REVIEW ARTICLE

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The Relationship Between Emotional Inhibition and Cardiovascular Disease: A Psychophysiological Review

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Abstract

Background: Cardiovascular disease (CVD) continues to be the foremost cause of death worldwide . Although established risk factors such as hypertension and smoking are well-recognized, a considerable amount of CVD risk remains unaccounted for, shifting focus toward psychosocial influences . Emotional suppression—the deliberate inhibition of emotional expression—constitutes an ineffective emotion regulation strategy with possible cardiotoxic effects.

Objective: This narrative review seeks to consolidate contemporary psychophysiological research to clarify the mechanisms through which habitual emotional suppression elevates CVD risk and contributes to adverse health outcomes.

Methods: A thorough literature review was performed using PubMed, PsycINFO, and Web of Science for publications between 1990 and 2025. Search terms included "emotional suppression," "expressive inhibition," "cardiovascular disease," "autonomic nervous system," "cortisol," "inflammation," and "endothelial function." Emphasis was placed on human studies exploring physiological pathways or clinical correlations.

Results: Current findings indicate that emotional suppression reliably initiates harmful psychophysiological processes: (1) amplified and sustained sympathetic nervous system activity alongside reduced parasympathetic function, (2) dysregulation of the hypothalamic-pituitary-adrenal axis, (3) heightened inflammatory responses, and (4) acute and potentially persistent endothelial impairment. Epidemiological research further connects trait suppression with greater incidence of hypertension, coronary artery disease, and cardiovascular mortality.

Conclusion: Chronic emotional suppression operates as a meaningful psychosocial stressor, fostering a pathogenic internal environment that accelerates atherosclerosis and cardiovascular incidents. Incorporating emotion regulation evaluation into cardiovascular risk assessment and implementing focused psychological interventions offer promising strategies for enhancing preventive cardiology within a biopsychosocial model.

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Introduction

Cardiovascular disease represents a critical worldwide health burden, responsible for an estimated 17.9 million deaths per year, constituting roughly 32% of global mortality [1]. This persistent challenge remains despite advances in the management of conventional biomedical risk factors including dyslipidemia, diabetes, and hypertension [2]. Notably, large-scale studies such as INTERHEART indicate that these traditional factors explain only a fraction of total population risk for myocardial infarction, underscoring a significant proportion of unexplained residual risk [3]. This gap has encouraged a paradigm shift in cardiology, fostering rigorous examination of psychosocial and behavioral contributors to cardiovascular pathology [4].

Within this evolving framework, psychosomatic research has demonstrated clear associations between chronic psychological stress, negative emotional states—such as depression and anxiety—and heightened CVD incidence and mortality [5,6]. Moving beyond broad psychological constructs, contemporary investigations have aimed to pinpoint specific psychological mechanisms that may translate emotional experience into physiological dysregulation. Central to this effort is emotion regulation—the diverse set of processes through which individuals modulate which emotions arise, when they occur, and how they are experienced and expressed [7].

Emotion regulation strategies range from adaptive to maladaptive. Cognitive reappraisal, which involves reinterpreting an emotion-eliciting event beforehand, is generally linked to positive psychological and physiological outcomes [8]. Conversely, emotional suppression—a response-focused tactic entailing the conscious restraint of ongoing emotional expression—is increasingly identified as a maladaptive pattern associated with substantial health consequences [9]. Although suppression may provide temporary social advantages, its habitual application correlates with heightened sympathetic arousal, elevated negative affect, diminished well-being, and compromised social functioning [10,11].

This article aims to deliver a thorough, mechanism-focused review of emotional suppression's role in CVD from an integrative psychophysiological standpoint. We propose that suppression is not solely a psychological characteristic but constitutes a persistent biobehavioral stressor. This review will outline the principal biological routes—autonomic, neuroendocrine, inflammatory, and vascular—by which suppression is hypothesized to produce cardiotoxic effects. We will integrate evidence from controlled experiments, ambulatory monitoring, and prospective cohort studies. Additionally, we will address moderating influences, clinical implications for cardiology and behavioral medicine, and vital directions for future interdisciplinary inquiry. By linking psychological science with cardiovascular pathophysiology, this review intends to fortify the conceptual basis for integrated biopsychosocial methods in CVD prevention and management.

Methods

A systematic search of relevant literature was conducted utilizing PubMed, PsycINFO, and Web of Science databases for articles released from 1990 through 2025. Key search terms comprised combinations of "emotional suppression," "expressive inhibition," "cardiovascular disease," "autonomic nervous system," "cortisol," "inflammation," and "endothelial function." The review prioritized human studies investigating physiological mechanisms or clinical endpoints.

Results and discussion

Cumulative evidence confirms that emotional suppression consistently triggers deleterious psychophysiological cascades: (1) exaggerated and prolonged sympathetic activation concurrent with vagal withdrawal, (2) disturbed hypothalamic-pituitary-adrenal axis function, (3) increased pro-inflammatory cytokine levels, and (4) acute and possibly chronic degradation of endothelial performance. Epidemiological data additionally correlate trait suppression with elevated rates of hypertension, coronary heart disease, and cardiovascular death.

The compiled evidence supports a persuasive, multifaceted case: chronic emotional suppression represents a notable independent psychosocial risk factor for CVD development and progression. Findings extend beyond correlation to reveal a coherent psychophysiological cascade whereby the behavioral inclination to inhibit emotional expression sets off a sequence of biological disruptions that collectively promote a pro-atherogenic and pro-hypertensive state [32,33]. This section synthesizes principal results, proposes an updated pathophysiological model, considers clinical applications and complications, and charts a course for subsequent research.

An Integrated Pathophysiological Framework

Reviewed data support an integrative model positioning emotional suppression as a central

component within a network of pathogenic events. The model originates with the act of suppression, which during emotional encounters demands considerable cognitive effort and self-monitoring [34]. This effortful restraint appears to disconnect the natural expressive-motor aspect of emotional response, resulting in physiological dissonance—where subjective emotional experience and physiological arousal become detached from behavioral expression [35].

This dissonance emerges acutely as autonomic nervous system rigidity: increased sympathetic cardiac and vascular activity paired with parasympathetic retreat [9,36]. Importantly, it is not the intensity of emotional reaction itself, but the pattern of prolonged activation following suppression that seems most harmful. Unlike adaptive regulation that supports physiological recovery, suppression entraps the cardiovascular system in a protracted state of heightened energy mobilization absent behavioral outlet [37]. When habitual, this pattern may contribute to sustained hypertension and left ventricular hypertrophy via repeated hemodynamic strain [38,39].

Simultaneously, suppression disrupts hypothalamic-pituitary-adrenal (HPA) axis regulation. Observed flattening of the diurnal cortisol rhythm [16,40] implies failure of negative feedback, possibly indicating glucocorticoid receptor resistance. This resistance is crucial as it weakens the body's primary anti-inflammatory signaling mechanism [41]. Hence, the pro-inflammatory state tied to suppression [20,42] may stem from a dual impact: sympathetic nervous system-mediated cytokine stimulation (e.g., via β-adrenergic receptors on immune cells) and concurrent insufficiency of cortisol to properly curb inflammatory responses [43]. This synergy establishes a persistent, low-grade inflammatory environment that directly encourages endothelial dysfunction, atherosclerotic plaque formation, and instability [24,44]. Laboratory demonstrations that suppression acutely reduces flow-mediated dilation [25,26] offer a direct, real-time connection between this psychological process and a fundamental event in atherogenesis.

Evaluation and Moderating Variables

While evidence is substantial, nuances and moderators warrant consideration. The relationship is likely bidirectional; subclinical cardiovascular impairment might affect emotion regulation capacity. Moreover, individual differences meaningfully moderate the suppression-CVD association. Cultural setting is critical: in societies where emotional restraint is normative and esteemed (e.g., certain East Asian cultures), the psychological and physiological toll of suppression may be reduced [45,46]. Gender may also influence outcomes, with some research indicating that men—often socialized toward emotional restraint—might display stronger cardiovascular reactivity during suppression tasks [47], though results are inconsistent.

The construct of Type D (Distressed) Personality, defined by high negative affectivity combined with high social inhibition (a conceptual relative of suppression), has supplied compelling epidemiological support [28,31]. The prognostic strength of Type D in cardiac patients emphasizes that it is the confluence of emotional distress and its behavioral restraint that proves especially detrimental, rather than either element alone. This underscores the need to examine suppression within its broader affective context.

Clinical Applications: Translating Research to Practice

Applying this knowledge clinically is a vital next step. First, routine psychosocial screening in cardiology should be extended. Beyond evaluating depression and anxiety, brief validated tools such as the Emotion Regulation Questionnaire [8] or the Type D Scale (DS-14) [48] could effectively detect patients relying on maladaptive regulatory strategies like suppression, enabling targeted intervention.

Second, evidence-based psychological interventions should be incorporated into standard cardiac rehabilitation and preventive care. These approaches should advance beyond general stress management to specifically address emotion regulation abilities.

- **Mindfulness-Based Interventions (MBIs)**, including Mindfulness-Based Stress Reduction (MBSR), foster non-judgmental awareness of present-moment experience, including emotions. This metacognitive stance lessens the perceived threat of negative emotions and the urge to suppress them, thereby lowering autonomic arousal and enhancing heart rate variability [49,50].
- **Cognitive-Behavioral Therapy (CBT)** can be tailored to help patients recognize automatic distressing thoughts and cultivate cognitive reappraisal skills as a healthier substitute for suppression [51].
- **Emotion-Focused Therapy (EFT)** offers a structured method to safely access, experience, and process restrained emotions, potentially alleviating the physiological stagnation linked to chronic suppression [52].

Third, promoting collaborative care models between cardiologists and health psychologists is essential. A genuine biopsychosocial approach requires concurrent assessment and management of biological and psychological risk factors.

Despite advances, several limitations persist. Much evidence remains correlational; longitudinal studies with repeated physiological measurements are

necessary to establish temporal sequence and causality. There is also variability in how suppression is assessed (state versus trait, laboratory induction versus self-report). Future studies should:

1. Implement ambulatory ecological momentary assessment (EMA) combined with wearable physiological sensors (e.g., for beat-to-beat blood pressure, heart rate variability) to capture real-world dynamics of suppression and cardiovascular function in daily life [53].
2. Employ multilevel assessment merging neural (fMRI to study prefrontal-amygdala connectivity during suppression) [54], endocrine (diurnal cortisol, inflammatory markers), and vascular (peripheral arterial tonometry) measures within single studies to clarify system-level interactions.
3. Explore biological embedding by investigating genetic (e.g., serotonin transporter polymorphisms) [55] and epigenetic (e.g., DNA methylation of stress-related genes like FKBP5) [56] moderators that may confer differential susceptibility.
4. Conduct mechanism-oriented randomized controlled trials (RCTs). These trials should examine whether reducing suppression via psychological interventions directly improves intermediate physiological outcomes (e.g., lowers inflammation, enhances endothelial function) in pre-hypertensive or stable CVD patients, confirming a clear pathway from intervention to biological benefit [57,58].

Conclusion

From a psychophysiological perspective, emotional suppression is considerably more than an interpersonal style or social tactic; it is a potent behavioral risk factor for cardiovascular disease. By sustaining a condition of autonomic imbalance, neuroendocrine dysregulation, systemic inflammation, and vascular damage, chronic suppression actively contributes to the pathophysiology it may unintentionally aim to control. Recognizing this hidden link marks a crucial evolution in comprehending heart disease. The future of cardiovascular medicine depends on seamlessly incorporating insights from psychology and neuroscience into preventive and therapeutic frameworks. By acknowledging the heart as an organ responsive not only to hemodynamics and biochemistry but also to the unspoken weight of emotional experience, we advance toward a more holistic, effective, and compassionate model of cardiovascular care.

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Authors Contributions

The authors contributed to the data analysis. Drafting, revising and approving the article, responsible for all aspects of this work.

Conflict of Interest

None

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