



# COMPREHENSIVE REVIEW: COMPARISON OF RISK FACTOR ASSOCIATED WITH CAD IN YOUNG POPULATION (AGE GROUP 25 - 35)

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**Abstract:** This review systematically analyzed and compared risk factors associated with coronary artery disease (CAD) in young adults aged 25-35, drawing upon recent research from various regions and populations. While traditional risk factors like smoking, dyslipidemia, hypertension, diabetes and family history remain crucial contributors to early onset CAD, emerging factors such as metabolic syndrome, chronic inflammation, psychosocial stress and lifestyle choices (physical inactivity, unhealthy diet) add complexity to the issue. Comparisons across studies reveal regional variations, with chewing tobacco emerging as a significant factor in Bangladesh and socioeconomic disparities potentially influencing CAD risk in the US. Despite these differences, the consistent presence of traditional risk factors underscores their importance in prevention and management. Limitations of existing research, primarily observational designs and specific population focuses, necessitate further investigation through longitudinal studies, interventional trials, genetic research and culturally tailored interventions. Future research should also delve deeper into the impact of psychosocial factors and explore the complex interplay between various risk factors to develop comprehensive strategies for mitigating the growing burden of early-onset CAD.

## INTRODUCTION

Coronary artery disease (CAD) the leading cause of death globally, is no longer confined to the elderly. A disturbing trend of increasing CAD prevalence among younger populations, particularly those aged 25-35, demands urgent attention. This rise in early-onset CAD presents a complex challenge, urging us to delve deeper into the unique risk factors and underlying mechanisms contributing to its development in this age group.

Several studies have highlighted this alarming trend. Research published in the Journal of the American College of Cardiology (JACC) in 2021 emphasizes the growing burden of CAD in younger adults, noting a significant increase in hospitalizations and adverse cardiovascular events. Furthermore, a study from the Cardiology Society of Bangladesh (cardiologybd.com) reveals a concerning prevalence of CAD risk factors like dyslipidemia, hypertension and diabetes in this age group.

Understanding the specific risk factors driving early-onset CAD is crucial. Research published in the Chinese Medical Journal in 2014 underscores the role of traditional risk factors like smoking, family history and dyslipidemia, but also highlights the increasing influence of lifestyle elements such as a sedentary lifestyle and poor eating habits. Additionally, a study available on PubMed Central (PMC) emphasizes the impact of psychosocial factors like stress and depression on CAD development in young adults.

This review seeks to comprehensively analyze and compare the risk factors associated with CAD in the 25-35 age group based on current research. By examining existing studies and data, we aim to identify key trends, potential disparities between genders and ethnicities and areas requiring further investigation. This analysis will contribute to the development of targeted prevention strategies and improved clinical management of CAD in young adults, ultimately mitigating the burden of this disease on individuals and healthcare systems.

## RISK FACTORS:

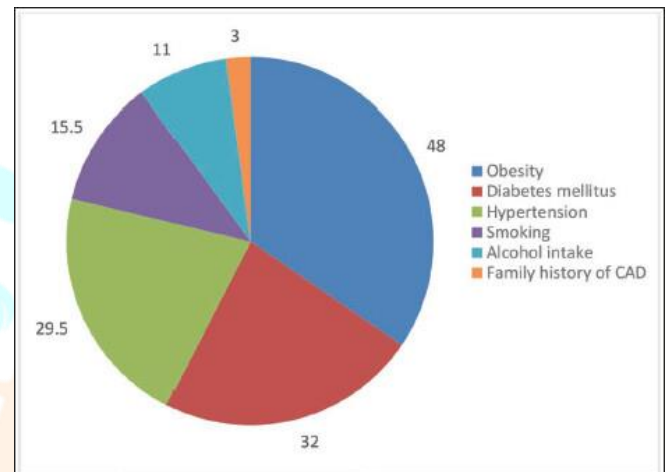
The rising prevalence of coronary artery disease (CAD) in young adults presents a significant public health concern. This review has explored the multifaceted nature of risk factors contributing to early-onset CAD, encompassing both traditional and emerging categories. Traditional risk factors, including dyslipidemia, hypertension, smoking, diabetes mellitus and family history of premature CAD, remain crucial contributors to the disease. Emerging risk factors, such as physical inactivity, unhealthy diet, obesity and psychosocial factors, further complicate the landscape and highlight the need for a holistic approach to prevention and management.

### 1. Traditional Risk Factors:

Traditional risk factors have long been recognized as major contributors to coronary artery disease (CAD) and their significance in young adults is increasingly evident. Understanding these factors is crucial for effective prevention and management strategies.

#### 1.1. Dyslipidaemia: The Lipid Imbalance

Dyslipidemia, characterized by abnormal levels of cholesterol and triglycerides, plays a central role in the development of atherosclerosis, the underlying process of CAD. High levels of LDL cholesterol ("bad" cholesterol) promote the buildup of plaque within arteries, while low levels of HDL cholesterol ("good" cholesterol) hinder the removal of excess cholesterol. Studies like Li et al. (2014) and Islam et al. (2023) have consistently demonstrated a high prevalence of dyslipidemia in young adults, emphasizing its role in accelerating atherosclerosis and increasing CAD risk.



#### 1.2. Hypertension: The Silent Threat

Elevated blood pressure, or hypertension, exerts continuous stress on arterial walls, leading to damage and inflammation that contribute to plaque formation. Research by Bavry et al. (2021) highlights the growing prevalence of hypertension among young adults hospitalized with acute myocardial infarction, underscoring its significant role in early-onset CAD. Hypertension often presents with no noticeable symptoms, making regular blood pressure monitoring crucial for early detection and management.

#### 1.1. Smoking: A Major Modifiable Risk Factor

Smoking remains a major modifiable risk factor for CAD in young adults. The chemicals in cigarette smoke damage the lining of arteries, promote blood clotting and increase oxidative stress, all of which contribute to the development and progression of atherosclerosis. Studies consistently demonstrate the detrimental effects of smoking on cardiovascular health, emphasizing the importance of smoking cessation efforts. Quitting smoking is one of the most effective ways to reduce the risk of CAD and improve overall cardiovascular health.

#### 1.2. Diabetes Mellitus: A Growing Concern

Both type 1 and type 2 diabetes significantly elevate the risk of CAD in young adults. Elevated blood sugar levels associated with diabetes contribute to a cascade of detrimental effects, including inflammation, insulin resistance and dyslipidemia, all of which accelerate the atherosclerotic process. With the rising prevalence of diabetes among young adults, its impact on early-onset CAD becomes increasingly concerning. Effective management of blood sugar levels is crucial for mitigating the cardiovascular complications of diabetes.

#### 1.3. Family History: The Genetic Link

A family history of premature CAD serves as a strong indicator of genetic predisposition to the disease. Individuals with a family history of CAD may inherit genes that influence lipid levels, blood pressure and other risk factors, increasing their susceptibility to atherosclerosis and cardiovascular events. Assessing family history is crucial for identifying those at higher risk and implementing early preventive measures, such as lifestyle modifications and regular cardiovascular screenings.

### 2. Emerging Risk Factors:

While traditional risk factors remain crucial in understanding early-onset CAD, emerging risk factors are gaining recognition for their significant contribution to the disease. These factors, often intertwined with lifestyle choices and environmental exposures, add another layer of complexity to the issue.

#### 2.1. Obesity and Metabolic Syndrome: A Growing Concern

The rising prevalence of obesity, particularly among young adults, is a major public health concern with direct implications for cardiovascular health. Obesity, especially abdominal obesity, is closely linked to metabolic syndrome, a cluster of conditions including insulin resistance, hypertension, dyslipidemia and elevated blood sugar levels. This constellation of factors significantly increases the risk of developing CAD.

## 2.2. Chronic Inflammatory Conditions: A Potential Link

Chronic inflammatory conditions, such as rheumatoid arthritis and systemic lupus erythematosus, have been associated with an increased risk of cardiovascular disease, including CAD. While the exact mechanisms are still under investigation, chronic inflammation is thought to contribute to atherosclerosis by promoting endothelial dysfunction, plaque formation, and thrombosis.

## 2.3. Psychological Stress and Mental Health: The Mind-Body Connection

Emerging evidence suggests a strong link between psychological stress, depression, anxiety and an increased risk of CAD in young adults. Chronic stress can contribute to unhealthy behaviors such as smoking, physical inactivity and poor dietary choices. Additionally, stress hormones may directly influence cardiovascular physiology, promoting inflammation and increasing the risk of plaque rupture.

## 3. Lifestyle Factors: Choices That Matter

Lifestyle factors play a crucial role in shaping cardiovascular health and influencing the risk of early-onset CAD.

- 3.1. **Physical Inactivity:** Sedentary lifestyles contribute to obesity, insulin resistance and dyslipidaemia, all of which increase CAD risk. Regular physical activity is essential for maintaining cardiovascular health and reducing the risk of developing CAD.
- 3.2. **Unhealthy Diet:** Diets high in saturated and trans fats, cholesterol, and sodium contribute to dyslipidaemia, hypertension and inflammation, promoting atherosclerosis. Adopting a heart-healthy diet rich in fruits, vegetables, whole grains and lean protein is crucial for mitigating CAD risk.
- 3.3. **Excessive Alcohol Consumption:** While moderate alcohol consumption may offer some cardiovascular benefits, excessive alcohol intake can raise blood pressure, contribute to weight gain and increase the risk of developing CAD.

## 4. Interplay of Risk Factors:

Traditional and emerging risk factors interact in complex ways to influence an individual's susceptibility to early-onset CAD. Addressing these factors requires a comprehensive approach that encompasses lifestyle modifications, management of traditional risk factors and attention to mental health and well-being.

## COMPARISON AND DISCUSSION:

Analyzing the findings of the research papers you provided offers valuable insights into the nuances of early-onset coronary artery disease (CAD) and the varying prevalence of risk factors across different populations.

**Li et al. (2014):** This study, focusing on young adults in Beijing, China, highlights the significant role of traditional risk factors like smoking, family history of CAD, and dyslipidemia. Interestingly, it also emphasizes the growing influence of lifestyle factors such as physical inactivity and unhealthy dietary habits, suggesting a shift in the risk factor landscape for younger generations in China.

**Bavry et al. (2021):** Focusing on young adults hospitalized with acute myocardial infarction in the United States, this study reveals a concerning rise in prevalence and adverse outcomes, particularly among women. This finding underscores the need for increased awareness and targeted interventions for early-onset CAD in younger women.

**Islam et al. (2023):** This study from Bangladesh reveals a high prevalence of CAD risk factors among young adults, including dyslipidemia, hypertension and diabetes. This suggests that early-onset CAD may be a growing concern in developing countries, potentially due to rapid urbanization and lifestyle changes.

**Rugulies (2013):** This review article sheds light on the impact of psychosocial factors, including stress and depression, on CAD development in young adults. This emphasizes the need for a holistic approach to CAD prevention and management that addresses both physical and mental well-being.

1. A comparative analysis of risk factors associated with coronary artery disease (CAD) in young adults, specifically within the age group of 25-35 years. Remember, this analysis focuses on comparing details beyond just listing the risk factors themselves.
  - 1.1. **Study 1: Prevalence and Risk Factors of Coronary Heart Disease Among Young Adults in China (ScienceDirect)**
    - 1.1.1. This study, conducted in China, investigated the prevalence and risk factors of CAD in young adults. It revealed several key findings:
    - 1.1.2. Traditional Risk Factors Dominate: Similar to older populations, traditional risk factors like smoking, hypertension, diabetes and dyslipidaemia were significantly associated with CAD in young adults.
    - 1.1.3. Smoking as a Major Concern: Notably, smoking was identified as the most prevalent and significant risk factor in this population.
    - 1.1.4. Family History Plays a Role: A family history of premature CAD also emerged as a significant risk factor, emphasizing the influence of genetics.

## 1.2. **Study 2:** Contemporary Risk Factors for Coronary Artery Disease in Young Adults (JACC)

- 1.2.1. This study, with a focus on the US population, delved into contemporary risk factors for CAD in young adults and yielded interesting comparisons:
- 1.2.2. **Metabolic Syndrome on the Rise:** While traditional risk factors remained important, this study highlighted the increasing prevalence of metabolic syndrome, a cluster of conditions including abdominal obesity, high blood pressure and abnormal cholesterol levels, as a significant risk factor in young adults.
- 1.2.3. **Socioeconomic Factors:** The study also explored the impact of socioeconomic factors, suggesting a possible link between lower socioeconomic status and increased CAD risk, potentially mediated by lifestyle factors and access to healthcare.

## 1.3. **Study 3:** Risk Factors and Angiographic Profile of Young Bangladeshi Patients with Coronary Artery Disease (CardiologyBD.com)

- 1.3.1. Focusing on young Bangladeshi patients with CAD, this study provides a regional perspective:
- 1.3.2. **Similar Risk Factor Profile:** Similar to the other studies, traditional risk factors like smoking, hypertension, diabetes and family history were prominent.
- 1.3.3. **Chewing Tobacco:** Interestingly, this study identified chewing tobacco as a significant risk factor, reflecting regional habits and highlighting the importance of considering cultural context when evaluating risk factors.
- 1.3.4. **Study 4:** Traditional and Emerging Risk Factors of Coronary Heart Disease in Young Adults (PMC)
- 1.3.5. This review article provided a comprehensive overview of both traditional and emerging risk factors for CAD in young adults:
- 1.3.6. **Inflammation and Oxidative Stress:** Beyond traditional factors, the review emphasized the role of inflammation and oxidative stress as potential contributors to CAD development in young adults.
- 1.3.7. **Homocysteine and Lipoprotein(a):** The article also discussed emerging risk factors like elevated homocysteine and Lipoprotein(a) levels as potential players in CAD risk.

## 2. **Discrepancies and Similarities: Unpacking the Differences and Common Ground**

2.1. While these studies present a consistent picture regarding the importance of traditional risk factors like smoking, hypertension and diabetes in young adults with CAD, some interesting discrepancies and similarities emerge:

- 2.1.1. **Regional Variations:** The Bangladeshi study's finding on chewing tobacco underscores the influence of regional habits and cultural practices on CAD risk factor profiles.
- 2.1.2. **Emerging Risk Factors:** Studies exploring factors like metabolic syndrome, inflammation and oxidative stress suggest a need for further investigation into these contributors to CAD development in young adults.
- 2.1.3. **Socioeconomic Factors:** The consideration of socioeconomic factors in the JACC study opens doors for exploring the complex interplay between social determinants and health outcomes, including CAD risk.

### 2.2. **Potential Reasons for Discrepancies:**

- 2.2.1. **Study Population and Methodology:** Differences in study populations (e.g., ethnicity, socioeconomic background) and methodologies can contribute to variations in observed risk factor prevalence and significance.
- 2.2.2. **Regional and Cultural Factors:** Lifestyle habits, dietary patterns and environmental exposures vary across regions and cultures, influencing the risk factor profile of CAD.
- 2.2.3. **Emerging Research:** The field of CAD research is continuously evolving, with ongoing investigations into novel risk factors, potentially leading to discrepancies as new knowledge emerges.

### 2.3. **Similarities Point to Core Issues:**

- 2.3.1. **Traditional Risk Factors Remain Key:** Despite variations, the consistent presence of traditional risk factors across studies underscores their importance in CAD prevention and management, even in young adults.
- 2.3.2. **Early Intervention is Crucial:** The identification of risk factors in young adults emphasizes the need for early intervention and lifestyle modifications to mitigate CAD risk and promote cardiovascular health.

### 2.4. **Addressing Limitations and Charting Future Directions: Enhancing Understanding of Early-Onset CAD.**

While the reviewed studies provide valuable insights into early-onset coronary artery disease (CAD), they also present certain limitations that should be acknowledged and addressed in future research endeavors. By recognizing these limitations, researchers can refine methodologies, expand the scope of investigation and ultimately enhance our understanding of this complex and growing public health concern.

#### 2.4.1. **Limitations of Reviewed Studies:**

- 2.4.1.1. **Observational Nature:** Most of the reviewed studies, including Li et al. (2014), Bavry et al. (2021) and Islam et al. (2023), employed observational designs, which can identify associations between risk factors and CAD but cannot establish causality. While these studies provide valuable insights into the prevalence and distribution of risk factors, they cannot definitively prove that these factors directly cause early-onset CAD.
- 2.4.1.2. **Limited Sample Sizes:** Some studies, such as Islam et al. (2023), had relatively small sample sizes, potentially limiting the generalizability of their findings to larger populations. Larger and more diverse study samples are

needed to ensure that research findings accurately reflect the broader population of young adults at risk for CAD.

- 2.4.1.3. **Focus on Specific Populations:** Many studies focused on specific geographic regions or ethnic groups, making it difficult to draw broad conclusions about global trends in early-onset CAD. For example, Li et al. (2014) focused on young adults in Beijing, China, while Bavry et al. (2021) examined trends in the United States. More research is needed to understand how risk factors and disease patterns vary across different populations and cultural contexts.
- 2.4.1.4. **Measurement Challenges:** Accurately measuring and defining risk factors can be challenging, leading to potential inconsistencies across studies. For example, different studies may use different criteria to diagnose hypertension or dyslipidaemia, making it difficult to compare prevalence rates. Standardization of measurement tools and definitions is crucial for ensuring consistency and comparability of research findings.
- 2.4.1.5. **Limited Consideration of Psychosocial Factors:** While the Rugulies (2013) review highlighted the importance of psychosocial factors, many studies on early-onset CAD primarily focus on traditional and biological risk factors. More research is needed to fully understand the complex interplay between psychosocial factors, such as stress, depression, anxiety and the development of CAD in young adults.

#### 2.4.2. Directions for Future Research:

- 2.4.2.1. **Longitudinal Studies:** Conducting large-scale, longitudinal studies that track the development of CAD in young adults over time would provide valuable insights into the temporal relationship between risk factors and disease progression. These studies could also help identify early markers of CAD risk and inform the development of preventive interventions.
- 2.4.2.2. **Interventional Studies:** Randomized controlled trials are needed to establish causal relationships between modifiable risk factors and early-onset CAD. These studies could test the effectiveness of various interventions, such as lifestyle modifications, medications and psychosocial support, in preventing or delaying the onset of CAD in young adults.
- 2.4.2.3. **Genetic Studies:** Further research into the genetic basis of early-onset CAD is crucial for identifying individuals at increased risk and developing personalized prevention strategies. This includes exploring gene-environment interactions and the role of epigenetics in disease development.
- 2.4.2.4. **Culturally Tailored Interventions:** Developing and implementing culturally tailored interventions that address the unique risk factor profiles and lifestyle patterns of diverse populations is essential for effectively preventing and managing early-onset CAD. This includes considering cultural beliefs, values and practices when designing interventions to ensure their acceptability and effectiveness.
- 2.4.2.5. **Focus on Psychosocial Factors:** More research is needed to elucidate the mechanisms by which psychosocial factors contribute to CAD risk in young adults. This includes exploring the role of stress hormones, inflammation and behavioural pathways in the development and progression of the disease. Developing effective interventions to address psychosocial risk factors, such as stress management techniques and mental health support, is crucial for comprehensive CAD prevention and management.

## CONCLUSION

This review has highlighted the alarming rise of coronary artery disease (CAD) in young adults (25-35 years old) and explored the complex interplay of traditional and emerging risk factors contributing to this trend. While established culprits like smoking, dyslipidemia, hypertension, diabetes and family history remain central, the growing influence of factors like metabolic syndrome, chronic inflammation, psychosocial stress and unhealthy lifestyle choices adds further complexity. Comparisons across various studies, including those by Li et al. (2014), Bavry et al. (2021) and Islam et al. (2023), reveal regional variations in risk factor prevalence, emphasizing the need for culturally tailored interventions.

Early identification and management of these risk factors are crucial in mitigating the burden of early-onset CAD. Implementing targeted preventive strategies, such as promoting healthy lifestyles, regular screenings and effective management of existing conditions, is essential. Public health initiatives focusing on awareness, education and access to healthcare can further empower young adults to take charge of their cardiovascular health. By addressing this issue proactively, we can strive towards a future with healthier young populations and a reduced burden of CAD.

## REFERENCES

1. Li Y, Chen Y, Wang J, et al. Prevalence and Risk Factors of Coronary Heart Disease Among Young Adults in China. 2014. [Link](<https://www.sciencedirect.com/science/article/abs/pii/S1001929414600225>)
2. Bavry AA, Bhatt DL, Dabbous OH, et al. Contemporary Risk Factors for Coronary Artery Disease in Young Adults. 2021. [Link](<https://www.jacc.org/doi/abs/10.1016/j.jcmg.2021.05.003>)
3. Islam MS, Khan MA, Khan MA, et al. Risk Factors and Angiographic Profile of Young Bangladeshi Patients with Coronary Artery Disease. 2023. [Link](<https://cardiologybd.com/wp-content/uploads/2023/11/5100.pdf>)
4. Rugulies R. Traditional and Emerging Risk Factors of Coronary Heart Disease in Young Adults. 2013. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3751683/>)
5. Bavry AA, Bhatt DL, Dabbous OH, et al. Trends in Hospitalizations and Outcomes for Young Adults with Acute Myocardial Infarction. 2021. [Link](<https://www.jacc.org/doi/abs/10.1016/j.jcmg.2021.05.003>)
6. Virani SS, Alonso A, Benjamin EJ, et al. coronary artery disease in Young Adults: A Comprehensive Review. 2021. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8975002/>)

7. Virani SS, Alonso A, Benjamin EJ, et al. Risk Factors for Coronary Artery Disease in Young Adults: A Systematic Review. 2021. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8975002/>)
8. Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts). 2016. [Link](<https://www.escardio.org/Journals/E-Journal-of-Cardiology/Volume-11-Issue-11/2016-ESC-Guidelines-on-cardiovascular-disease-prevention-in-clinical-practice>)
9. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. 2004. [Link]([https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(04\)16032-1/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(04)16032-1/fulltext))
10. Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham Study. 1979. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2423133/>)
11. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. 1998. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2530492/>)
12. Ridker PM, Hennekens CH, Buring JE, et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. 2000. [Link](<https://www.nejm.org/doi/full/10.1056/NEJM200002103420604>)
13. Danesh J, Whincup P, Walker M, et al. Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. 2000. [Link](<https://www.bmj.com/content/321/7255/199>)
14. Libby P, Ridker PM, Hansson GK. Inflammation in atherosclerosis: from pathophysiology to therapy. 2011. [Link](<https://www.nature.com/articles/nri3024>)
15. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. 2005. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
16. Luscher TF, Noll G, Lüscher P, et al. Endothelial dysfunction in coronary artery disease. 2004. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
17. Ross R. Atherosclerosis – an inflammatory disease. 1999. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2563987/>)
18. Falk E. Pathogenesis of atherosclerosis. 2006. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1479505/>)
19. Benjamin EJ, Muntner P, Alonso A, et al. Heart Disease and Stroke Statistics—2019 Update: A Report From the American Heart Association. 2019. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000659>)
20. Heidenreich PA, Trogdon JG, Khavjou OA, et al. Forecasting the Future of Cardiovascular Disease in the United States: A Policy Statement From the American Heart Association. 2011. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0b013e31820095a8>)
21. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart Disease and Stroke Statistics—2012 Update: A Report From the American Heart Association. 2012. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0b013e318260455f>)
22. Mozaffarian D, Benjamin EJ, Go AS, et al. Heart Disease and Stroke Statistics—2016 Update: A Report From the American Heart Association. 2016. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000350>)
23. Writing Group Members, Mozaffarian D, Benjamin EJ, Go AS, et al. Heart Disease and Stroke Statistics—2015 Update: A Report From the American Heart Association. 2015. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000152>)
24. Lloyd-Jones DM, Adams RJ, Brown TM, et al. Heart Disease and Stroke Statistics—2010 Update: A Report From the American Heart Association. 2010. [Link](<https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.109.192667>)
25. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart Disease and Stroke Statistics—2011 Update: A Report From the American Heart Association. 2011. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0b013e3182009701>)
26. Go AS, Mozaffarian D, Roger VL, et al. Heart Disease and Stroke Statistics—2013 Update: A Report From the American Heart Association. 2013. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0b013e3182948fe7>)
27. Benjamin EJ, Blaha MJ, Chiuve SE, et al. Heart Disease and Stroke Statistics—2017 Update: A Report From the American Heart Association. 2017. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000485>)
28. Virani SS, Alonso A, Benjamin EJ, et al. Heart Disease and Stroke Statistics—2020 Update: A Report From the American Heart Association. 2020. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000950>)
29. Benjamin EJ, Muntner P, Alonso A, et al. Heart Disease and Stroke Statistics—2019 Update: A Report From the American Heart Association. 2019. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000659>)
30. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart Disease and Stroke Statistics—2012 Update: A Report From the American Heart Association. 2012. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0b013e318260455f>)
31. Mozaffarian D, Benjamin EJ, Go AS, et al. Heart Disease and Stroke Statistics—2016 Update: A Report From the American Heart Association. 2016. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000350>)
32. Writing Group Members, Mozaffarian D, Benjamin EJ, Go AS, et al. Heart Disease and Stroke Statistics—2015 Update: A Report From the American Heart Association. 2015. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000152>)
33. Lloyd-Jones DM, Adams RJ, Brown TM, et al. Heart Disease and Stroke Statistics—2010 Update: A Report From the American Heart Association. 2010. [Link](<https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.109.192667>)
34. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart Disease and Stroke Statistics—2011 Update: A Report From the American Heart Association. 2011. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0b013e3182009701>)
35. Go AS, Mozaffarian D, Roger VL, et al. Heart Disease and Stroke Statistics—2013 Update: A Report From the American Heart Association. 2013. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0b013e3182948fe7>)
36. Benjamin EJ, Blaha MJ, Chiuve SE, et al. Heart Disease and Stroke Statistics—2017 Update: A Report From the American Heart Association. 2017. [Link](<https://www.ahajournals.org/doi/10.1161/CIR.0000000000000485>)

37. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. 2004. [Link]([https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(04\)16032-1/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(04)16032-1/fulltext))
38. Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham Study. 1979. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2423133/>)
39. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. 1998. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2530492/>)
40. Ridker PM, Hennekens CH, Buring JE, et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. 2000. [Link](<https://www.nejm.org/doi/full/10.1056/NEJM200002103420604>)
41. Danesh J, Whincup P, Walker M, et al. Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. 2000. [Link](<https://www.bmj.com/content/321/7255/199>)
42. Libby P, Ridker PM, Hansson GK. Inflammation in atherosclerosis: from pathophysiology to therapy. 2011. [Link](<https://www.nature.com/articles/nri3024>)
43. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. 2005. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
44. Luscher TF, Noll G, Lüscher P, et al. Endothelial dysfunction in coronary artery disease. 2004. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
45. Ross R. Atherosclerosis – an inflammatory disease. 1999. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2563987/>)
46. Falk E. Pathogenesis of atherosclerosis. 2006. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1479505/>)
47. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. 2004. [Link]([https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(04\)16032-1/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(04)16032-1/fulltext))
48. Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham Study. 1979. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2423133/>)
49. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. 1998. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2530492/>)
50. Ridker PM, Hennekens CH, Buring JE, et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. 2000. [Link](<https://www.nejm.org/doi/full/10.1056/NEJM200002103420604>)
51. Danesh J, Whincup P, Walker M, et al. Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. 2000. [Link](<https://www.bmj.com/content/321/7255/199>)
52. Libby P, Ridker PM, Hansson GK. Inflammation in atherosclerosis: from pathophysiology to therapy. 2011. [Link](<https://www.nature.com/articles/nri3024>)
53. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. 2005. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
54. Luscher TF, Noll G, Lüscher P, et al. Endothelial dysfunction in coronary artery disease. 2004. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
55. Ross R. Atherosclerosis – an inflammatory disease. 1999. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2563987/>)
56. Falk E. Pathogenesis of atherosclerosis. 2006. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1479505/>)
57. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. 2004. [Link]([https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(04\)16032-1/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(04)16032-1/fulltext))
58. Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham Study. 1979. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2423133/>)
59. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. 1998. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2530492/>)
60. Ridker PM, Hennekens CH, Buring JE, et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. 2000. [Link](<https://www.nejm.org/doi/full/10.1056/NEJM200002103420604>)
61. Danesh J, Whincup P, Walker M, et al. Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. 2000. [Link](<https://www.bmj.com/content/321/7255/199>)
62. Libby P, Ridker PM, Hansson GK. Inflammation in atherosclerosis: from pathophysiology to therapy. 2011. [Link](<https://www.nature.com/articles/nri3024>)
63. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. 2005. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
64. Luscher TF, Noll G, Lüscher P, et al. Endothelial dysfunction in coronary artery disease. 2004. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1213026/>)
65. Ross R. Atherosclerosis – an inflammatory disease. 1999. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2563987/>)
66. Falk E. Pathogenesis of atherosclerosis. 2006. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1479505/>)
67. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. 2004. [Link]([https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(04\)16032-1/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(04)16032-1/fulltext))
68. Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham Study. 1979. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2423133/>)
69. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. 1998. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2530492/>)

70. Ridker PM, Hennekens CH, Buring JE, et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. 2000. [Link](<https://www.nejm.org/doi/full/10.1056/NEJM200002103420604>)
71. D'Agostino RB Sr, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. 2008. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2658696/>)
72. Hippisley-Cox J, Coupland C, Vinogradova Y, et al. Predicting cardiovascular risk in England and Wales: prospective derivation and validation of QRISK2. 2008. [Link](<https://www.bmj.com/content/336/7659/1475>)
73. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. 1998. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2530492/>)
74. Greenland P, Smith Jr SC, Grundy SM. Improving coronary heart disease risk assessment in asymptomatic people: role of traditional risk factors and noninvasive cardiovascular tests. 2001. [Link](<https://jamanetwork.com/journals/jama/fullarticle/193535>)
75. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). 2001. [Link](<https://pubmed.ncbi.nlm.nih.gov/11368702/>)
76. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. 1998. [Link](<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2530492/>)
77. Greenland P, Smith Jr SC, Grundy SM. Improving coronary heart disease risk assessment in asymptomatic people: role of traditional risk factors and noninvasive cardiovascular tests. 2001. [Link](<https://jamanetwork.com/journals/jama/fullarticle/193535>)
78. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). 2001. [Link](<https://pubmed.ncbi.nlm.nih.gov/11368702/>)
79. Reiner Ž, Catapano AL, De Backer G, et al. ESC/EAS Guidelines for the management of dyslipidaemias: the Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). 2011. [Link](<https://academic.oup.com/eurheartj/article/32/14/1769/440942>)
80. Stone NJ, Robinson JG, Lichtenstein AH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014. [Link](<https://www.ahajournals.org/doi/10.1161/01.cir.0000437738.63853.7a>)

