**Review Article**

**Co-relating Hyperlipidaemia In School Going Children Due With Their Sedentary Lifestyle**

**ABSTRACT**

**Introduction:** Due to their increasingly sedentary lifestyles, school-age children are more at risk for hyperlipidaemia, which is defined by increased blood lipid levels. Children who have hyperlipidaemia are far more likely to be obese and have higher BMIs, which increases their risk of developing cardiovascular diseases (CVD) that may last into adulthood. This study examines the relationship between childhood sedentary behaviour and hyperlipidaemia, highlighting risk factors, pathophysiological mechanisms, and preventative strategies.

**Method:** To evaluate the connection between sedentary lifestyles, lipid metabolism, and the emergence of hyperlipidaemia in children, a thorough literature analysis was carried out. Important elements were examined, including lipoprotein profiles, lipid classes, and their connection to atherosclerosis. Pharmacological treatments, lifestyle changes, and screening techniques for hyperlipidaemia management were also assessed.

**Conclusion:** According to the results, controlling hyperlipidaemia and lowering children's long-term cardiovascular risks requires early detection through screening in addition to dietary changes and lifestyle changes such increased physical exercise. When lifestyle modifications are insufficient for severe cases, pharmaceutical treatment is advised. To reduce the increased prevalence of hyperlipidaemia and related CVD risks, public health interventions must emphasize encouraging youngsters to lead active lives and to be nutritionally aware.

*Keywords: Hyperlipidaemia, Cardiovascular disease, Sedentary lifestyle, School children, Body Mass Index, Atherosclerosis.*

**1. INTRODUCTION:**

Hyperlipidaemia, defined as elevated lipid levels in the blood, has emerged as a concerning health issue in school-going children, often linked to the rise in the sedentary lifestyles [[20]](https://medpulse.in/Pediatrics/Article/Volume14Issue2/Ped_14_2_2.pdf). Also, a pattern has emerged correlating hyperlipidaemia with frequent hospitalizations and treatments. In the age group of children in between 6-12 years, the mean levels of Triglycerides in boys are 85.3 mg/dL, LDL is 78.3 mg/dL, HDL is 57.1 mg/dL and Total Cholesterol is 151.9 mg/dL, and similarly in girls of the same age group, the levels of triglyceride are 95.8 mg/dL, LDL is 75.5 mg/dL, HDL is 63.8 mg/dL and Total Cholesterol is 159.0 mg/dL [[1]](https://pubmed.ncbi.nlm.nih.gov/28769178/)[[32]](https://pubmed.ncbi.nlm.nih.gov/34449807/). According to a study conducted in 2023, 15% of the children population globally are affected by this, due to at least one adverse lipid level, mainly due to the increase in BMI (Body Mass Index) with age [[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/). Children who are obese typically have an even greater prevalence of hyperlipidaemia – up to 42%, which means overweight and obesity both acts as significant factors which are also related to their sedentary lifestyle [[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/)[[31]](https://pubmed.ncbi.nlm.nih.gov/29174025/).

The frequency of cardiovascular disease (CVD) in children rises in tandem with this condition. These cardiovascular risk factors tend to follow throughout adulthood and predict a subsequent high risk for cardiovascular events, even if they may manifest at an early age [[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/). There is no doubt that cardiovascular risk increases with the number of cardiovascular risk factors. Children who come from low-income families can be vulnerable, and even children from middle-income families are also vulnerable, all due to the unhygienic food consumption, irregular nutritional intake or diet, and poor healthcare [[33]](https://pubmed.ncbi.nlm.nih.gov/32927656/).

We review the present scenario in terms of the elevated lipid levels that is observed in children, the reasons for which hyperlipidaemia is becoming prevalent in this particular population and whether its specific to a particular region or not. We also try and discuss the necessary pathways for the control and prevention of hyperlipidaemia, thus eliminating or minimizing the risk of cardiovascular diseases [[31]](https://pubmed.ncbi.nlm.nih.gov/29174025/)[[34]](https://pmc.ncbi.nlm.nih.gov/articles/PMC8996670/).

**2. THE RELATION BETWEEN THROMBOSIS, FIBRINOLYSIS AND HYPERLIPIDAEMIA**

Structural vascular wall damage and functional problems are encouraged by hyperlipidaemia. This is mostly mediated by the endo’s numerous roles.

Endothelium, a smooth surface that helps control blood flow and has anti-inflammatory, anti-thrombotic, and pro-fibrinolytic qualities is an intact and healthy endothelium.

Numerous of these processes are mediated by oxidation-sensitive pathways that affect nitric oxide’s bioavailability [[3]](https://pubmed.ncbi.nlm.nih.gov/34978528/)[[35]](https://pubmed.ncbi.nlm.nih.gov/34987194/).

Atherosclerotic plaque rupture or haemorrhage has been the subject of much of the attention about the connection between thrombosis and atherosclerosis (Figure 1). Thrombosis of the lumina. Both platelets and clotting factors contribute to this dynamic reaction, which leads to acute coronary syndromes.

Nevertheless, very severe atherosclerotic lesions are uncommon in children. However, it has been demonstrated that children with dyslipidaemia have decreased fibrinolytic activity [[35]](https://pubmed.ncbi.nlm.nih.gov/34987194/)[[37]](https://pubmed.ncbi.nlm.nih.gov/25810456/).



Figure 1: Thrombosis of the lumina

Adult research has demonstrated that treating hyperlipidaemia with hydroxymethylglutaryl (HMG) CoA reductase inhibitors or bstatinsQ can greatly reduce the production of thrombuses and enhances the fibrinolytic profile.

Reduced levels of tissue plasminogen activator antigen and plasminogen activator inhibitor 1 are indicators of this. A study conducted by Joukha Dar and associates demonstrated that prothrombin fragment 1+2 and von willibrand factor antigen plasma levels were considerably lowered by treatment with different statins. According to these research, hyperlipidaemia, endothelial dysfunction, and anomalies in children’s thrombosis and fibrinolysis may be related [[36]](https://pubmed.ncbi.nlm.nih.gov/30706683/)[[37]](https://pubmed.ncbi.nlm.nih.gov/25810456/).

**3. LIPID CLASSES**

To better understand lipid problems in children, it is essential to have a thorough grasp of how lipids are processed in the body. Maintaining metabolic homeostasis requires precise control of the balance between energy intake, storage and use.

The four primary classes of lipids in plasma that must be packaged and transported are phospholipids, cholesterol, cholesterol esters and triglycerides (TG).[[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/)[[21]](https://pmc.ncbi.nlm.nih.gov/articles/PMC9671590/#abstract1)[[23]](https://link.springer.com/article/10.1007/s00246-007-9149-0)

**3.1 Phospholipids**

A vital component of cell membranes, phospholipids are made up of a hydrophilic phosphate “head” and a hydrophobic fatty acid “tail”. Polar phospholipids include sphingomyelin and phosphatidylcholine. The phospholipids of the oxidized low-density lipoprotein (LDL) surface monolayers are hydrolyzed by lipoprotein-associated phospholipase A2 (Lp-FLA2), which is secreted by white blood cells [[5]](https://pubmed.ncbi.nlm.nih.gov/35373933/)[[38]](https://pubmed.ncbi.nlm.nih.gov/33516680/). This process produces endogenous inflammatory mediators, lysophosphatidylcholine and oxidizes nonesterified fatty acids, which are important in atherogenesis. Phospholipids can influence a number of cellular functions, such as the initiation of the innate immune response, because of their location on the surface of lipoproteins.[[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/) [[22]](https://pmc.ncbi.nlm.nih.gov/articles/PMC4110615/) [[24]](https://www.scielo.br/j/rbme/a/pXkfbdcwgNyR7sjjQ5BvtxN/?lang=en).

**3.2 Cholesterol**

A number of substances that are essential to our body’s necessary biological processes, including bile acids, vitamin D, steroid hormone and cell membrane integrity are synthesized in large part by cholesterol. Although cholesterol has several beneficial properties, excessive quantities of it are poisonous and many routes strictly regulate them[[40]](https://pubmed.ncbi.nlm.nih.gov/33942057/), They are principally represented by esterification through the enzyme acyl-coenzyme-A cholesterol acyltransferase (ACAT), LDL receptor-mediated endocytosis, cholesterol efflux from plasma membranes to cholesterol acceptor particles in the high-density class of lipoproteins and de novo synthesis in the smooth endoplasmic reticulum via 3-hydroxy-3methylglutryl-coenzyme-A (HMG-CoA) reductase. All of these pathways exhibit distinct regulation, with the first two and final pathways being influenced by environmental and inherited modulators that affect gene transcription, and the ACAT pathway being regulated by substrate interactions that are sensitive to the amount of cholesterol in the membrane.[[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/)[[3]](https://pubmed.ncbi.nlm.nih.gov/34978528/)[[39]](https://pubmed.ncbi.nlm.nih.gov/31475726/).

**3.3Cholesterol esters**

ACAT produces cholesterol esters in the liver and gut while lecithin cholesterol acyltransferase produces them in the plasma. Cholesterol esters are created when cholesterol is esterified with long-chain fatty acids. Not only is TG (triacylglycerol) carried by lipoproteins through the bloodstream, but cholesterol itself can also be retained in cells.

Therefore, these molecules play a critical role in metabolic processes that underpin homeostasis and cholesterol trafficking.[[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/)[[3]](https://pubmed.ncbi.nlm.nih.gov/34978528/)

**3.4Triglycerides**

Many organs, including the liver, gut and adipose cells, generate TGare esters, which are made from glycerol and three fatty acids. They are in the blood to facilitate the liver’s two-way translocation of blood glucose and adipose fat. Chylomicrons are responsible for transport of diet TG. During absorption, the byproducts of TG cleavage pass through the membranes of the epithelial cells in the villi of the small intestine before being transported to smooth endoplasmic reticulum, where TG resynthesis occurs although visceral and subcutaneous adipose cells typically retain TG, under stressful conditions, TGlipolysis take place, resulting in the generation of free fatty acids that are then taken up by muscle cells as a substrate for ATP synthesis and mitochondrial oxidation.[[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/)[[4]](https://pubmed.ncbi.nlm.nih.gov/30724974/)

**4. LIPOPROTEINS**

Lipoproteins are categorized based on the relative amounts of four main lipids serve as the body’s main storage respiratory and have a variety of distinct roles –

**4.1 Chylomicrons**

There are three primary chylomicron apoproteins: apoB48, apoE and apoC11. A plasma enzyme known as lipoprotein lipase (LPL) is activated by apoc11 if the preceding two work by attaching the chylomicron fragments to hepatocyte scavenger receptors. This enzyme recognizes recognizes the hydrolysis of lipoproteins that contain a lot of TG. [[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/)[[41]](https://pubmed.ncbi.nlm.nih.gov/30639555/)

**4.2 LDL-Cholesterol and VLDL-Cholesterol**

. LDL cholesterol (LDL-C) and VLDL cholesterol (VLDL-C), which are intricate protein-lipid supramolecular complexes that deliver polyunsaturated fatty acids and cholesterol to cells, respectively, are the most atherogenic lipoprotein classes.

 As the main causes of atherogenesis, particles change the properties of endothelium, improve blood cell adhesion, and trigger monocyte/macrophage chemotaxis, all of which lead to the growth of smooth muscle cells. According to Brown and Goldstein, receptor-mediated endocytosis is responsible for the movement of cholesterol into the cell as a component of LDL-C. Therefore, the receptor-mediated capture of LDL-C can maintain normolipidemia, ensuring that blood cholesterol levels stay within the normal range and preventing the development of atherosclerosis. Peroxide-modified LDL-C plays an even greater impact in atherogenesis and is quickly identified and taken up by the scavenger receptors of macrophages. [[2](https://pubmed.ncbi.nlm.nih.gov/36979789/)][[5]](https://pubmed.ncbi.nlm.nih.gov/35373933/)[[41]](https://pubmed.ncbi.nlm.nih.gov/30639555/)

**4.3 HDL-Cholesterol**

Phospholipids and apolipoproteins A-1 and A-2 make up the majority of nascent HDL particles, which are produced in the liver and gut. The ATP binding cassette transporter A1 (ABCA1) allows them to absorb cholesterol. HDL particles make an effort to primary function by recognizing the reverse movement of cholesterol from arterial walls and peripheral tissues to liver cells, earning it the moniker anti-atherogenic. HDL esterification is accomplished by the enzyme lecithin cholesterol acyltransferase (LCAT). Furthermore, HDL cholesterol (HDL-C) protects LDL-C by preventing their transformation into an atherogenic orientation and promoting the use of TG-rich lipoproteins [[2](https://pubmed.ncbi.nlm.nih.gov/36979789/)][[6](https://pubmed.ncbi.nlm.nih.gov/34637926/)][[42]](https://www.ahajournals.org/doi/10.1161/cir.0000000000000350). Since only two-thirds of plasma cholesterol is impacted by the esterification of VLDL, HDL and LDL carried out by cholesterol ester transfer protein (CETP), only a small percentage of circulating cholesterol is free. LDL causes atherosclerosis by depositing cholesterol in foam cells, where as HDL removes cholesterol from these cells. This is one of the main differences between the two types of cholesterol. Low plasma HDL-C concentration has been shown to be an independent risk factor for ASCVD; however, there is currently no proof that higher plasma HDL-C levels lower the risk of ASCVD [[2](https://pubmed.ncbi.nlm.nih.gov/36979789/)][[4](https://pubmed.ncbi.nlm.nih.gov/30724974/)][[43]](https://pubmed.ncbi.nlm.nih.gov/35583875/). How ever, characteristics associated with raised HDL-C levels include feminine gender, estrogens, increased physical activity, and weight loss. Conversely, masculine gender, progesterone, obesity, hypertriglyceridemia, type 2 diabetes. Since low HDL-C plasma concentrations are more frequently caused by drinking, tobacco usage and heavy carbohydrate consumption.

**4.4 Lipoprotein-a**

The LDL-C conjugate lipoprotein a (Lp(a)) has a distinct apoprotein (a) that resembles plasminogen in structure and can prevent fibrinolysis. Furthermore Lp(a) displays pro-inflammatory properties due to the transfer of oxidized phospholipids. The risk of ASCVD increases with increased plasma levels of Lp(a); however, in certain patients, the amount of risk may be less than that associated with LDL-C. However, it has recently been discovered that individuals with Lp(a) levels greater than 180 mg/dL are more likely to develop ASCVD, as if they had familial hypercholesterolemia. It is more plausible that an excessively high level of Lp(a) determines a novel and common hereditary dyslipidemia linked to an elevated risk of ASCVD because Lp(a) is generally transmitted in nearly 90% of instances all entire life.[[2]](https://pubmed.ncbi.nlm.nih.gov/36979789/)[[44]](https://pubmed.ncbi.nlm.nih.gov/34981790/)

**5. THE ATHEROSCLEROTIC PROCESS IN CHILDREN AND ADOLESCENTS**

An increasing amount of research indicates that the atherosclerosis process starts in childhood and advances in response to the presence of conventional risk factors for cardiovascular disease. A pathologic analysis of 30 young adults revealed a linear relationship between pre-mortem measurements of low-density lipoprotein (LDL) cholesterol levels and percentage of the aorta’s surface involvement with fatty streaks [[7](https://pubmed.ncbi.nlm.nih.gov/16709475/)][[25]](https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.118.032531)[[43]](https://pubmed.ncbi.nlm.nih.gov/35583875/).

According to a pathologic investigation, in young children with normal cholesterol levels, the rate of abdominal aortic fatty streaks were considerably more common in children born to mothers with high cholesterol. This study raises the notion that endothelial dysfunction may be caused by environmental prenatal programming. Berenson and colleagues’ additional research revealed that fibrous plaque lesions were directly associated with conventional cardiovascular risk factor and that their prevalence rose with age [[7](https://pubmed.ncbi.nlm.nih.gov/16709475/)][[9](https://pubmed.ncbi.nlm.nih.gov/9614255/)][[34]](https://ijponline.biomedcentral.com/articles/10.1186/s13052-022-01250-5). But they also demonstrated that when the number of cardiovascular risk factors rose exponentially. Pediatric patients have been studied using noninvasive in vivo indicators of atherosclerosis. Vascular ultrasound imaging has demonstrated aberrant endothelial function in children with familial hypercholesterolemia, as measured by provocative maneuvers that cause the brachial artery to dilate. Vascular ultrasonography has also demonstrated that hyperlipidaemia in children causes anomalies in the carotid intima’s medium thickness, distensibility and compliance. [[7](https://pubmed.ncbi.nlm.nih.gov/16709475/)][[10]](https://pubmed.ncbi.nlm.nih.gov/11733400/)[[45]](https://pubmed.ncbi.nlm.nih.gov/29858795/)

**6. DYSLIPIDEMIA**

Dyslipidemia is the term for lipoprotein abnormalities identified by laboratory testing, which typically manifest in childhood and early adulthood without any outward manifestations. Appropriate identification and handling potentially lower cardiovascular morbidity and death due to lipoprotein abnormalities. Conceptually, dyslipidemia can be classified as primary when it involves hereditary lipoprotein issues that result in abnormalities in the metabolic pathways and secondary when it comes to medication, lifestyle and environmental factors, metabolic or endocrine disorders, or coexisting conditions [[16]](https://pubmed.ncbi.nlm.nih.gov/20397826/)[[31]](https://pubmed.ncbi.nlm.nih.gov/29174025/). The most prevalent primary dyslipidemia in children and adolescents is familial hypercholesterolemia, which is an autonomic dominant condition with an incidence of 1 in 250–500 in the general population for heterozygous genotype. This is linked to a higher risk of early CVD and entails a functional change where there is a malfunction in either the LDL receptor or the LDL receptor's capacity to identify apo B. TC and LDL-C values rise noticeably as a result of this lipid condition. Compared to children whose parents had premature CVD, children with lipid abnormalities, such as an LDL-C concentration above 240 mg/dl and an HDL-C level below 40 mg/dl, were 1.7 and 1.8 times more at risk, respectively [[16]](https://pubmed.ncbi.nlm.nih.gov/20397826/) [[17]](https://repository.poltekkes-kaltim.ac.id/1157/1/Pediatric%20Nutrition%20in%20Practice%2C%202nd%20Edition.pdf)[[38]](https://pubmed.ncbi.nlm.nih.gov/33516680/). The study by Cortner et al. displayed the plasma lipid profiles of kids with and without primary dyslipidemia. Due to the high cost of tests and the challenges of using new technology to analyze lipid profiles in order to discover metabolic pathway deficiencies, the diagnosis of hereditary lipid abnormalities often happens after ruling out the most prevalent causes of secondary dyslipidemia.Obesity, which is characterized by low HDL-C concentration, elevated triglyceride levels, and alterations in the structure of LDL-C, which makes it a smaller, denser, and more atherogenic particle, is the most common secondary cause of dyslipidemia in children[[27]](https://pmc.ncbi.nlm.nih.gov/articles/PMC4107225/)[[20]](https://medpulse.in/Pediatrics/Article/Volume14Issue2/Ped_14_2_2.pdf). In affluent nations, an appropriate lipid profile was described as falling between the 5th and 95th percentiles, or between -2 and +2 standard deviations, based on population reference values for age and sex. Ethnicity and teenage sexual maturity, which are known to result in significant variations in lipoprotein and triglyceride levels because of the impact of sexual hormones, were not taken into account by these curves [[16]](https://pubmed.ncbi.nlm.nih.gov/20397826/) [[18]](https://pubmed.ncbi.nlm.nih.gov/15075702/). When one or more lipoproteins are greater than or equivalent to the 95th percentile for TC, LDL-C, triglycerides, and apo B, and lower than the 5th percentile for HDL-C and apo AI, dyslipidemia is diagnosed. The National Cholesterol Education Program (NCEP) released the first pediatric joint statement, which outlines two methods for identifying dyslipidemia in kids and teenagers. According to one of them, TC serum levels should be checked in kids with a family history of early CVD. Parents or grandparents who are 55 years of age or younger and exhibit signs of atherosclerosis, peripheral vascular disease, cerebrovascular illness, coronary artery surgery, acute myocardial infarction, or sudden cardiac death are considered to have a family history [[40]](https://pubmed.ncbi.nlm.nih.gov/33942057/)[[45]](https://pubmed.ncbi.nlm.nih.gov/29858795/). The other states that children should have their TC levels checked if their parents have a history of hypercholesterolemia (TC level ≥240 mg/dl). Repeat the measurement if the cholesterol level is borderline (170–200 mg/dl). Children should have their serum cholesterol fractions tested if the first measurement is more than 200 mg/dl or if two TC readings are 170 mg/dl or higher (Table 1). To confirm the diagnosis of dyslipidemia, serum cholesterol levels and fractions should be evaluated twice in the same clinical laboratory, always after a 12-hour overnight fast [[16]](https://pubmed.ncbi.nlm.nih.gov/20397826/) [[19]](https://pubmed.ncbi.nlm.nih.gov/30290407/).

**Table 1: Lipid Profile Table for Children and Adolescents**

|  |  |  |  |
| --- | --- | --- | --- |
| Lipoproteins | Acceptable (mg/dl) | Borderline (mg/dl) | Elevated (mg/dl) |
| TC | <150 | 150–169 | ≥170 |
| LDL-C | <100 | 100–129 | ≥130 |
| HDL-C | <45 | --- | --- |
| TG | <100 | 100–129 | ≥130 |

**7. SCREENING FOR HYPERLIPIDAEMIA**

Risk factors and risk behaviors that accelerate the development of atherosclerosis start in infancy, and there is mounting evidence that reducing risk may slow the progression of the disease and lower cardiovascular complications, even though there is rarely obvious cardiovascular damage in children and adolescents. Nonetheless, it is crucial to realize the differences and similarities among children and adolescents' risk categories, genders, ages, and ethnicities in order to fully appreciate the timeliness, relevance, and sufficiency of a broad population lipid screening. About 20% of pediatric patients have an increase in one or more blood lipid values, and this number rises to 40% in pediatric patients who are obese, which is defined as having a BMI more than the 95th percentile, according to data from the US and other nations [[2](https://pubmed.ncbi.nlm.nih.gov/36979789/)][[36]](https://pubmed.ncbi.nlm.nih.gov/30706683/). Increased TC, HDL-C, LDL-C, HDL-C, and TG levels in children are predictive of coronary artery calcium (CAC) and CIMT, two known indicators of severe atherosclerosis. Furthermore, compared to young people in good health, adolescents with elevated TCvalues had a five-fold higher chance of experiencing CVD events 40 years later. Given these assumptions, it should seem very easy to understand the crucial role and significant influence that appropriate screening may have in preventing CVD and lowering the incidence of cardiovascular mortality in both adult and pediatric patients [[15]](https://pubmed.ncbi.nlm.nih.gov/8345411/)[[40]](https://pubmed.ncbi.nlm.nih.gov/33942057/). It is well recognized that childhood hyperlipidaemia is on the rise, coupled with obesity and insulin resistance. In fact, age-based guidelines for lipid value screening in specific patients should be a typical component of routine pediatric therapy in order to identify issues at appropriate times that are pertinent to the course of the disease. A strong wide-population screening program, however, should influence the course of the disease and its long-term results and have good reproducibility, accuracy, and acceptability. Serum lipids and lipoproteins, in particular, are easily detected in blood samples, the costs are affordable, the accuracy is proven, the impact on patients is well recognized, and the technique relies heavily on the participation of qualified applicants. However, the majority of hyperlipidaemias are clinically asymptomatic, and selective screening—such as looking only for children with a positive family history—fails to detect a sizable percentage of children with lipid abnormalities, making it difficult to choose suitable kids and teenagers for lipid screening. According to the results of the 2010 "Coronary Artery Risk Detection in Appalachian Communities" (CARDIAC) Project, which involved over 20,000 fifth-graders, using family history to determine whether children needed cholesterol screening would have missed a large percentage of mild hyperlipidaemias and failed to identify a significant portion with likely hereditary hyperlipidaemias requiring pharmacological therapy [[2](https://pubmed.ncbi.nlm.nih.gov/36979789/)][[11]](https://pubmed.ncbi.nlm.nih.gov/21696080/). All children with severe hyperlipidaemia would be covered by universal cholesterol screening; however, there is currently no information on how cost-effective pediatric cholesterol screening methods are. Natural history study indicates that 25% of women and 50% of men with FH develop clinical CVD by the age of 50 due to elevated LDL-C levels, despite these acquisitions. A family history of CVD is recognized as a significant risk factor. However, there is no standardized method for assessing family history of CVD, which is often inaccurate and insufficient. Individual lipid screening is supported by the fact that between 25 and 55 percent of children have family history of early CVD [[28]](https://publications.aap.org/pediatrics/article/150/6/e2022057957/189891/Cardiovascular-Risk-Assessment-and-Management-for). This fact is incredibly relevant because, even though pediatric medication trials have relatively short follow-up periods, statin therapy significantly lowers LDL-C levels and delays the development of atherosclerosis and CVD in RCTs, including older children and adolescents with FH. Furthermore, since first-degree relatives of children with elevated LDL-C values also had higher LDL-C levels and a higher incidence of CV events, lipid screening ought to be expanded to the entire family. However, as stated in the same recommendations, economic evaluations play a significant role in screening decisions. To identify hyperlipidaemias in kids and teens, several screening techniques have been put forth, including the universal method, population screening for a particular age group, the selective method, which evaluates a particular high-risk population, the cascade method, which screens from an index case to family members, the reverse cascade method, which evaluates from pediatric affected patients to other family members, and the child-parent method, which screens from kids at a particular age to parents[[12]](https://pubmed.ncbi.nlm.nih.gov/18200807/)[[30]](https://pubmed.ncbi.nlm.nih.gov/38097375/). The strategy should be age-based if the patient has no risk factors: ----

 Children under 9: no screening

 Every youngster aged 9 to 11 should have their cholesterol levels evaluated once;

 Due to fluctuations in lipid levels throughout puberty, screening is not advised for children aged 12 to 16.

 One assessment of lipid levels was conducted for individuals aged 17 to 21.

 Lipid screening should be done when a kid or teenager acquires one or more risk factors, which is often after the age of two. However, depending on the patient's individual risk level, monitoring should continue until the risk factor is present, with lipid value assessments occurring every one to three years. Individualization is a key factor for correct lipid screening. Indeed, when a single mild risk factor is present, such as smoking exposure without other risk factors or mild obesity, it is permissible to start screening later and less often, while lipid tests should be performed sooner and more often for high risk children such as those with Kawasaki illness and coronary artery aneurysm (Table 2) [[2](https://pubmed.ncbi.nlm.nih.gov/36979789/)][[46]](https://pubmed.ncbi.nlm.nih.gov/35328769/).

**Table 2: Assessment of lipid levels was conducted for individuals aged 12 to 16 years**

|  |  |  |
| --- | --- | --- |
|  **Category** | **Cholesterol Level** |  **Next Steps** |
| Acceptable (Total) | < 170 mg/dL | Repeat in 5 years; provide diet and education |
| Borderline (Total) | 170–200 mg/dL | Repeat and average; if <170 mg/dL, follow “Acceptable” protocol; if ≥170 mg/dL, proceed to fasting lipoprotein analysis |
| High (Total) | > 200 mg/dL | Do fasting lipoprotein analysis (TChol, LDL, HDL, TGs) |
| Acceptable (LDL) | < 110 mg/dL | Repeat lipoprotein analysis within 5 years; provide education on eating patterns and risk factors |
| Borderline (LDL) | 110–130 mg/dL | Risk factor advice; provide Step-One Diet and other interventions; re-evaluate in 1 year |
| High (LDL) | ≥ 130 mg/dL | Clinical evaluation (history, exam, labs); evaluate for secondary/familial causes; intensive intervention; set LDL goals; Step-One then Step-Two Diet |

method for screening and management developed by the NCEP Expert Panel. NCEP stands for National Cholesterol Education Program; HDL stands for high density lipoprotein; LDL stands for low density lipoprotein; TG stands for triglycerides; TChol for total cholesterol.

**8. TREATMENT OF HYPERLIPIDAEMIA**

Every kid and adolescent should adopt healthy habits, but those with hyperlipidaemia in particular should. Although there are a number of published recommendations and guidelines for treating lipid abnormalities in children, most patients and physicians are not sufficiently aware of them. According to current guidelines, the main treatment for children hyperlipidaemia is a healthy lifestyle, in which parents are extremely important. In fact, parents play with their kids to help them develop the necessary skills in addition to providing a movement example. In particular, dietary changes, consistent exercise, weight loss, and quitting smoking in late adolescence should all be part of therapeutic lifestyle modifications. Therapeutic options for children with hyperlipidaemia have significantly increased since 2011, even if the food and lifestyle recommendations evaluated in the NHLBI guidelines issued in 2011 are generally still regarded as relevant and effective [[46]](https://pubmed.ncbi.nlm.nih.gov/35328769/)[[49]](https://www.ahajournals.org/doi/full/10.1161/CIRCGENETICS.116.001604).

**8.1 PHYSICAL ACTIVITY**

Sedentary lifestyles raise blood lipid levels, according to recent research.

In fact, watching too much television is linked to higher TG and lower HDL-C, most likely as a result of lower energy use. But among the many known benefits of exercise are improved cardiovascular, mental, behavioral, and musculoskeletal health. In particular, exercise improves blood pressure, lipid profiles, insulin sensitivity, serum glucose levels, and cardiorespiratory fitness. Additionally, exercise increases bone density and balance, which shields adults and children against harm and falls [[2](https://pubmed.ncbi.nlm.nih.gov/36979789/)][[48]](https://www.ahajournals.org/doi/10.1161/CIR.0000000000000618). Both healthy individuals and children with hyperlipidaemia or obesity should engage in regular physical activity to raise HDL-C, lower TC, TG, and LDL-C, and—above all—reduce body fat and improve BMI. Recent research has shown a dose-response relationship between increased physical activity duration and improved lipid concentrations (HDL-C and TG values), emphasizing once more the need for all patients aged 6 to 17 to engage in at least 60 minutes of moderate-to-intense physical activity daily and at least three days of bone- and muscle-strengthening activities. In the meanwhile, preschool-aged children between the ages of three and five should engage in physical play at various points during the day, per the 2018 Physical Activity Guidelines Advisory Committee. However, by assessing and documenting gross motor skills and physical activity during checkups, talking about the benefits of exercise for growth and development, and encouraging physical activity for all kids through health care, insurance, schools, and community organizations, pediatricians can advance toward recommended guidelines and encourage physical literacy and exercise in kids [[26]](https://pubmed.ncbi.nlm.nih.gov/34572264/)[[29]](https://pubmed.ncbi.nlm.nih.gov/27881102/). However, up to 80% of American adults and adolescents are deemed to be insufficiently active, and there is still more work to be done to raise public awareness and ensure that rules are followed. Parents should also encourage their kids to get enough sleep and to spend no more than two hours a day using screens like computers and cell phones. Shorter sleep durations are linked to a higher risk of overweight and obesity in children under the age of 18, according to recent meta-analyses. The American Academy of Sleep Medicine's most recent Consensus Statement states that in order to maintain good health, babies ages 4 to 12 months should sleep 12 to 16 hours every 24 hours, and toddlers ages 1 to 2 years should sleep 11 to 14 hours daily.[[13]](https://pubmed.ncbi.nlm.nih.gov/27592099/)[[41]](https://pubmed.ncbi.nlm.nih.gov/30639555/).

**8.2 DIETARY THERAPY**

Lipid-lowering dietary therapy has been promoted as the cornerstone of management due to its assumed safety (Table 3). Although many dietary supplements have also been promoted in various ways, the emphasis has been on a diet low in fat and cholesterol. A comprehensive multi-institution, randomized clinical trial examined the safety of the diet low in fat and cholesterol. 663 kids with mild to moderate hypercholesterolemia who didn't fit the requirements for medication lipid-lowering therapy were enrolled in this experiment. They were randomly assigned to either the usual care group or an intervention that involved extensive nutrition education with the goal of lowering the daily consumption of cholesterol to fewer than 150 mg, saturated fat to less than 8%, and total fat to less than 28% of calorie intake[[14]](https://pubmed.ncbi.nlm.nih.gov/8883496/)[[47]](https://pubmed.ncbi.nlm.nih.gov/34508626/). The LDL level decreased from 131 to 119 mg/dL in the usual care group and from 131 to 115 mg/dL in the diet intervention group over the course of a year. An additional trial demonstrated that the intervention was maintained for up to seven years of follow-up. Psychologic examination, growth and sexual development, and any nutritional safety measures did not reveal any abnormalities. In addition, 1062 infants aged 7 months were randomly assigned to either a control diet or a dietary intervention that intended to reduce the daily intake of cholesterol and the dietary fat composition ratio to less than 200 mg. At age five, males had lower LDL levels (15 mg/dL) than controls, whereas females had lower levels (6 mg/dL) [[7](https://pubmed.ncbi.nlm.nih.gov/16709475/)][[14]](https://pubmed.ncbi.nlm.nih.gov/8883496/).

**Table 3: Dietary Therapy**

|  |  |
| --- | --- |
| **Condition** | **Recommendations** |
| Elevated LDL-cholesterol | • Reduce food consumption with high saturated fat (meats, poultry and organ meats)• Limit fried foods and products with hydrogenated fat consumption• Limit egg yolk intake (2–3/week)• Use vegetable oils (soybean, canola and olive oil) and soft margarines• Eat more vegetables, fruits and legumes• Increase intake of high-fiber foods, whole-grains and cereals• Use low-fat or skim milk, and dairy products• Include fish at least twice a week |
| High triglycerides and low HDL-cholesterol | • Limit refined carbohydrates, including from industrialized products (juice and soft drink)• Eat more vegetables, fruits and legumes• Increase intake of high-fiber foods, wholegrain and cereals• Use vegetable oils (soybean, canola and olive oil)• Eat fish 2–3/week |

**8.3 NUTRACEUTICALS THERAPY**

When dietary measures, particularly in high-risk instances, are unable to attain target lipid levels, pharmacological therapy for hyperlipidaemia in children is taken into consideration. If a child's LDL cholesterol is still more than 190 mg/dL or above 160 mg/dL with a family history of early cardiovascular disease or other risk factors, the National Cholesterol Education Program (NCEP) advises starting medication therapy. Bile acid-binding resins, statins, niacin, and fibrates are the primary groups of medications that decrease cholesterol. Since bile acid-binding resins, such cholestyramine and colestipol, are not absorbed systemically, they are frequently used as first-line medications for pediatric patients, minimizing systemic side effects. However, the gastrointestinal side effects and poor palatability of these medications frequently make compliance difficult [[7]](https://pubmed.ncbi.nlm.nih.gov/16709475/)[[15]](https://pubmed.ncbi.nlm.nih.gov/8345411/). Statins that block HMG-CoA reductase, like simvastatin, pravastatin, lovastatin, fluvastatin, and atorvastatin, have been shown to significantly lower LDL cholesterol. They also have other advantages, like better endothelial function and a decrease in inflammatory indicators. Although there is currently a lack of long-term data, clinical trials in children have demonstrated their efficacy and overall safety. Because it causes flushing, glucose intolerance, and hepatotoxicity, niacin—which raises HDL while lowering LDL and triglycerides—is not as widely used [[7]](https://pubmed.ncbi.nlm.nih.gov/16709475/)[[27]](https://pubmed.ncbi.nlm.nih.gov/25061583/). Although there is little information on the safety and effectiveness of fibrates, like bezafibrate, in children, they mainly enhance HDL levels and decrease triglycerides. Throughout treatment, it is crucial to keep an eye on lipid profiles, muscle enzymes, and liver function tests. According to recent data, children with familial hypercholesterolemia may benefit from lipid-lowering medications, especially statins, which may lower cholesterol while also improving vascular function. Drug therapy, however, needs to be carefully adapted to each patient's risk profile and is only advised following lifestyle changes[[7]](https://pubmed.ncbi.nlm.nih.gov/16709475/)[[15]](https://pubmed.ncbi.nlm.nih.gov/7859485/)[[50]](https://pubmed.ncbi.nlm.nih.gov/31227123/).

**9. CONCLUSION**

It has been demonstrated that hyperlipidaemia and the athero-sclerotic process in kids and teenagers are related. Considering the prevalence of more weight and related cardiovascular risk factors, there is a compelling need to put in place a program for screening and control. Diets low in fat and cholesterol have had varying degrees of success in treating hyperlipidaemia. It is probable that the prevention and treatment of overweight and obesity should become the primary emphasis of nutritional management. Clinical trials of lipid-lowering medication therapy in children are indicating efficacy and safety comparable to those seen in adults. Early research indicates that treatment can help children with hyperlipidaemia by improving endothelial function.

 This could imply a preventive translation or delayed development of adult-onset atherosclerotic cardiovascular disease.

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