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Global Warming and Vascular Thrombosis: Mechanisms and Medical Implications

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Abstract

Global warming represents one of the most pressing health challenges of the 21st century, with far-reaching implications beyond infectious diseases and respiratory disorders. Emerging evidence suggests that rising global temperatures and associated environmental stressors can profoundly influence vascular health, particularly by promoting the development of thrombosis. Vascular thrombosis, encompassing venous thromboembolism, arterial thrombosis, and microvascular clotting, is a major contributor to cardiovascular morbidity and mortality. This review explores the mechanistic links between global warming and vascular thrombosis, including heat-induced hemoconcentration, systemic inflammation, endothelial dysfunction, and alterations in coagulation pathways. Special emphasis is placed on vulnerable populations such as the elderly, patients with cardiovascular risk factors, and individuals living in low-resource settings where climate adaptation is limited. The medical implications of these interactions are profound, underscoring the need for integrated preventive strategies, biomarker-driven monitoring, and climate-sensitive healthcare policies. By examining current evidence and highlighting research gaps, this review advocates for a holistic understanding of how global warming shapes thrombotic disease risk and for the urgent implementation of adaptive medical strategies.

Keywords: *Global warming, Vascular thrombosis, Hemostasis, Climate change, Hypercoagulability*

Introduction

Global warming is one of the defining challenges of the 21st century, with direct and indirect consequences for human health [1]. Traditionally, research has focused on infectious diseases, respiratory conditions, and heat-related illnesses, yet an equally significant concern lies in its effects on vascular health. Vascular thrombosis, a leading cause of morbidity and mortality worldwide, is increasingly being linked to climate-related environmental stressors. Understanding this intersection is crucial, as cardiovascular and hematologic diseases already impose a substantial burden on global healthcare systems [2-3]. Vascular thrombosis encompasses arterial, venous, and microvascular clotting events, with conditions such as myocardial infarction, ischemic stroke, deep vein thrombosis (DVT), and pulmonary embolism representing its most serious manifestations [4-5]. The pathophysiology of thrombosis has classically been explained through Virchow's triad—endothelial injury, stasis of blood flow, and hypercoagulability. Each of these elements can be exacerbated by environmental changes associated with global warming, thereby creating a perfect storm for thrombotic disease development [6-7]. Heat exposure and dehydration, common during prolonged heat waves, directly contribute to hemoconcentration and blood viscosity, heightening the risk of venous and arterial clot formation. Simultaneously, global warming intensifies exposure to air pollution, wildfires, and allergens, which trigger systemic inflammation and oxidative stress, leading to endothelial dysfunction. These physiological disruptions create a pro-thrombotic environment in populations exposed to extreme weather conditions [8-9]. Moreover, climate-driven extreme weather events such as hurricanes, floods, and wildfires disrupt healthcare services, medication supply chains, and access to timely medical care. Patients dependent on anticoagulation therapy may face interruptions in treatment, while populations displaced by disasters often endure prolonged immobility—both strong risk factors for venous thrombosis. This interplay between environmental disruption

and vascular pathology illustrates how global warming indirectly magnifies the burden of thrombotic diseases [10-11].

The influence of climate change on vascular thrombosis is not uniform across populations. Elderly individuals, patients with pre-existing cardiovascular or metabolic diseases, and socioeconomically disadvantaged communities are disproportionately affected. These groups are less able to adapt to extreme heat, more likely to experience dehydration, and often face barriers to healthcare access. Pregnant women also constitute a vulnerable group, given their heightened baseline risk of thromboembolic events and susceptibility to heat stress [12-13]. This review therefore aims to provide a comprehensive examination of the mechanisms linking global warming to vascular thrombosis and to discuss the medical implications of this evolving relationship. By analyzing physiological, molecular, and systemic pathways, we highlight how climate change exacerbates thrombotic risks. Furthermore, we address the impact on vulnerable populations, explore potential biomarkers for monitoring climate-associated vascular stress, and propose preventive and adaptive strategies. Ultimately, this review underscores the need for urgent interdisciplinary collaboration to mitigate the cardiovascular consequences of global warming.

Aim

The aim of this narrative review is to comprehensively examine the relationship between global warming and vascular thrombosis by integrating mechanistic, clinical, and epidemiological evidence

Methods

This narrative review employed a structured yet flexible approach appropriate for synthesizing emerging and interdisciplinary evidence on climate-driven vascular thrombosis. A comprehensive literature search was conducted across major scientific databases, including

PubMed, Scopus, Web of Science, and Google Scholar, covering publications from 2000 to 2025. Search terms included combinations of “global warming,” “climate change,” “heat exposure,” “vascular thrombosis,” “venous thromboembolism,” “ischemic stroke,” “myocardial infarction,” “air pollution,” “endothelial dysfunction,” and “coagulation.”

Both observational and experimental studies, including epidemiological reports, mechanistic investigations, climate–health modeling studies, and clinical outcome analyses, were considered. Grey literature sources such as WHO climate reports, IPCC assessments, and environmental health agency publications were reviewed to supplement peer-reviewed evidence. Articles were screened for relevance to temperature-related vascular effects and mechanisms of thrombogenesis. Priority was given to studies providing mechanistic insights, quantifiable risk estimates, or climate-sensitive health outcomes. Extracted data were synthesized thematically, focusing on mechanistic pathways, epidemiological associations, vulnerable populations, and medical implications. Given the narrative design, no formal meta-analysis or statistical pooling was performed. Instead, a qualitative integration of findings was used to identify convergent evidence, highlight research gaps, and construct an interdisciplinary understanding of how global warming influences thrombotic risk.

Mechanisms Linking Global Warming to Vascular Thrombosis

The relationship between global warming and vascular thrombosis is multifactorial, involving physiological, biochemical, and systemic pathways. Rising global temperatures, recurrent heat waves, worsening air pollution, and climate-related disasters all converge to create a pro-thrombotic environment. The mechanistic links can be understood by examining their impact on hemoconcentration, endothelial function, inflammation, coagulation cascades, platelet activity, and healthcare disruptions [14-15].

1. Heat Stress and Hemoconcentration

High ambient temperatures increase sweating and fluid loss, often leading to dehydration. This reduces plasma volume, elevates hematocrit, and thickens the blood, resulting in hemoconcentration. Increased blood viscosity slows venous return, fostering stasis—a key component of Virchow’s triad. Hemoconcentration also elevates shear stress in arteries, predisposing to endothelial injury and plaque rupture, which can trigger arterial thrombosis such as myocardial infarction or stroke [16].

2. Endothelial Dysfunction

Endothelial cells are central to vascular homeostasis, regulating vasodilation, anticoagulant activity, and platelet adhesion. Global warming contributes to oxidative stress, primarily through heat exposure, ozone formation, and particulate matter pollution. Reactive oxygen species (ROS) generated under these conditions impair nitric oxide bioavailability, disrupt vascular tone, and increase endothelial permeability. Dysfunctional endothelium expresses higher levels of adhesion molecules and tissue factor, tipping the hemostatic balance toward thrombosis [17-18].

3. Systemic Inflammation

Climate change is associated with higher levels of environmental allergens, wildfire smoke, and fine particulate matter, all of which induce systemic inflammation. Pro-inflammatory cytokines such as IL-6, TNF- α , and IL-1 β enhance tissue factor expression and stimulate hepatic synthesis of fibrinogen and clotting factors. This inflammatory milieu activates leukocytes, increases neutrophil extracellular trap (NET) formation, and propagates a hypercoagulable state. Thus, systemic inflammation acts as a major link between environmental stressors and thrombus development [19].

4. Coagulation Cascade Activation

Heat-induced stress and inflammatory mediators converge on the coagulation system. Elevated fibrinogen levels, increased factor VIII activity, and suppressed fibrinolysis via plasminogen activator inhibitor-1 (PAI-1) contribute to a pro-thrombotic profile. Studies have demonstrated that heat waves correlate with increased hospital admissions for venous thromboembolism, suggesting that coagulation system activation is a critical mediator of climate-associated thrombosis [20].

5. Platelet Hyperreactivity

Air pollution and oxidative stress enhance platelet activation and aggregation. Ultrafine particles inhaled from polluted air can translocate into circulation, directly interacting with platelets and endothelial cells. Activated platelets release thromboxane A₂, serotonin, and microparticles, all of which reinforce clot formation. This platelet-driven hyperreactivity is particularly concerning in individuals with pre-existing atherosclerosis, where plaque rupture combined with platelet aggregation precipitates arterial thrombosis [21].

6. Circadian and Behavioral Influences

Global warming alters human activity patterns, sleep quality, and circadian rhythms. Disrupted circadian regulation affects hemostasis, as clotting

factor levels and platelet activity are modulated by circadian cycles. Additionally, behavioral responses such as reduced mobility during heat waves increase venous stasis, while reliance on air-conditioned indoor environments may limit physical activity, further compounding thrombotic risk [22].

7. Healthcare Disruptions During Extreme Events

Climate-related disasters disrupt healthcare delivery, particularly for patients requiring long-term anticoagulation. Interruptions in warfarin or direct oral anticoagulant supply can precipitate recurrent thrombotic events. Displacement during floods or wildfires often results in immobility and inadequate hydration, both of which accelerate thrombus formation. These indirect effects highlight how systemic consequences of global warming can magnify thrombotic risks [23].

8. Genetic and Epigenetic Modulation

Emerging evidence suggests that environmental stressors associated with global warming may influence gene expression and epigenetic regulation of coagulation pathways. Heat shock proteins (HSPs), induced during heat stress, modulate endothelial survival and coagulation factor expression. Chronic exposure to pollutants may also alter DNA methylation patterns in genes regulating thrombosis, creating long-term susceptibility (Table 1) [24].

Table 1: Mechanisms Linking Global Warming to Vascular Thrombosis

Mechanism	Climate Factor Involved	Pathophysiological Impact	Clinical Implication
Hemoconcentration	Heatwaves, dehydration	Reduced plasma volume increases blood viscosity and platelet aggregation	Elevated risk of venous thromboembolism (VTE) and stroke
Endothelial dysfunction	Heat stress, air pollution, oxidative stress	Impaired nitric oxide signaling, vascular injury	Promotes thrombosis and atherosclerotic plaque rupture
Systemic inflammation	Wildfire smoke, ozone, particulate matter	Cytokine release (IL-6, TNF- α), leukocyte activation	Enhances coagulation cascade, increasing arterial thrombosis risk
Hypercoagulability	Prolonged immobility during extreme weather events	Elevated fibrinogen, D-dimer, tissue factor expression	Higher incidence of deep vein thrombosis (DVT)
Autonomic imbalance	Rapid temperature fluctuations	Sympathetic overactivation, vasoconstriction	Triggers acute coronary syndromes and ischemic stroke
Hypoxia	Heat-induced cardiorespiratory stress, pollution	Increased erythropoietin, red cell mass	Heightened clotting tendency in susceptible individuals

Vulnerable Populations

The impact of global warming on vascular thrombosis is not evenly distributed across societies. Certain populations are more susceptible to the combined effects of heat stress, dehydration, systemic inflammation, and healthcare disruptions, making them disproportionately vulnerable to thrombotic events. Identifying these groups is essential for targeted interventions and adaptive strategies [25].

1. Elderly Individuals

Older adults are among the most affected populations due to impaired thermoregulation, reduced thirst perception, and diminished renal concentrating ability. These physiological limitations make them more prone to dehydration and hemoconcentration during heat waves. Age-related endothelial dysfunction, arterial stiffness, and increased prevalence of comorbidities such as hypertension and diabetes further amplify their thrombotic risk. Epidemiological studies have

consistently shown higher mortality from cardiovascular causes during heat waves in older adults, underscoring their heightened vulnerability [26].

2. Patients with Cardiovascular and Metabolic Disorders

Individuals with pre-existing cardiovascular disease, diabetes, obesity, or chronic kidney disease already live with baseline endothelial injury and pro-thrombotic tendencies. Global warming worsens these conditions through oxidative stress, poor glycemic control, and heightened inflammation. Obesity is particularly concerning, as heat intolerance and restricted mobility exacerbate venous stasis. Diabetic patients are also at risk of impaired hydration due to osmotic diuresis, compounding the effects of heat-related hemoconcentration [27].

3. Cancer Patients

Cancer itself is a well-established pro-thrombotic condition, and treatments such as chemotherapy,

hormonal therapy, and targeted agents further increase coagulation activation. Global warming heightens these risks by worsening dehydration and restricting mobility during extreme weather events. Moreover, interruptions in cancer care during climate-related disasters may delay thromboprophylaxis or anticoagulation management, leaving patients more susceptible to thrombotic complications [28].

4. Pregnant and Postpartum Women

Pregnancy is a natural hypercoagulable state, evolved to prevent excessive bleeding during delivery. However, in the context of global warming, physiological adaptations can turn hazardous. Dehydration during pregnancy worsens blood viscosity, while reduced mobility due to heat exposure or displacement during disasters can promote venous stasis. Postpartum women are also at higher risk, especially if exposed to healthcare disruptions or poor hydration during recovery [29-30].

5. Children and Infants

Although vascular thrombosis is less common in younger populations, children and infants are not exempt. They are highly vulnerable to heat-related dehydration due to higher surface area-to-body mass ratios and limited ability to regulate temperature. Hospitalized children, particularly

those with congenital heart disease, sickle cell anemia, or cancer, face additional thrombotic risks when exposed to climate stressors [31].

6. Socioeconomically Disadvantaged Populations

Low-income populations often lack access to cooling systems, safe drinking water, and healthcare infrastructure, leaving them highly vulnerable to climate-related thrombosis. Displacement during floods, droughts, or wildfires is more common in these groups, leading to immobility and poor access to anticoagulants. Additionally, malnutrition and chronic infections prevalent in resource-limited settings may interact with climate stress to exacerbate pro-thrombotic conditions [31].

7. Outdoor Workers and Migrant Populations

Individuals who work outdoors—such as agricultural laborers, construction workers, and migrant populations—face prolonged exposure to heat stress, dehydration, and air pollution. Long working hours in high temperatures predispose them to venous stasis, while poor access to healthcare or protective policies amplifies risk. Migrant populations displaced by climate change also experience unstable healthcare access, increasing susceptibility to untreated thrombotic events (Table 2) [32].

Table 2: Vulnerable Populations and Preventive Strategies

Vulnerable Group	Risk Factors	Examples of Climate-Related Risks	Recommended Strategies
Elderly individuals	Age-related endothelial dysfunction, reduced thermoregulation	Dehydration, impaired cardiovascular compensation during heatwaves	Hydration programs, early warning systems, cooling centers
Patients with cardiovascular disease	Pre-existing atherosclerosis, anticoagulant use	Higher risk of myocardial infarction and stroke during heat stress	Medication review, remote monitoring, climate-adapted care plans
Cancer patients	Hypercoagulable state, immobility, chemotherapy effects	Increased risk of VTE during extreme heat or displacement	Prophylactic anticoagulation, hospital readiness during climate crises
Pregnant women	Physiological	DVT and preeclampsia	Prenatal care with hydration

	hypercoagulability, increased fluid demands	exacerbated by dehydration	focus, close monitoring in hot climates
Low-resource populations	Poor access to healthcare, limited infrastructure	High exposure to polluted environments, delayed treatment	Community health interventions, mobile clinics, policy-driven equity measures
Outdoor workers	Prolonged heat and pollution exposure	Increased dehydration, oxidative stress, thrombosis risk	Protective policies, hydration breaks, workplace safety regulations

Potential Biomarkers and Monitoring

The intersection of global warming and vascular thrombosis highlights the need for early detection tools that can identify individuals at elevated risk before clinical events occur. Biomarkers provide valuable insight into coagulation, inflammation, and endothelial integrity, offering a means to track thrombotic risk in populations exposed to climate stressors. In addition, innovative monitoring technologies can bridge the gap between environmental exposure and clinical management, facilitating timely interventions [33].

1. Traditional Coagulation Biomarkers

Conventional markers of coagulation activity remain fundamental in assessing thrombotic risk. D-dimer, a fibrin degradation product, is widely used as an indicator of fibrin turnover and is elevated in venous thromboembolism. Heat stress, dehydration, and systemic inflammation may drive persistent elevations in D-dimer, making it a sensitive though non-specific marker. Prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR) can also provide indirect measures of coagulation dynamics, particularly useful in patients receiving anticoagulation whose therapy may be interrupted during climate-related disasters [34].

2. Inflammatory Biomarkers

Given the strong role of systemic inflammation in climate-induced thrombosis, C-reactive protein (CRP) and interleukin-6 (IL-6) serve as important markers of vascular risk. Elevated CRP correlates

with endothelial activation and pro-thrombotic states, while IL-6 drives hepatic synthesis of fibrinogen and coagulation factors. Tumor necrosis factor-alpha (TNF- α) and interleukin-1 β (IL-1 β), though less commonly used clinically, are emerging as potential markers of climate-related vascular inflammation [35].

3. Endothelial Dysfunction Biomarkers

Endothelial health is a central determinant of thrombosis risk. Biomarkers such as von Willebrand factor (vWF) and soluble thrombomodulin reflect endothelial activation and injury. Circulating endothelial cells (CECs) and endothelial microparticles (EMPs) are more advanced indicators of endothelial stress, with evidence suggesting that their levels increase in response to oxidative damage from heat and air pollution. Monitoring these markers in populations exposed to recurrent climate stressors may help identify subclinical vascular injury before thrombotic events occur [36].

4. Platelet Activation Biomarkers

Platelet reactivity plays a pivotal role in climate-related thrombosis. Biomarkers such as soluble P-selectin, platelet factor 4 (PF4), and platelet microparticles reflect heightened platelet activation and aggregation. Flow cytometry-based assays measuring glycoprotein IIb/IIIa or P-selectin expression can provide additional insights into platelet activation status, though these remain largely confined to research settings [37].

5. Oxidative Stress and Heat Stress Markers

Global warming contributes significantly to oxidative imbalance. Malondialdehyde

(MDA) and 8-isoprostane serve as markers of lipid peroxidation, while heat shock proteins (HSPs) indicate cellular stress responses to elevated temperatures. These markers may not be specific to thrombosis but can complement coagulation and endothelial biomarkers in climate-sensitive risk assessments [38].

6. Novel and Emerging Biomarkers

Advances in omics technologies have identified potential novel markers for climate-related thrombosis. Transcriptomic studies suggest that climate stress can alter the expression of coagulation-related genes, while metabolomic profiling reveals changes in amino acids and lipids linked to thrombotic states. Epigenetic modifications, such as DNA methylation patterns in pro-coagulant genes, are being investigated as long-term indicators of environmental stress exposure [39].

7. Monitoring Approaches

In addition to biomarkers, innovative monitoring strategies are needed for effective prevention. Wearable sensors capable of tracking hydration status, body temperature, and heart rate variability can provide early warning signs of heat stress and dehydration—conditions that precede thrombosis. Integration of point-of-care testing (POCT) for D-dimer or CRP in community and disaster settings may allow rapid risk stratification. Furthermore, the use of telemedicine platforms can ensure continuity of anticoagulation monitoring during climate-related healthcare disruptions, reducing the risk of preventable thrombotic events [40].

Preventive and Adaptive Strategies

Addressing the rising burden of vascular thrombosis in the era of global warming requires a dual approach: clinical prevention to mitigate direct thrombotic risks, and adaptive public health strategies to build resilience against climate-induced health stressors. These measures must operate at the individual, community, healthcare system, and policy levels to be effective [41].

1. Individual-Level Strategies

Individuals can significantly reduce climate-related thrombotic risk by adopting preventive behaviors during extreme heat and environmental stress. Maintaining adequate hydration is critical to counteract hemoconcentration, particularly for the elderly and those with pre-existing cardiovascular conditions. Avoiding prolonged immobility during heat waves or natural disasters, wearing breathable clothing, and seeking cooler environments reduce venous stasis. For patients on long-term anticoagulation therapy, adherence and contingency planning for medication continuity during climate disruptions are essential. Education on early warning signs of thrombosis—such as limb swelling, chest pain, or sudden neurological deficits—empowers individuals to seek timely medical attention [42].

2. Clinical Strategies

Healthcare providers must integrate climate-sensitive risk factors into routine cardiovascular and hematology practice. Physicians should adjust anticoagulation regimens for patients exposed to extreme weather conditions, particularly those at high thrombotic risk. Prophylactic low-dose anticoagulation or antiplatelet therapy may be considered for vulnerable patients during prolonged immobility, such as disaster displacement. Regular biomarker monitoring—D-dimer, fibrinogen, CRP, and endothelial markers—can guide individualized management. Furthermore, educating patients about hydration, temperature regulation, and mobility during heat stress should become part of clinical counseling, particularly in high-risk populations [43].

3. Community-Level Strategies

Community adaptation measures play a crucial role in reducing exposure to climate stressors. Establishing cooling centers during heat waves, ensuring widespread access to potable water, and modifying work schedules for outdoor laborers are practical interventions. Community awareness programs should highlight the link between heat exposure, dehydration, and thrombotic events,

particularly for elderly populations living alone. Local healthcare facilities should be equipped with point-of-care diagnostic tools for rapid thrombotic risk assessment during emergencies [44].

4. Healthcare System Adaptation

Hospitals and clinics must strengthen climate resilience to maintain continuity of care during disasters. Ensuring reliable supply chains for anticoagulants and essential cardiovascular medications is paramount. Digital health solutions such as telemedicine and remote monitoring platforms can sustain anticoagulation management when physical access is disrupted. In disaster-prone regions, healthcare systems should establish mobile clinics or field units capable of delivering urgent thromboembolic care, including anticoagulation and thrombolysis, to displaced populations [45].

5. Policy-Level Interventions

Policy frameworks must incorporate vascular health into broader climate adaptation plans. Governments should enforce stricter air quality regulations to reduce particulate matter and ozone levels, which exacerbate vascular inflammation. Urban planning that increases green spaces and reduces heat islands can directly decrease heat-related thrombotic risks. Climate-adaptive occupational health policies, including mandated rest breaks, hydration schedules, and protective infrastructure for outdoor workers, are essential. In addition, governments should prioritize equitable healthcare access, ensuring that socioeconomically disadvantaged groups—often the most vulnerable—are not excluded from climate resilience measures [46].

6. Research and Innovation

Preventive strategies must be underpinned by ongoing research to clarify the molecular links between climate stressors and thrombosis. Development of novel biomarkers for climate-induced vascular injury, wearable hydration and coagulation monitors, and predictive models that integrate meteorological data with health

surveillance will enable precision prevention. Clinical trials evaluating prophylactic therapies during extreme heat or environmental stress can inform evidence-based guidelines [47].

7. Global and Multisectoral Collaboration

Because climate change is a transboundary challenge, international collaboration is critical. Multisectoral partnerships between climatologists, hematologists, cardiologists, and public health experts can foster integrated approaches to prevention. Global health organizations, including the WHO, should incorporate vascular thrombosis into climate-health frameworks, ensuring funding and policy alignment across nations. Collaboration with non-health sectors—such as urban planning, water management, and disaster response—will be essential in creating sustainable adaptation strategies [48].

Mapping of Global Warming and Vascular Thrombosis Pathways

Global warming initiates a complex chain of events that culminate in vascular thrombosis, acting through interconnected environmental, physiological, and molecular mechanisms. Rising ambient temperatures and more frequent heatwaves accelerate fluid loss, leading to dehydration and hemoconcentration, which in turn increase blood viscosity and platelet aggregation. This hyperviscous state provides the first trigger for venous thromboembolism [49]. Simultaneously, global warming fuels air pollution through increased ozone formation, wildfire smoke, and particulate matter release. These pollutants provoke oxidative stress and systemic inflammation, impairing endothelial function, reducing nitric oxide availability, and exposing subendothelial collagen that enhances platelet adhesion and activation. This endothelial dysfunction forms a central pathway connecting climate stress to thrombosis [50].

Another physiological stressor is autonomic imbalance, driven by rapid temperature fluctuations that trigger sympathetic nervous

system overactivation. The resultant vasoconstriction and increased blood pressure precipitate acute vascular events such as myocardial infarction and ischemic stroke. In addition, hypoxia secondary to heat-induced cardiorespiratory strain or pollution-related lung injury elevates erythropoietin and red cell mass, creating a prothrombotic milieu [51]. When combined, these physiological stressors converge on thrombosis pathways: increased coagulation factor activity, heightened platelet reactivity, and impaired fibrinolysis. Elevated biomarkers such

as D-dimer, fibrinogen, IL-6, and TNF- α reflect this heightened thrombotic risk [52]. The final outcome is an increased incidence of clinical thrombotic events, including deep vein thrombosis, pulmonary embolism, ischemic stroke, and acute coronary syndromes. These events disproportionately affect vulnerable populations such as the elderly, individuals with pre-existing cardiovascular disease, cancer patients, pregnant women, and socioeconomically disadvantaged groups (Figure 1) [53].

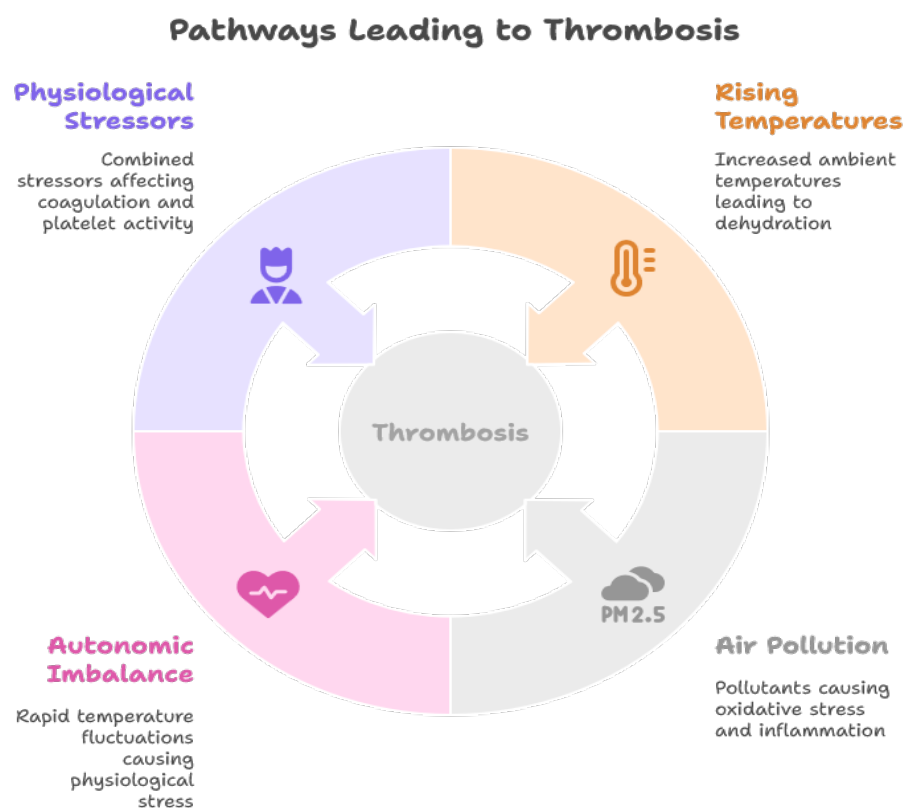


Figure 1: Mapping of Global Warming and Vascular Thrombosis Pathways

Future Directions

The relationship between global warming and vascular thrombosis is an emerging field that requires extensive exploration to strengthen causal evidence, refine clinical strategies, and guide policy development. While current knowledge suggests strong mechanistic links,

future research and global collaboration are essential to address unanswered questions and mitigate risks effectively [54].

1. Strengthening Epidemiological Evidence

Large-scale, longitudinal, and geographically diverse studies are needed to establish the direct associations between climate variables—such as

heat waves, air pollution, and extreme weather events—and thrombotic outcomes. Standardized data collection integrating meteorological information with hospital admissions for venous thromboembolism, stroke, and myocardial infarction will help define population-specific risks. Future studies should also investigate seasonal and regional variations in thrombosis rates to better understand the climate-health nexus [55].

2. Mechanistic and Molecular Research

Mechanistic research at the cellular and molecular level remains limited. Further studies should investigate how climate stressors alter endothelial biology, platelet function, and coagulation cascades. The role of oxidative stress, heat shock proteins, and neutrophil extracellular traps (NETs) in mediating thrombosis under environmental stress warrants special focus. Omics-based approaches—genomics, proteomics, metabolomics, and epigenomics—offer opportunities to discover novel biomarkers and therapeutic targets for climate-induced vascular injury [56].

3. Development of Predictive Tools

Future work should prioritize predictive models that integrate climate data with clinical risk factors. Machine learning and artificial intelligence (AI) can be harnessed to create climate-thrombosis risk maps, identifying populations at greatest risk during extreme events. Personalized prediction tools incorporating biomarkers, wearable sensor data (hydration status, body temperature, activity levels), and environmental exposure information could transform preventive care [57].

4. Innovation in Monitoring and Prevention

Advances in point-of-care diagnostics and wearable technologies offer promising avenues for early detection and prevention. Development of portable devices capable of monitoring coagulation markers, hydration status, and cardiovascular stress in real time would be

invaluable during heat waves and disaster displacement. Preventive trials testing hydration protocols, anticoagulation adjustments, and climate-sensitive lifestyle interventions could generate evidence-based guidelines for clinical practice [58].

5. Climate-Resilient Healthcare Systems

Research must also address health system resilience. Identifying strategies to maintain continuity of anticoagulation therapy during climate-related disasters is a pressing priority. Implementation studies should evaluate how digital health platforms, mobile clinics, and decentralized diagnostic tools can sustain care delivery for thrombotic patients during healthcare disruptions. Lessons learned from disaster medicine and pandemic response can inform future preparedness for climate-related thrombotic challenges [59].

6. Policy Integration and Global Collaboration

Future directions extend beyond biomedical research to include public health and policy. Thrombosis should be incorporated into climate-health frameworks of international organizations such as the World Health Organization (WHO). Policy-relevant research should assess the effectiveness of interventions such as cooling centers, occupational health regulations, and air quality controls in reducing thrombotic outcomes. Multinational collaborations can ensure equitable adaptation, particularly for low- and middle-income countries that face disproportionate climate vulnerabilities [60].

7. Addressing Vulnerable Populations

Dedicated research on how climate stressors uniquely affect vulnerable groups—including the elderly, patients with chronic diseases, cancer patients, pregnant women, and low-income populations—will guide targeted preventive strategies. Culturally sensitive interventions and equity-focused policies must be developed to ensure that adaptation strategies reach those most at risk [60].

Conclusion

Global warming represents more than an environmental challenge—it is a profound health threat that increasingly intersects with vascular medicine. Mounting evidence links rising temperatures, extreme weather events, dehydration, and air pollution with a higher incidence of vascular thrombosis, including venous thromboembolism, stroke, and myocardial infarction. The pathophysiological mechanisms involve endothelial dysfunction, hypercoagulability, hemoconcentration, and systemic inflammation, all of which are amplified under climate stressors. Particularly concerning is the disproportionate impact on vulnerable groups such as the elderly, individuals with pre-existing cardiovascular disease, cancer patients, pregnant women, and communities with limited access to healthcare. This underscores the urgent need for equitable strategies that combine biomedical advances with public health and climate adaptation policies.

Emerging research into biomarkers, real-time monitoring, and predictive modeling offers promising opportunities for early detection and prevention of climate-related thrombotic events. Furthermore, preventive interventions—ranging from hydration protocols to improved disaster preparedness and resilient healthcare delivery—will be vital in mitigating risks. However, significant gaps remain in epidemiological evidence, mechanistic understanding, and the integration of vascular health into climate resilience policies. Addressing these gaps requires interdisciplinary collaboration across medicine, environmental science, data science, and policy-making, ensuring that clinical insights translate into population-level protection.

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HSPs – Heat Shock Proteins
ICAM-1 – Intercellular Adhesion Molecule-1
IL-6 – Interleukin-6
IPCC – Intergovernmental Panel on Climate Change
MI – Myocardial Infarction
NO – Nitric Oxide
PM_{2.5} – Fine Particulate Matter $\leq 2.5 \mu\text{m}$
PM₁₀ – Particulate Matter $\leq 10 \mu\text{m}$
TLRs – Toll-Like Receptors
TNF- α – Tumor Necrosis Factor-Alpha
TME – Temperature-Mediated Exposure (contextual environmental exposures)
VCAM-1 – Vascular Cell Adhesion Molecule-1
vWF – von Willebrand Factor
VTE – Venous Thromboembolism

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Abbreviations

AF – Atrial Fibrillation
CO – Carbon Monoxide
CO₂ – Carbon Dioxide
CVD – Cardiovascular Disease
DOACs – Direct Oral Anticoagulants
DVT – Deep Vein Thrombosis