The Role of Some LPL Gene Polymorphisms with Cardiovascular Disease

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Abstract: Cardiovascular burden still holds the top position for global mortality rates. Still, how exactly small genetic differences tied to lipid metabolism affect personal risk varies between different groups. One factor involves changes in a gene called LPL - specifically HindIII, known as SNP rs320, and another named Ser447Ter, or SNP rs328. Data shows SNPrs320 tends to go with higher triglyceride numbers, along with differing degrees of harm depending on group origin. In contrast, SNP rs328 usually comes with healthier lipid profiles and a lower chance of blood vessel damage. Altogether, what we know so far shows how these gene changes act differently based on where people live and their ancestry. Because of this, looking closely at genetics in specific areas - like Georgia (South Caucasus), which hasn't been studied much - can sharpen how we judge cardiovascular risks while shaping prevention plans that actually fit the group they're meant for.

Key-words: cardiovascular disease, lipid metabolism genes, population study, inheritance, HindIII, Ser447Ter.

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1 Introduction

Across the globe, about every third person is currently battling a life-limiting illness that restricts their daily life and shortens their years. Whether it is heart attacks, different types of cancer, metabolic disorders, or the weight of mental health, these conditions are redefining the landscape of wellness and leaving behind a profound, long-term impact. These illnesses tend to cluster within families. We can say that this kind of disease "runs in the family". Because of this pattern, first-degree relatives have higher chances of getting ill than those found in the general population. Still, remember - they do not follow classic genetic rules; in other words, no solitary gene flaw causes them.

One cause behind most of these conditions lies in several gene changes that make someone more likely to develop illness. There is more polygenic inheritance and complex genetic or multifactorial disorders. Not just DNA matters - life exposures play a role too, shaping how risk unfolds. Genetic and nongenetic factors influence the clinical manifestations of the disease-associated symptoms. When genes and surroundings team up, symptoms may emerge faster - or sometimes that mix blocks sickness instead. These illnesses do not follow one path. Shared family patterns show up often, since relatives carry similar genetic codes (predisposition) by chance alone. Such overlap makes groups within families stand out when studying who gets sick. Living in much the same surroundings means exposure to matching outside influences. Things like habits, diet, different types of viral and microbial infections, daily pressures, living geographic area, traditions, socioeconomic condition, shared routines - these often line up closely among relatives. Because of this setup, close family ties tend to show closer links in how genes interact with one another or respond to life conditions compared to those unrelated by genetics.

We wanted to find existing studies on cardiovascular diseases tied to inherited traits. Jumping into hereditary patterns comes after a short look at what cardiovascular disease actually involves.

2 Review Methodology

In modern times, there are many technical supports for the diagnosis and treatment of various diseases. Despite major advances in prevention and treatment, diseases of the heart and blood vessels still cause more deaths worldwide than any other group of conditions and diseases. For the detailed study of disease genetic predisposition in important. There are some single-gene inheritance (SNP) and polygenic factors that are associated with promoting the symptoms and signs of cardiovascular disease. Knowing the associated genes earlier is most important for preventing disorders or reducing the time to clinical manifestation and the severity of symptoms. We aimed to synthesize and analyse information about LPL Gene Polymorphisms and their association with the general type of cardiovascular disease. Our review is intended for high-level educational students (especially master's and doctoral students).

This literature review of ours was conducted as part of a targeted grant project, "Association of LPL Gene Genotype with Cardiovascular Diseases", set at Batumi Shota Rustaveli State University between 2025 and 2027. Search work followed a clear plan, leaning mostly on PubMed for sourcing. PubMed is a good scientific platform for medical, biological, and health-related research. Here are reliable sources with a large database. Most importantly, it is free access and is very useful for students, researchers, etc. PubMed gives us the opportunity to find new scientific papers regularly, with updated information.

Terms like "cardiovascular disease", "lipid metabolism genes", "lipoprotein lipase", "HindIII", "rs320", "Ser447Ter", "Single nucleotide polymorphism", and "rs328" shaped each query, mixed in various ways. To narrow things down, Boolean operators (AND/OR) were used as filters. One step at a time, these phrases helped narrow down relevant studies without overlap. From beginning to end, methodical choices guided how information came together.

Preference was given to papers from 2020 to 2026. Yet some older papers were considered, as they showed core mechanisms or broad review data. Original studies came into view along with structured summaries. After screening and removing non-eligible records, 28 peer-reviewed articles were selected for analysis. We examined how these genetic variations function, affecting lipid levels. Also, regarding their role in cardiovascular diseases such as CAD, MI, and stroke. We gather information relative to different populations, too.

3 Cardiovascular Disease: A Syndrome of Genetic Predisposition and Environmental Risk

Even now, heart-related diseases take more lives than any other disease around the world. About 19.8 million people died from them in 2022 alone, says the World Health Organization's (WHO) latest report, [1]. That number adds up to roughly 30% of all deaths everywhere. Most of these are caused by blocked blood arteries in the heart or bleeding in the brain. Damage builds very slowly inside the blood vessel walls, way ahead of clinical symptoms. In most cases, the underlying pathology revolves around atherosclerosis, endothelial wall dysfunction, clot formation, and chronic inflammation. All these together compromise blood flow to vital organs. A range of illnesses falls under cardiovascular diseases, like blocked arteries, heart attacks, strokes, weak hearts, and poor circulation in limbs. These problems arise not from one single cause, yet through layers: genetic inherited risks mix with surrounding environmental factors plus daily habits such as diet, traditions, religion, or movement, [2], [3], [4].

Knowing which genes are involved helps make sense of how diseases start, plus it shapes better ways to forecast risks and take early steps. Researchers have found plenty of gene spots linked to higher chances, though each alone does little (complex inheritance). Heart problems aren't driven by one broken switch; they build up through layers of small shifts across different genes. A mix of inherited patterns works behind the scenes, never just one change pulling all the strings. Not everyone carries the same genetic blueprint - small differences stack up over time, nudged by routine choices and surroundings, inching risk upward.

Certain gene clusters tied to cardiovascular health fall into different categories. Some of them are as follows: 1. Lipid metabolism genes, 2. Thrombosis and blood clotting genes,3. Immune response genes 4. Blood pressure-regulating genes. The above grouping of genes is presented in Table 1(Appendix). In Table 1(Appendix), the primary functions of the

mentioned genes and gene clusters are also shown, along with their prevalence in cardiovascular disease and the disorder's pathophysiology.

The study of all genes provides more powerful information regarding the predisposition. Our area of interest was lipid metabolism genes and their association with cardiovascular diseases. As seen in the table below, multiple clusters of genes fall into this category. The primary focus is given to the lipid metabolism genes, as it plays a key role in lipid-driven vascular injury. These clusters together shape levels of plasma lipids, lipoproteins, and vascular lipid clearance.

3.1 LPL Gene Complex

One gene that stands out when talking about fat metabolism is LPL - it plays a big role in heart-related health issues. It modulates the activity of a key enzyme that is crucial for the hydrolysis of triglycerides in circulating chylomicrons and VLDL. Certain gene variants, such as HindIII (rs320) and Ser447Ter (rs328), modulate enzyme expression and activity, influencing the levels of plasma triglyceride and HDL-C, [5]. When certain impaired versions slow things down, fats build up in ways that harm arteries; on the flip side, stronger forms speed the breakdown of fats and tend to guard against damage.

Solid research shows how changes in LPL affect blood vessels. A big analysis combining data from over eighty separate reports found the D9N mutation raises the chances of heart artery problems. On the flip side, those carrying either HindIII markers or the active Ser447X form tend to have better fat levels in their blood - pointing toward lower threat. In another look across seven variations, Sagoo's team noted similar trends linking helpful LPL forms to healthier cholesterol patterns. Together, these results paint a clear picture: differences in the LPL gene shape how fats move through the body, placing each version somewhere on a line from harmful to guarding, [5], [6].

3.2 APOE Gene Complex

Different forms of the APOE gene - called ε2, ε3, ε4 - affect how the liver clears lipoprotein remnants from blood. When someone carries the ε4 version, their LDL cholesterol tends to rise, along with a higher risk for artery buildup. On the flip side, having ε2 often means lower danger, even though it sometimes leads to a rare form of lipid metabolism disorder called type III hyperlipoproteinemia. As APOE teams up with LPL enzyme activity, it helps clear chylomicron and VLDL debris from blood, showing how genetic differences in proteins can quietly shape heart disease likelihood, [7].

Not long ago, fresh findings spotlighted how APOE behaves when the body faces metabolic stress. In a study by Liu and team from 2019, people carrying the ε4 variant who also had type 2 diabetes showed stronger signs of heart-related problems - possibly tied to disrupted fat processing during bodily strain. Those with either ε3/ε4 or ε4/ε4 versions of the gene faced greater odds of narrowed arteries if they were living with type 2 diabetes, revealing how genes can amplify harm when metabolism falters, [7].

3.3 CETP Gene Complex

Cholesteryl ester transfer between HDL and apoB-containing particles is supported by CETP. When CETP works less due to certain changes, HDL-C often goes up - yet how well cholesterol moves backward might shift, [8]. These genetic differences in CETP act differently depending on the group studied; some versions work better if a person also has helpful LPL forms like Ser447Ter.

Recent studies help clarify this tangled biological process. Though the Taq1 B2 variant links to higher HDL-C, findings from a 2014 meta-analysis by Wu and Lou show it doesn't always mean less chance of heart disease - hinting that more HDL might not equal better protection. Changes in the CETP gene region affect how HDL works and shape heart-related risks. What we see now points to CETP playing a role deeper than just raising HDL numbers, possibly tied to how HDL operates and connects with other genes involved in lipid handling, [9].

3.4 LDLR Gene Complex

Now picture liver cells grabbing LDL bits from blood using special gates made by the LDLR gene. When some changes happen in that gene (for example, single-nucleotide mutations - missense, or nonsense of frame shift), those gates work poorly, so cholesterol sticks around longer. Because of this delay, more lipid residue can build up inside the arteries over time. How well these receptors team up with lipid - handling proteins decides how fast these residues get removed. That cleanup speed shapes how quickly trouble might start in vessel walls, [10].

Studies tracking large groups show certain LDLR gene types link to CAD at younger ages, pointing to how multiple genes together fuel plaque buildup, [11]. What's more, genetic data drawn from inheritance patterns reveal that people born with lower LDL cholesterol - often due to LDLR-related

mechanisms - face far less chance of heart issues over time. It turns out these LDLR variations do more than just signal risk - they help steer both lipid levels in blood and overall cardiovascular health.

3.5 LIPC Gene Complex

Hepatic lipase comes from the LIPC gene, working on reshaping HDL while turning IDL into LDL. Changes in its promoter adjust how active the enzyme is, shifting both the size and the weight of HDL particles. Alongside certain LPL forms, those shifts affect lipid processing and atherogenic risks, showing the interconnected nature of lipid metabolism systems, [12].

What happens in the liver with lipid processing ties into many moving parts. Work by Shohet and team showed a change in the LIPC gene linked to less enzyme function, along with more HDL cholesterol in men's blood; yet, people with or without heart vessel issues carried this form just as often, hinting that tweaking one enzyme might not shift real-world heart risks, [13]. Far from seeing HDL as always good, digging into LPL gene forms reveals risk shifts based on surrounding genes; take a fresh look at data from 80 reports - the D9N version raises chances of heart trouble, but HindIII slightly lowers it, pushing science to see liver enzymes as part of a larger DNA puzzle guiding lipids in the body, [6].

Packed into clusters, these genes build a finely tuned system managing plasma lipid levels, reshaping particles that carry cholesterol, along with where lipid molecules settle in vessel walls. What happens between them shows how genetic differences - mixed with lifestyle and food habits - influence heart-related risks person by person. So it goes - links between certain gene forms like HindIII and Ser447Ter aren’t fate, just shifts in odds for heart-related results. Looked at one way, changes in the LPL gene show how lipid processing, blood vessel function, and inherited traits tug at each other. It turns out minute gene variations can nudge health patterns, especially when mixed with eating habits, excess weight, or existing illnesses. From here, digging into these connections adds depth to how heart disease takes hold, while quietly pointing toward more tailored medical approaches.

Some genes tied to lipid handling - like LPL, APOE, LDLR, and CETP - heavily shape plasma lipid concentration and how likely artery damage becomes. When these systems go off track, they spark problems like stiffened vessel walls, fatty buildup, and even clots. Each gene tweak alone does little. Yet pile them up, mix in poor diet or smoking, danger climbs fast. Heart troubles then emerge not from one broken switch but many small nudges acting together. Blame rarely lands on a single genetic marker. Patterns matter more than a single gene fault, [11], [14].

Apart from genes, things like eating lots of processed fats, sitting too much, smoking, ongoing mental strain, plus health issues such as high blood pressure, extra body weight, and trouble managing sugar, push cardiovascular risks higher. These pieces tend to mix with inherited tendencies, shifting how cholesterol moves, how the body handles inflammation, and how blood vessels work. That is one reason people born with almost identical DNA might end up very differently when it comes to cardiovascular health, based on daily choices and surroundings, [15].

Heart problems show up in many ways. Some involve narrowed arteries that lead to heart attacks or strokes. Others tie into how body metabolism affects blood vessels when cholesterol is off balance or when weight adds strain. Irregular beats or weak pumping also count. All these pieces connect - each hints at how deeply linked the body's circulation system really is.

How common a condition is changes a lot between groups because of genes, what people eat, and where they live. Take heart problems in South Asians - these show up more often and start earlier, usually along with low good cholesterol and trouble using insulin, [16]. In a similar fashion, people from the Middle East and North Africa face steeper odds of unhealthy fats in blood and early hardening of arteries, shaped by inherited traits plus shifts in daily routines. On the flip side, some Europeans carry helpful gene variants like the Ser447Ter change in the LPL gene, tied to better lipid metabolism and stronger responses to treatment, [17]. Some African groups carry distinct genetic patterns, so standard risk markers might matter less there - maybe due to long-term adaptation, [18]. Because of these population-specific traits, heart disease studies need local context in both genetics and care.

Because things are so intertwined, giving special attention to certain genes like Lipoprotein Lipase matters more. This enzyme is the core controller of lipid breakdown, turning lipid-carrying particles into pieces the body can use, which shifts how much lipid floats in blood and whether it clogs arteries. Any changes to the LPL gene can alter the way in which the enzyme works. Either it slows down or speeds up, reshaping how lipids move through the bloodstream and affecting heart disease odds. Seeing these DNA

clues alongside many other factors helps scientists make sense of links between genes and illness - never set in stone, always one thread in a web that shapes heart well-being.

4 Lipoprotein Lipase: A Critical Enzyme in Lipid Homeostasis

Lipid concentration in the plasma is managed by a protein called lipoprotein lipase, which also helps to keep cardiovascular health stable. Mainly made in adipocytes, skeletal muscles, and heart tissue, this enzyme moves to tiny blood vessels where it sticks through a molecule named GPIHBP1. There, it breaks down lipid particles like chylomicrons and VLDL floating in the bloodstream. Once split apart, these FFAs move into nearby organs - either burned for fuel or tucked away as stored fat. How much goes in either direction depends on hormones calling the shots. After eating, insulin ramps up the enzyme's work in fat tissue, shifting incoming calories toward storage, yet at the same time dials back its effort in muscle areas. Midway through a fast or while moving your body, stress hormones kick in, turning up LPL activity in muscle and heart tissue to fuel ongoing effort, [19]. Suddenly, fat becomes the go-to source - not because it's trendy, but because biology adapts. What seems like a switch is really fine-tuned control: LPL adjusts based on whether you're fed or drained.

Beyond breaking down lipids, LPL shapes how lipoproteins change form. Phospholipids move toward young HDL particles because LPL makes it possible, along with certain proteins, once bound to fat-heavy leftovers. New HDL forms more easily this way, aiding the body's method for shuttling cholesterol away from blood vessels. Less plaque builds up inside the arteries since this process keeps circulation stable. If LPL slows, triglycerides pile up in blood, HDL dips, and leftover particles loaded with cholesterol stick around longer - together fueling artery damage, [20].

LPL might do more than just break down fats. Sometimes it helps particles stick to certain body structures - like docking stations on cells. This sticking process happens through links with heparan sulfate proteoglycans or similar receptors. When things run smoothly, that means better fat transport where needed. But inside damaged artery walls, immune cells make LPL too - and there, it backfires. Instead of helping, it piles up lipid deposits in those spots. That pile-up turns into dangerous clumps known as foam cells, [21].

LPL isn't just about breaking down triglycerides - its role stretches further, quietly shaping how lipids move through the body and influencing blood vessel function. When something interferes with how it works, or when genes shift its control, that steady rhythm falters, opening space for heart-related issues to take hold.

5 HindIII (rs320) and Ser447Ter (rs328): Functional Variants of Clinical Significance

A stretch of DNA on chromosome 8p21.3 holds the LPL gene, taking up around 30 thousand base pairs and built from ten separate coding sections. Among changes spotted here, one named HindIII (rs320), along with another called Ser447Ter (rs328), draws strong attention due to their clear links to how fats move through blood and affect heart health.

A single letter swap - cytosine for thymine - in stretch eight of the gene marks the rs320 spot, known as HindIII. This shift sits outside protein blueprints yet still trips up helper segments and messes with RNA editing, leading to less LPL production, [5]. People carrying the H^+ version often show skewed fat markers: higher triglycerides, lower HDL levels - a pattern seen among groups in the Middle East, [20].

Nucleotide sequence for rs320:

Wild-type allele: AGTCTCGAAT

Mutant allele: AGTCTTGAAT

A change in exon 9 known as Ser447X (rs328) comes from a DNA swap that halts protein building too soon, leading to a shorter version of the LPL enzyme missing its last two building blocks. Though cut short, this altered form works harder than usual, breaking down fats faster, which helps clear triglycerides more effectively - shaping a healthier fat pattern in blood, [22]. People who inherit this version tend to have less fat floating in their bloodstream, better levels of good cholesterol, and fewer artery problems over time.

Nucleotide sequence for rs328:

Starting off differently, the wild-type allele holds a TCA sequence right where serine shows up at spot 447

Mutant allele: …TGA (Stop codon) at position 447…

Certain genetic patterns tied to ethnicity add clarity about how gene changes behave. Noticing differences in LPL rs320 among people with heart issues compared to healthy individuals in South Asia hints at ancestry shaping how this variation acts,

affecting cholesterol-related risks, [23]. Studies from the Middle East find that distinct inherited blocks make rs320 more involved in unhealthy fat levels and early heart disease, [20]. Starting elsewhere - Europe - it's a different story: versions carrying rs328 tend to guard against problems, seen through better plasma lipids and stronger reactions to medication. From Africa, the layout of genes shifts again; there, rs320 matters less while advantages from rs328 appear unevenly across groups, [5].

6 Coronary Artery Disease: LPL Variants and Atherogenesis

CAD arises due to lipid deposits, ongoing inflammation, cell damage, and endothelial dysfunction. Changes in the LPL gene - like those at rs320 and rs328 - play into each of these factors. Because of that, they can shift how likely someone is to develop coronary problems. Sometimes it's protection; other times it raises risk.

Some large-scale studies found the HindIII gene variation tied to higher triglycerides and greater heart disease risk. Still, when combining many analyses, the link shifts - sometimes stronger, sometimes reversed - based on ancestry or how the study was set up. So this particular marker acts more like a shifting influence, shaped by surroundings, not a clear-cut danger sign. These lipid remnants slip into blood vessel walls, undergo oxidation, and then trigger inflammation signals. Once inside, immune cells swallow the transformed lipids, turning into bloated foam cells - the start of artery plaque buildup, [24]. People with the H^+ version tend to carry more triglycerides, produce denser LDL particles, and run lower HDL across repeated observations. Lipid imbalances together speed up how quickly plaque builds and breaks down. Research among people in Saudi Arabia and South Asia links rs320 closely to more cases of heart artery disease and blocked blood flow strokes, showing its effects reach arteries throughout the body, not just the heart's pathways, [20].

One form of the gene, called Ser447X (or rs328), boosts fat breakdown by activating an enzyme named LPL. Because of this effect, people who carry it tend to have lower levels of triglycerides in their blood. Their HDL cholesterol often runs higher than average, too. The type of LDL particles shifts - becoming bigger and likely less harmful. These changes appear linked to fewer cases of heart-related blockages in arteries. Recent data from many studies together confirm that risk drops noticeably for those with the variant. Still, there's no solid proof yet that it affects how blood vessels widen through nitric oxide. Inflammation markers do not clearly change either. Nor has any study shown its influence on the physical makeup of artery plaques, [22].

Carried along in longer DNA segments, rs320 links to worse cholesterol patterns and quicker buildup in arteries. On the flip side, versions carrying rs328 tend to support healthier blood lipids and respond better to therapy. What people eat plays a big part too - meals high in solid fats make the negative effects of rs320 even more pronounced. In contrast, eating foods with omega-3s boosts the ability of rs328 to lower triglycerides, something shown clearly when genes and diet are studied together under strict conditions, [5]. Environmental interactions matter just as much: heavy saturated fat intake worsens what rs320 can do, but omega-3 components strengthen how rs328 protects. Looking across populations through wide genetic scans shows both uncommon broken forms of LPL and frequent gene variants tied to higher triglycerides sharply raise the chances of heart vessel disease. One clear example: those who inherit faulty LPL copies face higher levels of circulating lipids and close to twice the likelihood of developing heart problems early in life - a sign of how vital LPL is in keeping lipid balance and preventing artery damage, [25]. LPL gene variations are just one of the several factors that contribute to CAD risk. Sometimes, their presence controls how well certain treatments work. That knowledge helps explain why people respond differently to the same treatment methods.

7 Myocardial Infarction: The Acute Face of LPL-Related Risk

MI, also known as a heart attack, is a medical emergency due to the sudden narrowing of the coronary arteries. In most cases, this vessel narrowing is the consequence of plaque rupture, thrombosis, and ischemia. New data points to changes in the LPL gene as key players, shaping how lipid particles behave and how vessels react. One look at a large group in China found people with the HindIII (rs320) faced much greater odds of heart attack, especially males. One group stood out with high triglyceride levels along with poor ApoB to ApoA1 balance - signs tied to fragile plaque, [26]. In Saudi Arabia, similar results appeared: people carrying the HindIII variant carried more tiny, dense LDL plus oxidized forms, setting conditions ripe for clots, [20].

Looking wider, Sagoo and team in 2008 pulled data showing LPL gene shifts affect long-term fat imbalances as well as sudden heart risks, revealing how deeply this gene shapes blood vessel health [5]. From another angle, He and colleagues in 2018 found that weaker LPL function leads to remnants of chylomicrons piling up, stirring inflammation through macrophage triggers and feeding fatty cell buildup inside arteries, [12].

People who carry the Ser447X gene change often show better plasma lipid profiles. This variation has been shown to boost the enzyme function, cut triglycerides, raises HDL, [25]. These shifts link to fewer heart problems in major studies. The connection varies, though - some groups see strong effects, others do not.

8 Stroke and Cerebrovascular Outcomes: Extending the Cardiovascular Map

It turns out LPL gene changes might matter more for stroke than was previously thought, even if research has mostly focused elsewhere. When it comes to pathologies related to the cerebrovascular network, trouble clearing lipids appears tied to damage in small vessels. Though strokes and heart attacks start differently, both involve abnormal lipid metabolism, cell stress, and harm to vessel walls. Clues are adding up that impaired lipid processing doesn't just clog arteries - it may also weaken the brain's blood supply.

Looking into stroke patterns across parts of South and East Asia, one variant - Ser447Ter (rs328) - shows signs of lowering the chance of blocked blood flow in the brain. This likely happens because lipids move out of the bloodstream faster, leaving fewer remnant particles behind, [26]. Among Han Chinese residing at middle and higher altitudes, changes in the APOE gene link more strongly to CAD. The mix of gene types found in sick people does not match that of those who stay healthy. When surroundings shift, so might how genes affect lipid levels, [27].

Several recent meta-analytic studies show that carriers of the LPL HindIII (rs320) polymorphism have a lower risk of stroke, specifically among Asian populations, a protective trend not steadily observed for Ser447Ter, but highlighting that certain LPL variants may regulate cerebrovascular risk depending on genetic makeup and lifestyle context, [28]. In South Indian cohorts, the LPL HindIII (rs320) variant has been tied with higher prevalence of MI and adverse lipid profiles, [23].

9 Dyslipidemia: The Metabolic Background to Cardiovascular Pathology

Some changes in the LPL gene are tied directly to how lipids move through the bloodstream, shaping heart disease odds. People with the HindIII H^+ variant tend to have higher triglyceride levels, lower HDL-C, and slower fat breakdown after meals. In South Asian groups, the rs320 marker keeps showing up alongside high triglycerides and unhealthy LDL-C patterns, especially as newer broad analyses revisit HindIII-related lipid imbalances, [21].

A change in the lipoprotein lipase gene, known as Ser447X (rs328), links to better plasma lipid profiles - especially lower triglycerides, along with slightly raised HDL cholesterol. People with this variation tend to have fewer heart problems over time, according to long-term study data, [22]. When people maintain a healthy weight and exercise regularly, the benefit seems stronger; under these conditions, the body may clear lipids from blood more efficiently, [22].

10 Regional Discrepancies and the Imperative for Georgian Data

Even though studies worldwide connect certain LPL gene forms to heart disease risk, big combined analyses show notable differences across groups. Take the HindIII and Ser447X variations - these often lower risk in people of European descent, yet their role isn't as clear elsewhere, suggesting real genetic differences tied to ancestry, [5], [6]. Such gaps might be due to influences other than genetics, like eating habits, daily routine, physical activity, socioeconomic factors, and epigenetics.

Situated at the intersection of Eastern Europe and West Asia, Georgia holds a mix of genetic backgrounds rarely seen together. From Slavic roots to Central Asian threads, its people carry layers shaped by centuries of movement. Because results differ so widely elsewhere, looking closely at Georgian DNA matters more than ever. What happens here might not reflect what's been found on distant shores. Curiosity drives the effort to map how certain genes spread among those feeling well and those facing illness.

11 Quantitative and Computational Perspectives on LPL Variants

Even though this analysis follows a narrative format, findings on LPL gene variants (rs320 and rs328) tied to cardiovascular outcomes are usually examined through logistic regression in the referenced studies. When the health outcome has two possible states, the genetic makeup's link to illness likelihood can take the form of:

(1)

$$\log [P(D=1) / (1 - P(D=1))] = \beta 0 + \beta 1 G(rs320) + \beta 2 G (rs328) + \beta 3(E) + \beta 4(G \times E)$$

where D represents disease status (e.g., CAD or MI), G(rs320) and G(rs328) denote genotype coding according to allele count (0, 1, or 2), E represents environmental exposure such as body mass index or dietary pattern, and G×E reflects gene–environment interaction. In many association studies cited in this review, the coefficients β1 and β2 correspond to odds ratios describing the effect of each polymorphism on cardiovascular risk. Allele frequency distributions are commonly evaluated under the Hardy–Weinberg equilibrium:

(2)

$$p^2 + 2pq + q^2 = 1$$

Where the variables, p and q, represent allele frequencies in the studied population. Deviations from equilibrium may indicate population stratification or selection effects.

One way forward might involve using simulations like Monte Carlo methods to gauge overall genetic influence. Repeated draws of genotype patterns from existing group data help shape these estimates. Odds ratios pulled from earlier reports feed into the process, too. Small shifts tied to each gene version can then show up clearly when linked to heart-related risk across large groups. The bigger picture emerges through many cycles of modeled variation.

12 Reference Summary of Selected Studies

The Table 2 (Appendix) shows some selected scientific literature related to LPL polymorphisms and cardiovascular disease, together with a brief overview of their core insights.

13 Conclusion

Cardiovascular system pathologies stem from many factors where genes and surroundings interact over time. Not far beneath the surface, changes in the LPL gene - like HindIII (SNPrs320) and Ser447Ter (SNPrs328) - affect how lipids move through blood. Triglyceride shift, HDL remodelling, and atherogenic risk depend on these markers. From one group to another, data shows SNPrs328 often links to healthier lipid profiles and fewer cardiac problems. Yet rs320 behaves differently - not always clear, its impact is shaped by lifestyle and location. Surroundings bend the outcome; genes alone do not decide the fate. Each variation alters likelihood, never guarantees illness. Alone they stand - small players in a much larger story.

Looking ahead, closer study of specific areas makes sense - especially among people in Georgia - to better understand how gene variations spread and respond to lifestyle factors. Integrating molecular genetics with quantitative modelling may further improve cardiovascular risk stratification and contribute to precision medicine approaches.

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APPENDIX

Table 1. Single genes associated with cardiovascular disease

Gene Group	Representative Genes	Primary Function / Pathway	Relevance to CVD Pathophysiology
Lipid Metabolism Genes	*LPL*, *APOE*, *APOB*, *LDLR*, *CETP*, *LIPC*, *LPA*	Regulate synthesis, transport, and clearance of lipoproteins; maintain cholesterol and triglyceride homeostasis.	Variants alter plasma lipid concentrations, remnant accumulation, and efficiency of reverse cholesterol transport, influencing atherogenesis.
Thrombosis and Coagulation Genes	*F5* (Factor V), *F2* (Prothrombin), *SERPINE1* (PAI-1), *ITGA2B*, *ITGB3*	Mediate coagulation, fibrinolysis, and platelet aggregation.	Mutations increase hypercoagulability and susceptibility to thrombosis, predisposing to acute coronary and cerebrovascular events.
Inflammatory and Immune Response Genes	*IL6*, *CRP*, *TNF-α*, *ICAM1*, *VCAM1*	Coordinate cytokine signalling and vascular inflammatory response.	Polymorphisms heighten systemic inflammation, endothelial activation, and plaque instability, aggravating atherosclerotic progression.
Blood Pressure-Regulating Genes	*ACE*, *AGT*, *AGTR1*, *NOS3*, *ADD1*	Control vascular tone, endothelial nitric oxide synthesis, and sodium–water balance.	Variants contribute to hypertension, arterial stiffness, and increased hemodynamic stress, amplifying cardiovascular risk.

Source: created by the authors.

Table 2. Summary of selected literature

No	Article Title	Authors	Year	Journal	Short Summary
1	Cardiovascular diseases (CVDs) fact sheet	WHO	2025	WHO	Provides updated global data on cardiovascular prevalence and mortality.
2	Global burden of cardiovascular diseases	GBD 2019 Cardiovascular Diseases Collaborators	2020	J. Am. Coll. Cardiol.	Summarizes worldwide trends and emphasizes prevention priorities.
3	Genetic risk, lifestyle, and coronary disease	Khera et al.	2016	N. Engl. J. Med.	Shows that healthy lifestyle habits reduce inherited CAD risk.
4	Lifestyle–genetic interactions in CAD	Said et al.	2019	Curr. Cardiol. Rep.	Examines how behavioral factors modify genetic susceptibility.
5	LPL polymorphisms meta-analysis	Sagoo et al.	2008	Am. J. Epidemiol.	Evaluates associations between multiple LPL SNPs and CAD.

6	Updated LPL meta-analysis	Ma et al.	2018	Biosci. Rep.	Confirms cumulative evidence linking LPL variants to CAD.
7	APOE polymorphism and CVD	Liu et al.	2019	BMC Cardiovasc. Disord.	Links APOE variants with cardiovascular and metabolic risk.
8	LPL genotype and triglycerides	Smith et al.	2010	Hum. Mol. Genet.	Demonstrates genotype influence on plasma triglyceride levels.
9	CETP polymorphism meta-analysis	Wu et al.	2014	BMC Med. Genet.	Uses genetic modeling to assess CETP contribution to CAD.
10	LPL haplotypes and statin response	Goodarzi et al.	2007	Pharmacogenomics J.	Shows LPL variation influences HDL response to statins.
11	GWAS meta-analysis of CAD	CARDIoGRAMplusC4D Consortium	2015	Nat. Genet.	Identifies multiple genomic loci associated with CAD.
12	LPL biosynthesis and disease	He et al.	2018	Clin. Chim. Acta	Reviews regulatory and functional aspects of LPL.
13	Hepatic lipase polymorphism in CAD	Shohet et al.	1999	Arterioscler. Thromb. Vasc. Biol.	Describes the impact of LIPC variants on HDL levels.
14	Genetic risk and statin benefit	Mega et al.	2015	Lancet	Demonstrates enhanced statin benefit in high-risk genotypes.
15	Lipoprotein(a) and ASCVD	Patel et al.	2025	J. Am. Heart Assoc.	Highlights Lp(a) as a major cardiovascular risk determinant.
16	INTERHEART study	Yusuf et al.	2004	Lancet	Identifies modifiable global risk factors for MI.
17	ESC/EAS dyslipidemia guidelines	Mach et al.	2020	Eur. Heart J.	Provides updated lipid management recommendations.
18	Dyslipidaemia in the elderly	Katsiki et al.	2018	Expert Rev. Clin. Pharmacol.	Discusses treatment considerations in older adults.
19	Regulation of LPL	Kersten	2014	Biochim. Biophys. Acta	Explains the physiological control of LPL activity.
20	HindIII variant and CAD	Bogari et al.	2020	Saudi J. Biol. Sci.	Links rs320 with increased CAD and stroke risk.
21	HindIII polymorphism review	Rojas et al.	2018	F1000Research	Summarizes evidence relating HindIII to dyslipidemia.
22	S447X variant and CHD	Jensen et al.	2009	Am. Heart J.	Reports the protective association of the S447X variant.

23	HindIII and MI (India)	Tanguturi et al.	2013	Indian Heart J.	Shows the association between rs320 and MI risk.
24	LPL structure and function	Mead et al.	2002	J. Mol. Med.	Provides foundational insight into LPL biology.
25	Rare LPL variants and CAD	Kera et al.	2017	JAMA Cardiol.	Identifies the impact of rare and common LPL variants.
26	HindIII and ischemic stroke	Cao et al.	2018	Medicine	Reports a protective association in stroke.
27	APOE polymorphisms in China	Zeng et al.	2024	Preprint	Examines regional APOE variation and CAD risk.
28	LPL polymorphisms and stroke	Nejati et al.	2018	J. Stroke Cerebrovasc. Dis.	Meta-analysis linking LPL variants with stroke susceptibility.

Source: created by the authors.

Abbreviations

ACE – Angiotensin-Converting Enzyme
ADD1 – Alpha-Adducin 1
AGT – Angiotensinogen
AGTR1 – Angiotensin II Receptor Type 1
APOA1 – Apolipoprotein A1
APOB – Apolipoprotein B
APOE – Apolipoprotein E
CAD – Coronary Artery Disease
CETP – Cholesteryl Ester Transfer Protein
CRP – C-Reactive Protein
CVD – Cardiovascular Disease
D9N – Aspartic Acid to Asparagine substitution at position 9
eNOS – Endothelial Nitric Oxide Synthase
F2 – Coagulation Factor II (Prothrombin) Gene
F5 – Coagulation Factor V Gene
FFA – Free Fatty Acids
GPIHBP1 – Glycosylphosphatidylinositol-Anchored High-Density Lipoprotein-Binding Protein 1
HDL – High-Density Lipoprotein
HDL-C – High-Density Lipoprotein Cholesterol
HindIII – Restriction Fragment Length Polymorphism detected by HindIII enzyme (LPL rs320 variant)
HuGE – Human Genome Epidemiology
ICAM1 – Intercellular Adhesion Molecule 1
IDL – Intermediate-Density Lipoprotein
IL6 – Interleukin 6
ITGA2B – Integrin Subunit Alpha 2b
ITGB3 – Integrin Subunit Beta 3
LDL – Low-Density Lipoprotein
LDL-C – Low-Density Lipoprotein Cholesterol
LDLR – Low-Density Lipoprotein Receptor
LIPC – Hepatic Lipase Gene (Lipase C, Hepatic Type)
LPA – Lipoprotein(a)
LPL – Lipoprotein Lipase
MI – Myocardial Infarction
NO – Nitric Oxide
NOS3 – Nitric Oxide Synthase 3
PAI-1 – Plasminogen Activator Inhibitor-1
rs320 – Reference Single-Nucleotide Polymorphism ID 320 (HindIII polymorphism of LPL gene)
rs328 – Reference Single-Nucleotide Polymorphism ID 328 (Ser447Ter polymorphism of LPL gene)
SER447X – Serine to Stop Codon substitution at amino acid position 447
SERPINE1 – Serpin Family E Member 1
Ser447Ter – Serine to Termination Codon substitution at position 447
SNP – Single-Nucleotide Polymorphism
T2DM – Type 2 Diabetes Mellitus
Taq1B – TaqI Restriction Fragment Length Polymorphism in the CETP gene
TNF-α – Tumor Necrosis Factor Alpha
VCAM1 – Vascular Cell Adhesion Molecule 1
VLDL – Very Low-Density Lipoprotein
WHO – World Health Organization

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Conflict of Interest

The authors have no conflicts of interest to declare that are relevant to the content of this article.