Journal of Advances in Drug Research

ISSN: 2230-7761
www.jadr.co.in

Volume 17, Issue 05 | Received 10.10.2026 | Accepted 11.10.2026 | Online 15.10.2026

CARDIOVASCULAR DISEASES AMONG YOUNG PEOPLE: AETIOLOGICAL DETERMINANTS, CONTEMPORARY TREATMENT STRATEGIES AND PREVENTIVE INTERVENTIONS

Sirojov Javohir Tohir o‘g‘li

Teacher, Department of Fundamental Medicine

Asia International University, Bukhara, Uzbekistan

Email: **javohirsirojov06@gmail.com**

ORCID iD: **0009-0000-3603-8332**

ABSTRACT

Cardiovascular disease (CVD) is conventionally regarded as a disorder of middle and old age, yet the decline in cardiovascular mortality achieved in older populations has not been matched among adolescents and young adults, and rising rates of obesity, hypertension, physical inactivity and substance use in early life threaten to reverse several decades of progress. This review synthesises current evidence on the causes of CVD in young people, the treatment strategies available to them and the preventive measures most likely to reduce their lifetime risk. A structured narrative review was conducted: landmark autopsy studies, prospective cohorts, registries of premature myocardial infarction, randomised trials, pooled global analyses and international guidelines published between 1998 and 2023 were purposively selected and synthesised thematically under four domains – burden, aetiology, treatment and prevention. Atherosclerosis begins in adolescence, and its extent is proportional to the number of conventional risk factors present. Childhood risk factor levels predict fatal cardiovascular events in adulthood, and blood pressure or LDL cholesterol elevations before the age of 40 confer excess risk independent of later exposure. Globally, 81% of school-going adolescents are insufficiently active, and the number of children and adolescents with obesity rose from 11 million in 1975 to 124 million in 2016. In registries of premature myocardial infarction, most patients carried at least one modifiable risk factor, but only a minority would have qualified for preventive pharmacotherapy beforehand, whereas early statin therapy in familial hypercholesterolaemia markedly reduced events by the age of 39. Cardiovascular disease in the young is therefore largely driven by modifiable, early-acquired exposures that current short-term risk models fail to capture; a life-course approach combining population-level policy, systematic screening for inherited and early-onset risk factors and timely treatment is warranted, and region-specific data, particularly from Central Asia, remain a research priority.

Journal of Advances in Drug Research

ISSN: 2230-7761
www.jadr.co.in

Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

Keywords: cardiovascular disease; young adults; premature atherosclerosis; risk factors; primary prevention; familial hypercholesterolaemia; lifetime risk.

INTRODUCTION

Cardiovascular diseases remain the leading cause of death worldwide. According to the World Health Organization, they were responsible for an estimated 17.9 million deaths in 2019, or roughly 32% of all global deaths, and more than three quarters of these occurred in low- and middle-income countries [1]. The Global Burden of Disease study further showed that prevalent cases of CVD almost doubled, from 271 million in 1990 to 523 million in 2019, while annual deaths rose from 12.1 million to 18.6 million [2]. Although these figures are dominated by older age groups, they conceal a worrying trend at the other end of the life course. In many high-income countries, age-standardised cardiovascular mortality has fallen steadily since the 1970s; however, this decline has slowed or stalled among younger adults [3]. In the Atherosclerosis Risk in Communities (ARIC) surveillance study, the proportion of hospital admissions for acute myocardial infarction attributable to patients aged 35 to 54 years increased from 27% in 1995–1999 to 32% in 2010–2014, and among women the share rose from 21% to 31% [4]. Similarly, Gupta et al. found no decline in hospitalisation rates for myocardial infarction among adults aged 30 to 54 years between 2001 and 2010, in contrast to the falling rates seen in older patients [5]. Consequently, a condition long considered rare before middle age now accounts for a growing share of the acute cardiac workload.

The consequences of early-onset disease are disproportionate. A cardiovascular event at the age of 35 removes decades of productive life, imposes prolonged treatment costs and frequently recurs: Collet et al., who followed 880 patients with coronary artery disease diagnosed at or before 45 years, reported that about one third experienced at least one further ischaemic event during long-term follow-up [6]. CVD in the young is therefore not only a clinical problem but also a social and economic one. The central problem is a mismatch between when cardiovascular risk is acquired and when it is addressed. Risk factors accumulate in childhood and adolescence, whereas prevention systems are designed around the ten-year risk of middle-aged adults. Because age is the strongest term in every short-term risk equation, a 30-year-old with several risk factors is almost always classified as low risk; as a result, young people are rarely screened, seldom treated and often unaware of their own risk. Moreover, the risk factor profile of this age group is changing: obesity, type 2 diabetes, sedentary behaviour, novel nicotine products and recreational drugs have become more prevalent, and their combined long-term effect is not yet fully understood.

The aim of this review is to provide an integrated, evidence-based synthesis of the causes, treatment and prevention of cardiovascular disease in young people. The object of the study is atherosclerotic cardiovascular disease and its antecedent risk factors in individuals aged approximately 15 to 45 years. The review addresses three research questions: which biological, behavioural and social factors explain the occurrence of CVD in young people; which treatment strategies are supported by evidence in this age group, and where that evidence falls short; and which preventive measures offer the greatest potential to reduce lifetime cardiovascular risk. The working hypotheses are that (H1) the majority of premature cardiovascular events are attributable to conventional modifiable risk factors acquired early in life; (H2) existing risk-assessment tools systematically under-

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

identify young people at high lifetime risk; and (H3) interventions initiated in childhood or early adulthood yield larger long-term benefit than equivalent interventions started later.

LITERATURE REVIEW

The scientific basis for early-life prevention rests on three lines of evidence. First, autopsy studies demonstrated that atherosclerosis starts young. The Bogalusa Heart Study showed that the extent of fatty streaks and fibrous plaques in the aorta and coronary arteries of children and young adults increased with body mass index, blood pressure and serum lipid levels, and rose sharply as the number of risk factors increased [7]. The Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study confirmed that intimal lesions were present in all aortas and in more than half of the right coronary arteries of individuals aged 15 to 19 years [8].

Second, prospective cohorts linked early exposures to later outcomes. Juonala et al. demonstrated that children with overweight who remained obese as adults had a higher risk of type 2 diabetes, hypertension, dyslipidaemia and carotid atherosclerosis, whereas those who became non-obese by adulthood had risks similar to people who had never been obese [9]. More recently, the International Childhood Cardiovascular Cohort (i3C) Consortium, with 38,589 participants, showed that childhood body mass index, systolic blood pressure, cholesterol, triglycerides and smoking were each associated with cardiovascular events in midlife [10], and analyses of cumulative exposure indicated that the same level of LDL cholesterol is more harmful when it is accumulated earlier in life [11,12].

Third, case–control and registry studies characterised the patients who actually develop premature disease. INTERHEART, conducted in 52 countries with 15,152 cases and 14,820 controls, showed that nine potentially modifiable factors accounted for about 90% of the population-attributable risk of myocardial infarction in men and 94% in women, and that these associations held in younger as well as older participants [13]. The Partners YOUNG-MI registry subsequently revealed that most patients with a first infarction at or before 50 years had at least one risk factor, yet few had been eligible for statin therapy under prevailing guidelines [14]. Despite this substantial body of work, several gaps remain. Most cohorts originate from North America, Western Europe and Finland, while data from Central Asia, including Uzbekistan, are sparse; definitions of “young” vary from under 35 to under 55 years, which hampers comparison; randomised trials of pharmacological prevention have largely excluded participants younger than 40; and the literature tends to treat aetiology, treatment and prevention separately, so an integrated account aimed at clinicians and educators is lacking.

MATERIALS AND METHODS

This study is a structured narrative review based on secondary analysis of published data. A narrative design was chosen in preference to a meta-analysis because the research questions span epidemiology, pathophysiology, therapeutics and public health, and because the available studies differ widely in design, age definitions and outcome measures. No new data were collected from human participants; therefore, ethical approval was not required. The reporting was guided by the principles of the Scale for the Assessment of Narrative Review Articles (SANRA) [15], and the transparency elements of the PRISMA 2020 statement were followed where they apply to a non-systematic review [16].

Journal of Advances in Drug Research

ISSN: 2230-7761
www.jadr.co.in

Volume 17, Issue 05 | Received 10.10.2026 | Accepted 11.10.2026 | Online 15.10.2026

The evidence base was assembled from peer-reviewed literature indexed in major biomedical databases (PubMed/MEDLINE, Scopus and Web of Science), supplemented by documents of the World Health Organization and by clinical practice guidelines of the European Society of Cardiology, the American College of Cardiology, the American Heart Association and the American Academy of Pediatrics. The conceptual search terms combined three groups: population (“young adults”, “adolescents”, “children”, “premature”, “early-onset”); condition (“cardiovascular disease”, “coronary artery disease”, “myocardial infarction”, “atherosclerosis”, “hypertension”); and domain (“risk factors”, “aetiology”, “treatment”, “statin”, “prevention”, “lifestyle”). Reference lists of key guidelines and state-of-the-art reviews were examined to identify additional landmark studies.

Sources were selected purposively rather than exhaustively. Priority was given to studies published between 1998 and 2023 in English; with a study population, or a clearly reported subgroup, aged 45 years or younger at baseline or at the index event, or a paediatric cohort followed into adulthood; with a design high in the evidence hierarchy for the question at hand (autopsy series and prospective cohorts for aetiology, registries for clinical characteristics, randomised trials or long-term controlled follow-up for treatment, and pooled global analyses for exposure prevalence); and reporting quantitative outcomes such as prevalence, hazard ratios or event rates. Small case series, studies of congenital or purely structural heart disease, and studies of cardiomyopathies and channelopathies were excluded, since their causes and management differ fundamentally from those of atherosclerotic disease. Trials conducted in older populations were retained only where no evidence in younger people existed, and such indirect evidence is identified explicitly in the Results. The final evidence set comprised 26 sources (Table 1). Because selection was purposive, no flow diagram of screened records is presented, and the set should not be read as a complete census of the literature.

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

Table 1. Composition of the evidence set by study type.

Study type	Number of sources	Main use in the review
Autopsy studies	2	Onset and extent of early atherosclerosis
Prospective cohort or pooled cohort analyses	7	Effect of early exposures on later events
Registry or surveillance studies of premature MI	4	Burden, trends and patient characteristics
International case–control study	1	Population-attributable risk of modifiable factors
Treatment studies	2	Evidence for early and lifestyle intervention
Pooled global analyses of exposure or burden	4	Prevalence of risk factors and disease burden
Clinical guidelines or scientific statements	4	Screening, treatment and risk assessment
Reviews	2	Context and emerging risk factors
Total	26	

For each source, the following items were extracted: design, setting, sample size, age range, exposure or intervention, outcome and principal effect estimate; extracted data were organised in a summary matrix. Analysis followed a thematic synthesis approach in three steps: findings were first coded deductively to the four predefined domains of burden, aetiology, treatment and prevention; within each domain, findings were then compared for direction and magnitude of effect; finally, the domains were integrated to test the three working hypotheses. Effect estimates are reported exactly as published; no pooling, re-analysis or statistical software was applied, and the figures below re-plot published values without modification. Where definitions of "young" differed between studies, the original age bands are stated.

RESULTS

The included surveillance studies consistently show that younger adults have not shared in the decline of coronary events. In the ARIC community surveillance, patients aged 35 to 54 years made up 27% of myocardial infarction admissions in 1995–1999 and 32% in 2010–2014; the increase was concentrated in women, from 21% to 31% (Figure 1), and over the same period the prevalence of hypertension and diabetes among these young patients increased [4]. In United States national data for 2001–2010, hospitalisation rates for myocardial infarction in adults aged 30 to 54 years did not fall, and young women had more comorbidity, longer hospital stays and higher in-hospital mortality than young men [5]. Furthermore, premature disease proved to be recurrent: among 880 patients with

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

coronary disease at or before 45 years, approximately one in three had a further ischaemic event during follow-up, and persistent smoking was among the strongest correlates of recurrence [6].

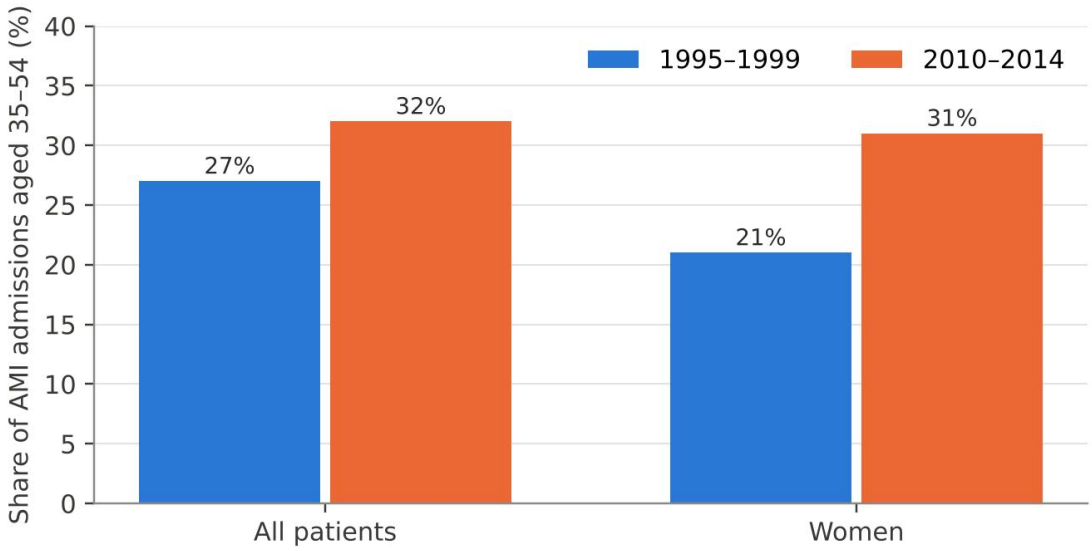


Figure 1. *Share of acute myocardial infarction admissions occurring in patients aged 35–54 years, ARIC Community Surveillance, 1995–1999 versus 2010–2014 (plotted from published data [4]).*

With regard to aetiology, both autopsy studies documented atherosclerotic lesions in the second decade of life. In the PDAY study, every aorta and more than half of the right coronary arteries examined in the 15–19 age group contained intimal lesions [8]; in Bogalusa, lesion extent correlated with body mass index, systolic blood pressure, LDL cholesterol and triglycerides, and it increased markedly with the number of coexisting risk factors [7]. Conventional risk factors dominated the clinical picture. In INTERHEART, smoking, abnormal lipids, hypertension, diabetes, abdominal obesity, psychosocial stress, low fruit and vegetable intake, lack of regular physical activity and absence of moderate alcohol intake together explained about 90% of the population-attributable risk in men and 94% in women [13], and in the YOUNG-MI registry 83% of patients with a first infarction at or before 50 years had at least one conventional risk factor [14].

The timing of exposure proved to be as important as its presence. In CARDIA, among 4,851 adults whose blood pressure was classified before the age of 40, the adjusted hazard ratios for subsequent cardiovascular events were 1.67 for elevated blood pressure, 1.75 for stage 1 hypertension and 3.49 for stage 2 hypertension, compared with normal blood pressure (Figure 2) [17]. In pooled cohort data, LDL cholesterol of 100 mg/dL or above during young adulthood was associated with a 64% higher risk of coronary heart disease, independent of later-life levels [12], and Domanski et al. likewise found that, for the same cumulative LDL exposure, risk was greater when the exposure occurred earlier [11]. The origins of this risk reach back into childhood: in the i3C consortium (38,589 participants; 319 fatal cardiovascular events), each unit increase in a combined childhood risk z-score

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

was associated with a hazard ratio of 2.71 for a fatal cardiovascular event in adulthood [10], whereas the excess risk associated with childhood overweight was largely absent in those who were not obese as adults [9].

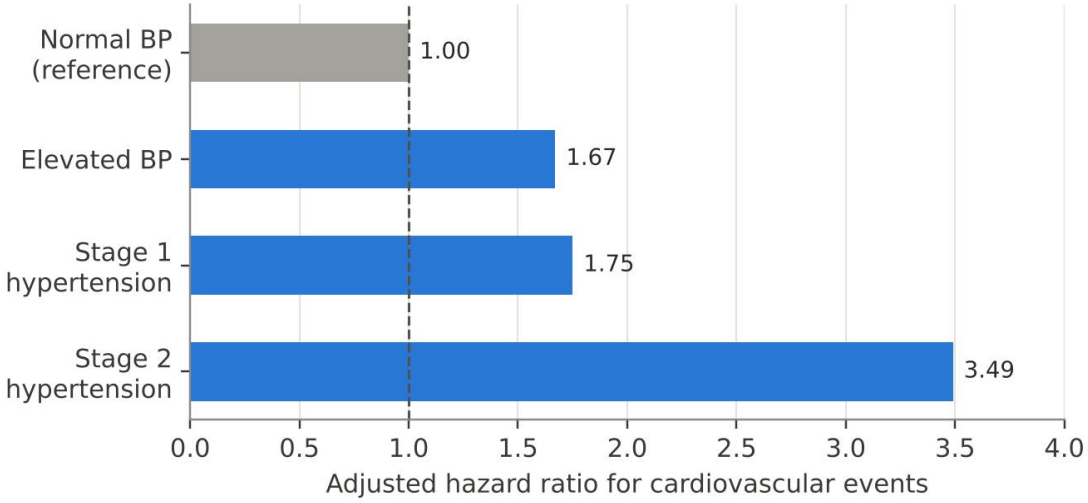


Figure 2. *Adjusted hazard ratios for later cardiovascular events by blood pressure category assessed before the age of 40 in the CARDIA cohort (n = 4,851; plotted from published data [17]).*

The pooled global analyses indicate how widespread these exposures have become. The number of children and adolescents aged 5 to 19 years with obesity increased from 11 million in 1975 to 124 million in 2016 [18]; in 2016, 81% of school-going adolescents aged 11 to 17 years did not meet physical activity recommendations, including 77.6% of boys and 84.7% of girls [19] (Figure 3); and the pooled prevalence of hypertension in children and adolescents was 4.0%, with a further 9.7% classified as prehypertensive [20]. Beyond these conventional exposures, heterozygous familial hypercholesterolaemia, which affects roughly 1 in 250 people, exposes carriers to high LDL cholesterol from birth and remains widely underdiagnosed [21], and the reviews identify recreational substances such as cocaine and cannabis, electronic nicotine products, chronic inflammatory conditions, adverse pregnancy outcomes and psychosocial stress as risk enhancers that are comparatively more important in the young [22]. Table 2 summarises the principal quantitative findings.

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

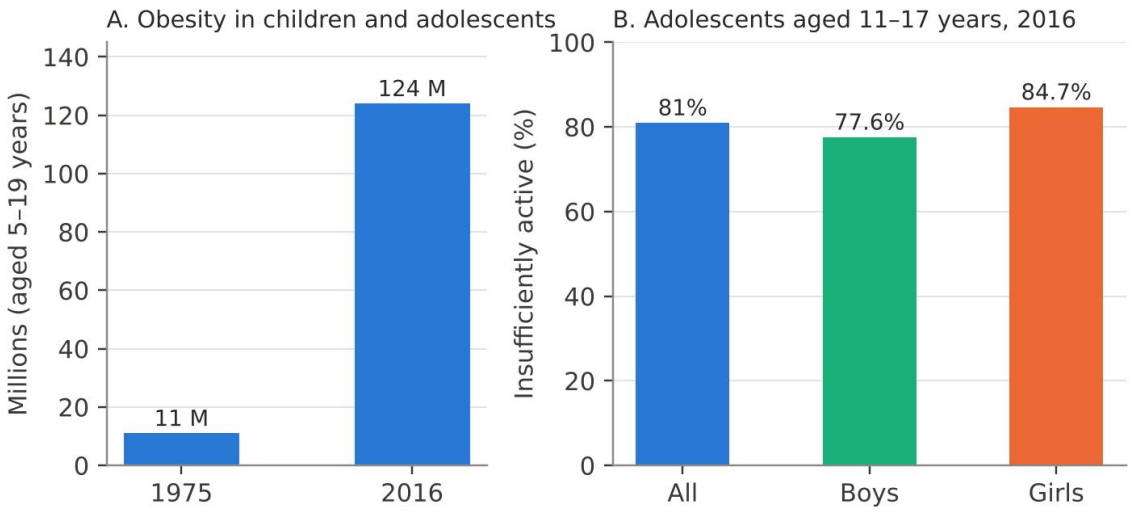


Figure 3. *Global prevalence of key exposures in the young: (A) number of children and adolescents aged 5–19 years with obesity, 1975 and 2016 [18]; (B) proportion of school-going adolescents aged 11–17 years not meeting physical activity recommendations, 2016 [19].*

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

Table 2. Key studies on cardiovascular risk in young people.

Study	Design and sample	Age group	Principal finding
Berenson et al., 1998 (Bogalusa) [7]	Autopsy series	2–39 years	Lesion extent rises with number of risk factors
Strong et al., 1999 (PDAY) [8]	Autopsy series	15–34 years	Aortic lesions in all subjects aged 15–19
Yusuf et al., 2004 (INTERHEART) [13]	Case–control, 52 countries, 15,152 cases	All ages, young subgroup	Nine factors explain about 90% of risk
Juonala et al., 2011 [9]	Four pooled cohorts	Childhood to adulthood	Risk reversed if obesity resolves by adulthood
Yano et al., 2018 (CARDIA) [17]	Cohort, n = 4,851	18–40 years	HR 3.49 for stage 2 hypertension
Singh et al., 2018 (YOUNG-MI) [14]	Registry of first MI	50 years or younger	83% had a risk factor; 12.5% on statin before MI
Arora et al., 2019 (ARIC) [4]	Community surveillance	35–54 years	Share of MI admissions rose from 27% to 32%
Luirink et al., 2019 [23]	20-year follow-up, n = 214	Statin started in childhood	Events by age 39: 1% vs 26% in affected parents
Guthold et al., 2020 [19]	298 surveys, 1.6 million participants	11–17 years	81% insufficiently active
Jacobs et al., 2022 (i3C) [10]	Seven pooled cohorts, n = 38,589	Childhood to midlife	HR 2.71 per unit of combined risk z-score

No source identified a treatment pathway specific to young patients; acute coronary syndromes are managed with the same reperfusion, antithrombotic and lipid-lowering strategies used in older adults. However, the registries reveal gaps before and after the event. In YOUNG-MI, only 12.5% of patients were taking a statin before their infarction, and only about half would have been eligible for one under the 2013 ACC/AHA criteria, falling to fewer than one in three under the 2016 USPSTF criteria [14]; after the event, continued smoking was strongly associated with recurrence [6]. The strongest direct evidence for early pharmacological treatment comes from familial hypercholesterolaemia: among 214 patients who started statins in childhood and were followed for 20 years, the cumulative incidence of cardiovascular events by the age of 39 was 1%, compared with 26% in their affected parents, and cardiovascular death occurred in none of the patients versus 7% of the parents (Figure 4) [23]. For young adults without the condition, the ACC/AHA guideline recommends statins at ages 20 to 39 when LDL cholesterol is 190 mg/dL or higher, and suggests considering them at 160 mg/dL or higher with a family history of premature disease [24].

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

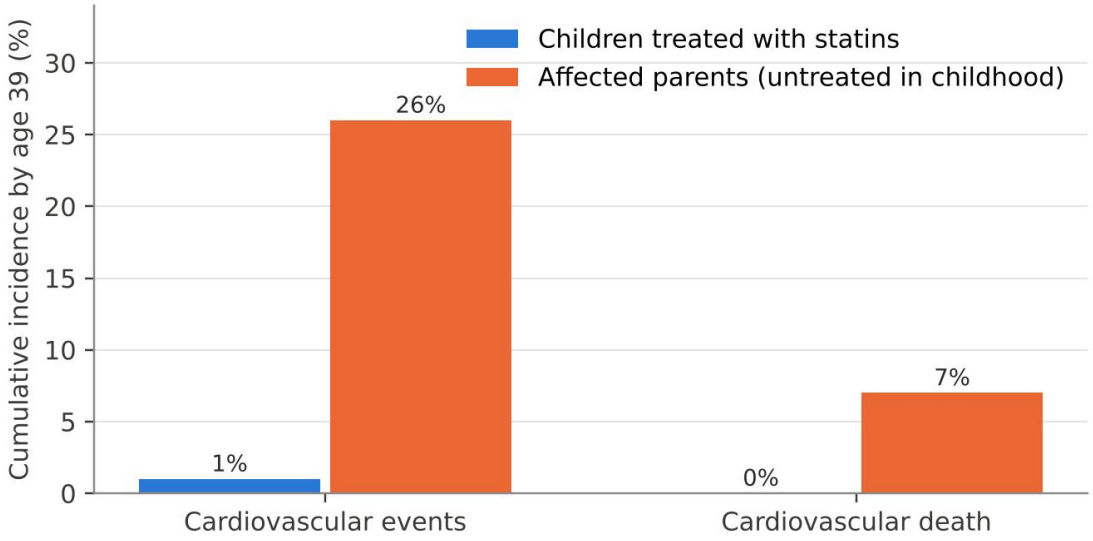


Figure 4. *Cumulative incidence of cardiovascular events and cardiovascular death by the age of 39 in patients with familial hypercholesterolaemia treated with statins from childhood (n = 214) compared with their affected parents (plotted from published data [23]).*

For blood pressure, the paediatric guideline recommends annual measurement from the age of three, lifestyle intervention as first-line treatment, and drug therapy with an ACE inhibitor, angiotensin receptor blocker, long-acting calcium channel blocker or thiazide diuretic when hypertension persists [25]. Both the European and American guidelines acknowledge that ten-year risk estimates are low in young people almost regardless of risk factor burden, and they recommend lifetime or 30-year risk estimation to guide discussion in adults under 40 [21,24].

Regarding prevention, the American Heart Association's Life's Essential 8 defines cardiovascular health by eight components – diet, physical activity, nicotine exposure, sleep, body mass index, blood lipids, blood glucose and blood pressure – each measurable from childhood [26]. Framingham data showed that lifetime risk of CVD at the age of 50 was 5.2% in men and 8.2% in women with an optimal risk factor profile, against 68.9% and 50.2% respectively in those with two or more major risk factors (Figure 5) [27], and Berry et al. confirmed this gradient across birth cohorts and ethnic groups [28].

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

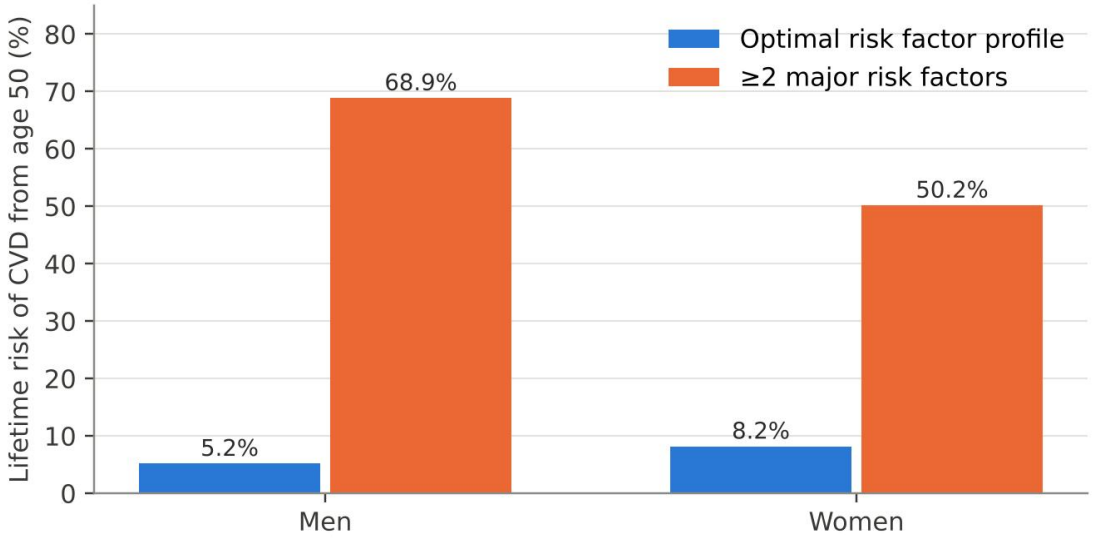


Figure 5. *Lifetime risk of cardiovascular disease from the age of 50 by risk factor burden in men and women, Framingham Heart Study (plotted from published data [27]).*

Guidelines recommend at least 60 minutes of moderate-to-vigorous activity daily for children and adolescents and 150 to 300 minutes weekly for adults, complete avoidance of tobacco, and a dietary pattern rich in vegetables, fruit, whole grains, legumes, nuts and fish [21]. The only large randomised trial of diet with hard outcomes, PREDIMED, reported about a 30% relative reduction in major cardiovascular events with a Mediterranean diet in 7,447 participants [29]; this evidence is indirect, however, because participants were aged 55 to 80 years. Finally, cascade or universal lipid screening in childhood is recommended to detect familial hypercholesterolaemia, and risk factor assessment every four to six years is advised from the age of 20 [21,24].

DISCUSSION

Taken together, the results support all three working hypotheses. With regard to H1, the evidence indicates that premature cardiovascular disease is not, in most cases, an unexplained or purely genetic phenomenon: more than four in five young patients with myocardial infarction carried at least one conventional risk factor, and the autopsy data show a dose–response relationship between the number of risk factors and the extent of arterial lesions as early as adolescence. In other words, the disease that presents at 40 has usually been developing for two or three decades. With regard to H2, the findings expose a structural blind spot. Only about half of the young patients in YOUNG-MI would have qualified for a statin on the day before their infarction. This is not a failure of clinicians but of the instruments they are given: because short-term risk is driven by age, a young person's risk score remains low even when several risk factors coexist. The lifetime risk data make the contrast explicit – a 50-year-old man with optimal risk factors faces a lifetime risk of about 5%, whereas one with two or more major risk factors faces a risk approaching 70%, yet both may be labelled "low risk" at 30. With regard to H3, three independent observations converge: earlier LDL exposure is more harmful than later

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

exposure of the same magnitude; obesity that resolves before adulthood leaves little residual risk; and statins started in childhood for familial hypercholesterolaemia almost abolished the events that had affected the previous generation. Accordingly, the timing of an intervention appears to matter as much as its intensity.

These conclusions are consistent with the view of Andersson and Vasan that the stagnation of cardiovascular mortality in young adults reflects the obesity and diabetes epidemics offsetting gains from reduced smoking and better acute care [3]. They also agree with Stone et al., who argue that risk in young adults should be judged by cumulative exposure and risk-enhancing factors rather than by a ten-year estimate [22]. Nevertheless, some differences deserve attention. INTERHEART suggested that the same nine risk factors operate across all ages [13], whereas the more recent reviews emphasise factors that are relatively specific to the young, such as recreational drug use, electronic nicotine products, inflammatory disease and adverse pregnancy outcomes [22]. These positions are complementary rather than contradictory: conventional factors explain most of the population burden, while emerging factors may explain a growing share of individual cases that conventional screening misses. Similarly, the rising proportion of young women among infarction patients, together with their worse in-hospital outcomes [4,5], departs from the traditional assumption that premenopausal women are protected and suggests that sex-specific risk factors have been underweighted. A further point of divergence concerns pharmacological prevention: the benefit of early statin therapy is well documented in familial hypercholesterolaemia, but it cannot be assumed to be equally large in young adults with moderately raised cholesterol, for whom no outcome trials exist. The cumulative exposure model predicts benefit; direct proof is still lacking.

Theoretically, the findings strengthen the life-course model of cardiovascular disease, in which risk is a function of both the level and the duration of exposure. This model implies that the conventional distinction between primary prevention in adults and health promotion in children is artificial, and that prevention is better conceived as a continuum beginning before risk factors appear (primordial prevention). Several practical implications follow. In clinical practice, blood pressure, body mass index, lipid profile and smoking status should be recorded routinely from adolescence, lifetime rather than ten-year risk should frame counselling of adults under 40, and a family history of premature disease or LDL cholesterol of 190 mg/dL or above should prompt evaluation for familial hypercholesterolaemia and cascade testing of relatives. In public health, since four in five adolescents are insufficiently active and childhood obesity has increased more than tenfold in four decades, individual counselling alone cannot be sufficient; school-based physical activity, restrictions on marketing of unhealthy food to children, tobacco and nicotine control, and salt reduction act on the whole distribution of risk. In medical education, students should be taught that atherosclerosis is a paediatric-onset disease, so that risk factor detection becomes part of routine care for young patients rather than an exception. Finally, in health systems of Central Asia such as Uzbekistan, where the population is young and lifestyles are changing rapidly, investment in early detection may yield a particularly large return, although local data are needed to quantify it.

Several limitations must be acknowledged. This is a narrative review with purposive selection of sources; selection bias cannot be excluded, and no formal risk-of-bias assessment or meta-analysis was performed. The studies define “young” inconsistently,

Journal of Advances in Drug Research
ISSN: 2230-7761
www.jadr.co.in
Volume 17, Issue 05 Received 10.10.2026 Accepted 11.10.2026 Online 15.10.2026

ranging from under 35 to under 55 years, so the findings are not strictly comparable. Most cohorts were recruited in North America and Europe decades ago, and their relevance to contemporary populations in Asia, and to exposures such as electronic cigarettes, is uncertain. Part of the prevention evidence, notably the dietary trial, was obtained in older adults and is applied to the young by extrapolation. Observational associations between early exposures and later events do not prove that modifying those exposures will prevent the events, even though the consistency and gradient of the associations make causality plausible. Finally, non-atherosclerotic causes of cardiovascular disease in the young, including cardiomyopathies, myocarditis and congenital lesions, were outside the scope of this review.

CONCLUSION

Cardiovascular disease in young people is predominantly the late manifestation of risk factors acquired in childhood and adolescence. Atherosclerosis is already present in the second decade of life, its extent tracks the number of conventional risk factors, and exposure to raised blood pressure or LDL cholesterol before the age of 40 carries a penalty that later control does not fully erase. At the same time, obesity, inactivity and hypertension are now common in the young, while the share of myocardial infarctions occurring in younger adults, especially women, has grown. Treatment of established disease in the young does not differ in principle from that in older patients, but opportunities for prevention are routinely missed because risk tools built on ten-year horizons classify most young people as low risk. The experience with familial hypercholesterolaemia demonstrates that early identification and treatment can change the natural history of the disease within a single generation. Therefore, prevention should follow a life-course strategy that combines population measures on diet, physical activity and nicotine with systematic screening and timely treatment of individuals at high lifetime risk. Future research should prioritise prospective cohort and registry studies of premature CVD in underrepresented regions, including Central Asia, with harmonised age definitions; randomised or pragmatic trials of lipid-lowering and blood pressure-lowering therapy in adults under 40 selected by lifetime risk or subclinical atherosclerosis; longitudinal studies of emerging exposures, particularly electronic nicotine products, recreational drugs and sex-specific risk factors; and implementation research on school-based and primary care screening models, including their cost-effectiveness in middle-income health systems.

REFERENCES:

1. World Health Organization. (2021). Cardiovascular diseases (CVDs): Fact sheet. https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds)
2. Roth, G. A., Mensah, G. A., Johnson, C. O., et al. (2020). Global burden of cardiovascular diseases and risk factors, 1990–2019: Update from the GBD 2019 study. Journal of the American College of Cardiology, 76(25), 2982–3021.
3. Andersson, C., & Vasan, R. S. (2018). Epidemiology of cardiovascular disease in young individuals. Nature Reviews Cardiology, 15(4), 230–240.
4. Arora, S., Stouffer, G. A., Kucharska-Newton, A. M., et al. (2019). Twenty year trends and sex differences in young adults hospitalized with acute myocardial

Journal of Advances in Drug Research

ISSN: 2230-7761
www.jadr.co.in

Volume 17, Issue 05 | Received 10.10.2026 | Accepted 11.10.2026 | Online 15.10.2026

infarction: The ARIC Community Surveillance Study. Circulation, 139(8), 1047–1056.

5. Gupta, A., Wang, Y., Spertus, J. A., et al. (2014). Trends in acute myocardial infarction in young patients and differences by sex and race, 2001 to 2010. Journal of the American College of Cardiology, 64(4), 337–345.
6. Collet, J.-P., Zeitouni, M., Procopi, N., et al. (2019). Long-term evolution of premature coronary artery disease. Journal of the American College of Cardiology, 74(15), 1868–1878.
7. Berenson, G. S., Srinivasan, S. R., Bao, W., Newman, W. P., Tracy, R. E., & Wattigney, W. A. (1998). Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. New England Journal of Medicine, 338(23), 1650–1656.
8. Strong, J. P., Malcom, G. T., McMahan, C. A., et al. (1999). Prevalence and extent of atherosclerosis in adolescents and young adults: Implications for prevention from the Pathobiological Determinants of Atherosclerosis in Youth Study. JAMA, 281(8), 727–735.
9. Juonala, M., Magnussen, C. G., Berenson, G. S., et al. (2011). Childhood adiposity, adult adiposity, and cardiovascular risk factors. New England Journal of Medicine, 365(20), 1876–1885.
10. Jacobs, D. R., Woo, J. G., Sinaiko, A. R., et al. (2022). Childhood cardiovascular risk factors and adult cardiovascular events. New England Journal of Medicine, 386(20), 1877–1888.
11. Domanski, M. J., Tian, X., Wu, C. O., et al. (2020). Time course of LDL cholesterol exposure and cardiovascular disease event risk. Journal of the American College of Cardiology, 76(13), 1507–1516.
12. Zhang, Y., Vittinghoff, E., Pletcher, M. J., et al. (2019). Associations of blood pressure and cholesterol levels during young adulthood with later cardiovascular events. Journal of the American College of Cardiology, 74(3), 330–341.
13. Yusuf, S., Hawken, S., Ôunpuu, S., et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. The Lancet, 364(9438), 937–952.
14. Singh, A., Collins, B. L., Gupta, A., et al. (2018). Cardiovascular risk and statin eligibility of young adults after an MI: Partners YOUNG-MI Registry. Journal of the American College of Cardiology, 71(3), 292–302.
15. Baethge, C., Goßner-Wiltsche, S., & Mertens, S. (2019). SANRA—a scale for the quality assessment of narrative review articles. Research Integrity and Peer Review, 4, 5.
16. Page, M. J., McKenzie, J. E., Bossuyt, P. M., et al. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. BMJ, 372, n71.
17. Yano, Y., Reis, J. P., Colangelo, L. A., et al. (2018). Association of blood pressure classification in young adults using the 2017 American College of Cardiology/American Heart Association blood pressure guideline with cardiovascular events later in life. JAMA, 320(17), 1774–1782.

Journal of Advances in Drug Research

ISSN: 2230-7761
www.jadr.co.in

Volume 17, Issue 05 | Received 10.10.2026 | Accepted 11.10.2026 | Online 15.10.2026

18. NCD Risk Factor Collaboration. (2017). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. The Lancet, 390(10113), 2627–2642.
19. Guthold, R., Stevens, G. A., Riley, L. M., & Bull, F. C. (2020). Global trends in insufficient physical activity among adolescents: A pooled analysis of 298 population-based surveys with 1.6 million participants. The Lancet Child & Adolescent Health, 4(1), 23–35.
20. Song, P., Zhang, Y., Yu, J., et al. (2019). Global prevalence of hypertension in children: A systematic review and meta-analysis. JAMA Pediatrics, 173(12), 1154–1163.
21. Visseren, F. L. J., Mach, F., Smulders, Y. M., et al. (2021). 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. European Heart Journal, 42(34), 3227–3337.
22. Stone, N. J., Smith, S. C., Orringer, C. E., et al. (2022). Managing atherosclerotic cardiovascular risk in young adults: JACC state-of-the-art review. Journal of the American College of Cardiology, 79(8), 819–836.
23. Luirink, I. K., Wiegman, A., Kusters, D. M., et al. (2019). 20-year follow-up of statins in children with familial hypercholesterolemia. New England Journal of Medicine, 381(16), 1547–1556.
24. Arnett, D. K., Blumenthal, R. S., Albert, M. A., et al. (2019). 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease. Circulation, 140(11), e596–e646.
25. Flynn, J. T., Kaelber, D. C., Baker-Smith, C. M., et al. (2017). Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics, 140(3), e20171904.
26. Lloyd-Jones, D. M., Allen, N. B., Anderson, C. A. M., et al. (2022). Life's Essential 8: Updating and enhancing the American Heart Association's construct of cardiovascular health. Circulation, 146(5), e18–e43.
27. Lloyd-Jones, D. M., Leip, E. P., Larson, M. G., et al. (2006). Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. Circulation, 113(6), 791–798.
28. Berry, J. D., Dyer, A., Cai, X., et al. (2012). Lifetime risks of cardiovascular disease. New England Journal of Medicine, 366(4), 321–329.
29. Estruch, R., Ros, E., Salas-Salvadó, J., et al. (2018). Primary prevention of cardiovascular disease with a Mediterranean diet supplemented with extra-virgin olive oil or nuts. New England Journal of Medicine, 378(25), e34.