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LONELINESS AS A RISK FACTOR FOR CARDIOVASCULAR DISEASE: A NARRATIVE REVIEW

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ABSTRACT

Cardiovascular diseases (CVDs) remain the leading cause of death worldwide, and growing evidence identifies loneliness as an independent psychosocial risk factor for cardiovascular health. This narrative review summarizes current evidence on the biological and behavioral mechanisms linking loneliness with CVD, based on peer-reviewed studies published between 2021 and 2026 retrieved from PubMed, Google Scholar, and Scopus. The literature indicates that loneliness acts as a chronic stressor, promoting activation of the hypothalamic-pituitary-adrenal axis and sympathetic nervous system, which contributes to systemic inflammation, impaired heart rate variability, and increased allostatic load. In addition, it encourages unhealthy behaviors such as smoking, poor diet, physical inactivity, and social withdrawal, further increasing cardiovascular risk. Overall, the evidence suggests that loneliness contributes to both the development and progression of CVD through interconnected biological and behavioral pathways. As a modifiable risk factor, loneliness should be identified early in clinical practice, and addressed through interventions such as social prescribing, cognitive behavioral approaches, and community-based programs.

KEYWORDS

Loneliness, Cardiovascular Disease, Isolation, Inflammation, HPA Axis

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1. Introduction

Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide, representing a major public health burden (Naghavi, 2025). Despite significant advances in medical treatment and prevention strategies, the incidence of cardiovascular events continues to rise, particularly in aging populations and socially vulnerable groups. This has led to increasing interest in psychosocial determinants of cardiovascular health.

Among these factors, loneliness has emerged as a significant risk factor (Cené et al., 2022). Loneliness can be described as a subjective, distressing experience that occurs when there is a discrepancy between an individual's desired and actual social relationships (Peplau & Perlman, 1982). Unlike objective social isolation, loneliness reflects an individual's perceived deficit in social relationships, which can have profound psychological and physiological consequences. It has been said that the mortality impact of loneliness is similar to smoking 15 cigarettes a day (Office of the Surgeon General (OSG), 2023). The influence of social interactions (and lack thereof) is comparable to well-established risk factors as obesity. Individuals who suffer from loneliness are at greater risk of not only cardiovascular events, but also stroke, dementia, depression, premature death. In today's "loneliness epidemic" it is important to highlight the potential harm and ways of prevention (Office of the Surgeon General (OSG), 2023). Recent research suggests that loneliness may contribute to cardiovascular disease through multiple pathways, including chronic activation of stress-related systems, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, increased inflammatory responses, and unhealthy behavioral patterns such as physical inactivity, smoking, and poor diet (Cene et al. 2022). These mechanisms collectively may increase the risk of hypertension, atherosclerosis, and coronary heart disease. Epidemiological studies and meta-analyses conducted in recent years have demonstrated a consistent association between loneliness and increased risk of cardiovascular events, including coronary heart disease and stroke (Cene et al 2022). Despite the growing number of studies on the impact of loneliness on cardiovascular health, this issue remains the subject of intensive research, with new findings continuously emerging in the scientific literature. Therefore, the aim of this narrative review is to synthesize current evidence on loneliness as a psychosocial risk factor for cardiovascular disease, with a particular focus on epidemiological findings and proposed biological and behavioral mechanisms.

2. Methodology

A literature search has been conducted using Google Scholar, PubMed and Scopus to identify relevant studies investigating the link between loneliness and cardiovascular health. The following search terms were used: “loneliness”, “cardiovascular disease”, “heart disease”, “isolation”, “psychosocial factors”. The research was primarily focused on peer-reviewed articles published between 2021 and 2026 to ensure the inclusion of the most recent evidence. As this is a narrative review the selection of sources was aimed at providing a broad synthesis of epidemiological findings and proposed biological and behavioral mechanisms linking social disconnection to cardiovascular risk. This review synthesizes evidence derived from high-level sources, including meta-analyses and systematic reviews, which provide the strongest level of empirical data, alongside insights from existing narrative reviews and large-scale cohort studies that facilitate a more comprehensive understanding of the underlying biological and behavioral mechanisms.

3. Results

Due to demographic and societal changes, many in modern societies are at risk of loneliness (Khairina & Ibrahim, 2024; Xia, Li, 2018). Along with longer life expectancy, number of people over the age of 60 has tripled since 1950s (Khairina & Ibrahim, 2024). Older age is associated with reduced number of social bonds and interactions and naturally age-related losses of close ones. However loneliness can be experienced at any age (Xia & Li, 2018). About 80% of individuals under the age of 18 and 40% of adults experience loneliness, while approximately 15-30% of the general population suffer from loneliness chronically (Khairina & Ibrahim, 2024). Another factor is the rise in one-person households, delayed marriage, delayed child-bearing or childlessness (Cohen, 2021). Additionally, the internet has changed how people live and interact. While it provides a platform for increased digital connectivity, this has paradoxically coincided with a rise in social isolation. Recent research suggests that rather than enhancing human well-being, social media can actually be harmful, potentially isolating individuals and replacing substantive "real-life" social contacts with digital interactions that do not sufficiently mitigate loneliness, as they often are superficial (Xia & Li, 2018). Chronic social isolation and loneliness are now recognized as critical health determinants, with their impact on morbidity and mortality being comparable to established clinical risk factors like hypertension, smoking, and obesity (Khairina & Ibrahim, 2024; Hawkey & Cacioppo 2010). Comprehensive research, a meta-analysis including 148 studies of over 300,000 participants, followed for 7.5 years on average, has shown that the influence of social isolation on health exists independently of other factors, whereas having strong social connections can boost survival odds by 50% (Holt-Lunstad et al., 2010). Loneliness has profound negative effects on an individual's cognitive, emotional, and behavioral health (Khairina & Ibrahim, 2024; Hawkey & Cacioppo, 2010). The overall mortality risk associated with loneliness and social isolation is estimated at 1.50, a figure that is comparable to light smoking and exceeds the risks posed by obesity or hypertension. Specific data indicates that social isolation, loneliness, and living alone increase the likelihood of death by 29%, 26%, and 32%, respectively. Within certain populations, individuals with "poor social health", defined by isolation, lack of support, and loneliness, are 42% more likely to develop cardiovascular disease (CVD) and face double the risk of CVD-related death within a five-year period. (Khairina & Ibrahim, 2024; Holt-Lunstad et al., 2015)

Research further distinguishes between the effects of objective isolation and subjective loneliness on specific diseases. Social isolation and low social support are strong predictors of the initial occurrence of CVD, whereas loneliness is more specifically associated with cardiovascular-related death. Large-scale population studies, such as those utilizing the UK Biobank, have demonstrated that while social isolation is linked to a higher 10-year risk of atherosclerotic cardiovascular disease (ASCVD) in both genders, loneliness shows a more significant association with ASCVD risk specifically in men. Ultimately, the risk of coronary heart disease increases by 29% and stroke by 32% due to poor social relationships, an effect size comparable to other psychological risk factors like anxiety and work-related stress. These findings emphasize that loneliness and social isolation are critical independent determinants of cardiovascular health in contemporary society (Khairina & Ibrahim, 2024; Freak-Poli, 2021).

4. Biological mechanisms linking loneliness and cardiovascular disease.

Loneliness strongly predicts negative physical and mental health effects throughout life (Morrish et al., 2023)

Thus far, there is a number of biological, behavioral and psychological pathways in loneliness that has been identified as contributors to cardiovascular disease (Xia & Li, 2018). These mechanisms do not operate in isolation but rather interact with and reinforce one another (Paul et al., 2021).

4.1. Stress response

Loneliness acts as a stressor to the body and activates the hypothalamic-pituitary-adrenal axis (HPA)- the stress response system. The hypothalamic pituitary adrenal gland secretes cortisol- the stress hormone. If the feeling of loneliness persists over a prolonged period of time it can lead to chronically elevated cortisol levels, which are associated with CVD incidence, and can contribute to CVD risk factors, such as elevated systolic blood pressure, higher cholesterol, elevated triglycerides, diabetes (Paul et al., 2021). Research shows that chronic stress can increase the risk of thrombosis, possibly resulting in strokes and heart attacks. Interestingly, the response to stress differs between sexes, among women, heightened HPA axis is typical, while men tend to demonstrate greater inflammatory reactivity (Warren, 2025).

4.2. Inflammation

The immune system serves as a primary biological bridge between loneliness and cardiovascular disease, primarily through the promotion of chronic systemic inflammation (Khairina & Ibrahim, 2024; Paul et al., 2021). Loneliness, acting as a potent chronic psychosocial stressor, triggers the sustained activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) (Khairina & Ibrahim, 2024; Warren, 2025). This physiological response leads to a state of low-grade systemic inflammation, often characterized by elevated levels of pro-inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6), both of which are central to the pathogenesis of atherosclerosis (Khairina & Ibrahim, 2024; Warren, 2025). Recent research highlights that these alterations do not occur in isolation but form an interconnected "immunometabolic syndrome". As proposed by Pourriyahi et al. (2021), loneliness functions as a chronic stressor that simultaneously drives low-grade inflammation and metabolic dysfunction including insulin resistance and visceral adiposity. These factors together disrupt physiological homeostasis and accelerate cardiovascular risk. Furthermore, cytokines coordinate the body's inflammatory reaction and initiate "sickness behavior," which includes symptoms such as fatigue and social withdrawal (Paul et al., 2021). While this response is an adaptive short-term defense against potential infection, its chronic persistence in lonely individuals contributes significantly to the cumulative physiological burden known as allostatic load, ultimately increasing cardiovascular morbidity and mortality (Warren, 2025).

At the molecular level, loneliness is associated with a specific genomic profile in leukocytes known as the Conserved Transcriptional Response to Adversity (CTRA) (Khairina & Ibrahim, 2024).. This profile is characterized by a significant upregulation of pro-inflammatory genes and a concurrent downregulation of genes involved in antiviral and antibody-related immune functions. These changes are largely driven by the selective expansion of immature CD14⁺⁺/CD16⁻ monocyte subsets in the bone marrow, a process stimulated by SNS-mediated β -adrenergic signaling in response to social stress. Despite the fact that the overall leukocyte count often remains stable, the higher percentage of these highly reactive, pro-inflammatory monocytes ensures a sustained inflammatory environment (Khairina & Ibrahim, 2024).. This pro-inflammatory state is further exacerbated by the development of glucocorticoid resistance. Chronic stress and perceived isolation lead to a downregulation of glucocorticoid receptor (GR) expression and an upregulation of the pro-inflammatory transcription factor NF- κ B. Consequently, circulating lymphocytes become less sensitive to the anti-inflammatory signaling of cortisol, preventing the body from effectively terminating the inflammatory response. The relationship between immune dysregulation and loneliness appears to be reciprocal; immature pro-inflammatory monocytes can cross the blood-brain barrier and enter the brain, potentially triggering anxiety and social behavioral alterations that further reinforce withdrawal (Khairina & Ibrahim, 2024). This feedback loop illustrates how loneliness "gets under the skin" to accelerate biological aging and cardiovascular risk through a complex interplay of neuroendocrine and genomic mechanisms (Khairina & Ibrahim, 2024; Warren, 2025).

4.3. Heart Rate Variability and dysregulated parasympathetic nervous system response

Heart Rate Variability (HRV) is a physiological marker that measures the variation in time intervals between consecutive heartbeats- specifically R-R intervals. In a healthy individual, the heart does not beat like a metronome, instead these intervals change dynamically, responding to internal and external factors, reflecting the overall balance and adaptability of the autonomic nervous system (Thayer et al., 2017). HRV serves as an essential indicator of the body's ability to regulate its activity and adapt to unknown, new, potentially threatening stimuli in the environment (Thayer et al., 2017). Therefore the heart rate variability is largely mediated through parasympathetic nervous system, which is responsible for relaxation and regeneration after the threat has passed. Parasympathetic response lowers heart rate, stabilizes heart rhythm, dilates blood vessels and lowers blood pressure. Empirical research indicates that in individuals experiencing high levels of chronic loneliness, parasympathetic regulation is significantly dysregulated (Piejka et al., 2024). Specifically, these individuals demonstrate a diminished capacity to "turn off" physiological stress responses and safely return to a state of homeostasis following exposure to a stressor. This autonomic blunting is frequently coupled with heightened activity within the hypothalamic-pituitary-adrenal (HPA) axis, keeping the organism in a prolonged, maladaptive "fight-or-flight" state. Research conducted by the Social Neuroscience Lab at the Polish Institute of Psychology of the Polish Academy of Sciences(PAN) provide robust empirical evidence linking HRV and loneliness. Their findings demonstrate that chronic perceived social isolation significantly associates with compromised resting vagal tone (Piejka et al., 2024). Furthermore, experimental evidence from this institution shows that even brief, laboratory-induced feelings of momentary

loneliness cause an immediate, measurable impairment in parasympathetic vagal regulation during social information processing (Okuszek, 2026; Piejka et al., 2021). It suggest that the heart reacts almost immediately to the "social pain" decreasing its natural variability and flexibility in responding to stimuli. From a clinical perspective, high baseline heart rate variability represents a resilient, flexible, and highly adaptable physiological system. Conversely, persistently low HRV indicates a severely restricted capacity for stress recovery and physiological regulation. Chronically suppressed HRV is widely recognized in contemporary literature as an independent and significant risk factor for the development of cardiovascular diseases and elevated all-cause mortality (Thayer et al., 2017).

5. Behavioral mechanisms

Although they are often used interchangeably, social isolation and loneliness are different concepts (Hawkley, 2022; Khairina & Ibrahim, 2024; Paul et al., 2021). Social isolation is an objective lack of social contacts and roles (World Health Organization [WHO], Commission on Social Connection, 2025), while loneliness is the painful, subjective feeling that one's relationships do not meet their needs (Khairina & Ibrahim, 2024; Paul et al., 2021; Peplau & Perlman, 1982).. In modern life, the quality of a relationship is a better protector of health than just the number of friends a person has (Khairina & Ibrahim, 2024). Strong social ties encourage healthy habits like regular exercise and a balanced diet (Khairina & Ibrahim, 2024; Paul et al., 2021). In contrast, loneliness acts as a major stressor that weakens a person's ability to self-regulate (Hawkley & Cacioppo, 2010; Khairina & Ibrahim, 2024; Warren, 2025).

This creates a dangerous "vicious cycle" (Warren, 2025), When someone feels lonely, their brain becomes hyper-alert to social threats and rejection. This hyper-vigilance makes them trust others less, which leads to further social withdrawal and even more isolation. To cope with this constant stress, lonely individuals often turn to maladaptive behaviors, such as smoking, alcohol abuse, or poor dietary choices, to get short-term relief (Khairina & Ibrahim, 2024; Paul et al., 2021; Warren, 2025).

Importantly, reduced physical activity in lonely individuals is not only a behavioral consequence but also an independent cardiovascular risk pathway (Manera et al., 2022; Paul et al., 2021; Warren, 2025).. Physical inactivity is strongly associated with reduced cardiorespiratory fitness, increased adiposity, and impaired metabolic health, all of which contribute to the development of cardiovascular disease (Khairina & Ibrahim, 2024; Paul et al., 2021).. Recent evidence suggests that socially isolated and lonely individuals are less likely to meet recommended physical activity guidelines, which may partially mediate the association between loneliness and cardiovascular outcomes (Manera et al., 2022; Wang et al., 2025). Conversely, regular participation in sport and exercise may act as a protective buffer, mitigating some of the physiological and psychological effects of loneliness through improved mood regulation, reduced stress reactivity, and enhanced social integration (Khairina & Ibrahim, 2024; Manera et al., 2022).

This cycle is not just psychological; it is deeply biological (Warren, 2025).. The immune system responds to loneliness by triggering "sickness behavior"—a state of fatigue and social withdrawal caused by

inflammation. This makes the person even less likely to interact with others (Khairina & Ibrahim, 2024; Warren, 2025).. Recent research from 2024 using daily surveys found that lonely people perceive everyday stressors as more severe and are less likely to ask for help during difficult moments (Kang et al., 2024).. Over time, these combined behavioral and biological factors increase allostatic load, which is the physical "wear and tear" on the heart and body caused by chronic stress. This interconnected model shows how loneliness directly drives the development and progression of cardiovascular disease (Khairina & Ibrahim, 2024; Warren, 2025; Cen   et al., 2022).

Furthermore, loneliness significantly impacts the 'social brain' by altering neural responses to social stimuli (Lieberz et al., 2021). Neuroimaging findings by Lieberz et al. (2021) reveal that lonely individuals exhibit diminished activation in key emotional and reward-processing regions during trust-based decision-making. This neurobiological disruption creates a self-perpetuating cycle: lonely individuals extend less trust to others and are, in turn, perceived as less trustworthy themselves. These patterns are further exacerbated by reduced connectivity between brain regions that integrate social and emotional data. By biasing cognitive processing toward social threats and decreasing responsiveness to positive interactions, loneliness systematically reduces the motivation for social engagement (Lieberz et al., 2021; Warren, 2025). This interplay between neural mechanisms and social withdrawal is illustrated in Figure 1.

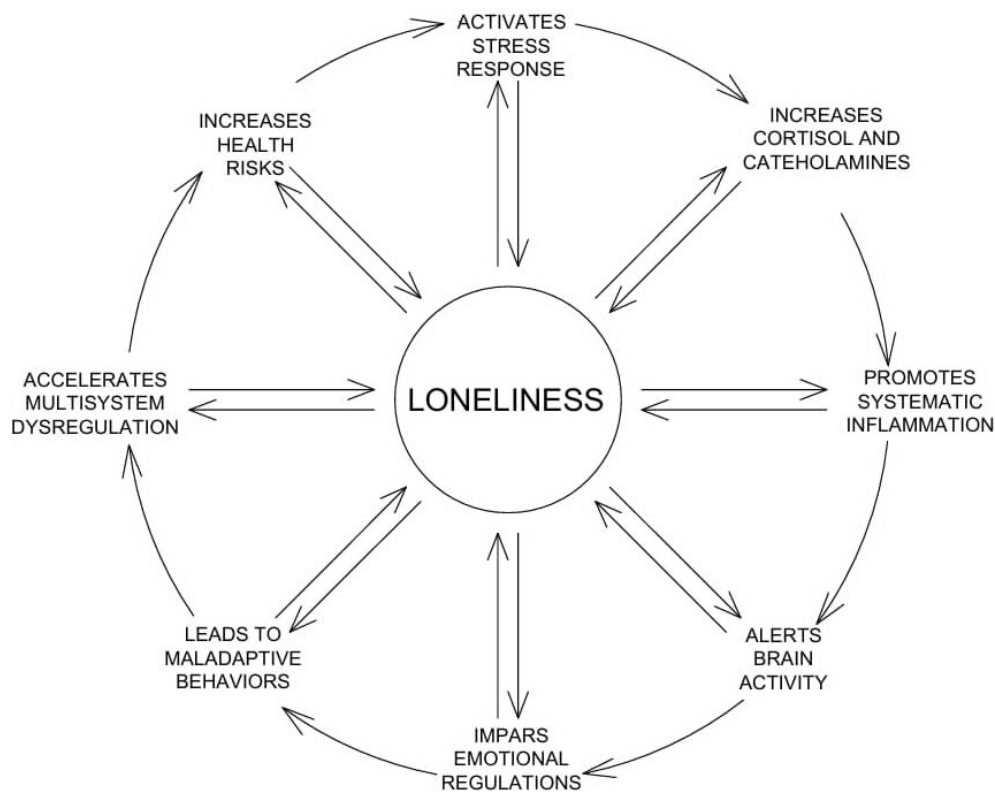


Fig. 1. The cycle of loneliness and health consequences. Adapted from Warren A. (2025)

6. Discussion

6.1. Clinical Implications

The growing evidence linking loneliness to cardiovascular disease has important clinical and public health implications (Khairina & Ibrahim, 2024; Paul et al., 2021; Warren, 2025). Loneliness should not be viewed solely as a psychosocial experience, but rather as a clinically relevant risk factor that contributes to the development and progression of cardiovascular conditions (Cené et al., 2022; Usama et al., 2024; Warren, 2025). From a clinical perspective, individuals experiencing chronic loneliness or social isolation may represent a high-risk group for adverse cardiovascular outcomes, including coronary artery disease, stroke, heart failure, and increased all-cause mortality (Holt-Lunstad et al., 2015; Khairina & Ibrahim, 2024; Liang et al., 2023;). Importantly, loneliness is associated not only with disease incidence but also with worse prognosis and higher mortality following cardiovascular events (Khairina & Ibrahim, 2024; Paul et al., 2021). This suggests that psychosocial factors may influence both the development and the clinical course of cardiovascular disease (Paul et al., 2021; Warren, 2025).

Despite this, loneliness is still rarely assessed in routine clinical practice (Cené et al., 2022; Usama et al., 2024). Unlike traditional risk factors, it is not typically included in standard cardiovascular risk scores, which may lead to underrecognition of patients at risk (Cené et al., 2022; Warren, 2025). Incorporating brief screening tools for social isolation and perceived loneliness into primary care and cardiology settings could improve early identification of vulnerable individuals (Cené et al., 2022; Warren, 2025). Furthermore, the recognition of loneliness as a modifiable risk factor opens new possibilities for intervention (Khairina & Ibrahim, 2024; Usama et al., 2024; Warren, 2025). Multidisciplinary approaches combining medical care with psychological support and social interventions may be particularly effective (Warren, 2025). Interventions such as social prescribing, community-based programs, and structured psychological therapies (e.g., cognitive-behavioral approaches targeting maladaptive social cognition) may help reduce perceived loneliness and improve overall health outcomes (Blodgett et al., 2025; Morrish et al., 2023; Reinhardt et al., 2021).

In addition, lifestyle-based interventions, including physical activity and group-based rehabilitation programs, may provide both direct cardiovascular benefits and indirect improvements through enhanced social engagement (Heaney et al., 2026; Manera et al., 2022; Paul et al., 2021). At a broader level, addressing loneliness requires public health strategies aimed at strengthening social connectedness within communities (Office of the Surgeon General [OSG], 2023; WHO Commission on Social Connection, 2025). Healthcare systems may play a key role in identifying at-risk individuals and facilitating access to social support resources (Usama et al., 2024; Warren, 2025). Given the complex and bidirectional relationship between loneliness and health, integrated interventions that target both biological and behavioral pathways are likely to be the most effective (Khairina & Ibrahim, 2024; Paul et al., 2021; Warren, 2025). Overall, recognizing loneliness as a significant cardiovascular risk factor shifts the focus from purely biomedical management of disease toward a more holistic model of care, in which social well-being is considered an essential component of cardiovascular health (Cené et al., 2022; Khairina & Ibrahim, 2024; Warren, 2025).

6.2. Prevention

Prevention strategies targeting loneliness are increasingly recognized as an important component of cardiovascular disease (CVD) prevention, particularly given its role as a modifiable psychosocial risk factor (Cené et al., 2022; Khairina & Ibrahim, 2024; Usama et al., 2024; Warren, 2025). Current evidence suggests that loneliness is not only treatable but also preventable through targeted individual, community, and healthcare-based interventions (Blodgett et al., 2025; Morrish et al., 2023; Warren, 2025). As loneliness is closely linked to social isolation, reduced social participation, and maladaptive health behaviors, effective prevention requires a multidimensional approach addressing both social environment and individual vulnerability (Khairina & Ibrahim, 2024; Paul et al., 2021; Warren, 2025). A growing body of literature highlights the effectiveness of social prescribing models, in which healthcare professionals connect individuals to non-clinical community resources such as group activities, volunteering, or exercise programs (Cené et al., 2022; Paul et al., 2021; Reinhardt et al., 2021; Warren, 2025). These interventions aim to strengthen social connectedness and reduce perceived loneliness while simultaneously improving physical and mental health outcomes (Cené et al., 2022; Paul et al., 2021). Systematic reviews indicate that social prescribing initiatives can lead to reductions in loneliness and improvements in wellbeing, although the strength of evidence varies depending on study design and population (Cené et al., 2022; Reinhardt et al., 2021; Warren, 2025). In addition to healthcare-led approaches, group-based and community interventions have been identified as particularly effective (Blodgett et al., 2025; Morrish et al., 2023; Warren, 2025). Recent systematic reviews show that interventions involving group participation, active engagement, and repeated social interaction are more likely

to reduce loneliness compared to purely individual or passive approaches (Blodgett et al., 2025; Morrish et al., 2023; Warren, 2025). These findings suggest that sustained social contact and meaningful interaction are key protective factors against loneliness-related health deterioration (Morrish et al., 2023; Paul et al., 2021; Warren, 2025). For older adults and individuals with cardiovascular disease, tailored interventions appear especially relevant (Heaney et al., 2026; Warren, 2025). A recent systematic review focusing on this population found that non-pharmacological interventions such as group exercise, educational sessions, and structured social activities may improve social support, psychological well-being, and quality of life, all of which are indirectly linked to better cardiovascular outcomes (Heaney et al., 2026; Paul et al., 2021; Warren, 2025).

From a broader public health perspective, prevention of loneliness also involves addressing upstream determinants such as living arrangements, community infrastructure, and access to social participation (Cené et al., 2022; Warren, 2025). Urban planning that encourages social interaction, policies supporting active ageing, and workplace or school-based programs promoting inclusion may all contribute to reducing population-level loneliness risk (Warren, 2025). Importantly, evidence suggests that there is no single “quick fix” for loneliness (Morrish et al., 2023). Instead, sustained prevention requires long-term engagement, personalised approaches, and integration across healthcare, social care, and community systems (Blodgett et al., 2025; Morrish et al., 2023; Warren, 2025). Given the bidirectional relationship between loneliness and cardiovascular health, preventive strategies that enhance social connection may also contribute to reducing cardiovascular morbidity and mortality over time (Khairina & Ibrahim, 2024; Paul et al., 2021; Warren, 2025).

7. Conclusions

Loneliness and social isolation have been established as significant, independent risk factors for cardiovascular disease (CVD), with an impact on morbidity and mortality comparable to traditional risks such as smoking, obesity, and hypertension (Holt-Lunstad et al., 2015; Paul et al., 2021; Khairina & Ibrahim, 2024;). This narrative review highlights that loneliness is a complex, multisystem stressor that "gets under the skin" through an interconnected web of biological, behavioral, and psychological pathways (Warren, 2025). The biological impact is characterized by the accumulation of allostatic load, driven by chronic activation of the HPA axis and the sympathetic nervous system, which leads to systemic inflammation, impaired heart rate variability (HRV), and altered genomic profiles in immune cells (Khairina & Ibrahim, 2024; Warren, 2025).

Behaviorally, loneliness triggers a "vicious cycle" where impaired self-regulation and increased threat vigilance lead to maladaptive habits—such as physical inactivity and smoking—which further exacerbate cardiovascular damage (Paul et al., 2021; Warren, 2025). Importantly, the evidence suggests that loneliness is not just a by-product of objective social isolation but a distinct subjective experience that requires specific clinical attention (Cené et al., 2022; Khairina & Ibrahim, 2024).

From a clinical and public health perspective, loneliness should be recognized as a modifiable risk factor (Cené et al., 2022; Usama et al., 2024). Future strategies must shift toward a more holistic model of care that includes routine screening for social health in primary care and cardiology settings (Cené et al., 2022; Warren, 2025). Interventions such as social prescribing, cognitive-behavioral therapies, and community-based group activities offer promising pathways to reduce social disconnection and improve long-term cardiovascular outcomes (Morrish et al., 2023; Blodgett et al., 2025; Warren, 2025). In conclusion, addressing loneliness is no longer just a social necessity but a critical public health priority for reducing the global burden of cardiovascular disease (Khairina & Ibrahim, 2024; Warren, 2025).

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