QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

CIUPAK, Klaudia, GARBACKI, Klaudiusz, DUDEK, Bartosz, POPEK, Lena, BŁAŻUKIEWICZ, Jan, DUDEK, Karolina, KOWALCZYK, Natalia, KOWALEWSKI, Piotr and KUCHTA, Eliza. Lifestyle Behaviors and Physical Activity as Modifiable Determinants of Chronic Disease. Quality in Sport. 2026;56:72424. https://doi.org/10.12775/QS.2026.56.72424

ARTICLE TIMELINE
Received: 22.05.2026 Revised: 26.05.2026
Accepted: 26.05.2026 Published: 30.05.2026

INDEXING & EVALUATION
MEiN points: 20 Unique ID: 201398
Disciplines: Economics & Finance; Management & Quality Sciences

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Economics and Finance (Social Sciences); Management and Quality Sciences (Social Sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Lifestyle Behaviors and Physical Activity as Modifiable Determinants of Chronic Disease

Klaudia Ciupak

Medical University of Lublin Aleje Racławickie 1, 20-059 Lublin, Poland

https://orcid.org/0000-0003-4785-9819

klaudia.c200119@gmail.com

Klaudiusz Garbacki

Medical University of Lublin Aleje Racławickie 1, 20-059 Lublin, Poland

https://orcid.org/0000-0002-3319-6025

garbackiklaudiusz@gmail.com

Bartosz Dudek

University of Rzeszów Tadeusza Rejtana 16C, 35-959 Rzeszów, Poland

https://orcid.org/0009-0004-5067-6460

dudekbartosz8@gmail.com

Lena Popek

University of Rzeszów Tadeusza Rejtana 16C, 35-959 Rzeszów, Poland

https://orcid.org/0009-0006-8530-6798

lenapopek@gmail.com

Jan Błażukiewicz

Medical University of Lublin Aleje Racławickie 1, 20-059 Lublin, Poland

https://orcid.org/0009-0008-6498-5810

j.blazukiewicz@gmail.com

Karolina Dudek

Medical University of Lublin Aleje Racławickie 1, 20-059 Lublin, Poland

https://orcid.org/0009-0009-8346-6204

kaladudek226@gmail.com

Natalia Kowalczyk

Medical University of Lublin Aleje Racławickie 1, 20-059 Lublin, Poland

https://orcid.org/0009-0001-9271-4339

natalia.kowalczyk@icloud.com

Piotr Kowalewski

Medical University of Lublin Aleje Racławickie 1, 20-059 Lublin, Poland

https://orcid.org/0009-0002-6687-2226

pkowalewski222@gmail.com

Eliza Kuchta

Medical University of Lublin Aleje Racławickie 1, 20-059 Lublin, Poland

https://orcid.org/0009-0009-2408-4900

eliza-kukhta@wp.pl

Corresponding author: Klaudia Ciupak
klaudia.c200119@gmail.com

Abstract

Background

Cardiovascular diseases remain one of the main causes of morbidity and mortality worldwide. Their development is strongly associated with modifiable lifestyle factors, including unhealthy diet, physical inactivity, chronic stress, sleep disturbances, and substance use. Recent research has increasingly focused on lifestyle as an integrated construct, emphasizing the interplay between individual behaviors and their cumulative effects on metabolic, inflammatory, and vascular pathways.

Aim

The aim of this study was to evaluate the association between lifestyle factors, including diet, physical activity, sleep, stress, and substance use, and the risk of cardiovascular disease.

Materials and Methods

This narrative review was based on a structured analysis of 31 peer-reviewed publications, including cohort studies, randomized clinical trials, systematic reviews, and current international guidelines addressing chronic disease prevention and cardiometabolic health. The analysis focused on major lifestyle factors, such as diet, physical activity, sleep, stress, and substance use, together with their effects on metabolic, inflammatory, and vascular mechanisms. Contemporary research methods, including metabolomic analyses, were also considered to improve understanding of the biological consequences of lifestyle patterns and their association with long-term health outcomes.

Results

A healthy lifestyle, particularly dietary patterns based on plant-derived foods, is associated with a lower risk of cardiovascular disease, with studies reporting an approximate 14% risk reduction. Observed benefits include improvement in lipid profile, lower levels of inflammation, and better metabolic regulation. Physical activity, adequate sleep, and effective stress management may further strengthen these effects. Emerging evidence also suggests that metabolic biomarkers may reflect the influence of diet and lifestyle on disease risk more accurately than traditional self-reported measures.

Conclusions

Lifestyle plays a central role in the prevention of cardiovascular disease. Its individual components interact with one another and influence metabolic, inflammatory, and vascular pathways. Even partial improvement in health behaviors may lead to measurable clinical benefits, highlighting the value of a comprehensive approach to both prevention and treatment.
Keywords: *lifestyle; chronic diseases; cardiovascular diseases; diet; physical activity; mental health; sleep; stress; obesity;*

1. Introduction

Chronic diseases, including diabetes, cardiovascular diseases, stroke, and cancer, are among the leading causes of morbidity and mortality worldwide. Their rising prevalence reflects ongoing epidemiological transition, marked by a shift from infectious to non-communicable diseases, together with increasing life expectancy. Lifestyle, particularly the level of physical activity, is now regarded as one of the main determinants of chronic disease development.

Sedentary behavior contributes to metabolic disturbances, obesity, and cardiovascular disease, whereas regular physical activity exerts protective effects through improved metabolic regulation, better cardiovascular function, and reduction of chronic inflammation. The growing burden of lifestyle-related diseases is also linked to substantial economic costs, projected to reach USD 47 trillion by 2030. Preventive efforts nevertheless remain inadequate, emphasizing the need for stronger public health interventions. [1]

Population differences in the prevalence of lifestyle-related diseases are strongly associated with patterns of physical activity and overall lifestyle. Physical inactivity is one of the most important modifiable risk factors for chronic diseases, particularly cardiovascular disease, type 2 diabetes, and obesity. In contrast, regular physical activity lowers disease risk by improving metabolic health, attenuating inflammatory processes, and supporting cardiovascular function. For this reason, physical activity should constitute a core component of contemporary preventive strategies at both individual and population levels, with broader integration into clinical practice and health policy. [2]

In aging populations, preserving functional capacity and independence has become especially important. Physical activity remains a major determinant of health in older adults. Its absence is associated with impaired mobility, frailty, and progression of chronic disease. Regular exercise may delay age-related decline by improving physical performance, muscle strength, and cognitive function. It also affects metabolic and inflammatory pathways linked to complications and premature mortality. Despite these established benefits, physical activity is still not routinely incorporated into therapeutic strategies for older patients. When appropriately adapted and implemented on a long-term basis, it may serve as an important adjunct to treatment and a central element of chronic disease prevention. [3]

Against this background, lifestyle medicine has emerged as a comprehensive approach that integrates behavioral interventions with clinical care. It focuses on the identification and modification of lifestyle-related risk factors, including physical inactivity, unhealthy diet, smoking, and chronic stress. Its effectiveness depends on interdisciplinary cooperation involving healthcare professionals, patients, and the broader social environment, facilitating sustainable health-related changes. The aim of this paper is to present current evidence supporting lifestyle medicine as an important tool in the prevention and treatment of chronic diseases. [4]

2. Diseases of affluence as the leading cause of death

Current epidemiological evidence indicates that population health is influenced less by single diseases than by clusters of coexisting risk factors that interact and reinforce one another. The most important of these include unhealthy diet, physical inactivity, smoking, and metabolic disorders such as hypertension and obesity. Together, they contribute to progressive systemic damage through mechanisms involving chronic inflammation and endothelial dysfunction, thereby increasing the likelihood of multiple chronic conditions developing concurrently. As a result, increasing attention is being directed not only toward the treatment of individual diseases, but also toward reducing the shared risk factors that underlie them. [5]

Recent studies have shown that lifestyle modification can meaningfully affect both life expectancy and mortality risk. The benefits are cumulative, with the greatest effects observed when multiple healthy behaviors are combined. Individuals who engage in regular physical activity, follow a healthy diet, avoid smoking, and maintain appropriate body weight demonstrate lower mortality from both cardiovascular disease and cancer. Importantly, lifestyle improvement remains beneficial even later in life, indicating that preventive interventions may be effective at any age. These findings support the view that lifestyle is not merely an adjunct to treatment, but an integral component of disease prevention and long-term health management. [6]

2.1 The Role of Diet in the Prevention of Chronic Diseases

The rising incidence of cancer is associated not only with population aging, but also with changes in lifestyle, including dietary habits. Evidence suggests that high consumption of ultra-processed foods (UPFs) may increase the risk of several malignancies, including breast and colorectal cancer. The magnitude of this association appears comparable to, or in some cases greater than, that reported for other dietary risk factors such as alcohol, processed meat, and excessive sugar intake. The strength of these findings is supported by large study populations and adjustment for variables including body weight and total energy intake. At the same time, many available studies are observational, and inconsistencies in the classification of foods according to processing level remain an important limitation. Despite these constraints, the findings are relatively consistent and indicate that reducing UPF consumption may play a role in cancer prevention, although further studies are needed to clarify the underlying mechanisms. [7]

Consumption of sugar-sweetened beverages (SSBs) remains high worldwide and exceeds recommended levels in many populations. A large body of evidence indicates that regular intake contributes to weight gain and increases the risk of cardiometabolic disorders, including type 2 diabetes, coronary artery disease, and nonalcoholic fatty liver disease. Even one serving per day may have clinically relevant effects. Proposed mechanisms include low satiety from liquid calories, rapid postprandial glycemic responses, and the metabolic effects of fructose, which favor lipid accumulation and metabolic dysregulation. Although associations with some other diseases remain less certain, SSBs are widely recognized as an important modifiable risk factor. Reducing their consumption through educational and regulatory interventions may improve dietary quality and population health, particularly when incorporated into broader strategies aimed at preventing lifestyle-related diseases. [8]

Analysis of data from the UK Biobank showed that health outcomes are influenced less by whether a diet is plant-based and more by the quality of the foods consumed. The protective effects of a healthy plant-based diet were observed regardless of genetic predisposition to cardiovascular disease and may even be more pronounced among individuals at higher genetic risk. Another notable finding was the contrasting effect of different plant-based dietary patterns. Diets centered on minimally processed foods, such as vegetables and whole grains, were associated with lower disease risk, whereas plant-based diets rich in highly processed products, including sugary beverages and refined carbohydrates, were linked to higher morbidity and mortality. These observations suggest that disease prevention depends less on the exclusion of animal products and more on limiting processed foods while improving overall dietary quality. [9]

2.2 Sleep and Metabolic Health and Diseases of Lifestyle

Within the framework of lifestyle-related prevention of chronic diseases, sleep represents an important but often overlooked factor. Sleep quality influences both physical and mental health, while persistent sleep disturbances are associated with increased risk of cardiovascular and metabolic diseases, as well as impaired overall functioning. Greater patient engagement in healthcare is known to encourage beneficial behaviors such as healthy eating and physical activity; however, its effect on sleep quality appears less pronounced. One of the main contributors to impaired sleep is the presence of chronic disease itself. Symptoms including

pain, dyspnea, and hormonal disturbances may directly disrupt restorative processes during sleep. Current evidence suggests that improving sleep as part of lifestyle modification requires a multidimensional approach. Alongside education and promotion of healthy behaviors, appropriate management of comorbid conditions and symptom control remain essential. Sleep should therefore be regarded as an integral component of chronic disease prevention, alongside diet and physical activity. [10]

In addition to sleep quality, adequate sleep duration is also critical in the prevention of chronic disease. Studies on chronic sleep restriction indicate that the body may partially adapt to insufficient sleep, with some parameters such as sleep continuity and depth appearing to improve. These changes, however, do not reflect true physiological recovery. At the same time, cognitive performance, attention, and reaction time deteriorate and may remain impaired even after several days of recovery sleep. This issue is particularly relevant in the context of lifestyle-related diseases, as chronic sleep deprivation disrupts hormonal regulation, including leptin and ghrelin signaling, promotes insulin resistance and hypertension, and increases the risk of obesity and type 2 diabetes. Cognitive impairment may also contribute to unfavorable health behaviors, including poorer dietary choices and reduced physical activity, further amplifying disease risk. These findings indicate that sleep quality alone is insufficient if total sleep duration remains inadequate. Adequate sleep duration should therefore be considered one of the principal elements of a healthy lifestyle, comparable in importance to diet and exercise, whereas chronic sleep restriction should be recognized as a major and frequently underestimated risk factor. [11]

Beyond sleep duration, sleep continuity also plays an important role in chronic disease prevention. Recurrent sleep fragmentation, characterized by frequent nocturnal awakenings, has been associated with hypertension, increased arterial stiffness, and endothelial dysfunction, all of which represent early stages in the development of cardiovascular disease. Importantly, this phenomenon is not limited to obstructive sleep apnea. It may also result from common lifestyle-related factors such as shift work, insomnia, and circadian rhythm disruption. Proposed mechanisms include chronic inflammation, oxidative stress, and heightened sympathetic nervous system activity. Consequently, even individuals with adequate sleep duration may remain at increased risk of lifestyle-related diseases if sleep quality is poor. Maintaining regular and uninterrupted sleep should therefore be considered an important component of preventive healthcare. [12]

2.3 Stress, Mental Health, and Lifestyle Diseases

Stress and mental health represent important, although frequently underestimated, components of lifestyle-related chronic disease prevention. Stress directly influences immune function through activation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system. Acute stress may temporarily enhance immune responsiveness, whereas chronic stress exerts the opposite effect, contributing to sustained cortisol elevation, altered cytokine production, and impaired immune cell activity. These changes are associated with greater susceptibility to infections, persistent low-grade inflammation, and increased risk of lifestyle-related diseases, including cardiovascular disease, diabetes, and obesity. Chronic stress also adversely affects mental health, promoting depressive and anxiety disorders that may further impair health behaviors and reduce adherence to preventive strategies. The complexity of these interactions indicates that effective chronic disease prevention should include stress management as a core element of a healthy lifestyle. Alongside diet, physical activity, and sleep, mental well-being plays an important role in maintaining immune homeostasis and reducing disease risk. [13]

Mental health may also modify the effects of other lifestyle factors on chronic disease risk. Findings from the cited study suggest that the benefits of health-promoting behaviors persist across groups differing in mental health status, including individuals with depressive symptoms. This observation indicates that healthy lifestyle patterns may partially mitigate some of the adverse consequences associated with impaired psychological well-being. At the same time, mental health strongly influences the ability to initiate and sustain beneficial behaviors such as healthy eating, regular physical activity, and appropriate sleep habits. Depression and anxiety may reduce adherence to preventive recommendations and contribute to the persistence of unhealthy behaviors. For this reason, mental health should be considered an integral component of lifestyle, influencing both direct and indirect pathways related to chronic disease development. [14]

Available evidence further indicates that both mental and physical health affect the effectiveness of preventive interventions. Individuals with chronic diseases are often more likely to engage with healthcare services; however, poorer overall health status may limit participation in screening programs and reduce adherence to medical recommendations. Mental health therefore plays a dual role. It may function both as a risk factor for chronic disease and as a determinant of engagement in health-promoting behaviors. Mood disorders and chronic fatigue can interfere with maintaining a healthy lifestyle, whereas appropriate psychological

support may improve participation in preventive care. These findings highlight the importance of a comprehensive approach that combines medical management with attention to mental well-being in order to improve the prevention of lifestyle-related diseases. [15]

2.4 Physical Activity

Physical activity in older adults is closely associated with the burden of chronic disease. Individuals with multimorbidity generally demonstrate lower levels of physical activity, particularly at moderate and vigorous intensities, while spending more time in sedentary behaviors. Although these differences are not always pronounced, they may contribute to progressive health decline over time. Reduced activity in this population reflects not only the effects of aging itself, but also symptoms related to chronic disease, including pain, fatigue, and reduced physical capacity. As a consequence, daily low-intensity activities tend to predominate. In older adults, reducing prolonged sedentary time and gradually increasing physical activity, even at low intensity, may therefore represent a practical and effective preventive strategy. [16]

Environmental conditions also play an important role in shaping physical activity patterns and chronic disease risk. Evidence indicates that individuals living in walkable neighborhoods with greater access to green spaces are more physically active, have lower rates of obesity, and demonstrate reduced risk of diabetes. These benefits appear to result not only from higher activity levels, but also from indirect effects on body weight and broader lifestyle patterns. Such findings emphasize the importance of population-level interventions, including urban infrastructure that supports walking, access to parks and recreational spaces, and reduced dependence on car transport, as components of chronic disease prevention strategies. [17]

Available studies show that physical activity levels among individuals with chronic diseases remain insufficient. In the analyzed populations, only approximately one-third of participants met recommendations of at least 150 minutes of moderate-to-vigorous physical activity per week, with activity levels declining further with age. Physical inactivity is a well-established health risk factor associated with increased all-cause and cardiovascular mortality. Individuals with low activity levels demonstrate substantially higher mortality risk, while sedentary behavior contributes to the development of numerous lifestyle-related diseases. These findings underline the need for interventions aimed at increasing physical activity, particularly among older adults and patients with chronic conditions. [18]

Interventions based on physical activity coaching may effectively increase exercise participation in individuals with chronic disease. Studies indicate that approaches incorporating education, goal setting, progress monitoring, and motivational support can lead to a measurable increase in daily step count. The greatest effects have been observed with strategies emphasizing self-monitoring and regular review of goals. At the same time, the influence of these interventions on broader health outcomes, including quality of life and symptom burden, appears more limited. [19]

Physical activity is increasingly recognized not only as supportive therapy, but also as a factor capable of modifying disease course and treatment response. During the perioperative period, aerobic capacity and exercise tolerance often decline substantially, particularly in patients with comorbidities such as chronic obstructive pulmonary disease (COPD). Insufficient physical stimulation may prolong these impairments and contribute to long-term functional decline. Early introduction of targeted exercise training can attenuate reductions in functional capacity by affecting adaptive physiological mechanisms, including oxygen utilization, skeletal muscle performance, and tolerance to physical load. Although these effects are not always fully captured by conventional clinical measures, they may translate into improved functional recovery and faster return to independence. Physical activity also influences several coexisting pathophysiological processes, including chronic inflammation, metabolic disturbances, and reduced physiological reserve. [20]

2.5 Smoking, Alcohol, and Lifestyle Diseases

Smoking remains one of the leading modifiable risk factors for chronic disease and premature mortality. Exposure to tobacco smoke promotes chronic inflammation, oxidative stress, and endothelial dysfunction, thereby contributing to the development of cardiovascular disease, cancer, and respiratory conditions, including chronic obstructive pulmonary disease (COPD). Smoking also adversely affects metabolic function, increasing the risk of insulin resistance and type 2 diabetes. Its harmful effects are further amplified through interaction with other unfavorable lifestyle factors. Smoking cessation, in turn, is associated with gradual reduction in complication risk and improvement in overall physiological function, underscoring its importance in both prevention and treatment of lifestyle-related diseases. [21]

Interest in the health effects of e-cigarettes has increased substantially; however, evidence regarding their long-term consequences remains limited. Although many studies have been

published, the overall quality of available data is relatively low, and the predominance of cross-sectional designs limits conclusions about causality. Current findings do not clearly demonstrate major differences in cardiovascular or respiratory function between e-cigarette users and non-smokers, which may partly reflect the relatively short duration of exposure assessed in existing studies. At the same time, e-cigarette users generally appear to have poorer health outcomes than non-smokers, but more favorable profiles than conventional cigarette smokers. Interpretation of these findings remains challenging due to confounding from prior tobacco exposure and variability in the composition of nicotine-containing products. Further studies with stronger methodology, particularly among exclusive e-cigarette users, are therefore needed to clarify their role in the development of chronic diseases. [22]

Smoking not only contributes to disease onset, but also modifies treatment response and disease progression. Smokers often demonstrate poorer tolerance to therapy, delayed recovery, and greater susceptibility to complications. These effects are linked, among other mechanisms, to impaired respiratory epithelial function and persistent inflammation. Coexisting conditions such as COPD may further intensify these effects by reducing physiological reserve. In this context, smoking should be regarded not simply as a risk factor for disease development, but as an active contributor to worse clinical outcomes and reduced therapeutic effectiveness. [23]

The relationship between alcohol consumption and chronic disease is influenced not only by the amount of alcohol consumed, but also by the pattern and regularity of drinking. Evidence suggests that drinking behavior itself may shape disease course independently of total alcohol intake. This relationship is particularly evident in chronic pancreatitis, where abstinence or substantial reduction in alcohol consumption is associated with milder disease progression, fewer complications, and lower relapse rates. Alcohol functions not only as an initiating factor, but also as a driver of disease progression through enhancement of inflammatory processes and tissue damage. Its harmful effects may be further amplified by coexisting lifestyle factors, especially smoking, resulting in more aggressive disease progression. These observations indicate that modification of alcohol-related behaviors, even without complete abstinence, may influence disease course and represent an important component of therapeutic management rather than prevention alone. [24]

Alcohol may therefore be viewed less as an isolated risk factor and more as a modifier of disease progression. Both quantity and drinking pattern appear relevant, with episodic heavy alcohol consumption capable of triggering acute exacerbations and accelerating clinical deterioration. Conversely, reduction in alcohol intake is often associated with stabilization of disease status. Reported benefits of moderate alcohol consumption in some observational

studies are likely attributable to accompanying lifestyle characteristics rather than alcohol itself. From a clinical perspective, alcohol use may shift the balance between disease stability and decompensation. Contemporary approaches increasingly recognize alcohol reduction not only as a preventive recommendation, but also as a practical strategy for influencing the course of chronic disease. [25]

In patients with chronic diseases, behavioral change is often driven less by knowledge alone than by direct experience of symptoms. When individuals begin to associate alcohol consumption with worsening health, such as pain, dyspnea, or disease exacerbation, motivation to reduce intake tends to increase. Studies indicate that even brief and appropriately targeted interventions may then produce sustained behavioral changes, particularly among individuals already engaging in risky drinking patterns. These findings suggest that the timing of intervention is important, and that preventive efforts may be most effective when patients perceive a clear connection between their behavior and disease progression. [26]

2.6 Obesity and Metabolic Disorders

Obesity has become one of the principal drivers of chronic disease, and its rapidly increasing prevalence, particularly among younger populations, suggests that the burden will continue to rise in the coming decades. Excess body weight is often not a transient condition, but rather the beginning of persistent metabolic disturbances that intensify with age. At the biological level, obesity is associated with chronic low-grade inflammation and metabolic dysregulation, contributing to the development of type 2 diabetes, cardiovascular disease, and chronic kidney disease. Its consequences may also extend beyond the individual, influencing the risk of health disorders in subsequent generations. Increasing evidence indicates that obesity cannot be explained solely by personal lifestyle choices, but is also shaped by environments that promote excessive caloric intake and low physical activity. Effective prevention therefore requires not only individual behavioral change, but also broader systemic interventions. [27]

The health consequences of obesity are not uniform and appear to depend partly on metabolic phenotype. Obesity characterized by visceral fat accumulation and adipose tissue dysfunction is particularly strongly associated with insulin resistance, dyslipidemia, and diseases such as type 2 diabetes, cardiovascular disease, and metabolic fatty liver disease. In some individuals, obesity may initially occur without marked metabolic abnormalities, suggesting that body weight alone does not fully determine disease risk. The functional properties of adipose tissue,

together with its influence on hormonal and inflammatory regulation, appear to play a central role. From a clinical perspective, obesity should therefore be evaluated not only quantitatively, but also in terms of metabolic function, as these abnormalities largely determine the onset and progression of chronic disease. [28]

Growing evidence further suggests that obesity, particularly abdominal obesity, contributes to the formation of an interconnected metabolic axis involving the liver, kidneys, and broader metabolic system. Within this framework, metabolic dysfunction–associated fatty liver disease (MAFLD) has emerged as an important link between obesity and chronic diseases, especially chronic kidney disease. Visceral and ectopic fat accumulation, including hepatic fat deposition, appears to be more relevant to disease progression than overall body weight alone. These changes promote lipotoxicity, chronic inflammation, and metabolic dysregulation, leading to simultaneous injury across multiple organs. Consequently, the coexistence of abdominal obesity and MAFLD is associated with higher risk of complications and mortality, particularly those related to renal disease. Traditional measures such as body mass index (BMI) may therefore be less informative than markers of visceral adiposity, including waist circumference. This underscores the importance of adipose tissue distribution and function rather than quantity alone. In clinical practice, these findings support the need for early identification and monitoring of metabolic abnormalities before irreversible organ damage develops. [29]

2.7 Lifestyle and Cardiovascular Disease

Lifestyle, particularly dietary habits, plays a major role in determining cardiovascular disease risk. Cohort studies indicate that dietary patterns based predominantly on plant-derived foods, including the so-called dietary portfolio, are associated with a lower incidence of cardiovascular disease and stroke, with risk reductions reaching approximately 10% among individuals with the highest adherence compared with those with the lowest. The underlying mechanisms appear multifactorial. Such dietary patterns improve lipid profile, especially by lowering atherogenic cholesterol fractions, while also reducing inflammatory activity, a central process in the development of atherosclerosis. Favorable changes in metabolic and hormonal markers have also been observed, suggesting broader cardiometabolic effects. Comparable benefits have been reported for other dietary models, including the Mediterranean and DASH diets, indicating that the overall dietary approach may be more important than adherence to a single specific pattern. Increased consumption of plant-based foods, dietary fiber, and

unsaturated fats appears to be a common protective feature. Even partial implementation of these changes may contribute to measurable reduction in cardiovascular risk. [30]

Recent evidence suggests that cardiovascular risk may be reflected more accurately by the body's metabolic response to diet than by self-reported dietary intake alone. Analyses of circulating metabolites indicate that specific metabolic profiles, particularly those associated with diets rich in processed foods and simple sugars, may predict cardiovascular disease more effectively than traditional dietary questionnaires. Importantly, the metabolome reflects not only dietary composition, but also individual variation in metabolism, gut microbiota, and inflammatory status. As a result, metabolomic assessment may provide a more comprehensive representation of the relationship between lifestyle and cardiovascular health.

[31]

Lifestyle factor	**Main mechanisms**	**Health impact**	**Estimated effect**
Diet (plant-based)	↓ inflammation, ↓ LDL, ↑ metabolic health	Reduced CVD and metabolic disease risk	~14% ↓ CVD risk
Physical activity	↑ insulin sensitivity, ↓ obesity	Lower mortality, improved cardiovascular health	Significant ↓ risk
Sleep (quality & duration)	Hormonal regulation, circadian rhythm	↓ diabetes, ↓ hypertension risk	Moderate ↓ risk
Stress & mental health	HPA axis activation, ↑ cortisol	↑ inflammation, ↑ chronic disease risk	Moderate ↑ risk
Smoking	Oxidative stress, endothelial damage	↑ CVD, cancer, respiratory diseases	Strong ↑ risk
Alcohol consumption	Metabolic disruption, inflammation	Disease progression depending on pattern	Variable effect
Obesity / metabolic status	Chronic inflammation, insulin resistance	↑ diabetes, CKD, CVD	Strong ↑ risk

Results

Analysis of the 31 included studies confirmed that lifestyle factors substantially influence the risk of chronic diseases, particularly cardiovascular disease. Healthy dietary patterns, especially those based on plant-derived foods, were associated with an approximate 14% reduction in cardiovascular risk, accompanied by favorable metabolic and inflammatory

changes. Regular physical activity demonstrated a clear protective effect, whereas low activity levels were linked to increased mortality and greater disease burden. Sleep disturbances and chronic stress were associated with metabolic dysregulation and enhanced inflammatory activity. Smoking and alcohol consumption both contributed to disease development and progression, with smoking demonstrating the most pronounced adverse effects.

Taken together, the findings indicate that lifestyle factors interact synergistically, and that combined modification of multiple behaviors yields the greatest health benefits.

Discussion

The presented findings confirm that lifestyle is one of the most important and modifiable determinants of chronic disease development, particularly in relation to cardiovascular and metabolic disorders, as well as selected cancers. The cumulative interaction of multiple lifestyle factors across the lifespan appears more important than the effect of any single behavior in isolation. Diet, physical activity, sleep, stress, and substance use influence shared pathophysiological pathways, including inflammation, oxidative stress, carbohydrate and lipid metabolism, and endothelial function.

Dietary patterns centered on plant-derived foods appear particularly important because of their anti-inflammatory and lipid-lowering effects, which are associated with reduced cardiovascular risk. At the same time, diet alone is unlikely to provide optimal benefit without support from other health-related behaviors. Regular physical activity improves insulin sensitivity, body composition, and lipid metabolism, while also reducing inflammatory activity independently of weight reduction. In contrast, chronic stress and sleep disturbances contribute to activation of the hypothalamic–pituitary–adrenal axis, elevation of cortisol levels, and development of insulin resistance and hypertension.

The analysis also highlights the importance of environmental and social determinants of health. Access to green spaces, infrastructure supporting physical activity, and availability of healthy food options may substantially influence health behaviors and disease risk. These observations support the need for a broader systemic approach that extends beyond individual-level interventions.

Increasing attention is also being directed toward modern diagnostic approaches such as metabolomics, which may allow more accurate assessment of lifestyle-related biological effects than traditional self-reported measures. Metabolomic profiles reflect not only dietary intake, but also individual physiological responses, including the influence of the gut

microbiome. This may have important implications for the development of personalized prevention and treatment strategies.

In the context of chronic disease, the findings further emphasize the complexity of obesity-related mechanisms. Disease risk depends not only on the quantity of adipose tissue, but also on its distribution, particularly visceral fat accumulation, and its metabolic activity. These factors are closely linked to complications such as type 2 diabetes, metabolic fatty liver disease, and chronic kidney disease, highlighting the need for multidimensional risk assessment.

Despite the growing body of evidence, several limitations should be acknowledged. Many available studies are observational and rely on self-reported data, which may introduce measurement bias and residual confounding. Lifestyle also represents a dynamic exposure that changes over time, making its long-term effects difficult to assess precisely. Further longitudinal and interventional studies addressing the multifactorial nature of lifestyle are therefore needed.

Conclusions

Lifestyle plays a central role in the prevention and progression of chronic diseases through its influence on key biological processes, including inflammation, metabolic regulation, and cardiovascular function. The effects of individual lifestyle factors are interconnected, with the greatest health benefits observed when multiple behaviors, such as diet, physical activity, sleep, and stress management, are modified simultaneously.

Even partial lifestyle improvement may lead to meaningful reductions in disease risk and better clinical outcomes. Effective prevention therefore requires not only individual behavioral change, but also environmental and systemic strategies that facilitate healthier choices at the population level.

The development of modern approaches, including metabolomic analysis, may provide new opportunities for evaluating the biological impact of lifestyle and for tailoring preventive and therapeutic interventions more precisely. These methods may represent an important direction for future research and clinical practice.

Conceptualization: Klaudia Ciupak, Klaudiusz Garbacki, Natalia Kowalczyk, Karolina Dudek

Methodology: Klaudia Ciupak, Klaudiusz Garbacki, Lena Popek, Jan Błażukiewicz

Software: Klaudia Ciupak, Klaudiusz Garbacki, Bartosz Dudek

Check: Klaudia Ciupak, Klaudiusz Garbacki

Formal analysis: Klaudia Ciupak, Natalia Kowalczyk, Karolina Dudek

Investigation: Klaudia Ciupak, Klaudiusz Garbacki, Natalia Kowalczyk, Lena Popek, Jan Błażukiewicz, Karolina Dudek, Bartosz Dudek

Resources: Natalia Kowalczyk, Klaudia Ciupak

Data curation: Klaudia Ciupak, Klaudiusz Garbacki, Natalia Kowalczyk

Writing – rough preparation: Klaudia Ciupak, Klaudiusz Garbacki, Jan Błażukiewicz, Lena Popek

Writing – review and editing: Klaudia Ciupak, Klaudiusz Garbacki, Natalia Kowalczyk, Karolina Dudek, Klaudiusz Garbacki

Visualization: Natalia Kowalczyk, Bartosz Dudek, Lena Popek, Jan Błażukiewicz

Supervision: Klaudia Ciupak, Klaudiusz Garbacki

Project administration: Klaudia Ciupak, Klaudiusz Garbacki, Natalia Kowalczyk

Funding statement: The study did not receive special funding.

Institutional review board statement: Not applicable.

Informed consent statement: Not applicable.

Data availability statement: Not applicable.

Conflict of interest: The authors declare no conflict of interest.

References:

1. Hacker K. The Burden of Chronic Disease. Mayo Clin Proc Innov Qual Outcomes. 2024 Jan 20;8(1):112-119. doi: 10.1016/j.mayocpiqo.2023.08.005. Erratum in: Mayo Clin Proc Innov Qual Outcomes. 2024 Dec 13;9(1):100588. doi: 10.1016/j.mayocpiqo.2024.11.005. PMID: 38304166; PMCID: PMC10830426.
2. Freihat O, Sipos D, Aamir M, Kovacs A. Global burden and future projections of non-communicable diseases (2000-2050): Progress toward SDG 3.4 and disparities across regions and risk factors. PLoS One. 2025 Dec 10;20(12):e0336036. doi: 10.1371/journal.pone.0336036. PMID: 41370213; PMCID: PMC12694828.
3. Izquierdo M, de Souto Barreto P, Arai H, Bischoff-Ferrari HA, Cadore EL, Cesari M, Chen LK, Coen PM, Courneya KS, Duque G, Ferrucci L, Fielding RA, García-Hermoso A, Gutiérrez-Robledo LM, Harridge SDR, Kirk B, Kritchevsky S, Landi F, Lazarus N, Liu-Ambrose T, Marzetti E, Merchant RA, Morley JE, Pitkälä KH, Ramírez-Vélez R, Rodriguez-Mañas L, Rolland Y, Ruiz JG, Sáez de Asteasu ML, Villareal DT, Waters DL, Won Won C, Vellas B, Fiatarone Singh MA. Global consensus on optimal exercise recommendations for enhancing healthy longevity in older adults (ICFSR). J Nutr Health Aging. 2025 Jan;29(1):100401. doi: 10.1016/j.jnha.2024.100401. Epub 2025 Jan 1. PMID: 39743381; PMCID: PMC11812118.
4. Sadiq IZ. Lifestyle medicine as a modality for prevention and management of chronic diseases. J Taibah Univ Med Sci. 2023 Apr 15;18(5):1115-1117. doi: 10.1016/j.jtumed.2023.04.001. PMID: 37187803; PMCID: PMC10176046.
5. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020 Oct 17;396(10258):1204-1222. doi: 10.1016/S0140-6736(20)30925-9. Erratum in: Lancet. 2020 Nov 14;396(10262):1562. doi: 10.1016/S0140-6736(20)32226-1. PMID: 33069326; PMCID: PMC7567026.
6. Zhu C, Lian Z, Arndt V, Thong MSY. Combined lifestyle factors on mortality and cardiovascular disease among cancer survivors: a systematic review and meta-analysis of cohort studies. Support Care Cancer. 2024 Dec 2;32(12):846. doi: 10.1007/s00520-024-09049-2. PMID: 39623080; PMCID: PMC11611996.
7. Isaksen IM, Dankel SN. Ultra-processed food consumption and cancer risk: A systematic review and meta-analysis. Clin Nutr. 2023 Jun;42(6):919-928. doi: 10.1016/j.clnu.2023.03.018. Epub 2023 Mar 30. PMID: 37087831.

8. Malik VS, Hu FB. The role of sugar-sweetened beverages in the global epidemics of obesity and chronic diseases. Nat Rev Endocrinol. 2022 Apr;18(4):205-218. doi: 10.1038/s41574-021-00627-6. Epub 2022 Jan 21. PMID: 35064240; PMCID: PMC8778490.
9. Thompson AS, Tresserra-Rimbau A, Karavasiloglou N, Jennings A, Cantwell M, Hill C, Perez-Cornago A, Bondonno NP, Murphy N, Rohrmann S, Cassidy A, Kühn T. Association of Healthful Plant-based Diet Adherence With Risk of Mortality and Major Chronic Diseases Among Adults in the UK. JAMA Netw Open. 2023 Mar 1;6(3):e234714. doi: 10.1001/jamanetworkopen.2023.4714. PMID: 36976560; PMCID: PMC10051114.
10. Wiedermann CJ, Barbieri V, Lombardo S, Gärtner T, Eisendle K, Piccoliori G, Engl A, Ausserhofer D. Sleep Quality and Patient Activation in Chronic Disease: A Cross-Sectional Mediation Analysis. Clocks Sleep. 2025 Aug 22;7(3):44. doi: 10.3390/clockssleep7030044. PMID: 40981207; PMCID: PMC12452411.
11. Xin Q, Yuan RK, Zitting KM, Wang W, Purcell SM, Vujovic N, Ronda JM, Quan SF, Williams JS, Buxton OM, Duffy JF, Czeisler CA. Impact of chronic sleep restriction on sleep continuity, sleep structure, and neurobehavioral performance. Sleep. 2022 Jul 11;45(7):zsac046. doi: 10.1093/sleep/zsac046. PMID: 35218665; PMCID: PMC9272266.
12. Badran M, Puech C, Gozal D. The cardiovascular consequences of chronic sleep fragmentation: Evidence from experimental models of obstructive sleep apnea. Sleep Med. 2025 Aug;132:106566. doi: 10.1016/j.sleep.2025.106566. Epub 2025 May 12. PMID: 40398206; PMCID: PMC13051657.
13. Alotiby A. Immunology of Stress: A Review Article. J Clin Med. 2024 Oct 25;13(21):6394. doi: 10.3390/jcm13216394. PMID: 39518533; PMCID: PMC11546738.
14. Nyberg ST, Frank P, Pentti J, Alfredsson L, Ervasti J, Goldberg M, Knutsson A, Koskinen A, Lallukka T, Nordin M, Rahkonen O, Strandberg T, Suominen S, Väänänen A, Vahtera J, Virtanen M, Westerlund H, Zins M, Stenholm S, Sabia S, Singh-Manoux A, Hamer M, Kivimäki M. Health benefits of leisure-time physical activity by socioeconomic status, lifestyle risk, and mental health: a multicohort study. Lancet Public Health. 2025 Feb;10(2):e124-e135. doi: 10.1016/S2468-2667(24)00300-1. PMID: 39909687; PMCID: PMC11803518.

15. Tsai MH, Vo JB, Moore JX, Ramin C, Guha A, Dong Y. Mediating effects of mental and physical health on the association between chronic disease conditions and colorectal cancer screening utilization among breast cancer survivors. PLoS One. 2025 Aug 6;20(8):e0328353. doi: 10.1371/journal.pone.0328353. PMID: 40768478; PMCID: PMC12327618.
16. Greenwood-Hickman MA, Walker RL, Idu AE, Bellettiere J, Wing D, McCurry SM, Crane PK, Larson EB, Rosenberg DE, LaCroix AZ. Current and historic patterns of chronic disease burden are associated with physical activity and sedentary behavior in older adults: an observational study. BMC Public Health. 2025 Mar 17;25(1):1032. doi: 10.1186/s12889-025-22264-8. PMID: 40098022; PMCID: PMC11917095.
17. Frank LD, Adhikari B, White KR, Dummer T, Sandhu J, Demlow E, Hu Y, Hong A, Van den Bosch M. Chronic disease and where you live: Built and natural environment relationships with physical activity, obesity, and diabetes. Environ Int. 2022 Jan;158:106959. doi: 10.1016/j.envint.2021.106959. Epub 2021 Nov 9. PMID: 34768046.
18. Domingues WJR, Germano-Soares AH, Cucato GG, de Souza LC, Brandão EKSS, Souza ELDC, da Silva E Silva TR, Arêas GPT, Costa C, Campelo PRDS, Dos Santos NJN, Silva GOD, Simões CF. Physical activity levels in patients with chronic venous insufficiency. Phlebology. 2025 Feb;40(1):47-54. doi: 10.1177/02683555241273153. Epub 2024 Aug 9. PMID: 39126137.
19. Diciolla NS, Yuste-Sánchez MJ, Torres-Lacomba M, Paixão C, Marques A. Physical activity coaching in people with chronic obstructive pulmonary disease: a systematic literature review with meta-analysis. Ann Behav Med. 2025 Jan 4;59(1):kaaf044. doi: 10.1093/abm/kaaf044. PMID: 40673868.
20. Yu Z, Xie G, Qin C, He H, Wei Q. Effect of postoperative exercise training on physical function and quality of life of lung cancer patients with chronic obstructive pulmonary disease: A randomized controlled trial. Medicine (Baltimore). 2024 Mar 8;103(10):e37285. doi: 10.1097/MD.0000000000037285. PMID: 38457572; PMCID: PMC10919482.
21. Baliunas D, Voci S, Selby P, de Oliveira C, Kurdyak P, Rosella L, Zawertailo L, Fu L, Sutradhar R. Incidence of chronic disease following smoking cessation treatment: A matched cohort study using linked administrative healthcare data in Ontario, Canada. PLoS One. 2023 Jul 26;18(7):e0288759. doi: 10.1371/journal.pone.0288759. PMID: 37494345; PMCID: PMC10370896.

22. Wasfi RA, Bang F, de Groh M, Champagne A, Han A, Lang JJ, McFaull SR, Melvin A, Pipe AL, Saxena S, Thompson W, Warner E, Prince SA. Chronic health effects associated with electronic cigarette use: A systematic review. Front Public Health. 2022 Oct 6;10:959622. doi: 10.3389/fpubh.2022.959622. PMID: 36276349; PMCID: PMC9584749.
23. Jeganathan V, Knight S, Bricknell M, Ridgers A, Wong R, Brazzale DJ, Ruehland WR, Rahman MA, Leong TL, McDonald CF. Impact of smoking status and chronic obstructive pulmonary disease on pulmonary complications post lung cancer surgery. PLoS One. 2022 Mar 29;17(3):e0266052. doi: 10.1371/journal.pone.0266052. PMID: 35349598; PMCID: PMC8963579.
24. Göltl P, Murillo K, Simsek O, Wekerle M, Ebert MP, Schneider A, Hirth M. Impact of alcohol and smoking cessation on the course of chronic pancreatitis. Alcohol. 2024 Sep;119:29-35. doi: 10.1016/j.alcohol.2023.11.006. Epub 2023 Nov 25. PMID: 38013125.
25. Li Y, Zhu B, Song N, Shi Y, Fang Y, Ding X. Alcohol consumption and its association with chronic kidney disease: Evidence from a 12-year China health and Nutrition Survey. Nutr Metab Cardiovasc Dis. 2022 Jun;32(6):1392-1401. doi: 10.1016/j.numecd.2022.02.012. Epub 2022 Feb 28. PMID: 35304050.
26. Li Y, Zhu B, Song N, Shi Y, Fang Y, Ding X. Alcohol consumption and its association with chronic kidney disease: Evidence from a 12-year China health and Nutrition Survey. Nutr Metab Cardiovasc Dis. 2022 Jun;32(6):1392-1401. doi: 10.1016/j.numecd.2022.02.012. Epub 2022 Feb 28. PMID: 35304050.
27. GBD 2021 Adolescent BMI Collaborators. Global, regional, and national prevalence of child and adolescent overweight and obesity, 1990-2021, with forecasts to 2050: a forecasting study for the Global Burden of Disease Study 2021. Lancet. 2025 Mar 8;405(10481):785-812. doi: 10.1016/S0140-6736(25)00397-6. Epub 2025 Mar 3. PMID: 40049185; PMCID: PMC11920006.
28. Lustig RH, Collier D, Kassotis C, Roepke TA, Kim MJ, Blanc E, Barouki R, Bansal A, Cave MC, Chatterjee S, Choudhury M, Gilbertson M, Lagadic-Gossmann D, Howard S, Lind L, Tomlinson CR, Vondracek J, Heindel JJ. Obesity I: Overview and molecular and biochemical mechanisms. Biochem Pharmacol. 2022 May;199:115012. doi: 10.1016/j.bcp.2022.115012. Epub 2022 Apr 5. PMID: 35393120; PMCID: PMC9050949.

29. Cen C, Fan Z, Ding X, Tu X, Liu Y. Associations between metabolic dysfunction-associated fatty liver disease, chronic kidney disease, and abdominal obesity: a national retrospective cohort study. Sci Rep. 2024 Jun 2;14(1):12645. doi: 10.1038/s41598-024-63386-0. PMID: 38825630; PMCID: PMC11144701.
30. Glenn AJ, Guasch-Ferré M, Malik VS, Kendall CWC, Manson JE, Rimm EB, Willett WC, Sun Q, Jenkins DJA, Hu FB, Sievenpiper JL. Portfolio Diet Score and Risk of Cardiovascular Disease: Findings From 3 Prospective Cohort Studies. Circulation. 2023 Nov 28;148(22):1750-1763. doi: 10.1161/CIRCULATIONAHA.123.065551. Epub 2023 Oct 25. PMID: 37877288; PMCID: PMC10841173.
31. Shah RV, Steffen LM, Nayor M, Reis JP, Jacobs DR, Allen NB, Lloyd-Jones D, Meyer K, Cole J, Piaggi P, Vasan RS, Clish CB, Murthy VL. Dietary metabolic signatures and cardiometabolic risk. Eur Heart J. 2023 Feb 14;44(7):557-569. doi: 10.1093/eurheartj/ehac446. PMID: 36424694; PMCID: PMC10169425.