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Exercise-Induced Hemostatic Changes: A Review of Acute Responses and Chronic Adaptations in Endurance Athletes.

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Abstract

Background. Endurance exercise exerts complex effects on the human hemostatic system. Acute bouts of strenuous exercise may transiently induce a prothrombotic state, while habitual endurance training is associated with improved vascular health and reduced baseline thrombotic risk.

Aim. To synthesize current evidence on acute and chronic exercise-induced hemostatic responses in endurance athletes and to evaluate their implications for venous thromboembolism (VTE) risk.

Material and methods. A narrative literature review was conducted using searches of PubMed, Scopus, Web of Science, and the Cochrane Library up to March 2026 using combinations of keywords related to hemostasis, coagulation, fibrinolysis, endurance training, and athletes.

Results. Acute endurance exercise increases platelet activation, thrombin generation, and coagulation factor activity, accompanied by parallel but often insufficient activation of fibrinolysis. In contrast, long-term endurance training promotes adaptive antithrombotic mechanisms, including improved endothelial function, enhanced fibrinolytic capacity, and reduced platelet reactivity. Factors such as genetic predisposition, dehydration, environmental stressors, travel, and age further modulate these responses and may increase thrombotic susceptibility in certain athletes. Preventive strategies, including optimized hydration, training periodization, biomarker monitoring, and targeted pharmacological prophylaxis for high-risk individuals, may mitigate exercise-associated thrombotic events.

Conclusions. Endurance exercise induces a biphasic hemostatic pattern characterized by transient procoagulant responses superimposed on long-term protective adaptations. Understanding this physiological paradox is essential for developing evidence-based recommendations that support safe participation in endurance sports.

Keywords: hemostasis, endurance athletes, venous thromboembolism, coagulation, fibrinolysis, platelet activation, endurance training

1. Introduction

Hemostasis represents a tightly regulated physiological system that preserves blood fluidity under normal conditions while enabling rapid clot formation following vascular injury. This balance is maintained through interactions between procoagulant mechanisms - such as platelet activation, thrombin generation, and fibrin formation - and anticoagulant and fibrinolytic pathways involving tissue plasminogen activator (tPA), plasmin, antithrombin, and the protein C system [1-3]. Disruption of this equilibrium toward excessive coagulation can lead to pathological thrombosis, including venous thromboembolism (VTE), which encompasses deep vein thrombosis (DVT) and pulmonary embolism (PE) [4,5].

Physical activity is widely recognized as a protective factor against cardiovascular disease and metabolic disorders in the general population. However, accumulating evidence suggests that strenuous exercise, particularly prolonged endurance activity, may transiently shift hemostatic balance toward a procoagulant state [7,8,19]. This paradox has attracted increasing interest in sports medicine and thrombosis research because endurance athletes frequently engage in physiological conditions known to influence coagulation, including dehydration, hemoconcentration, systemic inflammation, and endothelial shear stress [9-11].

Endurance athletes - including marathon runners, ultra-distance competitors, cyclists, and triathletes - represent a unique population exposed to extreme metabolic and hemodynamic demands. Prolonged exercise events lasting several hours can induce substantial plasma volume loss, elevated catecholamine concentrations, and inflammatory responses characterized by increased cytokines such as interleukin-6 (IL-6) [12-14]. These physiological stressors can activate platelets, increase coagulation factor activity, and stimulate thrombin generation. At the same time, exercise also stimulates fibrinolytic activity, particularly through enhanced endothelial release of tPA [15].

Several clinical and experimental studies have attempted to quantify these responses. For example, investigations involving marathon runners demonstrated significant post-race increases in markers of thrombin generation and platelet activation, suggesting that prolonged endurance exercise may transiently enhance coagulation potential [7,9,19]. Conversely, longitudinal studies examining trained endurance athletes indicate that habitual aerobic exercise is associated with enhanced fibrinolytic activity and improved endothelial function at rest [18,27].

The net effect of these opposing processes varies depending on exercise intensity, duration, training status, and individual susceptibility. Epidemiological observations indicate that although overall cardiovascular risk is lower among physically active individuals, isolated cases of exercise-associated VTE have been reported in endurance athletes, particularly after prolonged competitions combined with dehydration, travel, or inherited thrombophilia [6,16,17].

Current evidence therefore suggests a bimodal or biphasic model of exercise-induced hemostatic responses. Acute bouts of strenuous exercise tend to induce transient hypercoagulability, whereas chronic endurance training promotes long-term antithrombotic adaptations including improved endothelial function, reduced platelet reactivity, and enhanced fibrinolytic capacity [8,18-20].

The aim of this review is to provide a comprehensive synthesis of current knowledge regarding the acute and chronic effects of endurance exercise on the hemostatic system. Particular emphasis is placed on mechanisms underlying exercise-induced coagulation activation, long-term adaptive responses to endurance training, and clinical implications for thrombotic risk management in athletes.

2. Materials and Methods

This study was conducted as a narrative literature review aimed at synthesizing current evidence on exercise-induced hemostatic responses in endurance athletes.

2.1 Literature Search Strategy

Electronic databases including PubMed, Scopus, Web of Science, and the Cochrane Library were searched from database inception until March 2026. The search strategy combined Medical Subject Headings (MeSH) and free-text terms related to hemostasis and endurance exercise. Core search terms included: ("hemostasis" OR "coagulation" OR "fibrinolysis" OR "thrombosis" OR "platelet activation" OR "venous thromboembolism") AND ("exercise" OR "endurance training" OR "aerobic exercise" OR "marathon" OR "triathlon" OR "ultra-endurance") AND ("athletes" OR "sports" OR "runners").

2.2 Study Selection

Studies were included if they:

- investigated hemostatic, coagulation, or fibrinolytic responses to exercise
- included endurance athletes or endurance-type exercise protocols
- reported biomarkers such as platelet activation, coagulation factors, thrombin markers, D-dimer, or fibrinolytic indicators.

Both experimental studies and observational cohort studies were considered. Relevant review articles were screened to identify additional primary literature. In total, several dozen studies investigating exercise-induced hemostatic responses were reviewed, with particular focus on controlled exercise experiments and investigations involving endurance competitions such as marathons or ultra-endurance races.

3. Results

3.1 Physiological Foundations of Hemostasis

Hemostasis involves two interconnected processes: primary hemostasis and secondary hemostasis. Primary hemostasis is initiated by platelet adhesion to exposed subendothelial collagen through von Willebrand factor, followed by platelet activation and aggregation mediated by ADP, thromboxane A2, and fibrinogen bridges [1,2]. Secondary hemostasis involves activation of the coagulation cascade through intrinsic and extrinsic pathways that ultimately lead to thrombin generation and conversion of fibrinogen to fibrin [3].

Thrombin serves as the central enzyme within the coagulation cascade, amplifying clot formation through activation of multiple coagulation factors and stabilization of fibrin networks via factor XIII cross-linking. In parallel, anticoagulant mechanisms-including antithrombin, tissue factor pathway inhibitor (TFPI), and the protein C system-limit excessive clot propagation [4].

The fibrinolytic system provides an additional regulatory mechanism that removes fibrin deposits and restores vascular patency. Endothelial release of tissue plasminogen activator converts plasminogen into plasmin, which degrades fibrin into soluble fragments such as D-dimer. Plasminogen activator inhibitor-1 (PAI-1) regulates this process by inhibiting tPA activity, thereby maintaining a controlled balance between clot formation and clot dissolution [15].

3.2 Evidence from Exercise Studies

Several experimental and observational studies have evaluated changes in hemostatic biomarkers following endurance exercise. Rock et al. [23] investigated hematological responses in marathon runners and reported significant increases in coagulation factors and fibrinolytic markers immediately after competition. Their findings suggested that endurance events activate both coagulation and fibrinolysis simultaneously, reflecting a dynamic regulatory response of the hemostatic system.

Similarly, Lippi et al. [7] analyzed platelet activation and coagulation responses in endurance athletes and demonstrated increased expression of platelet activation markers, including P-selectin, following prolonged endurance exercise. These findings support the hypothesis that exercise-induced sympathetic activation and elevated shear stress contribute to platelet activation during strenuous physical activity.

Schobersberger et al. [8] reported significant increases in thrombin-antithrombin complexes (TAT) and other coagulation markers following long-distance trail running competitions.

Hilberg et al. [18] demonstrated that endurance training enhances fibrinolytic capacity through increased endothelial release of tissue plasminogen activator.

3.3 Acute Effects of Exercise on Hemostasis

Acute bouts of strenuous endurance exercise induce measurable activation of several components of the hemostatic system. Circulating platelet counts increase due to splenic contraction and catecholamine-mediated mobilization of platelets. Studies conducted in marathon runners and triathlon athletes have reported increases in platelet counts of approximately 15-25% immediately after competition, accompanied by elevated expression of platelet activation markers such as P-selectin and glycoprotein IIb/IIIa activation [7,9,24]. Several studies have reported substantial post-exercise increases in trombin-antitrombin complexes and D-dimer concentrations following marathon events [7,9].

Exercise duration appears to be a critical determinant of the magnitude of hemostatic activation. Short exercise bouts lasting less than one hour generally produce mild and rapidly reversible changes [7,19]. Experimental studies comparing different exercise intensities indicate that high-intensity interval training may induce transient activation of coagulation and platelet aggregation similar to that observed after moderate continuous exercise, although these responses typically normalize within several hours after recovery [22]. In contrast, prolonged efforts exceeding 2-4 hours - such as marathon or Ironman segments - can sustain elevations of thrombin generation markers for several hours [7,23]. Some studies report that TAT concentrations may remain elevated for 4-6 hours after full marathon events [19]. These prolonged responses may be partially explained by metabolic acidosis associated with lactate accumulation, which lowers blood pH and enhances factor Xa activity, as well as by cytokine release such as interleukin-6 that stimulates plasminogen activator inhibitor-1 (PAI-1) production.

Hydration status also strongly modifies hemostatic responses to exercise. Even moderate dehydration corresponding to reduction in body mass may concentrate coagulation factors by approximately 15-20%, increase blood viscosity, and reduce circulating antithrombin levels [9,10]. Athletes experiencing substantial dehydration during endurance events demonstrate significantly higher D-dimer and factor VIII levels compared with adequately hydrated participants [9,10]. Controlled hydration strategies appear capable of attenuating these responses, with some reports indicating that oral rehydration during endurance events reduces post-exercise fibrinolytic activation by nearly half.

Environmental and demographic variables further influence the magnitude of acute hemostatic responses. Age and sex are another relevant factors.

Classic studies conducted between the late 1990s and early 2010s consistently demonstrate that endurance exercise produces duration-dependent increases in coagulation factors such as FVII and FVIII. Importantly, well-trained athletes tend to recover from these transient prothrombotic changes approximately twice as fast as untrained individuals, suggesting that repeated exposure to endurance exercise promotes adaptive regulatory mechanisms [23]

3.4 Chronic Hemostatic Adaptations to Endurance Training

While acute bouts of endurance exercise transiently activate coagulation pathways, long-term training produces several protective adaptations within the hemostatic system. Regular aerobic training enhances endothelial function primarily through increased nitric oxide (NO) bioavailability. Studies examining trained endurance athletes demonstrate greater endothelial-dependent vasodilation compared with sedentary controls, reflecting improved vascular reactivity and reduced platelet adhesion [25].

Chronic endurance training is also associated with enhanced fibrinolytic potential and structural modifications of fibrin clot properties observed in habitual runners [18,26]. Hilberg and colleagues reported that physically trained individuals exhibit significantly higher resting tissue plasminogen activator (tPA) activity and lower baseline plasminogen activator inhibitor-1 (PAI-1) concentrations compared with sedentary populations [18]. These changes facilitate more rapid fibrin clot degradation and may partially explain the lower baseline thrombotic risk observed in habitually active individuals.

Platelet function is also modified by repeated endurance exercise. Cross-sectional studies comparing trained athletes with sedentary controls demonstrate reductions in platelet aggregability ranging from 10-25%, particularly in response to ADP and collagen stimulation [21,27].These adaptations are believed to result from repeated exposure to increased shear stress and enhanced endothelial signaling, which collectively suppress platelet activation pathways.

Recent studies also suggest that habitual endurance training may influence the structural properties of fibrin clots. Analyses conducted in recreational runners indicate that regular physical activity can modify clot microstructure and mechanical stability, potentially contributing to improved fibrinolytic efficiency and reduced thrombotic risk [26].

Long-term endurance training also influences systemic inflammatory status. Regular exercise has been associated with reductions in circulating inflammatory markers such as C-reactive protein (CRP) and interleukin-6 under resting conditions [12,13]. Because inflammatory mediators can stimulate procoagulant pathways and PAI-1 expression, this anti-inflammatory effect contributes indirectly to improved hemostatic balance.

Another important adaptation concerns recovery kinetics after acute exercise. Trained endurance athletes typically return to baseline hemostatic parameters more rapidly than untrained individuals. Some studies report normalization of thrombin-antithrombin complexes and fibrinolytic markers within 12-24 hours in trained athletes, whereas sedentary individuals may require up to 48 hours for full recovery after comparable exercise stress [18,19]. However, these adaptations are partially reversible. Periods of detraining lasting 3-6 weeks have been shown to reduce endothelial nitric oxide availability and increase platelet reactivity, suggesting that continuous training stimulus is necessary to maintain the protective hemostatic phenotype [11].

3.5 Risk Factors and Modifiers in Endurance Athletes

Despite the overall protective cardiovascular profile associated with regular endurance exercise, several factors may increase thrombotic susceptibility in athletes. Genetic thrombophilias represent one of the most important non-modifiable risk factors. For example, individuals carrying the Factor

V Leiden mutation demonstrate a three- to seven-fold increased lifetime risk of venous thromboembolism compared with the general population [29]. When combined with physiological stressors such as dehydration or prolonged immobilization during travel, this predisposition may substantially increase thrombotic risk.

Environmental stressors may further amplify these responses. High temperatures (>30°C) can increase sweat-induced fluid losses and exacerbate hemoconcentration, while exposure to altitude (>2000 m) may stimulate hypoxia-related pathways that increase hemoglobin and hematocrit levels after acclimatization. These physiological changes may contribute to increased blood viscosity and procoagulant activity during endurance exercise performed in extreme environments [24].

Overall, the interaction between exercise-induced hemostatic activation and individual risk factors determines whether endurance exercise produces beneficial physiological adaptation or transiently increases thrombotic susceptibility.

Although endurance athletes typically demonstrate favorable cardiovascular profiles, certain physiological and environmental conditions may increase their susceptibility to thrombotic events. One of the most important modifiers is genetic predisposition. Inherited thrombophilias such as Factor V Leiden mutation, prothrombin G20210A mutation, and deficiencies in natural anticoagulants including protein C, protein S, or antithrombin can significantly elevate baseline thrombotic risk. When combined with exercise-induced hemoconcentration and endothelial activation, these genetic factors may further amplify hypercoagulability [17,30].

Dehydration represents another important contributor to thrombotic risk in endurance sports. Prolonged exercise, particularly in hot environments, can result in substantial fluid loss through sweating. Reduced plasma volume leads to increased hematocrit, higher blood viscosity, and concentration of coagulation proteins. These changes promote platelet interactions and may facilitate clot formation under certain conditions.

Travel associated with major endurance events may also play a role in thrombotic risk. Long periods of immobility during flights or car travel can contribute to venous stasis in the lower extremities. When combined with dehydration and post-exercise inflammatory responses, these factors may create conditions favorable for venous thrombosis. For this reason, athletes traveling long distances to participate in competitions may require specific preventive strategies such as maintaining hydration and periodic movement during travel [16,17]. These mechanisms correspond with the classical Virchow triad describing thrombosis development through venous stasis, endothelial injury, and hypercoagulability.

Age-related changes in hemostatic regulation may represent another modifier. Masters athletes often maintain high levels of physical fitness but may exhibit age-associated alterations in coagulation factor concentrations and endothelial function. They often demonstrate moderately elevated baseline levels of coagulation factors such as fibrinogen and factor VIII. Age-related endothelial stiffening and reduced nitric oxide bioavailability may also prolong recovery from exercise-induced coagulation activation. Some studies suggest that older athletes may experience prolonged post-exercise coagulation activation compared with younger individuals, potentially increasing transient thrombotic risk during recovery periods. [11,26]

Hormonal factors may also influence hemostatic responses in female athletes. Use of combined oral contraceptives is associated with increased levels of certain coagulation factors and reduced anticoagulant activity, which can elevate the risk of venous thromboembolism. When combined with intense endurance exercise, this procoagulant tendency may warrant careful clinical evaluation and individualized risk assessment [30].

3.6 Preventive Strategies and Clinical Recommendations

Given the complex interaction between exercise, environmental conditions, and individual risk factors, preventive strategies aimed at reducing thrombotic risk in endurance athletes are increasingly discussed in the sports medicine literature [6,19]. One of the most fundamental approaches involves maintaining adequate hydration before, during, and after prolonged exercise. Proper fluid intake helps preserve plasma volume, reduces hemoconcentration, and supports normal circulation [7,20]

Training structure and recovery strategies also play an important role. Gradual progression of training intensity and duration allows the cardiovascular and hemostatic systems to adapt to repeated physiological stress. Adequate recovery between intense training sessions or competitions may help minimize cumulative endothelial stress and systemic inflammation [18,11]

Another emerging approach involves monitoring hemostatic biomarkers in athletes who participate in extreme endurance events. Measurements of markers such as D-dimer, thrombin-antithrombin complexes, platelet activation indicators, and fibrinolytic proteins may help identify individuals who demonstrate exaggerated coagulation responses to exercise. Although routine screening is not currently recommended for all athletes, targeted monitoring may be beneficial for those with known thrombophilia or a history of thrombotic events [6,19].

In selected high-risk situations, temporary pharmacological prophylaxis may be considered under medical supervision. For example, athletes with prior venous thromboembolism or confirmed hereditary thrombophilia may require individualized preventive strategies during periods of prolonged immobility, major competitions, or travel. Such approaches must carefully balance the benefits of thrombosis prevention with the potential risk of bleeding associated with anticoagulant therapy [16,17].

Importantly, current evidence suggests that appropriately supervised physical activity after a previous episode of venous thromboembolism is generally safe and may even support recovery and vascular function, provided that training intensity is carefully managed and medical guidance is followed [28].

Education also represents an important preventive tool. Athletes, coaches, and sports medicine professionals should be aware of symptoms associated with venous thrombosis and pulmonary embolism, including limb swelling, unexplained pain, chest discomfort, or shortness of breath. Early recognition and prompt medical evaluation are essential for preventing serious complications [6].

4. Conclusions

Endurance exercise exerts complex and sometimes paradoxical effects on the hemostatic system. Acute strenuous exercise promotes transient activation of coagulation pathways and platelet function, potentially increasing short-term thrombotic risk during and shortly after intense physical activity. At the same time, exercise also stimulates fibrinolysis and vascular responses that help maintain physiological balance within the hemostatic system.

Over the long term, regular endurance training induces a range of protective adaptations including improved endothelial function, enhanced fibrinolytic capacity, reduced platelet reactivity, and improved metabolic health. These changes collectively contribute to the well-documented cardiovascular benefits of regular physical activity.

Nevertheless, specific circumstances - including dehydration, prolonged travel, genetic predisposition, and extreme endurance events - may increase the likelihood of thrombotic complications in certain athletes. Understanding the interplay between these factors and exercise-induced hemostatic responses is therefore essential for developing effective prevention strategies.

Future research should focus on large prospective studies examining hemostatic responses across different endurance disciplines, training intensities, and athlete populations. Identification of reliable biomarkers capable of predicting thrombotic risk in athletes may allow for more individualized preventive approaches while preserving the substantial health benefits associated with endurance exercise.

Understanding this biphasic response is essential for clinicians, sports physicians, and researchers seeking to optimize athlete safety while preserving the substantial cardiovascular benefits of regular physical activity. Future research should focus on large prospective studies examining thrombotic risk across different endurance disciplines and identifying biomarkers that may enable individualized risk assessment.

Disclosures:

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