviewed publications and its frequent use by A.S.P.E.N. members and other healthcare professionals in clinical practice, academia, research, and industry. The guidelines inform professional clinical activities, serve as educational tools, and influence institutional practices and resource allocation.[24]

These clinical guidelines are formatted to promote the ability of the end user of the document to understand the strength of the literature used to grade each recommendation. Each guideline recommendation is presented as a clinically applicable definitive statement of care and should help the reader make the best patient care decision. The best available literature was obtained and carefully reviewed. Chapter authors completed a thorough literature review using Medline, the Cochrane Central Register of Controlled Trials, the Cochrane Database of Systematic Reviews, and other appropriate reference sources. The results of the literature search and review formed the basis of an evidence-based approach to the clinical guidelines. Chapter editors work with the authors to ensure compliance with the authors' directives regarding content and format. The initial draft is reviewed internally to ensure consistency with the other A.S.P.E.N. Guidelines and Standards and reviewed externally (either by experts in the field within our organization or outside of our organization) for appropriateness of content. Finally, the draft is reviewed and approved by the A.S.P.E.N. Board of Directors.

The system used to categorize the level of evidence for each study or article used in the rationale of the guideline statement and to grade the guideline recommendation is outlined in Table 1.[25]

The grade of a guideline is based on the levels of evidence of the studies used to support the guideline. A randomized controlled trial (RCT), especially one that is double-blind in design, is considered to be the strongest level of evidence to support decisions regarding a therapeutic intervention in clinical medicine.[26] A systematic review (SR) is a specialized type of literature review that analyzes the results of several RCTs. A high-quality SR usually begins with a clinical question and a protocol that addresses the methods to answer this question. These methods usually state how the literature is identified and assessed for quality, what data are extracted, how they are analyzed, and whether there were any deviations from the protocol during the course of the study. In most instances, meta-analysis (MA), a mathematical tool to combine data from several sources, is used to analyze the data.

However, not all SRs use MA. SR is considered among the most important level of evidence in the field of evidence-based medicine. A level of I, the highest level, will be given to large RCTs where results are clear and the risk of alpha and beta error is low (well-powered). A level of II will be given to RCTs that include a relatively low number of patients or are at moderate to high risk for alpha and beta error (under-powered). Meta-analyses can be used to combine the results of studies to further clarify the overall outcome of these studies but will not be considered in the grading of the guideline. A level of III is given to cohort studies with contemporaneous controls and to validation studies, whereas cohort studies with historic controls will receive a level of IV. Case series, uncontrolled studies, and articles based on expert opinion alone will receive a level of V.

Practice Guidelines and Rationales

Table 2 provides the entire set of guideline recommendations for nutrition support of hospitalized pediatric patients with obesity.

Practice Guidelines

1. BMI is the preferred practical method to screen children for obesity. (Grade: D)

Rationale—Although BMI (kg/m^2) does not directly measure body fat, it has been recognized as a useful predictor of adiposity and medical complications of obesity. BMI is a measure of relative weight rather than adiposity.[27] Tracking studies from childhood to adulthood provide the best available evidence to support the validity of BMI as a screening criterion for obesity in children and adolescents.[28] There is increasing evidence that ≥95th percentile on BMI for sex and age charts in childhood predicts adult BMI, obesity, adiposity, and mortality[29–37] (Table 3); however, more tracking (longitudinal) data are needed, especially on clinical risks associated with obesity.[10,28] Although BMI is an adequate screening method for older children and at a group level, its strength as an indicator of adiposity decreases at younger ages (<13 years) and may vary by ethnicity and race.[10,38] There is no current valid measure for children younger than 2 years[10,39–40] or for severe obesity at any age.[10,38,41–43]

2. Pediatric obese inpatients may be at increased nutrition risk. Testing for potential laboratory abnormalities is recommended for safety reasons (eg, fasting blood sample, including lipid profile, glucose, phosphorus, and complete blood count). (Grade: E)

Rationale—Although the prevalence of pediatric obesity (based on BMI ≥95th percentile) is elevated, studies of obesity prevalence and nutrition support outcomes among obese compared with nonobese children in the hospital setting have not been evaluated. Nevertheless, we believe that hospitalized pediatric patients should undergo nutrition screening to identify those who require formal nutrition assessment with development of a nutrition care plan. Obese children are at increased risk for anemia,[44–45] low fat-soluble vitamins levels (such as vitamin D),[8] low vitamin B status,[9] hyperlipidemia, insulin resistance, and hyperglycemia.[6,7,10,46,47] The presence of the metabolic syndrome in children is not well defined and may not predict obesity in adulthood.[48] There is some evidence from adult studies that tight control of hyperglycemia may affect morbidity and mortality, and there are anecdotal reports of hypophosphatemia following glucose provision in long-term fasting.[49]

3. When possible, energy requirements of obese hospitalized children should be assessed using indirect calorimetry rather than predictive equations. (Grade: D)

Rationale—Resting energy expenditure (REE) varies with obesity status but is best explained by differences in lean body mass. The percentage of lean body mass for each additional kilogram of weight above ideal weight is highly variable. Therefore, the calculation of excess weight to estimate ideal body weight is imprecise. As there is no practical and valid tool to evaluate lean body mass in order to estimate ideal weight in hospitalized patients, assessment of REE using indirect calorimetry is an alternative to the imprecision of equations (Table 4).[13–15,50–54]

4. There is not adequate evidence to assess the clinical outcomes of hypocaloric or hypercaloric feeding during hospitalization of obese children. Therefore, the goals for the provision of energy to pediatric obese inpatients should be similar to the goals for their nonobese counterparts until more evidence is available. (Grade: E)

Rationale—Although hypocaloric solutions are used in the outpatient setting, there is no evidence that these solutions should be initiated during hospitalizations. There are anecdotal reports of use of hypocaloric solutions in patients who are hospitalized for major obesity-related complications such as heart failure, pseudotumor cerebri, and sleep apnea. Finally, note that the use of old guidelines may result in overfeeding (recommended dietary allowances may overestimate needs by up to 20%, depending on the age group) and further complications.[55]

Acknowledgments

We thank Kathleen Gura, PharmD, for her guidance at the initiation of these guidelines in 2007. We are very thankful for the contribution of Howard Bauchner, MD, in the review of the manuscript, with support from his 5 K24 HD042489-5.

References

1. Ogden C, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. JAMA. 2006; 295:1549–1555. [PubMed: 16595758]
2. Wang G, Dietz WH. Economic burden of obesity in youths aged 6 to 17 years: 1979–1999. Pediatrics. 2002; 109:E81. [PubMed: 11986487]
3. Dietz WH, Robinson TN. Clinical practice: overweight children and adolescents. N Engl J Med. 2005; 352:2100–2109. [PubMed: 15901863]
4. Barlow SE, Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007; 120(suppl 4):S164–S192. [PubMed: 18055651]
5. Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Low-grade systemic inflammation in overweight children. Pediatrics. 2001; 107:E13. [PubMed: 11134477]
6. Sinha R, Fisch G, Teague B, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. N Engl J Med. 2002; 346:802–810. [PubMed: 11893791]
7. Berenson GS, Srinivasan SR, Bao W, Newman WP III, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults: the Bogalusa Heart Study. N Engl J Med. 1998; 338:1650–1656. [PubMed: 9614255]
8. Smotkin-Tangorra M, Purushothaman R, Gupta A, Nejati G, Anhalt H, Ten S. Prevalence of vitamin D insufficiency in obese children and adolescents. J Pediatr Endocrinol Metab. 2007; 20:817–823. [PubMed: 17849744]
9. Pinhas-Hamiel O, Doron-Panush N, Reichman B, Nitzan-Kaluski D, Shalitin S, Geva-Lerner L. Obese children and adolescents: a risk group for low vitamin B12 concentration. Arch Pediatr Adolesc Med. 2006; 160:933–936. [PubMed: 16953016]
10. Whitlock EP, Williams SB, Gold R, Smith PR, Shipman SA. Screening and interventions for childhood overweight: a summary of evidence for the US Preventive Task Force. Pediatrics. 2005; 116:e125–e144. [PubMed: 15995013]

11. [Accessed January 21, 2009] Weight-for-length and BMI growth charts. http://www.cdc.org/growthcharts.
12. Argall JA, Wright N, Mackway-Jones K, Jackson R. A comparison of two commonly used methods of weight estimation. Arch Dis Child. 2003; 88:789–790. [PubMed: 12937099]
13. Derumeaux-Burel H, Meyer M, Morin L, Boirie Y. Prediction of resting energy expenditure in a large population of obese children. Am J Clin Nutr. 2004; 80:1544–1550. [PubMed: 15585766]
14. McDuffie JR, Adler-Wailes DC, Elberg J, et al. Predictive equations for resting energy expenditure in overweight and normal-weight black and white children. Am J Clin Nutr. 2004; 80:365–373. [PubMed: 15277157]
15. Kaplan AS, Zemel BS, Neiswender KM, Stallings VA. Resting energy expenditure in clinical pediatrics: measured versus predictive equations. J Pediatr. 1995; 127:200–205. [PubMed: 7636642]
16. Koplan, JP.; Liverman, CT.; Kraak, VA., editors. Preventing Childhood Obesity: Health in the Balance. The National Academy Press; Washington, DC: 2004.
17. Fisler JS, Drenick EJ. Calcium, magnesium, and phosphate balances during very low calorie diets of soy or collagen protein in obese men: comparison to total fasting. Am J Clin Nutr. 1984; 40:14–25. [PubMed: 6540047]
18. Hulst JM, Joosten KF, Tibboel D, van Goudoever JB. Causes and consequences of inadequate substrate supply to pediatric ICU patients. Curr Opin Clin Nutr Metab Care. 2006; 9:297–303. [PubMed: 16607132]
19. Solomon SM, Kirby DF. The refeeding syndrome: a review. JPEN J Parenter Enteral Nutr. 1990; 14:90–97. [PubMed: 2109122]
20. A.S.P.E.N. Board of Directors. Guidelines for use of total parenteral nutrition in the hospitalized adult patient. JPEN J Parenter Enteral Nutr. 1986; 10:441–445. [PubMed: 3093699]
21. A.S.P.E.N. Board of Directors. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. JPEN J Parenter Enteral Nutr. 1993; 17(4 suppl):1SA–52SA. [PubMed: 8264077]
22. A.S.P.E.N. Board of Directors; the Clinical Guidelines Task Force. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. JPEN J Parenter Enteral Nutr. 2002; 26(1 suppl):1SA–138SA. [PubMed: 11841046]
23. Committee to Advise Public Health Service on Clinical Practice Guidelines (Institute of Medicine). Clinical Practice Guidelines: Directions for a New Program. National Academy Press; Washington, DC: 1990. p. 58
24. Seres D, Compher C, Seidner D, Byham-Gray L, Gervasio J, McClave S, A.S.P.E.N. Standards and Guidelines Committees. 2005 American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Standards and Guidelines survey. Nutr Clin Pract. 2006; 21:529–532. [PubMed: 16998152]
25. Dellinger RP, Carlet JM, Masur H, et al. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. Crit Care Med. 2004; 32:858–873. [PubMed: 15090974]
26. Guyatt GH, Haynes RB, Jaeschke RZ, et al. Users' guides to the medical literature, XXV: evidence-based medicine: principles for applying the users' guides to patient care. JAMA. 2000; 284:1290–1296. [PubMed: 10979117]
27. Dietz WH, Robinson TN. Use of the body mass index (BMI) as a measure of overweight in children and adolescents. J Pediatr. 1998; 132:191–193. [PubMed: 9506622]
28. Lenders, C.; Willett, W. Nutrition epidemiology. In: Duggan, C.; Walker, W.; Watkins, J., editors. Nutrition in Pediatrics: Basic Science and Clinical Applications. 4th ed.. B.C. Decker Inc.; Hamilton, Ontario, Canada: 2007. p. 167-178.
29. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Racial differences in the tracking of childhood BMI to adulthood. Obes Res. 2005; 13:928–935. [PubMed: 15919847]
30. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. Pediatrics. 2005; 115:22–27. [PubMed: 15629977]

31. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. Am J Clin Nutr. 2002; 76:653–658. [PubMed: 12198014]
32. Guo SS, Huang C, Maynard LM, et al. Body mass index during childhood, adolescence and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. Int J Obes Relat Metab Disord. 2000; 24:1628–1635. [PubMed: 11126216]
33. Casey VA, Dwyer JT, Coleman KA, Valadian I. Body mass index from childhood to middle age: a 50-y follow-up. Am J Clin Nutr. 1992; 56:14–18. [PubMed: 1609751]
34. Bjørge T, Engeland A, Tverdal A, Smith GD. Body mass index in adolescence in relation to cause-specific mortality: a follow-up of 230,000 Norwegian adolescents. Am J Epidemiol. 2008; 168:30–37. [PubMed: 18477652]
35. Engeland A, Bjørge T, Søgaard AJ, Tverdal A. Body mass index in adolescence in relation to total mortality: 32-year follow-up of 227,000 Norwegian boys and girls. Am J Epidemiol. 2003; 157:517–523. [PubMed: 12631541]
36. Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Davey Smith G. Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd Orr cohort. Am J Clin Nutr. 1998; 67:1111–1118. [PubMed: 9625081]
37. Nieto FJ, Szklo M, Comstock GW. Childhood weight and growth rate as predictors of adult mortality. Am J Epidemiol. 1992; 136:201–213. [PubMed: 1415142]
38. Krebs NF, Himes JH, Jacobson D, Nicklas TA, Guilday P, Styne D. Assessment of child and adolescent overweight and obesity. Pediatrics. 2007; 120(suppl 4):S193–S228. [PubMed: 18055652]
39. Gardner DS, Hosking J, Metcalf BS, Jeffery AN, Voss LD, Wilkin TJ. Contribution of early weight gain to childhood overweight and metabolic health: a longitudinal study (EarlyBird 36). Pediatrics. 2009; 123:e67–e73. [PubMed: 19117849]
40. Wilkin TJ, Metcalf BS, Murphy MJ, Kirkby J, Jeffery AN, Voss LD. The relative contributions of birth weight, weight change, and current weight to insulin resistance in contemporary 5-year-olds: the EarlyBird Study. Diabetes. 2002; 51:3468–3472. [PubMed: 12453901]
41. Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Study. J Pediatr. 2007; 150:12–17. e2. [PubMed: 17188605]
42. Freedman DS, Khan LK, Serdula MK, Ogden CL, Dietz WH. Racial and ethnic differences in secular trends for childhood BMI, weight, and height. Obesity (Silver Spring). 2006; 14:301–308. [PubMed: 16571857]
43. Lenders CM, Wright JA, Apovian CM, et al. Weight loss surgery eligibility according to various BMI criteria among adolescents. Obesity (Silver Spring). 2009; 17:150–155. [PubMed: 19008870]
44. Nead KG, Halterman JS, Kaczorowski JM, Auinger P, Weitzman M. Overweight children and adolescents: a risk group for iron deficiency. Pediatrics. 2004; 114:104–108. [PubMed: 15231915]
45. Pinhas-Hamiel O, Newfield RS, Koren I, Agmon A, Lilos P, Phillip M. Greater prevalence of iron deficiency in overweight and obese children and adolescents. Int J Obes Relat Metab Disord. 2003; 27:416–418. [PubMed: 12629572]
46. Cali AM, Caprio S. Obesity in children and adolescents. J Clin Endocrinol Metab. 2008; 93(11 suppl 1):S31–S36. [PubMed: 18987268]
47. Nguyen QM, Srinivasan SR, Xu JH, Chen W, Berenson GS. Changes in risk variables of metabolic syndrome since childhood in pre-diabetic and type 2 diabetic subjects: the Bogalusa Heart Study. Diabetes Care. 2008; 31:2044–2049. [PubMed: 18628566]
48. Goodman E, Daniels SR, Meigs JB, Dolan LM. Instability in the diagnosis of metabolic syndrome in adolescents. Circulation. 2007; 115:2316–2322. [PubMed: 17420347]
49. Corredor DG, Sabeh G, Mendelsohn LV, Wasserman RE, Sunder JH, Danowski TS. Enhanced postglucose hypophosphatemia during starvation therapy of obesity. Metabolism. 1969; 18:7547–7563.
50. Lazzer S, Agosti F, De Col A, Sartorio A. Development and cross-validation of prediction equations for estimating resting energy expenditure in severely obese Caucasian children and adolescents. Br J Nutr. 2006; 96:973–979. [PubMed: 17092390]

51. Schmelzle H, Schröder C, Armbrust S, Unverzagt S, Fusch C. Resting energy expenditure in obese children aged 4 to 15 years: measured versus predicted data. Acta Paediatr. 2004; 93:739–746. [PubMed: 15244220]

52. Tverskaya R, Rising R, Brown D, Lifshitz F. Comparison of several equations and derivation of a new equation for calculating basal metabolic rate in obese children. J Am Coll Nutr. 1998; 17:333–336. [PubMed: 9710841]

53. Molnár D, Jeges S, Erhardt E, Schutz Y. Measured and predicted resting metabolic rate in obese and nonobese adolescents. J Pediatr. 1995; 127:571–577. [PubMed: 7562278]

54. Maffeis C, Schutz Y, Micciolo R, Zoccante L, Pinelli L. Resting metabolic rate in six- to ten-year-old obese and nonobese children. J Pediatr. 1993; 122:556–562. [PubMed: 8463900]

55. Butte NF, Wong WW, Hopkinson JM, Heinz CJ, Mehta NR, Smith EO. Energy requirements derived from total energy expenditure and energy deposition during the first 2 y of life. Am J Clin Nutr. 2000; 72:1558–1569. [PubMed: 11101486]

Table 1

Grading of Guidelines and Levels of Evidence

Grading of Guidelines	
A	Supported by at least two level I investigations
B	Supported by one level I investigation
C	Supported by at least one level II investigation
D	Supported by at least one level III investigation
E	Supported by level IV or V evidence

Levels of Evidence	
I	Large randomized trials with clear-cut results; low risk of false-positive (alpha) and/or false-negative (beta) error
II	Small, randomized trials with uncertain results; moderate to high risk of false-positive (alpha) and/or false-negative (beta) error
III	Nonrandomized cohort with contemporaneous controls
IV	Nonrandomized cohort with historical controls
V	Case series, uncontrolled studies, and expert opinion

Reproduced from Dellinger RP, Carlet JM, Masur H. Introduction. *Crit Care Med.* 2004;32(11suppl):S446 with permission of the publisher. Copyright 2004 Society of Critical Care Medicine.

Table 2

Nutrition Support Guideline Recommendations of Hospitalized Pediatric Patients With Obesity

Guideline Recommendation	Grade
1. Body mass index is the preferred practical method to screen children for obesity.	D
2. Pediatric obese inpatients may be at increased nutrition risk. We recommend testing for potential laboratory abnormalities for safety reasons (eg, fasting blood sample, including lipid profile, glucose, phosphorus, and complete blood count).	E
3. When possible, energy requirements of obese hospitalized children should be assessed using indirect calorimetry rather than predictive equations.	D
4. There is no adequate evidence to assess the clinical outcomes of hypocaloric or hypercaloric feeding during hospitalization of obese children. Therefore, the goals for the provision of energy to the pediatric obese inpatient should be similar to their nonobese counterparts.	E

Table 3

Adult Outcomes of Childhood Obesity

Study	Population	Intervention	Outcome
Tracking studies of BMI and other adiposity measures from childhood to adulthood			
Freedman[29] 2005 Level III	n = 2,392	Tracking childhood overweight to adult obesity by race	Tracking differs by race, with 65% (white girls) to 84% (black girls) of "overweight" children becoming obese adults
	Age 5–14 y in 1973–1974, 17 y follow-up	Longitudinal models for repeated measure data	
	Louisiana (USA)		
Freedman[30] 2005 Level III	n = 2,610	Child vs adult BMI, adiposity, adiposity by TSF	Childhood BMI strongly predicts adult adiposity
	Age 2–17 y in 1973–1974 and 1992–1994, age 18–37 y in 1982–1996	Spearman correlation, simple linear regression	Childhood overweight strongly associated with adult overfat
	Louisiana (USA)		
Guo[31] 2002 Level III	In 1929 (n = 347)	Predict adult overweight/obesity from childhood/adolescent BMI cutoffs	Half of children and adolescents with BMI ≥75th %tile overweight as adults
	Annual measures ages 3–18 y, age 20 y, age 30–39 y (mean 35 y)	ROC, logistic regression	With BMI ≥95th %tile, children 62%–98% more likely to be overweight at age 35 y
	White race only		
	Ohio, Indiana, Kentucky (USA)		
Guo[32] 2000 Level III	In 1929 (n = 338), age 2–25 y; age 35–45 y (n= 159)	BMI patterns during childhood, puberty, postpuberty vs overweight and body fatness at age 35–45 y	Change in childhood BMI related to adult overweight and adiposity, especially in females
	Hydrostatic weight data (n = 85)	Pediatric BMI gain/y at BMI rebound, puberty, postpuberty	Early BMI rebound associated with maximum BMI velocity and BMI as adult
	White race only	General linear models	Maximum BMI velocity strong predictor of total and percent body fat
	Ohio, Indiana, Kentucky (USA)		
Casey[33] 1992 Level III	At birth in 1930s (n = 296), age 18 y (n = 134), at age 50 y (n = 91)	Tracking BMI with early and late adolescence defined as 2 y before or 2 y after peak height velocity	Starting in late adolescence, BMI predictive of adult obesity, especially for males
	White race only	Age categories: childhood, early and late adolescence, ages 18, 30, 40, and 50 y	With Foulkes-Davis tracking index,[a] subjects at age 18 y unlikely to change BMI category
	Massachusetts (USA)	Pearson correlation, simple linear regression	
Tracking studies of BMI and mortality from childhood or adolescence to adulthood			
Bjørge[34] 2008 Level III	In 1963–1974 (n = 226,678), age 14–19 y, 34.9 y follow-up	Risk of death according to categories of adolescent BMI %tiles: <3; 3–4; 5–9; 10–24; 25–74; 75–84; 85–94; ≥95th or <25; 25–74; 75–84, and >85th, adult BMI categories <18.5; 18.5–22.49; 22.5–24.99; 25–27.49; 25–29.99; ≥30 kg/m^2	Adolescent BMI >75th %tile predicts increased mortality in middle age

Study	Population	Intervention	Outcome
	Race not specified	Multivariate Cox proportional hazards models, spline analysis	
	Norway		
Engelanc[35] 2003 Level III	In 1963–1975 (n = 128,121), age 10–19 y; follow-up ≥10 y (up to 29 y)	Risk for death as adult associated with adolescent "obesity" using adolescent BMI %tiles: <25; 25–74; 75–85; ≥95th at ages 14–15 y, 16–17 y, 18–19 y vs adult ages 25–29, 30–34, 35–40, 40–54 y	Adolescent BMI >75th %tile at higher risk for mortality of all causes in adulthood
	Race not specified	Logistic regression, multivariate Cox proportional hazards model	Adjustment for adult BMI reduces excess mortality observed for men, to a lesser extent for women
	Norway		
Gunnell[36] 1998 Level III	In 1937–1939 (n = 2,990), age 2–14 y, Follow-up to 1995, to age 57 y	BMI in adolescents and adults vs mortality from all causes, from CVD	Nonlinear association BMI vs overall mortality
	Race not specified	BMI *z* scores at BMI <25; 25–49; 50–74; ≥75 kg/m^2	Reference BMI with lowest mortality
	England	Hazard ratios with reference = BMI 25 th–49th %tile; BMI >90th %tile vs >90th %tile; Cox proportional hazards model	Greater mortality risk in older children, those with BMI >90th %tile
			Age and gender differences in all-cause and CVD mortality
Nieto[37] 1992 Level III	In 1933–1945 (n = 13,146), age 5–18 y, follow-up to 1985	Hypothesized that body weight and rate of growth during school-age y directly associated with middle-age mortality from all causes	Higher mortality with higher relative weight, at both prepubertal and postpubertal age
	Race not specified	Internally defined relative weight USA, National Standards (1979) Quintiles of growth parameters, nested case-control (1:10)	
	Maryland (USA)	Cox proportional hazards model	

BMI, body mass index; CDC, Centers for Disease Control and Prevention; CVD, cardiovascular disease; %tile, percentile; OR, odds ratio; ROC, receiver operating characteristic; TSF, triceps skinfold thickness. Studies with measured weight and height rather than self-report have been included.

[a]Foulkes-Davis tracking index determines probability that mean of the curves of 2 individuals (with repeated measures) selected at random will not cross over time.

Table 4

Energy Expenditure in Children with Obesity

Study	Population	Intervention	Outcome
Lazzer[50] 2006 Level III	Children with BMI >99th %tile	Develop and cross-validate new equations for severely obese children and adolescents using indirect calorimetry	First equation based on age, gender, weight, and height; second equation based on age, gender, FM, and FFM
	Age 7–18 y (n = 574), age 12–18 y (n = 53)	BMI >99th %tile by Italian growth charts, 1997, body fat by BIA	Both predict REE with mean difference >2%
	Study period not defined	Multiple linear regression, Bland-Altman	
	White race		
	Italy		
Schmelzle[51] 2004 Level III	Children with BMI >95th %tile	Compare measured and calculated REE using 14 published equations	Published equations for obese children yield scattered data
	In 1999–2000 (n = 82), age 4–15 y	DXA scan for body composition	LBM improves accuracy of predicted REE
	Race not defined	Simple linear regression, bootstrap analysis	
	Germany		
Derumeaux-Burel[13] 2004 Level III	Children with BMI *z* score ≥2 (n = 471 derivation), (n = 211 validation), age 3–18 y	Establish new equations using indirect calorimetry, compare with HB, Schofield, WHO, Tverskaya equations; FM from BIA	FFM explained >75% of REE in both genders
	Race not defined	ANOVA, regression, Bland-Altman	All predictive equations miscalculated REE
	France		
McDuffie[14] 2004 Level III	Children with BMI >95 %tile (n = 502), age 6–11 y	Compare measured REE with FAO/WHO/UNU, Schofield, Molnar, Maffetis, Tverskaya equations, by race; DXA for body composition	After adjusting for race, gender, and overweight status, no equation accurately predicted REE
	Study period not specified		Authors propose new equation
	Pennsylvania, Louisiana, Washington, DC (USA)		
Tverskaya[52] 1998 Level III	Children with BMI >28 kg/m^2 (n = 110), age 3–18 y in 1992–1996	Compare measured BMR to equations, create new equations	Former equations do not predict BMR accurately
	New York (USA)	Multiple regression, Bland-Altman	New equation predicts within 4% of measured BMR
Kaplan[15] 1995 Level III	Children with 76% as FTT, 19% obesity in 1988, (n = 102), age 2–10 y in 1990–1994	Measured vs FAO/WHO/UNU, HB, Schofield equations; paired *t* test	Predictive equations *closely* predict REE in only 40% of subjects
	Pennsylvania (USA)		
Molnár[53] 1995 Level III	Children ≥120% expected weight for height (n = 371), age 10–16 y	Measured vs calculated REE by FAO/WHO/UNU, Robertson and Reid, Fleisch, Mayo equations	Equations overestimate REE
	Race not defined	ANOVA, simple linear regression	LBM may explain up to 80% of REE
	Hungary		
Maffeis[54] 1993 Level III	25% of children ≥120% expected weight for height (n = 130), age 6–10 y	Measured vs calculated REE by FAO/WHO/UNU, Robertson and Reid, Fleisch, Talbot, and Mayo equations skinfold measurement	FFM is best predictor of REE
	Study period not defined	ANOVA, regression	Most equations overestimate REE
	Race not defined		
	Italy		

ANOVA, analysis of variance; BIA, bioelectrical impedance analysis; BMI, body mass index; BMR, basal metabolic rate; CDC, Centers for Disease Control and Prevention; DXA, dual-energy x-ray absorptiometry; FAO/WHO/UNU, Food and Agriculture Organization/World Health Organization/United Nations University equation; FFM, fat-free mass; FM, fat mass; FTT, failure to thrive; HB, Harris-Benedict equation; LBM, lean body mass; REE, resting energy expenditure; SD, standard deviation; WHO, World Health Organization equation.