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Nutrition interventions for youth with dyslipidemia: a National Lipid Association clinical perspective

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Abstract: A heart-healthy lifestyle, beginning at an early age and sustained throughout life, may reduce risk for cardiovascular disease in youth. Among youth with moderate to severe dyslipidemia and/or those with familial hypercholesterolemia, lipid-lowering medications are often needed for primary prevention of cardiovascular disease. However, lifestyle interventions are a foundation for youth with dyslipidemia, as well as those without dyslipidemia. There are limited data supporting the use of dietary supplements in youth with dyslipidemia at this time. A family-centered approach and the support of a multi-disciplinary healthcare team, which includes a registered dietitian nutritionist to provide nutrition counseling, provides the best opportunity for primary prevention and improved outcomes. While there are numerous guidelines that address the general nutritional needs of youth, few address the unique needs of those with dyslipidemia. The goal of this National Lipid Association Clinical Perspective is to provide guidance for healthcare professionals caring for youth with disorders of lipid and lipoprotein metabolism, including nutritional guidance that complements the use of lipid lowering medications.

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This NLA clinical perspective will address the following key clinical questions

1. **Why are targeted nutritional interventions for youth necessary and is there evidence that they improve outcomes? How do the needs of youth with dyslipidemia differ from those of their peers? (Sections II and III)**
2. **Are there specific nutritional interventions for youth with dyslipidemia caused by a genetic variant? Acquired causes of dyslipidemia? (Section IV)**
3. **Does being under- or overweight alter the nutritional management of youth with dyslipidemia? (Section V)**
4. **Is there a role for use of dietary supplements in the management of youth with dyslipidemia? If so, is there evidence to support their safety and efficacy? (Section VI)**
5. **What are the psychosocial implications of dyslipidemia in youth and the need for adherence to a heart-healthy lifestyle, including proper nutrition? Is there evidence that altering dietary intake early in life may be harmful? (Section VII)**

Introduction

It is well known that atherosclerosis begins in childhood and accelerates by age 20.[1] Lifestyle interventions, including a heart-healthy dietary pattern, daily moderate-to-vigerous physical activity, maintaining a healthy body weight, and avoiding tobacco use, are the cornerstone of cardiovascular disease (CVD) risk reduction in youth with acquired and genetic dyslipidemia.[2-4] When adopted early and sustained over a lifetime, heart-healthy lifestyle habits are critical in maintaining overall health and reducing risk of premature CVD and CVD-related events. Acceptable and elevated levels of blood lipids in youth are shown in Table 1. Several professional guidelines address the nutritional needs of youth but few provide detailed recommendations, particularly for those with disorders of lipid and lipoprotein metabolism. This National Lipid Association (NLA) Clinical Perspective provides practical recommendations for healthcare professionals for nutrition interventions for youth with a variety of both acquired and genetic lipid/lipoprotein disorders. A summary of nutrition interventions for youth by lipid/lipoprotein disorder is provided in Table 2.

Implementation and efficacy of nutrition interventions

Nutrition interventions in youth with dyslipidemia provide short- and long-term benefits without adverse effects on growth or maturation.[5-9] Recent guidelines, including the Dietary Guidelines for Americans (DGAs), outline a variety of heart-healthy dietary patterns, including the Mediterranean diet, Dietary Approaches to Stop Hypertension (DASH), Healthy US-style diet, and vegetarian-style dietary patterns.[3] counseling by a registered dietitian nutritionist (RDN) is strongly recommended to help youth and their families successfully alter dietary intake to meet nutritional needs, provide ongoing support, and encourage long-term adherence to healthy nutrition habits.[2,4,10] A shared decision-making model of family-centered care is critical which, ideally, includes the child. Nutrition interventions include detailed recommendations for dietary changes based upon age- and gender-specific nutrient needs, dietary patterns, cultural norms, the family's food preferences, as well as food allergies or sensitivities.[2] Assessing willingness to change, identifying potential barriers, including food cost and access, and setting realistic goals are also fundamental for successful nutrition changes.[11]

Components of a heart-healthy lifestyle

Saturated and unsaturated fatty acids

Saturated fatty acids (SFAs) have a substantial effect on plasma lipids.[12-14] Table 3 illustrates the SFA content of several common foods. In the United States (U.S.), the leading sources of SFA for youth 1 year of age and older include sandwiches (e.g., breakfast sandwiches, hamburgers,

Table 1 Acceptable and elevated levels of blood lipids in youth <18 years of age[2].

Test	Acceptable	Borderline[a]	High[b]
TC	<170	170–199	≥200
LDL-C	<110	110–129	≥130
TG			
0–9 yrs	<75	75–99	≥100
10–19 yrs	<90	90–129	≥130
	Acceptable	**Borderline low**	**Low[c]**
HDL-C	>45	40–45	<40

***All values are listed in mg/dL**
Abbreviations: TC=total cholesterol; LDL-C=low-density lipoprotein cholesterol; TG=triglycerides; HDL-C=high-density lipoprotein cholesterol
Percentiles: [a]75th.
[b]95th.
[c]10th.

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Table 2 Summary of nutrition interventions for youth by disorder.

Lipid Disorder	Nutrition Intervention
Familial Hypercholesterolemia (FH)	• FH is a common genetic disorder characterized by moderate-to-severe elevations of LDL-C that increase the likelihood of premature CHD. • While LLM is generally required to reduce LDL-C and non-HDL-C, nutrition interventions to reduce SFA to <7% daily caloric intake, avoidance of TFA, <200 mg/day of dietary cholesterol, and increased dietary soluble fiber provides additional benefits. Supplements, such as soluble fiber or plant sterols, may also be beneficial. • All diet modifications should exist in the context of an age-appropriate dietary pattern, with adequate calorie and macro- and micronutrient intake to promote normal growth and development.
Familial Combined Hyperlipidemia (FCHL)	• FCHL is a common metabolic disorder characterized by: (a) elevated levels of Apo B that may present as either mixed hyperlipidemia, isolated hypercholesterolemia, HTG, or as normal serum lipid levels with an elevated level of Apo B; (b) intra-individual and intra-familial variability of the lipid phenotype; (c) an increased risk of premature CHD; and (d) a polygenic inheritance. • Nutrition interventions are similar to those for FH, with additional recommendations for TG lowering, such as reduction in foods containing simple carbohydrates and sugar sweetened beverages, as needed.
Elevated Lipoprotein(a) [Lp(a)]	• Serum Lp(a) reaches adult levels by school age and remains relatively constant into adulthood. • Elevated Lp(a) is recognized as a causal, independent risk factor for premature CVD. • While nutrition interventions do not significantly lower Lp(a), a lifelong heart-healthy lifestyle is helpful in minimizing additional CVD risk factors.

Sitosterolemia	• Hyperabsorption and decreased biliary excretion of cholesterol and non-cholesterol sterols leads to accumulation of serum sterols, such as campesterol and sitosterol.
	• Effective nutrition intervention includes:
	  ○ Dietary restriction of cholesterol and plant-based non-cholesterol sterols.
	  ○ Limiting intake or avoidance of shellfish (e.g., clams, scallops, oysters) and plant foods that are high in fat (e.g. vegetable oils, olives, margarine, nuts, seeds, avocados, and chocolate).
	  ○ Fruits, vegetables, and refined cereal products (not whole grain) may be used.
	• Margarines/spreads and other sterol- or stanol-fortified products are contraindicated.

Cerebrotendinous Xanthomatosis (CTX)	• CTX, characterized by impaired bile acid synthesis, leads to accumulation of cholestanol and cholesterol in many tissues, including the brain. • The treatment of choice for CTX is oral chenodeoxycholic acid therapy, although it is currently not approved by the FDA for this indication. • A dietary pattern low in cholestanol (egg yolks, meat, fish/shell fish and poultry, and high fat dairy), especially when implemented at a young age, may also be helpful.
Lysosomal Acid Lipase Deficiency (LAL-D)	• LAL-D is a rare autosomal recessive disease, the manifestations of which include a clinical continuum from infancy through adulthood. • The infantile form generally presents with severe failure to thrive, may require a low-fat, amino acid-based formula and, in the absence of timely enzyme replacement, is most often fatal. • Patients with childhood/adult-onset LAL-D may benefit from a dietary pattern with <25–30% daily caloric intake from fat and <200 mg dietary cholesterol daily. Fat-soluble vitamin supplementation may also be helpful in those who have malabsorption and malnutrition. • Enzyme replacement therapy with sebelipase alfa is recommended for the treatment of LAL-D. • Nutrition intervention is an important supportive measure to medical intervention and not a primary therapy to promote changes in lipid levels.

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Table 2 (*continued*)

Lipid Disorder	Nutrition Intervention
Hypobetalipoproteinemias	Abetalipoproteinemia (ABL) Homozygous ABL • A rare, inherited, autosomal-recessive disorder resulting from a microsomal triglyceride transfer protein deficiency characterized by the absence, or near absence, of LDL-C. • Disruption of cellular fat transport causes symptoms of fat malabsorption (steatorrhea, diarrhea) and failure to thrive, which often present in infancy or early childhood. • Dietary fat, cholesterol, and fat-soluble vitamins, such as A, E, D, and K, are poorly absorbed, leading to deficiencies. • A low-fat diet (20–30% daily caloric intake), adequate intake of EFAs (2–4% daily caloric intake) with supplementation as needed, and vitamin supplementation, are critical in nutritional management. These interventions are most effective when started at a young age, • Prognosis is variable, but early diagnosis and strict adherence to treatment can improve neurological function and halt disease progression Patients with heterozygous ABL usually have normal lipids. Hypobetalipoproteinemia (HBL) Homozygous HBL • A rare, inherited, autosomal co-dominant disorder resulting from mutations in both alleles of the *APOB;* characterized by the absence, or near absence, of LDL-C. • Disruption of cellular fat transport causes symptoms of fat malabsorption (steatorrhea, diarrhea) and failure to thrive, which often present in infancy or early childhood. • Dietary fat, cholesterol, and fat-soluble vitamins such as A, E, D, and K are poorly absorbed, leading to deficiencies. • A low-fat diet (20–30% daily caloric intake), adequate intake of EFAs (2–4% daily caloric intake) with supplementation as needed, and vitamin supplementation, are critical in management. These interventions are most effective when started at a young age, • Prognosis is variable, but early diagnosis and strict adherence to treatment can improve neurological function and halt disease progression Patients with heterozygous HBL typically have half-normal levels of Apo B-containing lipoproteins. Some may be at-risk of steatohepatitis.

Familial Chylomicronemia Syndrome (FCS) and Multifactorial Chylomicronemia Syndrome (MCS)	• Individuals with FCS have impaired or absent LPL activity caused by a monogenic variant; MCS, which is 50–100 times more common, occurs in individuals with co-existence of genetic and secondary causes.
	• Both FCS and MCS lead to severe elevations in TG (>1000 mg/dL).
	• The mainstay of treatment is a specialized dietary pattern:
	  ○ Very-low-fat <15–20 g per day (<10%–15% of total daily caloric intake) while meeting EFA needs (2–4% daily caloric intake).
	  ○ MCT oil to increase overall caloric intake and balance macronutrients in the dietary pattern, as needed.
	  ○ Emphasis on complex carbohydrate foods (e.g., oatmeal, brown rice, quinoa, beans) while limiting simple and refined carbohydrate foods.
	  ○ Avoidance of alcohol.
	  ○ Fat-soluble vitamin and mineral supplementation, as needed.

Familial Hypertriglyceridemia (FHTG)	• FHTG may be present in youth, typically in those with overweight or obesity and/or insulin resistance. • A low-fat diet (<30% calories from fat), limited intake of foods and beverages with added sugars, and the addition of complex carbohydrate foods and dietary sources of O3FAs is helpful in lowering TGs. • Promotion of a healthy weight is especially helpful in youth with overweight or obesity and/or insulin resistance.

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Table 2 (*continued*)

Lipid Disorder	Nutrition Intervention
Acquired Hypertriglyceridemia (HTG)	• Acquired HTG is closely associated with adverse lifestyle behaviors. • Nutrition intervention with lifestyle behaviors designed to achieve and maintain healthy body weight are the primary treatments for acquired HTG. • For youth with mild- to moderate-HTG, emphasis on vegetables, fruits, and lean protein intake with reduced intake of refind carbohydrate foods and foods/beverages with added sugar can be effective. • For youth with severe HTG (fasting TGs >1000 mg/dL), fat intake should be decreased to <10–15% of total daily caloric intake (<15–20 g per day) with avoidance of simple carbohydrates, such as foods and beverages with added sugars and 100% fruit juice.

Abbreviations: FH=familial hypercholesterolemia; LDL-C=low density lipoprotein cholesterol; CHD=coronary heart disease; LLM=lipid-lowering medication; non-HDL-C= non high density lipoprotein cholesterol; SFA=saturated fatty acids; TFA=*trans* fatty acids; FCHL=familial combined hyperlipidemia; Apo B=apolipoprotein B; HTG=hypertriglyceridemia; CVD=cardiovascular disease; TG=triglycerides; Lp(a)=lipoprotein(a); CTX=cerebrotendinous xanthomatosis; FDA=Food and Drug Administration; LAL-D=lysosomal acid lipase deficiency; ABL=abetalipoproteinemia; EFA=essential fatty acids; HBL=hypobetalipoproteinemia; FCS=familial chylomicronemia syndrome; LPL=lipoprotein lipase; MCS=multifactorial chylomicronemia syndrome; MCT=medium chain triglyceride; FHTG=familial hypertriglyceridemia; O3FAs=omega-3 fatty acids.

Table 3 Saturated fat content in select foods[15].

Food (serving size)	SFA (g/serving)
Lentils (1 cup, boiled)	0.1
Skim milk (1 cup)	0.1
Boneless, skinless chicken breast (3 oz, cooked)	0.9
Salmon, wild Atlantic (3 oz, cooked)	1.1
Low-fat yogurt, plain (6 oz)	1.7
Olive oil (1 Tbsp)	1.9
Sirloin steak (3 oz, lean only, cooked)	1.9
Egg (1 large, scrambled)	2.0
Ground beef (93% lean/7% fat) (3 oz, cooked)	3.3
Whole milk yogurt, plain (6 oz)	3.6
Field Roast® plant-based burger (Field Burger™) (one)	4.0
Whole milk (1 cup)	4.5
Ground beef (80% lean/20% fat) (3 oz, cooked)	5.6
Cheddar cheese (1 oz)	6.0
Palm oil (2 Tbsp)	6.7
Beyond Meat® plant-based burger (Classic Cookout™) (one)	7.0
Butter (1 Tbsp)	7.3
Bacon (3 oz, cooked)	11.7
Coconut oil (1 Tbsp)	11.8

Abbreviation: SFA=saturated fatty acid.

and tacos), and desserts and sweet snacks (e.g., ice cream and brownies).[3]

While there are subtle differences in the effect on levels of LDL-C by different types of SFAs, it is more helpful to discuss their effects as a class.[16] For youth older than 2 years with dyslipidemia, the recommended intake of SFA is <7% of daily caloric intake (approximately 8–20 g/day, depending on the child's caloric needs).[2,4] Use of plant-based, minimally processed foods can help to reduce SFA intake and assist with lowering total cholesterol (TC) and LDL-C.[17] It is important to note that coconut oil, while plant-based, is high in SFAs and is not recommended as part of a heart-healthy diet.[18]

Replacing foods rich in SFAs with foods rich in unsaturated fatty acids (UFAs) lowers TC and LDL-C by increasing the number of LDL receptors (LDLR) and increasing LDL clearance from the plasma.[13,16] Patients should be encouraged to replace foods rich in SFAs with plant- and marine-based foods rich in monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs).[4,11,19-20] (Table 4)

Trans fatty acids

Elimination of dietary artificial *trans* fatty acids (TFAs) is important in reducing health risks, especially in patients

Table 4 Unsaturated fat content in select foods[14].

Food (serving size)	PUFAs (g/serving)	MUFAs (g/serving)	Total UFAs (g/serving)
Nuts, average all varieties (1 oz)	6.2	6.3	12.5
Canola oil (1 Tbsp)	4.1	8.2	12.3
Peanuts, roasted (1 oz)	4.4	7.0	11.4
Olive oil (1 Tbsp)	1.4	9.8	11.2
Seeds, average all varieties (1 oz)	7.0	3.9	10.9
Avocado (1/2 medium)	1.2	6.7	7.9
Nut or seed butter, any variety (1 Tbsp)	2.1	4.7	6.8
Granola (1/4 cup)	3.3	2.3	5.6
Hummus (2 Tbsp)	1.0	1.1	2.1

Abbreviations: PUFA=polyunsaturated fatty acid; MUFA=monounsaturated fatty acid; UFA=unsaturated fatty acid.

Table 5 Cholesterol content of select foods[15].

Food (serving size)	Cholesterol (mg/serving)
Cheddar cheese (1 oz)	30
Butter (1 Tbsp)	31
Boneless, skinless chicken breast (3 oz, cooked)	42
Sirloin steak (3 oz, lean only, cooked)	49
Salmon, wild Atlantic (3oz, cooked)	60
Ground beef (80% lean/20% fat) (3 oz, cooked)	76
Ground beef (93% lean, 7% fat) (3 oz, cooked)	76
Bacon (3 oz, cooked)	94
Egg yolk (17 g)	184

with dyslipidemia. Replacement of SFAs and UFAs with TFAs increases LDL-C, triglycerides (TGs), apolipoprotein B (Apo B), and lipoprotein(a) [Lp(a)] and decreases high-density lipoprotein cholesterol (HDL-C).[21] Consumption of TFAs increases risk of atherosclerosis, although the mechanism(s) are incompletely understood.[14] While legislation has eliminated most dietary sources of artificial TFAs in the U.S., families should be taught to read nutrition facts labels and encouraged to avoid products that contain "partially hydrogenated" oils, which indicates the food contains articial TFAs.

Dietary cholesterol

Evidence supporting reduction in CVD risk by limiting dietary cholesterol is the subject of ongoing debate. In 2015, the NLA recommended a dietary pattern containing reduced amounts of cholesterol (<200 mg/day) to decrease LDL-C and non-HDL-C, especially for persons at high risk for CVD or with diabetes.[4] Reducing consumption of dietary cholesterol was included in recent recommendations from the American College of Cardiology and the American Heart Association, as well as the 2020–2025 DGAs.[3,11,22] Heart-healthy dietary patterns are typically low in dietary cholesterol because they are low in animal products rich in SFA, which may also be high in dietary cholesterol. Choosing plant-based protein sources also limits cholesterol intake.[23] Table 5 illustrates the cholesterol content of select foods.

Dietary soluble fiber

Viscous soluble fiber, which is not absorbed by the intestine, binds cholesterol and facilitates its excretion. For all individuals older than 2 years of age, the DGAs recommends a total daily fiber intake of 14 g per 1000 calories.[3] For youth with dyslipidemia, soluble fiber should account for at least 6 g/day for those 2–12 years old and 12 g/day or more for those older than 12 years of age.[2-4] Foods high in soluble fiber are shown in Table 6. Youth with inadequate dietary intake may benefit from soluble fiber supplementation.

Dietary omega-3 fatty acids

The impact of omega-3 fatty acids (O3FAs) on lipid levels and CVD risk reduction has been extensively investigated in adults; however, less is known of the benefits and potential side effects in youth. While there is debate on the benefit of dietary supplement O3FA, it is important to note that consumption of foods rich in O3FAs, particularly docosahexaenoic acid (DHA), is important for cerebral development and retinal function in young children, and is recommended as part of an overall heart-healthy dietary pattern.[3,24] While there are several O3FAs available in foods, most of the research has focused on the plant-derived alpha-linolenic acid (ALA) and the marine-derived O3FAs eicosapentaenoic acid (EPA) and DHA. Plant sources of ALA include chia seeds, walnuts, and soybean, flaxseed, and canola oils. Foods rich in EPA and DHA include fatty/oily fish (herring, fresh

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Table 6 Fiber content of select foods[15].

Food (serving size)	Soluble fiber (g/serving)	Total dietary fiber (g/serving)
Flaxseed (1/2 cup)	6.9	12.8
Kidney beans (1 cup, cooked)	5.7	11.4
Vegetarian baked beans (1 cup)	4.8	12.6
Avocado (1/2)	3.4	9.2
Fresh asparagus (1 cup, cooked)	3.4	5.6
Fresh broccoli (1 cup, cooked)	2.3	4.6
Pear (1 medium)	2.2	4.0
Fresh beets (1 cup, cooked)	2.0	3.4
Oats, rolled, uncooked (1/2 cup)	2	4
Pistachios (1/2 cup)	1.7	6.9
Fresh green beans (1 cup, cooked)	1.6	3.7
Kale (1 cup, cooked)	1.4	2.6
High-fiber whole wheat bread (1 slice)	1.3	3.0
Apple (one medium)	1.0	3.7

Table 7 Recommended fish serving sizes by age[26].

Age	Serving size*
2–3 years	1 oz
4–7 years	2 oz
8–10 years	3 oz
≥11 years	4 oz

*2 servings per week recommended.

water trout, salmon, and sardines), seaweed, algae, fortified foods, and cod liver oil.[25] Recommended daily intake for EPA and DHA has not been specified in youth.[3] However, the U.S. Food and Drug Administration (FDA) provides guidance on seafood serving sizes for individuals 2 years of age and older, as well as guidance on fish varieties low in mercury.[26] (Table 7) Two servings of low-mercury fish per week is recommended, with serving sizes based on an individual's age. Fish that contain high levels of mercury, such as king mackerel, orange roughy, and swordfish, should be avoided by most individuals.[26] Mozaffarian and Rimm reviewed studies that indicated a 36% lower risk of CVD death and 17% reduction in total mortality in adults who consumed 1–2 servings of fish per week, and noted 250 mg of EPA and DHA daily appears to be sufficient for primary prevention of coronary heart disease mortality.[27] Similar evidence is not available for youth. Guidance on use of O3FA supplementation in youth is discussed in section VI.

Refined carbohydrates and sugar-sweetened beverages

Current guidelines recommend replacing simple, refined carbohydrate foods with complex carbohydrate foods while limiting added sugar to <10% of daily caloric intake.[2-3,18] Individuals should limit the consumption of beverages high in sugar, including soda, flavored milk, sweet tea, and other sweetened drinks, as well as 100% fruit juice. In adults, consumption of ≥1 sugary beverage/day was associated with high TGs and low HDL-C.[28] Similarly, consumption of sugar-sweetened beverages in youth has been associated with development of obesity.[29-30]

Physical activity

Although the focus of this NLA Clinical Perspective is nutrition interventions, one of the foundations of a heart-healthy lifestyle is physical activity, which is discussed briefly. Physical activity lowers the risk for early death, coronary heart disease (CHD), stroke, hypertension (HTN), an abnormal lipid profile, metabolic syndrome, and type 2 diabetes (T2D).[31] Physical activity also assists in achieving and maintaining a healthy body weight and lowers serum lipid levels in youth.[32] At least 60 min of moderate-to-vigorous physical activity daily is recommended for all youth, including aerobic and bone- and muscle-strengthening activities.[2-3]

Nutrition intervention in disorders of lipid and lipoprotein metabolism

Cholesterol disorders

Familial hypercholesterolemia (FH)

Overview FH is a common, autosomal codominant genetic disorder with an estimated prevalence of 1:250 and is characterized by moderate to severe elevations of LDL-C, most commonly due to variants in the *LDLR, APOB*, and proprotein convertase subtilisin/kexin 9 (*PCSK9*) genes.[33] Lifelong exposure to increased levels of atherogenic lipoprotein particles significantly increases the development of CHD at a young age.[34] FH includes homozygous and heterozygous forms, with individuals affected with homozygous FH characteristically exhibiting more severe LDL-C elevation, which requires urgent, aggressive intervention starting in childhood. In addition to adopting a lifelong heart-healthy lifestyle, individuals with heterogyzous FH benefit from

Table 8 Nutrition and physical activity recommendations for youth with FH[2-3].

Nutrient	Recommended daily intake	Comments
Total fat	25–30% of total daily calories	
Saturated fatty acids	<7% of total daily calories	
Unsaturated fatty acids (MUFA and PUFA)	18–23% of total daily calories	Increase consumption of PUFAs
Trans fatty acids	0 g	Eliminate from diet
Cholesterol	200 mg or less	
Dietary fiber	14 g per every 1000 calories consumed	Increase consumption of viscous soluble fiber, with at least 6 g/day for those <12 years; 12 g or more/day for those >12 years
Plant sterols/stanols	2 g	Supplementation is likely necessary; care must be taken in purchasing a reliable supplement
Physical activity	>2 years of age – 60 min of moderate-to-vigorous physical activity or active play time/day	Limit sedentary activities to <2 h/day

LLM, ideally initiated in childhood and sustained thoughout adulthood.

Nutrition intervention The majority of individuals with FH require LLM to achieve optimal lipid goals and effectively lower lifetime exposure to atherogenic particles and CVD risk.[35] Nutrition intervention provides additional benefits, especially when implemented early in life and maintained over a lifetime. Detailed nutrition and physical activity recommendations for youth with FH are listed in Table 8.

A cholesterol-lowering dietary pattern (<7% daily caloric intake from SFAs, replacing SFAs with MUFAs and PUFAs, and ≤200 mg/day dietary cholesterol), with adequate macro- and micronutrients to promote growth and development, can safely be used in youth 2 years of age and older.[2,4] While a strict vegetarian or vegan diet is not required to achieve LDL-C lowering, inclusion of plant-based protein sources and less processed food in the dietary pattern can assist in decreasing intake of SFA and dietary cholesterol. For youth who choose to follow a strict vegetarian or vegan dietary pattern, care must be taken to ensure all macro- and micronutrients requirements are met, particularly B vitamins, EPA, and DHA, which are largely obtained from animal sources.[3,17,36]

Familial combined hyperlipidemia (FCHL)

Overview FCHL is a metabolic disorder characterized by: (a) elevated levels of Apo B, which may present as either mixed hyperlipidemia, isolated hypercholesterolemia, hypertriglyceridemia (HTG), or as a normal serum lipid levels with an abnormally high Apo B; (b) intra-individual and intra-familial variability of the lipid phenotype; (c) increased risk of premature CHD; and (d) a polygenic inheritance. FCHL is one of the most common genetic causes of hyperlipidemia in the general population with an estimated prevalence of 0.5%−2.0%. Because of the peculiar variability of laboratory parameters and the frequent overlapping with the features of metabolic syndrome, FCHL is often not recognized nor appropriately treated in youth.[37-38]

Identifying FCHL during childhood can be difficult because of the lack of long-term data linking lipid values measured before age 12 to the expression of the disease in adulthood.

FCHL may present in childhood in association with other metabolic conditions, such as T2D, non-alcoholic fatty liver disease (NAFLD), steatohepatitis, and metabolic syndrome.[39] Youth with FCHL are considered to be at increased risk of future CVD given adults with FCHL have higher cardiovascular (CV) mortality.[40]

Nutrition intervention plays a key role in effective management of FCHL, although LLM may also be needed to achieve lipid level goals.

Nutrition intervention While specific guidelines are not available for youth with FCHL, suggested lifestyle interventions include a heart-healthy dietary pattern and 60 min of moderate-to-vigorous physical activity. Reduced intake of SFAs, TFAs, and dietary cholesterol may be beneficial to lower LDL-C and Apo B. Additional nutrition recommendations may be needed if TGs are elevated (discussed in the TG Disorders section below). Achieving a 5% weight loss in overweight adults has been shown to reduce TGs and non-HDL levels, thus, individuals should be educated on lifestyle interventions to achieve and maintain an appropriate body weight.[41]

Elevated lipoprotein(a) [Lp(a)]

Overview Lp(a) is a lipid-rich apo B-containing lipoprotein with proatherogenic and prothrombotic properties. Elevated levels are recognized as a causal, independent risk factor for CVD and serve as a significant CVD risk enhancer.[42] Levels of Lp(a) are largely genetically determined, reach adult levels before school age, and remain relatively constant throughout life.[42-47] As such, there has been growing interest in screening and intervention strategies beginning at an early age.

Nutrition intervention Although targeted Lp(a)-lowering drugs are in development, currently none are commercially avail-

Table 9 Phytosterol content of select foods[15].

Food (serving size)	Phytosterol (mg/serving)
Olive oil, extra-virgin (100 mL)	168
Lentils, pink or red, raw (1 cup)	109
Pistachio nuts (1 oz, 49 kernels)	60
Soybean oil (1 Tbsp)	40
Safflower oil (1 Tbsp)	39
Nuts, almonds, unroasted (1 oz, 23 whole kernels)	39
Avocado, raw, California (1 fruit without skin and seed)	35
Cashew nuts, raw (1 oz)	35
Nuts, walnuts, English (1 oz, 14 halves)	26
Seeds, pumpkin and squash seed kernels, roasted without salt (1 oz)	5

able, thus nutrition intervention for lowering Lp(a) is of interest. However, available evidence shows a variable effect of nutrition intervention on Lp(a) levels. Well-controlled studies in adults found that reducing total and SFA intake resulted in beneficial lowering of plasma TC and LDL-C levels, yet were accompanied by a stepwise increase in plasma Lp(a) concentrations.[48-49] Physical activity appears to have a modest, if any, effect on Lp(a) levels. While the results of a number of studies did not demonstrate an association between Lp(a) levels and physical activity, others noted an inverse correlation in children and adolescents.[50-54] Given the inconsistent effects of nutrition intervention and physical activity on Lp(a) levels, these interventions should focus on minimizing additional CVD risk factors in patients with elevated Lp(a), such as other dyslipidemias, obesity, insulin resistance, and T2D.

Sitosterolemia

Overview Sitosterolemia is a rare autosomal recessive disorder of non-cholesterol sterol metabolism, caused by mutations of the *ABCG5* or *ABCG8* transporter genes. Hyperabsorption and decreased biliary excretion of non-cholesterol sterols leads to accumulation of sterols in the serum, such as campesterol and sitosterol.[55] While the presenting signs and symptoms of this disorder are heterogeneous, extensor tendon xanthomas may be present in the first decade of life and death from CAD can occur in severely affected youth. Some adults are asymptomatic and TC levels are often normal or only moderately elevated, although severe hypercholesterolemia can occur, simulating FH.

Nutrition intervention Effective dietary management of sitosterolemia includes limiting or avoiding dietary sources of cholesterol and plant sterols, though specific limits have not been outlined in previous guidelines. Common dietary sources of sterols include high-fat plant foods (avocado, nuts, seeds) and vegetable fats and oils (palm kernel oil, coconut oil, and other vegetable oils).[56-59] (Table 9) It is also recommended to avoid shellfish.[60] However, vegetables, fruits, and cereal products without germ may be consumed. Stanol-fortified foods typically recommended for managing hypercholesterolemia, such as spreads, juices, and supplements, are contraindicated, as they can exacerbate sterol accumulation and absorption.[61]

Youth receiving total parenteral nutrition (TPN) who develop cholestasis may have plasma concentrations of plant sterols that mimic those seen in hereditary sitosterolemia. Intralipid, a TPN fat emulsion, typically contains plant sterols (sitosterol, campesterol, and stigmasterol) and should be avoided in patients with hereditary and acquired sitosterolemia.[57]

Cerebrotendinous xanthomatosis (CTX)

Overview CTX is a rare, autosomal-recessive disorder of bile acid metabolism and lipid storage, which results in impaired bile acid synthesis and leads to accumulation of cholestanol and other sterols in numerous tissues, including the brain.[62] Clinical manifestations usually begin in infancy. Neonatal cholestatic jaundice and infantile-onset chronic diarrhea with failure to thrive are often the earliest clinical manifestations.[63] The clinical presentation may be mistaken for homozygous FH, as youth will occasionally present with xanthomas as the initial clinical sign. However, while plasma and serum cholestanol levels are elevated, cholesterol levels are low or normal. Although not currently approved by the FDA for this indication, oral chenodeoxycholic acid therapy is the current treatment of choice for CTX.[64]

Nutrition intervention There is limited literature regarding nutrition recommendations for individuals with CTX. Pharmacologic intervention is the preferred treatment, along with a dietary pattern low in cholesterol and cholestanol (low in egg yolks, meat, fish/shell fish and poultry, and high-fat dairy foods, such as whole milk or full-fat yogurt and cheese).[65] In the absence of pharmacologic intervention, chronic diarrhea and fat malabsorption may lead to nutrient deficiencies, including fat-soluble vitamin deficiency and essential fatty acid (EFA) deficiency. Therefore, fat-soluble vitamin supplementation may be beneficial in patients with CTX.[66]

Lysosomal acid lipase deficiency (LAL-D)

Overview LAL-D is a rare, autosomal recessive, lysosomal storage disorder caused by pathogenic variants in the *LIPA* gene resulting in decreased or total absence of LAL activity.[67] Infantile-onset LAL-D (previously known as Wolman's

disease) typically presents in the first months of life as a rapidly progressive disease with hepatosplenomegaly, malabsorption, and growth failure, and is generally fatal in the first year of life. Childhood/adult-onset LAL-D (previously known as cholesteryl ester storage disease) has a more variable course, which commonly involves hepatosplenomegaly, chronic liver injury, and severe dyslipidemia and is sometimes confused with FH. The most common biochemical abnormalities seen with LAL-D include elevated liver transaminases, elevated TC, LDL-C, and TG levels, and decreased HDL-C levels.[68]

Nutrition intervention Enzyme replacement therapy with sebelipase alfa is recommended for the treatment of LAL-D.[68] Although infantile-onset LAL-D is often treated with a specialized, amino acid-based, low-fat formula or TPN due to malabsorption and malnutrition, these do not alter the clinical course of the condition.[68-71] Patients with childhood/adult-onset LAL-D may benefit from a dietary pattern with <25–30% daily caloric intake from total fat and <200 mg/day dietary cholesterol, similar to the heart-healthy diet for FH (Table 8). Fat-soluble vitamin supplementation may be required for some patients based upon the degree of malnutrition or malabsorption.[68,72-73] Failure to thrive and chronic diarrhea are signs of malabsorption.[68] The patient's growth and nutrition status should be closely monitored by the healthcare team.

Hypobetalipoproteinemia

Overview This rare, heterogeneous group of disorders is characterized by plasma levels of TC, LDL-C, or Apo B concentrations less than the 5th percentile.[74]

Chylomicron retention disease and homozygous mutations of microsomal triglyceride transfer protein (abetalipoproteinemia) and apo B (familial homozygous hypobetalipoproteinemia) are characterized by the absence, or near absence, of LDL-C.[75] Clinical findings include steatorrhea, failure to thrive, and fat-soluble vitamin deficiencies, sometimes with retinopathy, coagulopathy, and progressive neurologic abnormalities.[76] Conversely, individuals who are heterozygous have very low LDL-C and apo B levels, yet are usually asymptomatic. Individuals with familial heterozygous hypobetalipoproteinemia are at risk for NAFLD.[75]

Nutrition intervention Strict adherence to a low-fat (20–30% daily caloric intake from fat) dietary pattern and fat-soluble vitamin supplementation is recommended for patients with hypobetalipoproteinemia.[74] Intake of foods rich in long-chain fatty acids should be limited, while ensuring adequate intake (2–4% daily caloric intake) of ALA, an omega-3 FA discussed previously, and linoleic acid (LA), an omega-6 FA.[77] Medium-chain triglyceride (MCT) oil may be used in infants to provide additional calories if the infant is underweight; however, MCT oil does not appear essential for treatment.[74,78] It is important to note that MCT oil does not contain EFAs.

Fat-soluble vitamin supplementation is critical to prevent and improve vitamin deficiency-related complications, which include progressive vision loss and blindness, as well as ataxia with loss of mobility.[74] Recommended fat-soluble vitamin dosing includes: 100–400 IU/kg/day vitamin A, 800–1200 IU/day vitamin D, 100–300 IU/kg/day vitamin E, and 5–35 mg/week vitamin K.[74] To circumvent intestinal malabsorption and the inability to transport vitamin A in the plasma, water-soluble vitamin A can be given intramuscularly.

Triglyceride disorders

Familial chylomicronemia syndrome (FCS) and multifactorial chylomicronemia syndrome (MCS)

Overview FCS is a rare autosomal recessive disorder with loss-of-function mutations involving the lipoprotein lipase (LPL) complex, which results in severe elevations in TGs and accumulation of chylomicrons in plasma.[79] While the most common mutations for FCS involve the *LPL* gene, others include *ApoA5, GPIHBP1, ApoC2, LMF1*. TG levels in individuals with FCS are often 10-fold to more than 100-fold above the upper limit of normal and may range from 1500 to 15000 mg/dL or higher, placing the individual at high risk for pancreatitis. Clinical presentation of FCS commonly includes eruptive xanthoma, lipemic plasma, and acute pancreatitis.[79]

In contrast to FCS, the co-existence of genetic and secondary causes of severe HTG is referred to as multifactorial chylomicronemia syndrome (MCS). MCS is at least 50–100 times more common than FCS and results from two different types of genetic variants: (1) rare heterozygous variants with variable penetrance in the five causal genes for FCS; and (2) common variants whose individually small phenotypic effects are quantified using a polygenic risk score.[80] While polygenic risk scores help to inform research, at present they are of limited benefit in clinical practice. Because susceptibility alleles and secondary factors tend to cluster in families, biochemical screening and counseling of family members of patients with MCS is important, although routine genetic testing is not recommended for these patients or their families.[81]

Nutrition intervention The pillar of treatment for FCS and MCS is a very-low-fat diet (<10–15% daily caloric intake from fat or <15–20 g/day total fat) while meeting EFA needs by ensuring adequate intake (2–4% daily caloric intake) of ALA and LA.[82,77] Simple carbohydrate foods should be avoided in individuals with FCS and MCS. Adolescents should be advised to avoid alcohol. MCT oil can be used in small amounts to increase overall caloric intake; however, MCT oil does not contain EFAs. MCT oil also aids in balancing macronutrients in the diet with the goal of limiting total carbohydrate to <60% daily caloric intake.[82-83] It is important to note that high-quality MCT oil is a prescription-grade oil containing only capric and caprylic acids. Dietary supplement, coconut oil-based MCT oil should not be used as a replacement

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Table 10 Dietary recommendations for treatment of children and adolescents with hypertriglyceridemia[2,82–84,87].

Nutrient	Moderate Hypertriglyceridemia	Severe Hypertriglyceridemia (FCS and MCS)
Fat	25–30% daily caloric intake • Increase dietary sources of O3FAs – fish high in EPA while low in methyl-mercury recommended	<10–15% daily caloric intake (<15–20 g/day) • Ensure adequate intake of EFA • MCT oil, as needed, to increase caloric intake and daily percent caloric intake from fat
Cholesterol	200 mg or less per day*	200 mg or less per day*
Carbohydrates	Decrease sugar intake by limiting foods and beverages with added sugars Choose foods that contain complex carbohydrates and reduce those with simple carbohydrates	Limit to <60% daily caloric intake Avoid foods that contain simple carbohydrates and avoid alcohol
Additional Recommendations	Supplementation with prescription O3FAs may be beneficial for further TG-lowering Encourage 60 min moderate-to-vigorous physical activity or active play time daily for children 2 years of age and older Limit sedentary screen time to <2 h daily	Monitor for EFA deficiency and fat-soluble vitamin deficiency – supplement as needed Encourage 60 min moderate-to-vigorous physical activity or active play time daily for children 2 years of age and older Limit sedentary screen time to <2 h daily

*Note: Recent guidelines recommend lowering dietary cholesterol, but do not provide a specific limit.[3].

Abbreviations: FCS=familial chylomicronemia syndrome; MCS=multifactorial chylomicronemia syndrome; O3FAs=omega-3 fatty acids; EPA=eicosapentaenoic acid; EFA=essential fatty acids; MCT=medium chain triglycerides.

for high-quality, prescription-grade MCT oil because it may contain higher amounts of lauric acid and smaller amounts of capric and caprylic acids. Recommendations for MCT oil use should be individualized based upon patient needs for additional calories, macronutrient needs, and overall tolerance. A summary of the nutrition recommendations for patients with FCS and MCS is included in Table 10.

Despite adherence to the diet, some patients may have persistently elevated or episodic severely elevated TGs and experience pancreatitis.[84] Patients should be monitored for EFA deficiency or the need for supplemental fat-soluble vitamins.[82] Age-specific recommendations, including recommended intake for infants, young children, and adolescents, have been published previously,[82] and interested readers are encouraged to review them.

Familial hypertriglyceridemia (FHTG)

Overview FHTG is primarily seen in adults and can be exacerbated by lifestyle habits. However, the clinical presentation of FHTG in youth is increasingly common due to an increase in the prevalence of childhood obesity and glucose intolerance in those with a genetic predisposition for FHTG. A risk factor for CVD in adulthood, FHTG is usually characterized by moderate-to-markedly elevated serum TGs with low-to-normal levels of LDL-C and HDL-C.[85]

Nutrition intervention Nutrition interventions for youth with significant TG elevation are shown in Table 10. Briefly, youth with FHTG should follow a dietary pattern with 25–30% daily caloric intake from fat and adequate intake of foods rich in O3FAs, while limiting simple carbohydrate foods, such as foods and beverages with added sugar and 100% fruit juice.[2] With severe HTG (fasting TG >1000 mg/dL), total fat intake should be decreased to <10–15% daily of total caloric intake (<15–20 g/day total fat) and simple carbohydrate foods, such as foods, and beverages with added sugar, and 100% fruit juice should be avoided.[83,86-87]

Physical activity and weight management are also important components of managing FHTG, with a goal of at least 60 min of moderate-to-vigorous physical activity daily.[2,3] In addition to food sources of O3FAs, dietary supplements may be beneficial for some individuals. Recommendations for O3FAs supplementation are reviewed in Section VI.

Acquired hypertriglyceridemia (HTG)

Overview Acquired HTG is impacted by unhealthy lifestyle behaviors, particularly decreased physical activity and excessive intake of refined carbohydrate foods, sugar-sweetened beverages, and alcohol, if consumed in youth. The increased prevalence of obesity has contributed to a growing number of youth with insulin resistance and T2D, HTN, and dyslipidemia. According to NHANES data, 42.9% of youth with a body mass index (BMI) ≥95th percentile had TG levels between 100 and 400 mg/dL.[88]

Nutrition intervention Nutrition intervention along with other lifestyle behaviors designed to achieve and maintain a healthy body weight, reduce insulin resistance, and prevent hyperglycemia are the primary treatments for acquired HTG (refer to "Special Considerations" in Section V and "Discussions on Weight" in Section VII).[87] If present, insulin resistance and T2D should be treated appropriately. Dietary recommendations for youth with HTG are shown in Table 10. A dietary pattern that reduces intake of refined carbohydrate foods and foods/beverages with added sugar, as well as emphasis on increased consumption of lower processed vegeta-

Table 11 Sources of calorie-dense foods for underweight youth with lipid disorders[15].

Food (with serving size)	Calories (per serving)	UFA (g/serving)		SFA (g/serving)
		PUFA	MUFA	
Nuts, average all varieties (1 oz)	173	6.2	6.3	2.3
Peanuts, roasted (1 oz)	166	4.4	7.0	2.0
Salmon and other oily fish (3 oz)	160	2.1	2.7	1.3
Seeds, average all varieties (1 oz)	158	7.0	3.9	1.9
Granola (1/4 cup)	149	3.3	2.3	1.4
Canola oil (1 Tbsp)	124	4.1	8.2	1.0
Olive oil (1 Tbsp)	119	1.4	9.8	1.9
Avocado (1/2 medium)	113	1.2	6.7	1.4
Chia seeds (2 Tbsp)	112	6	1.6	0.75
Dried fruit, average all varieties (1/4 cup)	108	0	0	0
Nut or seed butter, any variety (1 Tbsp)	97	2.1	4.7	1.3
Ground flaxseed (2 Tbsp)	75	4	1	0.5
Hummus (2 Tbsp)	46	1.0	1.1	0.4

bles and fruits, and lean protein, can be effective for those with mild-to-moderate HTG, in addition to increasing physical activity to at least 60 min per day.[4,87-91] With severe HTG (fasting TGs >1000 mg/dL), fat intake should be decreased to <10–15% of total caloric intake (<15–20 g per day) with avoidance of simple carbohydrates, such as foods and beverages with added sugars and 100% fruit juice.[83,86,87]

Special considerations

Underweight youth

For youth with lipid disorders other than severe HTG who are underweight (BMI <5th percentile for age and gender), nutrient-dense, calorie-dense, high-fat foods rich in UFAs can provide additional calories for weight gain while remaining within guidelines for lipid lowering as previously outlined. Nuts, nut butters, seeds, avocados, and oils may be included in the dietary pattern; nutritional shakes that are low in SFAs, low in added sugars, and adequate in fiber may also be appropriate (Table 11). Increased caloric intake from foods rich in SFAs, such as fatty and processed meats (hot dogs, bacon, sausage, and pepperoni), full-fat dairy (whole milk, regular cheese, and full-fat yogurt), and butter and coconut oil, increases LDL-C and should be avoided in underweight youth with dyslipidemia.

Calorie-dense foods low in SFAs and TFAs may be added to an individual's usual dietary pattern. For example, adding a trans-fat free, oil-based butter alternative to a bowl of oatmeal adds calories and UFAs without affecting the flavor/texture. Some cholesterol-lowering spreads and other food products that contain plant sterols should be avoided in youth with suspected or known sitosterolemia.[61] Other calorie-dense items rich in UFAs, such as avocado, nuts, nut butters, and flaxseed, can also be incorporated into an individual's usual food intake. For youth with severe HTG, MCT oil can be utilized to provide additional calories to the dietary pattern (refer to discussion of MCT oil above).[82]

An RDN can develop an individualized plan for youth who are underweight to assist with weight gain while adhering to the recommended nutrition plan for the individual's dyslipidemia diagnosis and needs.

Youth with overweight or obesity

Youth with overweight or obesity are at elevated risk for cardiometabolic diseases, and an estimated 39% of youth with obesity also demonstrate lipid abnormalities.[91-92] Similar to adults, the most common pattern of dyslipidemia in youth with overweight or obesity is characterized by elevated TGs, decreased HDL-C, and increased levels of small, dense LDL. Levels of LDL-C measured on routine lipid panels are generally normal or mildly elevated in youth with overweight or obesity.[93] Other comorbid conditions are common, and include insulin resistance, T2D, NAFLD, polycystic ovary syndrome, obstructive sleep apnea, and mental health disorders.[93]

BMI percentiles and z-scores are tools that are readily available and easy to use as a measure to determine if a child meets criteria for overweight or obesity. Youth with BMI at or above the 85th percentile for age and sex can benefit from a comprehensive pediatric weight management program, if available. It is important to note that programs may have varying referral criteria. In all age categories, short- and long-term decreases in BMI are more likely to be achieved in the context of a multi-component, family-centered weight management intervention that included nutrition counseling by an RDN, physical activity, and behavioral components managed by a psychologist or mental health provider.[94,95] A staged approach to weight management has previously been outlined by the American Academy of Pediatrics (AAP) (Table 12).[96]

Although some behavioral and pharmacotherapy studies report modest success, additional research into accessibil-

Table 12 Suggested staged approach to weight management for children and adolescents[96].

Stage 1: Prevention Plus	
Components	**Implementation Details**
• ≥5 vegetables and fruits • ≤2 h of recreational screen time (none if <2y of age) • No TV in child's bedroom • ≥60 min of physical activity (may need to be gradual progression toward this goal) • Involve entire family and consider cultural differences	• Primary care office-based • Administered by trained office support: physician, PNP, PA, RN • Schedule follow-up frequency tailored to family • Advance to stage 2 if no improvement in BMI status after 3–6 months, if congruent with family's readiness to change
Stage 2: Structured Weight Management	
Components	**Implementation Details**
• Reduced-calorie eating plan • Increased structure of daily meals and snacks • ≤60 min of recreational screen time • ≥60 min of physical activity • Implement monitoring of screen time, physical activity, etc. • Perform medical screening	• Primary care office-based • Administered by RDN, physician, PNP with training in assessment and counseling • Support from referrals • Monthly follow-up visits • Advance to stage 3 if no improvement in BMI status after 3–6 months, if congruent with family's readiness to change
Stage 3: Comprehensive Multidisciplinary Intervention	
Components	**Implementation Details**
• More frequent contact • More structured monitoring, goal-setting, feedback • More behavioral strategies used with more formal monitoring and feedback. • Components include: • parental involvement for children <12 years old (decrease as adolescents age) • assessment of diet, physical activity, weight (body fat) before treatment and at specified intervals • behavioral program including food monitoring, short-term nutrition and activity goal setting and contingency management • parent/caregiver training to improve home food and activity environments • structured dietary and physical activity interventions that improve dietary quality and result in negative energy balance	• Multidisciplinary team (behavioral counselor, RDN, exercise specialist) or RDN and behavioral specialist paired with an outside physical activity program if a multidisciplinary team unavailable • Structured physical activity • Weekly follow-up for 8–12 weeks, then monthly. Advance to stage 4 if no improvement in BMI, if congruent to family's readiness to change.
Stage 4: Tertiary Care	
Components	**Implementation Details**
All components of Stage 3, but also: • Consider obesity treatment medication • Consider bariatric surgery • Consider meal replacements • Ongoing behavior change	• Pediatric weight management center • Multidisciplinary team with expertise in childhood obesity, including physician, behavioral counselor, RDN and exercise specialist • Clinical or research protocol

Abbreviations: PNP=Pediatric Nurse Practitioner; PA=physician assistant; RN=registered nurse; RDN=registered dietitian nutritionist.

ity and efficacy for treating obesity in children and adolescents is limited.[97] The FDA has approved a select number of weight loss medications, and bariatric surgery can also be considered for youth who meet published criteria and who have been unsuccessful achieving satisfactory weight loss with lifestyle modification.[97-98] The 2017 Endocrine Society Clinical Practice Guideline recommends that adolescents undergoing lifestyle therapy, medication regimens, or bariatric surgery for obesity need cohesive planning to help them effectively transition to adult care, with continued monitoring, support, and intervention.[97] Further study is needed of the genetic and biological factors that increase the risk of weight gain and influence the response to therapeutic interventions in youth.[97] Encouragement and ongoing support for youth by

the primary care provider that focuses on early intervention is essential.[96-97,99]

Extensive recommendations for weight management in youth are beyond the scope of this review. A brief discussion on weight in youth is provided in Section VII.

Eating disorders

Alterations in the levels of blood lipids have been reported in individuals with eating disorders, such as anorexia nervosa and bulimia. However, there are limited data in youth on the extent, mechanism, and normalization of dyslipidemia with weight restoration. In adults with eating disorders, the results of studies have been inconsistent; for example, both increased and decreased lipid concentrations have been reported in anorexia nervosa compared with healthy controls.[100-103] Recently, Hussain et al. conducted a systematic literature review and meta-analysis that demonstrated aggregate evidence for elevated lipid concentrations in acutely ill anorexia nervosa patients compared with healthy controls, some of which persisted after partial weight restoration.[104] The authors suggested that this finding could signal an underlying adaptation or dysregulation not fully reversed by weight restoration. Although concentrations differed between subjects with anorexia and healthy controls, most lipid levels remained within the reference range and meta-analyses were limited by the number of available studies.

A detailed discussion on nutrition interventions for eating disorders is beyond the scope of this paper. Screening for and treatment of eating disorders are discussed in Section VII.

Dietary supplements

A limited number of dietary supplements are included in national guidelines for youth and discussed in this section. With all patients, it is important to inquire about use of dietary supplements, including herbal products, and provide appropriate guidance. Parents and caregivers should be informed that some dietary supplements may have harmful side effects, particularly if started at a young age and taken for a prolonged period of time. Dietary supplements should also be discussed with the medical care team, which ideally includes a pharmacist, to ensure that no drug-drug interactions exist for dietary supplements and any prescribed medications used by patients.

Plant sterols and stanols

Research findings demonstrate that plant sterols and stanols lower LDL-C in youth who have been unable to achieve their lipid-lowering goals with dietary modification alone.[2,105-107] For those 2 years of age and older, a dose of 2 g/day is recommended as a replacement for usual dietary fat sources.[2] Results of limited studies in youth 4.5–16 years of age have demonstrated 6–10% lowering of TC, LDL-C, and apo B in individuals with hypercholesterolemia or FH, and up to a 15% lowering of similar parameters in individuals with FCHL.[106-107] Plant sterols and stanols in fortified food, such as margarines, capsules, or chewable supplements, appear to be more effective when administered throughout the day and with meals, rather than as a single dose.[108-109] While studies have demonstrated lipid-lowering and general safety, there is insufficient evidence in youth of long-term CV risk reduction.[4] Plant sterols and stanols supplements are contraindicated in youth with sitosterolemia.[4,110]

Soluble fiber

While soluble fiber intake from food sources is imperative, supplemental soluble fiber may be beneficial to glucose tolerance and assist in lowering atherogenic lipid levels.[2] Viscous soluble fiber has been shown to be effective in reducing TC, LDL-C, and non-HDL-C.[4,111-113] The recommend dose for youth is currently 6 g/day for those 2-12 years of age, and 12 g/day for those $\geq$12 years of age.[2] Studies with a range of soluble fiber doses and sources in youth 2–18 years of age have demonstrated up to a 23% lowering of TC and LDL-C.[114-115]

Soluble fiber supplements come in a variety of preparations, including powders, gummies, capsules, and fortified food products. To be effective, the gel-forming properties of a fiber supplement should remain intact throughout manufacturing and processing.[4] Ample fluid intake should be encouraged to reduce the risk of intestinal blockage or constipation.[4] Powdered supplements should be consumed with an 8 oz glass of water to reduce choking risk and may not be appropriate for those with swallowing dysfunction. Additional research is needed to support recommendations for dosing, long-term safety, and efficacy of fiber supplementation in the pediatric population to treat dyslipidemia.

Omega-3 fatty acids (O3FAs)

While numerous studies have investigated the effects of O3FA supplementation on dyslipidemia in adults, clinical trials in youth are limited and the results are inconclusive.[116-118] A pediatric trial comparing high-dose, prescription O3FA supplementation (3.4 g EPA+DHA/day) to placebo demonstrated no statistically significant TG lowering, while two similar trials in youth (one using 500–1000 mg non-prescription fish oil daily and one using 4 g prescription omega-3 ethyl esters daily) demonstrated slight, though not statistically significant, decreases in TG.[119-121] Trials in youth younger than 8 years of age are lacking. Thus, while O3FAs are sometimes used in clinical practice to lower TG in youth, additional research is needed to help determine dosing recommendations, long-term efficacy, and safety of these supplements.

If O3FA supplementation is used, prescription formulations are recommended, as dietary supplement preparations are not regulated by the FDA, may have an inaccurate repre-

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Table 13 AAP recommendations to mitigate weight stigmatization in clinical practice[128].

1. **Role Modeling.** Demonstrate and model behavior that is supportive and non-stigmatizing to children and their families. Avoid placing blame and judgment solely on individuals with excess weight. Recognize and acknowledge the many contributors to obesity (i.e., genetic, socioeconomic, environment, family and cultural traditions, individual choices).
2. **Language and Word Choice.** When referring to patients, use people first language, which places the person before the medical condition (i.e., child with obesity). Use neutral words like "weight" or "body mass index" over words that may be more emotionally laden (i.e., "obese," "extremely obese," "fat")
3. **Clinical Documentation.** Convey information about weight in a sensitive and supportive manner by using neutral words, such as "unhealthy weight" and "very unhealthy weight," when speaking to families and when documenting in the medical record.
4. **Behavior Change Counseling**. Use a collaborative, patient-centered approach, such as motivational interviewing, to encourage families to make healthy and sustainable behavioral changes.
5. **Clinical Environment.** Provide a non-stigmatizing clinic space for children and their families by creating a setting that accommodates patients of varying body sizes.
6. **Behavioral Health Screening.** Assess for psychosocial comorbidities associated with negative exposures associated with obesity (i.e., bullying, poor school performance, low self-esteem, depression, anxiety).

sentation of EPA and DHA content, and may contain significant amounts of cholesterol and SFAs or oxidized FAs.[87,122]

Coenzyme Q10 (CoQ10)

While CoQ10 has been studied in adult populations, limited research exists that support the use of CoQ10 in the pediatric population. CoQ10 is often used, without proof of benefit, to help ameliorate statin-associated muscle symptoms in adults; however, such symptoms are rare in youth taking statins.[123] One small, short-term study of adolescents with FH treated with rosuvastatin reported a decrease in CoQ10 levels in peripheral blood mononuclear (PBM) cells, but without a decrease in adenosine triphosphate synthesis. The authors recommended the need for additional long-term studies to determine the effects of CoQ10 supplements on youth treated with statins.[124] Others have reported that PBM cell or plasma levels of CoQ10 may have little correlation with levels in myocytes or mitochondrial function in myocytes. Thus, there is insufficient evidence that the use of CoQ10 in youth treated with LLM either prevents or decreases statin-related myopathies.

Psychosocial aspects of nutrition counseling in youth

Discussions on weight

Discussion about modifying a child's weight and recommendations for behavioral changes should be approached in a non-stigmatizing way by emphasizing modifiable lifestyle behaviors rather than weight modification. Weight stigmatization has been associated with maladaptive eating behaviors and decreased physical activity.[125-127] Clinicians should attempt to mitigate peer, family, and clinician weight-related stigma by utilizing the AAP practice recommendations to guide interactions with youth, choice of mental health screening tools, clinical documentation, and decisions about how to structure the clinical environment[128] (Table 13).

Efficacy of family-based interventions

Dietary choices and habits of youth are often made within the context of the family. As such, parent or caregiver involvement is critical to successful implementation and sustained healthful lifestyle changes for youth.[129] Family-based interventions have been shown to be efficacious by encouraging behavior changes in both the child and parent through teaching the parent behavioral skills.[130] A meta-analysis found that the greater the parent involvement, the better the treatment effects.[131] Clinicians are encouraged to utilize cognitive behavioral interventions to encourage family-level behavioral changes[99] (Table 14).

Disordered eating

Youth with a history of overweight/obesity and individuals on restricted or weight-loss diets are at greater risk for developing an eating disorder.[132-135] While weight-related counseling is unlikely to lead to significant eating disordered-behaviors, it is important to mitigate risk when providing dietary counseling[136-137] (Table 15). Since youth with eating disorders often have a normal or elevated BMI, identification of eating disorders can be more challenging.[133,138] Clinicians should inquire about use of weight-control behaviors and screen youth for eating disorders[139-140] Table 16 illustrates an example of a screening tool for eating disorders. If an eating disorder is suspected or confirmed in youth, referral for a multi-disciplinary team approach with specialized and intensive treatment

Table 14 Cognitive behavioral techniques to assist with family lifestyle behavior modification[99].

1. Set concrete and achievable goals with families.
2. Encourage families to keep track of progress toward their goals.
3. Encourage families to reward their child for achieving behavioral goals (as opposed to rewarding weight change). Reinforcers could include verbal praise or non-food related privileges.
4. Help families to be aware of their progress, even if their execution is imperfect. Encourage them to focus on their successes and not their failures.

Table 15 Recommendations to prevent obesity and eating disorders in adolescents[141].

1. Inform adolescents that dieting and unhealthy weight control behaviors may be counterproductive. Instead, encourage positive eating and physical behaviors that can be maintained on a regular basis.
2. Do not use body dissatisfaction as a motivator for change. Instead, help teens care for their bodies so that they will want to nurture them through healthy eating, activity, and positive self-talk.
3. Encourage families to have regular and enjoyable family meals.
4. Encourage families to avoid weight talk: Talk less about weight and do more to help teens achieve a weight that is healthy for them.
5. Assume overweight teens have experienced weight mistreatment and address this with teens and their families.

Table 16 SCOFF Questionnaire: Eating Disorder Assessment Tool[140].

1. Do you make yourself **S**ick because you feel uncomfortably full?
2. Do you worry you have lost **C**ontrol over how much you eat?
3. Have you recently lost more than **O**ne stone (i.e., 14 pounds) in a 3-month period?
4. Do you believe yourself to be **F**at[a] when others say you are too thin?
5. Would you say that **F**ood dominates your life?

One point for every "yes"; a score $\geq$ 2 indicates a likely case of anorexia nervosa or bulimia
[a] Consider using alternative language that may be considered less stigmatizing (i.e., too big)

is indicated, including a psychotherapist, pediatrician, and dietitian.[139]

Additional psychosocial considerations

The family's ability to understand and implement recommendations for lifestyle behavioral changes that affect dyslipidemia in youth are affected by modifiable and non-modifiable individual, family, community, and healthcare system-level factors.[142] It is important to be aware of the patient's psychosocial state when counseling families regarding the need for interventions. Interventions should be patient-centered and developmentally appropriate (i.e., considering the ages and cognitive abilities of both the child and family members) and sensitive to cultural differences. There is no universal agreement as to when youth can be considered competent to make decisions, as contextual factors (i.e., brain development maturity, personality, situational factors) significantly influence decisions.[143] Clinicians are encouraged to utilize strategies for shared decision-making with a patient and their family that have been published previously.[144] A clear explanation of disease processes with the parents and the patient is important to ensure both have an accurate understanding. Knowledge gaps can be assessed and addressed by encouraging parents and youth to utilize the teach back method, sharing information previously learned.[145] Children and adolescents may present with mental health concerns, which can create barriers for successful implementation of lifestyle changes.[146-150] Some youth may require counseling and/or treatment with medications, including antipsychotics, many of which have been associated with weight gain and unhealthy metabolic profiles.[151] Additionally, families may struggle with food insecurity, imposing a barrier to adherence with treatment recommendations.[152-153] Ideally, care should be provided within the context of a multidisciplinary team, with clinicians who can address concomitant mental health and social barriers. When this is not possible, it is important to screen for comorbid psychoso-

cial risk factors, and seek out collaborative, specialist care clinicians.[154-156]

Conclusion

Atherosclerosis, the origin of CVD, begins at a very early age and is significantly influenced by lifestyle behaviors. When started at an early age and maintained over a lifetime, nutrition interventions and other heart-healthy lifestyle behaviors can delay the onset or prevent the occurrence of CVD-related events. Even in those who require LLM, lifestyle interventions provide additional benefits and enhance lipid-lowering. Changes in lifestyle habits are best achieved with a multidisciplinary, family-based approach. Referral to an RDN knowledgeable about the needs of youth with lipid disorders is recommended to help youth and their families successfully meet nutritional needs, provide ongoing nutritional support, and encourage long-term adherence. Referral to a social worker and/or a mental health professional is recommended to resolve treatment barriers.

Credit authorship contribution statement

LW, JC, SLL, and DPW contributed to the initial draft of the article. All authors contributed to the concept, design, revision, and approval of the article.

Conflicts of interest

Disclosures for CMBS during the prior 24 months include a speaker honorarium from Cardiometabolic Congress. ALP received consulting fees paid to institution from Novartis during the prior 24 months. DPW received consulting fees from Alexion during the prior 24 months. LW, JB, JC, CK, SLL, ASS, GS, and ALW have no relevant disclosures.

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