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Childhood Obesity and Exercise

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Introduction

Childhood obesity is the most prevalent nutritional problem among children and adolescents in the United States.[1] Data from the third National Health and Nutrition Examination Survey (NHANES III) suggest that 22% of children and adolescents are overweight, and 11% are obese.[2] Table 70.1 shows the unadjusted prevalence of overweight by body mass index (BMI) for ages 6 to 17 from NHANES III and sex, age, and race-specific 85th and 95th percentile cutoff points from the second and third National Health Examination Surveys (NHES II and NHES III).[2] As shown in Table 70.2, the age-adjusted prevalence of childhood overweight has increased among all sex and ethnic groups since the mid-1960s.[2] Thus, an ever-increasing number of young people are experiencing the host of undesirable social, emotional, and medical implications of obesity.

Definition and Clinical Evaluation of Childhood Obesity

Defining childhood obesity is difficult, and no generally accepted definition has yet emerged.[2] Table 70.3 shows commonly encountered anthropometric definitions of childhood obesity.[2-5] Use of BMI has been recommended for most clinical settings due to its high reliability and ease of measurement.[6-7] A clinical decision about whether a child with a given BMI is truly overfat may require additional information, such as skinfold thickness measurements, comorbidity, family history, and recent health history.[8] A careful family history and physical examination can readily diagnose most of the hormonal causes and genetic syndromes associated with childhood obesity.[1] Table 70.4 lists common hormonal and genetic causes of childhood obesity, and Table 70.5 provides a differential diagnosis of childhood obesity. Although genetic and hormonal disorders are responsible for less than 10% of childhood obesity, they must be ruled out, as they require different modes of therapy.[3]

Age, Gender, Ethnicity, and Socioeconomic Status

Tables 70.6 and 70.7 provide age-, sex-, and race-specific percentiles for BMI and triceps skinfold thickness for ages 6 to 18.[4] NHANES III data (Table 70.8) indicate that lower

TABLE 70.1

Unadjusted Prevalence of Overweight for NHANES III, from Sex- and Age-Specific 85th and 95th Percentile Cutoff Points of NHES II and NHES III[a]

Category	No.	Percentile	
		85th	95th
Sex and Age, Y			
Both sexes	2920	22.0 ± 1.1	10.9 ± 0.8
6-11	1817	22.3 ± 1.5	11.0 ± 1.0
12-17	1103	21.7 ± 1.9	10.8 ± 1.3
Boys			
6-8	442	21.3 ± 4.3	11.7 ± 3.6
9-11	467	22.7 ± 2.7	10.9 ± 2.3
12-14	253	23.5 ± 3.3	12.0 ± 2.6
15-17	289	20.7 ± 2.9	13.5 ± 2.8
Girls			
6-8	450	24.2 ± 3.6	13.7 ± 2.7
9-11	458	21.4 ± 4.1	8.2 ± 2.1
12-14	288	21.5 ± 2.8	8.5 ± 2.0
15-17	273	21.4 ± 3.1	9.0 ± 1.4
Sex, Age, Y, Race			
Boys age 6 to 11			
Total[b]	909	21.9 ± 2.4	11.3 ± 1.8
Non-Hispanic white	267	20.5 ± 2.8	10.4 ± 2.4
Non-Hispanic black	257	26.5 ± 2.7	13.4 ± 2.3
Mexican American	350	33.3 ± 3.0	17.7 ± 2.3
Boys age 12 to 17			
Total[b]	542	22.0 ± 2.2	12.8 ± 1.9
Non-Hispanic white	155	23.1 ± 3.1	14.4 ± 2.7
Non-Hispanic black	163	21.1 ± 3.7	9.3 ± 2.4
Mexican American	203	26.7 ± 4.6	12.8 ± 3.2
Girls age 6 to 11			
Total[b]	908	22.7 ± 2.4	10.6 ± 1.3
Non-Hispanic white	270	21.5 ± 3.7	9.8 ± 2.0
Non-Hispanic black	224	31.4 ± 4.0	16.9 ± 2.8
Mexican American	389	29.0 ± 2.1	14.3 ± 1.7
Girls age 12 to 17			
Total[b]	561	21.4 ± 2.7	8.8 ± 1.4
Non-Hispanic white	191	20.3 ± 3.5	8.3 ± 1.6
Non-Hispanic black	147	29.9 ± 4.5	14.4 ± 3.1
Mexican American	198	23.4 ± 3.0	8.7 ± 2.5

[a] Values are prevalences ± SEMs. NHANES indicates National Health and Nutrition Examination Survey; NHES, National Health Examination Survey.

[b] Includes data for race-ethnicity groups not shown separately.

From Troiano RP, Flegal KM, Kuczmarski RJ, Campbell M, Johnson CL. *Arch Pediatr Adolesc Med* 149: 1085-1089; 1995. With permission.

TABLE 70.2

Age-Adjusted Prevalence[a] of Overweight from National Surveys (1963 to 1991) by Two Percentile Cutoff Point Definitions

	Boys		Girls	
Population Group	**85th Percentile**	**95th Percentile**	**85th Percentile**	**95th Percentile**
Ages 6 to 11 y				
All Races[b]				
NHES	15.2	5.2	15.2	5.2
NHANES I	18.2	6.5	13.9	4.3
NHANES II	19.9	7.9	15.8	7.0
NHANES III	22.3	10.8	22.7	10.7
White				
NHES	16.0	5.6	15.7	5.1
NHANES I	19.5	6.7	13.4	4.5
NHANES II	20.8	7.9	15.4	6.4
NHANES III	22.3	10.4	22.0	10.2
Black				
NHES	10.3	2.0	12.1	5.3
NHANES I	12.3	5.6	16.8	3.5
NHANES II	15.1	7.9	18.4	11.3
NHANES III	27.2	13.4	30.7	16.2
Ages 12 to 17 y				
All races[b]				
NHES	15.1	5.2	15.2	5.2
NHANES I	14.9	5.3	19.7	7.2
NHANES II	16.3	5.4	15.5	6.0
NHANES III	21.7	12.8	21.2	8.8
White				
NHES	15.8	5.4	15.0	5.0
NHANES I	15.3	5.5	19.7	6.6
NHANES II	16.6	5.4	15.2	5.3
NHANES III	22.6	14.4	20.3	8.4
Black				
NHES	10.4	3.7	16.5	6.6
NHANES I	12.3	4.3	20.8	11.2
NHANES II	14.5	6.3	18.2	10.4
NHANES III	23.3	9.4	29.9	14.4

[a] Based on sex- and age-specific percentile cutoffs derived from NHES II and III. NHES indicates National Health Examination Survey; 1963 to 1965 for ages 6 to 11 years, and 1966 to 1970 for ages 12 to 17 years for III; NHANES, National Health and Nutrition Examination Survey; 1971 to 1974 for I, 1976 to 1980 for II, and 1988 to 1991 for III.

[b] Includes data for race groups not shown separately.

From Troiano RP, Flegal KM, Kuczmarski RJ, Campbell M, Johnson CL. *Arch Pediatr Adolesc Med* 149: 1085-1089, 1995. With permission.

TABLE 70.3

Anthropometric Definitions of Childhood Overweight/Obesity

Definition	Ref.
Body weight >120% of the value predicted from height	3
Body mass index (BMI) >85% percentile	2
Body mass index (BMI) >95% percentile	2
Triceps skinfold thickness >85% percentile	4
Triceps skinfold thickness >95% percentile	4
Body fat >25% for boys and 30% for girls as estimated from sum of subscapular and triceps skinfolds	5

TABLE 70.4

Hormonal and Genetic Causes of Childhood Obesity

Hormonal Causes	Diagnostic Clues
Hypothyroidism	Increased TSH, decreased thyroxine (T_4) levels
Hypercortisolism	Abnormal dexamethasone test; increased 24-hour free urinary cortisol level
Primary hyperinsulinism	Increased plasma insulin, increased C-peptide levels
Pseudohypoparathyroidism	Hypocalcemia, hyperphosphatemia, increased PTH level
Acquired hypothalamic	Presence of hypothalamic tumor, infection, syndrome trauma, vascular lesion
Genetic Syndromes	**Associated Characteristics**
Prader-Willi	Obesity, unsatiable appetite, mental retardation, hypogonadism
Laurence-Moon-Biedel	Obesity, mental retardation, spastic paraplegia
Alstrom	Obesity, retinitis pigmentosa, deafness, diabetes mellitus
Cohen	Truncal obesity, mental retardation, hypotonia
Turner's	Short stature, undifferentiated gonads, cardiac abnormalities, obesity, X genotype
Weaver	Infant overgrowth syndrome, accelerated skeletal maturation

Adapted from Moran R. Evaluation and treatment of childhood obesity, *Am Fam Phys* 59: 861; 1999. With permission.

TABLE 70.5

Differential Diagnosis of Childhood Obesity

	Hormonal/Genetic	Idiopathic
Family	Obesity not common	Obesity common in family
Height	Short child	Tall child (>50%)
IQ	IQ often low	Normal IQ
Bone age	Bone age retarded	Normal bone age
Physical	Defects common	Normal physical exam

Adapted from Williams CL, Campanaro LA, Squillace M, Bollella M. *Ann NY Acad Sci* 817: 225; 1997. With permission.

TABLE 70.6

Smoothed 85th and 95th Percentiles of Body Mass Index from NHANES I[a] Male Subjects 6 to 18 Years

	Whites						Blacks						Population					
Age	**n**	**5th**	**15th**	**50th**	**85th**	**95th**	**n**	**5th**	**15th**	**50th**	**85th**	**95th**	**n**	**5th**	**15th**	**50th**	**85th**	**95th**
Males																		
6	117	12.93	13.46	14.62	16.52	17.75	47	12.68	13.66	14.49	16.83	18.58	165	12.86	13.43	14.54	16.64	18.02
7	122	13.30	13.88	15.15	17.31	18.98	40	13.11	14.03	14.98	17.29	19.56	164	13.24	13.85	15.07	17.37	19.18
8	117	13.67	14.31	15.70	18.10	20.22	30	13.54	14.41	15.49	17.76	20.51	149	13.63	14.28	15.62	18.11	20.33
9	121	14.04	14.75	16.24	18.88	21.45	55	13.98	14.81	16.00	18.26	21.45	177	14.03	14.71	16.17	18.85	21.47
10	146	14.42	15.19	16.79	19.67	22.66	29	14.41	15.21	16.53	18.78	22.41	177	14.42	15.15	16.72	19.60	22.60
11	122	14.81	15.64	17.35	20.47	23.87	44	14.86	15.62	17.06	19.32	23.42	169	14.83	15.59	17.28	20.35	23.73
12	153	15.21	16.11	17.93	21.28	25.01	50	15.36	16.06	17.61	19.85	24.39	204	15.24	16.06	17.87	21.12	24.89
13	134	15.69	16.65	18.57	22.12	26.06	42	15.89	16.64	18.28	20.62	25.26	177	15.73	16.62	18.53	21.93	25.93
14	131	16.16	17.22	19.25	22.97	27.02	42	16.43	17.22	18.94	21.54	26.13	173	16.18	17.20	19.22	22.77	26.93
15	128	16.57	17.79	19.94	23.82	27.86	43	16.97	17.79	19.56	22.50	27.05	175	16.59	17.76	19.92	23.63	27.76
16	131	17.00	18.35	20.63	24.63	28.69	40	17.51	18.37	20.19	23.45	27.95	172	17.01	18.32	20.63	24.45	28.53
17	133	17.29	18.72	21.13	25.44	29.50	33	17.86	18.77	20.70	24.41	28.89	167	17.31	18.68	21.12	25.28	29.32
18	91	17.50	18.95	21.46	26.08	29.89	28	18.05	19.03	21.09	25.06	29.35	120	17.54	18.89	21.45	25.92	30.02
Females																		
6	118	12.81	13.37	14.33	16.14	17.49	42	12.52	13.40	13.83	16.24	18.58	161	12.83	13.37	14.31	16.17	17.49
7	126	13.18	13.82	15.00	17.16	18.93	47	12.88	13.79	14.55	17.36	19.56	174	13.17	13.79	14.98	17.17	18.93
8	118	13.57	14.27	15.68	18.19	20.36	35	13.25	14.17	15.26	18.49	20.51	153	13.51	14.22	15.66	18.18	20.36
9	125	13.96	14.72	16.35	19.21	21.78	47	13.63	14.57	15.98	19.64	21.45	173	13.87	14.66	16.33	19.19	21.78
10	152	14.36	15.18	17.02	20.23	23.20	41	14.02	14.96	16.69	20.79	22.41	194	14.23	15.09	17.00	20.19	23.20
11	117	14.76	15.64	17.69	21.24	24.59	43	14.41	15.36	17.39	21.96	23.42	163	14.60	15.53	17.67	21.18	24.59
12	129	15.17	16.11	18.36	22.25	25.95	47	14.83	15.77	18.11	23.15	24.39	177	14.98	15.98	18.35	22.17	25.95
13	151	15.59	16.55	18.91	23.13	27.07	47	15.33	16.23	18.78	24.41	25.26	199	15.36	16.43	18.95	23.08	27.07
14	141	15.89	16.89	19.29	23.87	27.97	49	15.77	16.66	19.24	25.46	26.13	192	15.67	16.79	19.32	23.88	27.97
15	117	16.21	17.23	19.69	24.28	28.51	47	16.20	17.07	19.67	26.04	27.05	164	16.01	17.16	19.69	24.29	28.51
16	142	16.55	17.59	20.11	24.68	29.10	30	16.65	17.48	20.11	26.68	27.95	173	16.37	17.54	20.09	24.74	29.10
17	114	16.76	17.84	20.39	25.07	29.72	44	16.92	17.81	20.45	27.38	28.89	159	16.59	17.81	20.36	25.23	29.72
18	109	16.87	18.01	20.58	25.34	30.22	29	17.04	18.06	20.78	27.92	29.35	140	16.71	17.99	20.57	25.56	30.22

[a] NHANES is National Health and Nutrition Examination Survey.

From Must A, Dallal G, Dietz W. *Am J Clin Nutr* 53: 839; 1991. With permission.

TABLE 70.7

Smoothed 85th and 95th Percentiles of Triceps Skinfold Thickness from NHANES I[a] Male Subjects 6 to 18 Years

	Whites						Blacks						Population					
Age	**n**	**5th**	**15th**	**50th**	**85th**	**95th**	**n**	**5th**	**15th**	**50th**	**85th**	**95th**	**n**	**5th**	**15th**	**50th**	**85th**	**95th**
Males																		
6	117	5.26	6.09	8.74	11.63	14.47	47	4.01	4.86	6.85	9.35	12.86	165	5.04	6.19	8.36	11.10	14.12
7	122	5.28	6.12	8.94	12.78	15.95	40	4.01	4.88	6.85	10.09	14.11	164	5.01	6.14	8.59	12.38	15.61
8	117	5.28	6.15	9.12	13.95	17.51	30	4.00	4.88	6.84	10.76	15.35	149	4.96	6.08	8.79	13.66	17.18
9	121	5.27	6.17	9.27	15.10	19.11	55	3.99	4.88	6.83	11.37	16.50	177	4.91	6.02	8.96	14.93	18.81
10	146	5.24	6.18	9.40	16.29	20.96	29	3.98	4.88	6.81	11.52	17.79	177	4.84	5.95	9.10	16.02	20.68
11	122	5.20	6.20	9.51	17.32	22.53	44	3.97	4.89	6.81	11.31	18.68	169	4.78	5.88	9.23	16.87	22.20
12	153	5.15	6.23	9.59	17.79	23.53	50	3.97	4.91	6.80	10.79	18.74	204	4.69	5.79	9.35	17.26	23.25
13	134	5.01	6.21	9.42	17.63	23.87	42	3.94	4.88	6.72	10.23	18.67	177	4.56	5.65	9.17	17.12	23.71
14	131	4.91	6.15	9.26	16.88	23.42	42	3.86	4.84	6.66	9.92	18.58	173	4.47	5.60	8.93	16.35	23.46
15	128	4.81	6.10	9.12	16.11	22.42	43	3.81	4.80	6.62	9.96	18.99	175	4.40	5.59	8.70	15.75	22.34
16	131	4.69	6.05	8.95	15.81	22.05	40	3.76	4.77	6.58	10.30	20.18	172	4.33	5.55	8.45	15.75	21.53
17	133	4.61	6.02	8.92	15.95	21.99	33	3.69	4.72	6.63	10.73	21.12	167	4.29	5.58	8.38	15.95	21.51
18	91	4.53	6.01	9.02	16.69	22.28	28	3.60	4.64	6.79	11.34	21.95	120	4.25	5.63	8.53	16.59	21.83
Females																		
6	118	5.65	6.96	10.19	13.48	15.47	42	4.90	6.10	7.99	13.71	14.94	161	6.00	6.76	10.01	13.44	15.57
7	126	6.09	7.42	10.89	14.93	18.08	47	5.09	6.33	8.60	15.27	17.20	174	6.24	7.17	10.68	14.94	17.89
8	118	6.52	7.86	11.60	16.35	20.60	35	5.29	6.57	9.22	16.82	19.41	153	6.47	7.58	11.36	16.41	20.18
9	125	6.94	8.31	12.31	17.74	23.07	47	5.51	6.83	9.85	18.40	21.65	173	6.71	8.01	12.05	17.85	22.47
10	152	7.37	8.77	13.02	18.84	24.84	41	5.73	7.09	10.47	19.63	23.76	194	6.95	8.44	12.74	19.01	24.38
11	117	7.80	9.23	13.74	19.82	26.23	43	5.96	7.36	11.08	20.72	25.84	163	7.20	8.87	13.43	20.13	26.15
12	129	8.17	9.68	14.44	20.97	27.73	47	6.21	7.62	11.68	21.58	27.53	177	7.45	9.31	14.13	21.25	27.98
13	151	8.49	10.19	15.14	22.00	29.08	47	6.50	8.05	12.22	21.86	29.17	199	7.78	9.84	14.87	22.25	29.51
14	141	8.78	10.76	15.77	22.99	30.22	49	6.81	8.53	12.56	21.71	30.48	192	8.15	10.37	15.47	23.27	30.86
15	117	9.06	11.29	16.39	24.08	31.48	47	7.11	8.94	12.95	21.77	30.54	164	8.46	10.85	16.03	24.32	32.22
16	142	9.34	11.83	17.03	24.85	32.35	30	7.41	9.35	13.36	22.06	30.07	173	8.78	11.34	16.62	25.12	33.22
17	114	9.55	12.18	17.45	25.48	32.95	44	7.67	9.70	13.75	23.03	30.46	159	9.03	11.66	17.02	25.80	33.83
18	109	9.66	12.29	17.67	26.22	33.51	29	7.87	10.03	14.19	24.94	31.42	140	9.21	11.79	17.24	26.51	34.26

[a] NHANES is National Health and Nutrition Examination Survey.

From Must A, Dallal G, Dietz W. *Am J Clin Nutr* 53: 839; 1991. With permission.

TABLE 70.8

Regression Coefficients (β) and p Values from Linear Models for Body Mass Index (BMI) in Black, Mexican-American, and White Youths Age 6 to 24 Years, Third National Health and Nutrition Examination Survey (NHANES III), 1988 to 1994

	β (SE)	P
Girls		
Main Effects		
Ethnicity		
Black	1.20 (0.26)	<0.001
Mexican-American	0.85 (0.33)	0.01
SES[a]	–0.23 (0.06)	<0.001
Age	0.46 (0.04)	<0.001
Boys		
Main Effects		
Ethnicity		
Black	–0.10 (0.21)	0.66
Mexican-American	0.48 (0.25)	0.06
SES[a]	–0.13 (0.09)	0.15
Age	0.44 (0.04)	<0.001

[a] Socioeconomic status as measured by years of education of head of household

Adapted from Winkleby MA, Robinson TN, Sundquist J, Kraemer HC. *JAMA* 281: 1006; 1999.

TABLE 70.9

Visceral Adipose Tissue in White and African-American Youth

Age	Boys	Girls	Boys	Girls	Significant Effects[a]	Ref.
4-10 y	27 ± 16 cm^2 (n = 16)	54 ± 27 cm^2 (n = 20)	22 ± 17 cm^2 (n = 27)	28 ± 17 cm^2 (n = 38)	race × sex	10[b]
13-16 y	433 ± 105 cm^3 (n = 10)	355 ± 151 cm^3 (n = 15)	279 ± 107 cm^3 (n = 16)	263 ± 109 cm^3 (n = 38)	race	11[c]

[a] Significant effects by two-way ANOVA, $p < 0.05$.

[b] Visceral adipose tissue measured as single-slice computed tomography scan at the level of the umbilicus and expressed in cm^2.

[c] Visceral adipose tissue measured as the sum of five abdominal MRI scans and expressed in cm^3.

Source: Goran MI, Nagy TR, Treuth MS, et al. *Am J Clin Nutr* 65: 1703; 1997. With permission.

socioeconomic status is predictive of greater BMI in girls but not boys.[9] White children appear to accumulate greater quantities of deleterious visceral adipose tissue than do African-American children (Table 70.9).[10-11]

Health Risks of Childhood Obesity

Although obesity-related health problems such as coronary artery disease, hypertension, and diabetes tend to present their clinical manifestations in adulthood, these disorders

TABLE 70.10

Health-Related Risk Factors Associated with Childhood Obesity

Risk Factor	References
Elevated triglycerides	13,14
Elevated LDL cholesterol	15
Reduced HDL cholesterol	13,5
Elevated blood pressure	17
Elevated insulin	14,15
Elevated left ventricular mass	18
Endothelial dysfunction	19
Orthopedic abnormalities (e.g., Blount's disease)	20,21
Gallstone formation	22,23
Asthma and reduced pulmonary function	24,25
Sleep disorders	26,27

TABLE 70.11

Visceral Adiposity and Increased Health Risk in Childhood Obesity

Health Risk	References
Elevated LDL cholesterol and triglycerides associated with visceral but not subcutaneous fat in obese children	28
Elevated triglycerides and insulin and lowered HDL cholesterol associated with visceral fat in obese but not in non-obese adolescents	29
Visceral fat, but not subcutaneous or total body fat, explained significant proportions of the variance in triglycerides, HDL cholesterol, and LDL particle size in obese children	30

have their beginnings in childhood,[12] with obese children tending to have a poorer risk profile than their normal-weight counterparts. A variety of additional health problems occur at significantly greater frequencies in obese children (Table 70.10).[13-27] Children with increased visceral adiposity may be at especially high risk (Table 70.11).[28-30]

Etiology of Childhood Obesity

Heredity

Feeding studies with identical twins have helped to elucidate the role of genetic background in determining obesity. When groups of identical twins are exposed to overfeeding, significantly more variance occurs in the response between pairs of twins than within pairs for the changes in body weight, suggesting a genetic component for susceptibility to weight gain.[31] Still, results from family studies suggest that the maximal heritability of obesity phenotypes ranges from about 30 to 50%,[32] indicating that non-genetic factors also play an important role. Quantifying the influence of the non-genetic factors has proven difficult, however.

Diet

Definitive data on the relationship between free-living energy intake and childhood obesity are lacking. Interestingly, as the prevalence of childhood obesity has increased over the past two decades, the reported mean energy intakes of 6- to 11-year-olds showed a

TABLE 70.12

Mean Energy Intake by Age, 1976-1980 and 1988-1991

	NHANES II (1976-1980)		NHANES III (1988-1991)		
Age	n	Energy kj (kcal)	n	Energy kj (kcal)	Percent Change
1-2 y	1417	5385 (1287)	1231	5393 (1289)	<1
3-5 y	2345	6565 (1569)	1547	6657 (1591)	1
6-11 y	1725	8201 (1960)	1745	7937 (1897)	–3

Adapted from Briefel RR, McDowell MA, Alaimo K, Caughman CR, Bischof AL, Carroll MD, Johnson CL. *Am J Clin Nutr* 62: 1072S; 1995.

slight decline from NHANES II (1976-1980) to NHANES III (1988-1991) (Table 70.12).[33] With respect to fat intake, most,[34-38] but not all, juvenile studies[39,40] support the idea that diets high in fat are associated with body fatness or gain in weight. As early as age three, children of obese parents demonstrate an increased preference for high-fat foods.[41] Data also suggest that food preferences developed in childhood tend to track into aduldhood.[42]

Resting Metabolism

Some studies that measured resting metabolic rate in children utilizing 30-minute measures of oxygen consumption support the notion that a lower than normal resting metabolism contributes to childhood obesity.[43,44] On the other hand, two recent reviews of the isotope dilution (doubly-labeled water) method for measuring energy expenditure concluded that low levels of resting energy expenditure are probably not responsible for obesity in most children.[45,46]

Physical Inactivity

Several lines of evidence suggest the importance of physical inactivity in childhood obesity. Preschoolers who were classified as inactive were 3.8 times as likely as active children to have increasing triceps skinfold slopes during the average 2.5 years of followup.[47] An analysis of doubly-labeled water studies concluded that low levels of physical activity were associated with higher levels of body fatness.[45] Time–motion studies have shown that inactive children are more fat than active children,[48,49] even while ingesting less energy.[50]

Treatment of Childhood Obesity

Family Involvement

Studies that have examined successful five-year[51] and ten-year[52] outcomes for childhood obesity interventions emphasize the importance of family involvement in the weight loss process and recommend a treatment model that integrates improved dietary habits, increased physical activity, and behavior modification. Active parental participation is a crucial component of the model, given that parenting styles not only influence the development of food preferences but establish the type of family environment that may be conducive to overeating or a sedentary lifestyle. Parents also function as role models and reinforcers for eating and exercise behaviors. Family-based behavioral interventions for childhood obesity typically

include an initial, short-term (8 to 16 weeks) treatment phase followed by a longer-term (1 year) maintenance or continued improvement phase.[53] During initial treatment, children typically meet in group settings once per week for 45 to 90 minutes.[51,54] Follow-up sessions usually occur once or twice per month for up to one year.[54,55] Facilitators for treatment sessions may be pediatricians, child psychologists, or nutritionists.[53]

Dietary Intervention

A variety of dietary approaches have been utilized in family-based childhood obesity interventions; some focus on healthy eating habits rather than energy restriction, while others prescribe significant caloric reduction. From a behavior change perspective, the specific content of the diet may be less important than how it is presented. The diet should be simple, explicit, and unambiguous, so that it is easy to implement and monitor, and not subject to confusion or easy rationalization of exceptions.[56] A trained nutritionist can assist the family in evaluating their cooking and eating patterns and by making suggestions relative to the purchasing and preparing of foods. The nutritionist should also help the family understand the concepts of portion size, nutritional contents of foods, and the use of food exchange lists such as the *Exchange Lists for Weight Management*.[57] It is recommended that obese children practice maintaining a food diary. Although in most cases the diary will be inaccurate in figuring total energy intake, it can prove useful for reviewing problem foods and eating patterns.[1,58] Dietary fiber is also useful in the treatment of childhood obesity, as it increases satiety and often displaces fat in the diet.[3,59] The recommended daily dietary intake of fiber for children aged 3 to 20 years is equivalent to their age in years plus 5 g per day, as the minimum, and increases to age in years plus 10 g per day as the maximum.[60] The "age + 10" upper level of fiber intake is the recommended goal for the obese child.[3] Table 70.13 summarizes the dietary component from several successful family-based behavioral interventions for childhood obesity. By way of note, The Traffic Light Diet was designed to maximize healthy food choices, decrease energy intake, and encourage individual control over the diet.[55] Foods are categorized as red, yellow, or green on the basis of their kcalorie and nutrient content. Green foods (primarily vegetables) are very low in kcalories. Yellow foods (skim milk, apple) are higher in

TABLE 70.13

Selected Family-Based Interventions for Childhood Obesity

Ages	n	Diet	Exercise	Outcome	Ref
12-16	36	Nutrition education, emphasis on low sugar, salt, and fat	Increasing physical activity encouraged	(1 y) % Overweight declined by 10.7%	54
7-17	4	Hypocaloric diets (600 kcal/d for 10 wk); 1200 kcal/d next 42 wk	Gradual increase in physical activity using aerobic points system	(1 y) % Overweight declined by 20%	61
8-12	18	Traffic Light Diet, target of 1000-1200 kcal/d	Children reinforced for decreasing sedentary activities	(1 y) % Body fat (electrical impedance) declined 4.7%	55
7-16	60	Nutrition education, emphasis on healthy eating rather than eating less	Moderate intensity exercise for 30 min/d, lifestyle changes to increase activity	(1 y) % Overweight declined by 11%	63
8-16	12	Modified Traffic Light Diet	Moderate intensity exercise for up to 45 min/d, 5-7 d/wk	(5 y) % Overweight declined by 23%	51

TABLE 70.14

Two Studies of After-School Exercise (without Dietary Intervention) in the Treatment of Childhood Obesity

Subjects (n)	Obesity Status[a]	Exercise Program	Results	Ref.
7-11 y girls (n = 25)	Ex = 42.8% fat C = 43.3% fat	Ex = aerobics, 5 d/wk, 12 wk C = lifestyle education only	-1.4% fat[b] + 0.4% fat[c]	68
7-11 y boys and girls (n = 74)	Ex = 44.5% fat C = 44.1% fat	Ex = aerobics, 5 d/wk, 17 wk C = maintain normal activities	-2.2% fat[b] no change	69

[a] Ex is exercise group, C is control group. % Fat is percentage of body fat measured with dual-energy x-ray absorptiometry.
[b] Significant change ($p<0.05$) from pre-test.
[c] Non-significant change ($p>0.05$) from pre-test.

kcalories and include the dietary staples needed for a balanced diet. Red foods (potato chips, candy) are foods higher in kcalories, with low nutrient density.

Exercise

Although energy restriction can result in significant short-term weight loss in obese children, it tends to reduce fat-free mass and resting metabolic rate,[64] thus setting the stage for regain of the lost fat when the dieting stops.[65] Recent data suggest that exercise (walking 2 to 3 miles per day, 5 days per week) offers metabolic benefits to obese children during diet-induced weight loss by reversing the potentially adverse changes in protein turnover consequent to the hypocaloric diet.[66] Data also suggest that adding exercise to a diet restriction regimen can result in a total daily energy expenditure above that predicted by the addition of the exercise.[67] Within the context of after-school physical activity programs, two recent studies demonstrated the efficacy of exercise alone (no dietary intervention) in reducing childhood obesity (Table 70.14).[68,69] The study by Owens et al.[69] also showed the efficacy of exercise alone in attenuating the increase in visceral adipose tissue in obese children.

Optimal Exercise Dose

Little is known about the optimal dose of exercise for the treatment of childhood obesity. The National Association for Sport and Physical Education recommends that children obtain at least one to two hours of exercise each day.[70] Examination of Tables 70.15 and 70.16 suggests that the exercise component of successful obesity treatment programs can vary widely and may fall short of the recommended one to two hours of daily exercise. For the obese child, increasing the level of physical activity gradually may be an important consideration, so that failure and discouragement do not sabotage both exercise and dietary resolve.[3] Adding 20 to 30 minutes per day of moderate physical activity to the obese child's routine would appear to be a reasonable initial goal. Exercise programs should be designed to increase the interaction between parents and children or with other children.[1] Behavioral research also suggests that obese children are more likely to continue being physically active over time if they perceive themselves as having choice and control over being more physically active, rather than attributing control to their parents.[55]

TABLE 70.15

Behavior Modification Components for Treatment of Childhood Obesity

Component	Comment
Self-monitoring	Utilize food and physical activity log books; monitor body weight changes (usually daily or weekly)
Stimulus control	Limit amount of high-kcalorie foods kept in house; establish set routines for meals and snacks; model physically active lifestyle
Reinforcement	Reward targeted behaviors by child with verbal praise by parents; include predetermined (with input from child) tangible rewards that encourage further physical activity (sporting equipment, trips to recreational areas) or healthy eating habits (favorite fruits)
Eating behavior	Avoid second helpings, take smaller bites, put down fork between bites, leave food on plate at meal's end, avoid TV watching while eating
Goal setting and contracting	Parent and child establish realistic goals for physical activity, weight loss, and eating behaviors; use contracts to help maintain focus and provide structure for rewarding desired changes
Managing high-risk situations	Pre-plan management strategies for holidays, birthdays, parties, and eating in restaurants; practice using role playing

Behavior Modification

The work of Epstein et al.[52,71] represents the current state of the art in behavioral treatment of childhood obesity. They have systematically studied a progression of behavioral methods and demonstrated beneficial long-term effects on weight control.[56] Table 70.15 summarizes components of behavior modification commonly utilized in the treatment of childhood obesity.

Resources on Childhood Obesity

Table 70.16 provides a topical list of sources of information on childhood obesity.

TABLE 70.16

Selected Resources on Childhood Obesity

Topic	Resource
Recent review articles on childhood obesity	References 1, 3, 53, 56, 58,
Recent books on childhood obesity	1. Smith, J.C., *Understanding Childhood Obesity,* University Press of Mississippi, 1999 2. Williams, C.L. (ed), *Prevention and Treatment of Childhood Obesity,* New York Academy of Sciences, 1993 3. Krasnegor, N.A., *Childhood Obesity: A Biobehavioral Perspective,* The Telford Press, 1990
Obesity-related professional journals	1. *International Journal of Obesity and Related Metabolic Disorders* 2. *Obesity Research*
Obesity-related research organizations	1. Center for Child and Adolescent Obesity (University of California, San Francisco) 2. Weight Control Information Network (National Institutes of Health, Bethesda, MD) 3. The National Institute of Child Health and Human Development (National Institutes of Health, Bethesda, MD) 4. European Childhood Obesity Group (Lyon, France)
Childhood exercise and fitness	1. *Pediatric Exercise Science* 2. American College of Sports Medicine (Indianapolis, IN) 3. American Alliance for Health, Physical Education, Recreation and Dance (Reston, VA) 4. President's Council on Physical Fitness and Sports (Washington, D.C.)
Childhood nutrition	1. The American Dietetic Association (Chicago, IL) 2. Food and Nutrition Information Center, U.S. Department of Agriculture 3. American Academy of Pediatrics

References

1. Strauss R. *Curr Prob Pediatr* 29: 5; 1999.
2. Troiano RP, Flegal KM, Kuczmarski RJ, et al. *Arch Pediatr Adolesc Med* 149: 1085; 1995.
3. Williams CL, Campanaro LA, Squillace M, Bollella M. *Ann NY Acad Sci* 817: 225; 1997.
4. Must A, Dallal G, Dietz W. *Am J Clin Nutr* 53: 839; 1991.
5. Williams D, Going S, Lohman T, et al. *Am J Pub Health* 82: 358; 1992.
6. Power C, Lake JK, Cole TJ. *Int J Obes Relat Metab Disord* 21: 507; 1997.
7. Himes JH, Dietz WH. *Am J Clin Nutr* 59: 307; 1994.
8. Bellizzi MC, Dietz WH. *Am J Clin Nutr* 70: 173S; 1999.
9. Winkleby MA, Robinson TN, Sundquist J, Kraemer HC. *JAMA* 281: 1006; 1999.
10. Goran MI, Nagy TR, Treuth MS, et al. *Am J Clin Nutr* 65: 1703; 1997.
11. Owens S, Gutin B, Barbeau P, et al. *Obes Res* [in press].
12. Berenson GS, Srinivasan SR, Bao W. *Ann NY Acad Sci* 817: 189; 1997.
13. Gidding SS, Bao W, Srinivasan SR, Berenson GS. *J Pediatr* 127: 868; 1995.
14. Gutin B, Islam S, Manos T, et al. *J Pediatr* 125: 847; 1994.
15. Kikuchi DA, Srinivasan SR, Harsha DW, et al. *Prev Med* 21: 177; 1992.
16. Gutin B, Owens S, Treiber F, et al. *Arch Pediatr Adolesc Med* 151: 462; 1997.
17. Lauer RM, Burns TL, Clarke WR, Mahoney LT. *Hypertension* 18: I74S; 1991.
18. Gutin B, Treiber F, Owens S, Mensah G. *J Pediatr* 132: 1023; 1998.
19. Treiber F, Papavassiliou D, Gutin B, et al. *Psychosom Med* 59: 376; 1997.
20. Loder RT, Aronson DD, Greenfield ML. *J Bone Joint Surg* 75: 1141; 1993.
21. Dietz WH, Gross WL, Kirkpatrick JA. *J Pediatr* 101: 735; 1982.

22. Friesen CA, Roberts CC. *Clin Pediatr* 7: 294; 1989.
23. Honore LH. *Arch Surg* 115: 62; 1980.
24. Unger R, Kreeger L, Christoffel KK. *Clin Pediatr* 29: 368; 1990.
25. Kaplan TA, Montana E. *Clin Pediatr* 32: 220; 1993.
26. Mallory GB, Fiser DH, Jackson R. *J Pediatr* 115: 892; 1989.
27. Marcus CL, Curtis S, Koerner CB, et al. *Pediatr Pulmonol* 21: 176; 1996.
28. Brambilla P, Manzoni P, Sironi S, et al. *Int J Obes Relat Metab Disord* 18: 795; 1994.
29. Caprio S, Hyman LD, McCarthy S, et al. *Am J Clin Nutr* 64: 12; 1996.
30. Owens S, Gutin B, Ferguson M, et al. *J Pediatr* 133: 41; 1998.
31. Bouchard C, Tremblay A, Despres JP, et al. *N Engl J Med* 322: 1477; 1990.
32. Perusse L, Bouchard C. *Ann Med* 31: 19; 1999.
33. Briefel RR, McDowell MA, Alaimo K, et al. *Am J Clin Nutr* 62: 1072S; 1995.
34. Eck LH, Kleges RC, Hanson CL, Slawson D. *Int J Obes Relat Metab Disord* 16: 71; 1992.
35. Gazzaniga JM, Burns T. *Am J Clin Nutr* 58: 21; 1993.
36. Maffeis C, Schutz Y. *Int J Obes Relat Metab Disord* 20: 170; 1996.
37. Nguyen VT, Larson DE, Johnson RK, Goran MI. *Am J Clin Nutr* 63: 507; 1996.
38. Tucker LA, Seljaas GT, Hager RL. *J Am Dietetic Assn* 97: 981; 1997.
39. Davies PSW, Wells JCK, Fieldhouse CA, Day JME, Lucas A. *Am J Clin Nurtr* 61: 1026; 1995.
40. Muecke L, Simons-Morton B, Huang IE, Parcel G. *J School Health* 62: 19; 1992.
41. Fisher JO, Birch LL. *J Am Diet Assoc* 95: 759; 1995.
42. Kelder SH, Perry CL, Klepp KI, Lytlle LL. *Am J Pub Health* 84: 1121; 1994.
43. Kaplan AS, Zemel BS, Stallings VA. *J Pediatr* 129: 643; 1996.
44. Morrison JA, Alfaro MP, Khoury P, et al. *J Pediatr* 129: 637; 1996.
45. DeLany JP. *Am J Clin Nurtr* 68: 950S; 1998.
46. Goran MI, Sun M. *Am J Clin Nutr* 68: 944S; 1998.
47. Moore LL, Nguyen UDT, Rothman KJ, et al. *Am J Epidemiol* 142: 982; 1995.
48. Goran MI, Hunter G, Nagy TR, Johnson R. *Int J Obes Relat Metab Disord* 21: 171; 1997.
49. Maffeis C, Zaffanello M, Schutz Y. *J Pediatr* 131: 288; 1997.
50. Deheeger M, Rolland-Cachera MF, Fontvielle AM. *Int J Obes Relat Metab Disord* 21: 372; 1997.
51. Johnson WG, Hinkle LK, Carr RE, et al. *Obes Res* 5: 257; 1997.
52. Epstein LH, Valoski A, Wing RR, McCurley J. *Health Psychol* 13: 373; 1994.
53. Owens S, Gutin B. In: *Lifestyle Medicine* Rippe, JM, Ed, Blackwell Science, Malden, MA, 1999; ch 50.
54. Brownell KD, Kelman JH, Strunkard AJ. *Pediatrics* 71: 515; 1983.
55. Epstein LH, Valoski AM, Vara LS, et al. *Health Psychol* 14: 109; 1995.
56. Robinson TN. *Int J Obes Relat Metab Disord* 23: 52S; 1999.
57. American Dietetic Association and American Diabetes Association, *Exchange Lists for Weight Management*, Revised ed, Chicago, 1995.
58. Moran R. *Am Fam Phys* 59: 861; 1999.
59. Kelsay JL, Behall KM, Prather ES. *Am J Clin Nutr* 31: 1149; 1978.
60. Williams CL. *Pediatrics* 5: 985S; 1995.
61. Figueroa-Colon R, Almen K, Franklin F, et al. *Am J Dis Child* 147: 160; 1993.
62. Epstein LH, Valoski AM, Vara LS, et al. *Health Psychol* 14: 109; 1995.
63. Braet C, van Winckel M, van Leeuwen K. *Acta Paediatr* 86: 397; 1997.
64. Maffeis C, Schutz Y, Pinelli L. *Int J Obes Relat Metab Disord* 16: 41; 1992.
65. Schwingshandl J, Borkenstein M. *Int J Obes Relat Metab Disord* 19: 752; 1995.
66. Ebbeling CB, Rodriguez NR. *Med Sci Sports Exerc* 31: 378; 1999.
67. Blaak EE, Westerterp KR, Bar-Or O, et al. *Am J Clin Nutr* 55: 777; 1992.
68. Gutin B, Cucuzzo N, Islam S, et al. *Obes Res* 3: 305; 1995.
69. Owens S, Gutin B, Allison J, et al. *Med Sci Sports Exerc* 31: 143; 1999.
70. National Association for Sport and Physical Education (NASPE), Physical activity guidelines for pre-adolescent children, 1998.
71. Epstein LH, Valoski A, Wing RR, McCurley J. *JAMA* 264: 2519; 1990.