



**МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
НАЦІОНАЛЬНИЙ ФАРМАЦЕВТИЧНИЙ УНІВЕРСИТЕТ
КАФЕДРА КЛІНІЧНОЇ ЛАБОРАТОРНОЇ ДІАГНОСТИКИ,
МІКРОБІОЛОГІЇ ТА БІОЛОГІЧНОЇ ХІМІЇ**



**MINISTRY OF HEALTH OF UKRAINE
NATIONAL UNIVERSITY OF PHARMACY
DEPARTMENT OF CLINICAL LABORATORY DIAGNOSTICS,
MICROBIOLOGY AND BIOLOGICAL CHEMISTRY**



**ЗБІРНИК
публікацій
II Міжнародної науково-практичної
online конференції
«СУЧАСНІ ДОСЯГНЕННЯ ЕКСПЕРИМЕНТАЛЬНОЇ,
КЛІНІЧНОЇ, ЕКОЛОГІЧНОЇ БІОХІМІЇ ТА
МОЛЕКУЛЯРНОЇ БІОЛОГІЇ»**

**BOOK
of publications
of II International scientific and practical
online conference
"MODERN ACHIEVEMENTS OF EXPERIMENTAL,
CLINICAL, ENVIRONMENTAL BIOCHEMISTRY AND
MOLECULAR BIOLOGY"**

**07 листопада 2025 р.
м. Харків, Україна
November 07, 2025
Kharkiv, Ukraine**

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THE EFFECT OF COFFEE DRINKS ON THE BIOCHEMICAL PROCESSES OF BLOOD CLOTTING

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Abstract. Coffee is one of the most widely consumed beverages worldwide, containing a complex mixture of biologically active compounds such as caffeine, polyphenols, diterpenes, and antioxidants. While numerous studies have explored its cardiovascular and metabolic effects, its influence on the biochemical mechanisms underlying blood coagulation remains an evolving field of investigation. This article aims to analyze and summarize the biochemical interactions between coffee components and the hemostatic system, focusing on platelet function, plasma coagulation factors, fibrinolysis, and oxidative stress modulation. Both experimental and clinical evidence indicate that coffee exerts dual effects on blood clotting: moderate intake may enhance endothelial function and inhibit platelet aggregation through antioxidant and nitric oxide-mediated mechanisms, whereas excessive consumption may increase pro-coagulant activity via catecholamine stimulation and dehydration. Understanding these mechanisms is crucial for evaluating the risks and benefits of coffee consumption in individuals with thrombotic or bleeding disorders.

Key Words: Coffee, caffeine, blood coagulation, platelets, fibrinogen, hemostasis, antioxidants, cardiovascular health, polyphenols.

Introduction. Coffee has been an integral part of human culture for centuries, serving not only as a social beverage but also as a potent stimulant with multiple physiological effects. The global popularity of coffee and its inclusion in various dietary patterns have prompted scientific interest in its potential health benefits and risks. Beyond its well-known impact on alertness and metabolism, coffee has been linked to several biochemical processes affecting the cardiovascular and hematologic systems [1, 2].

Blood coagulation, or hemostasis, is a finely balanced physiological process involving vascular, platelet, and plasma components. Disruptions in this system can lead to either thrombosis (excessive clot formation) or hemorrhage (inadequate clot formation). Given that cardiovascular diseases remain the leading cause of death globally, understanding how dietary factors such as coffee influence coagulation mechanisms has significant clinical implications.

Coffee contains over one thousand chemical compounds, including caffeine, chlorogenic acids, cafestol, kahweol, trigonelline, and various polyphenols. These compounds can influence endothelial function, platelet activation, oxidative stress, and inflammatory pathways – all of which are integral to coagulation dynamics. Despite numerous epidemiological studies linking coffee consumption to cardiovascular outcomes, the direct biochemical effects of coffee on blood clotting remain partially understood and occasionally contradictory [3].

This article provides an analytical review and synthesis of current knowledge on

the biochemical impact of coffee drinks on the blood clotting process, highlighting molecular mechanisms, dose-dependent effects, and clinical implications.

The Aim of the Study. The aim of this study is to evaluate the biochemical effects of coffee drinks on the processes of blood clotting by analyzing available literature data and experimental evidence. Specific objectives include: to identify the active coffee components that influence hemostatic mechanisms; to describe the molecular interactions of these compounds with coagulation factors and platelets; to evaluate the balance between pro-coagulant and anti-coagulant effects of coffee consumption; to discuss potential health implications of regular coffee intake concerning thrombotic and bleeding risks.

Materials and Methods. This article is based on a comprehensive literature analysis using data from peer-reviewed journals and databases including PubMed, Scopus, ScienceDirect, and Google Scholar. The search terms used were “coffee and blood coagulation,” “caffeine and platelet function,” “chlorogenic acid and hemostasis,” and “coffee polyphenols and thrombosis.”

Inclusion criteria comprised studies published between 2000 and 2025 that examined biochemical, molecular, or physiological effects of coffee or its constituents on coagulation parameters in humans or experimental animals. Both in vitro and in vivo investigations were considered. Exclusion criteria included articles focused solely on epidemiological associations without biochemical data.

Data were classified according to the following parameters: Platelet function: aggregation, adhesion, activation markers (P-selectin, thromboxane A₂).

- Plasma coagulation factors: fibrinogen, prothrombin time (PT), activated partial thromboplastin time (aPTT), factor VII, and thrombin generation.
- Endothelial markers: nitric oxide (NO), prostacyclin, von Willebrand factor.
- Oxidative and inflammatory parameters: reactive oxygen species (ROS), cytokines, and antioxidant enzyme activity.

This methodological approach allows for a balanced evaluation of both mechanistic and clinical perspectives regarding coffee’s effect on hemostasis.

Results and Discussion.

Biochemical Composition of Coffee Relevant to Coagulation.

Coffee contains caffeine, a methylxanthine known for its adenosine receptor antagonism and phosphodiesterase inhibition, which enhances cyclic AMP levels in platelets and vascular cells. Polyphenolic compounds, particularly chlorogenic acids, exert antioxidant and anti-inflammatory effects. Diterpenes (cafestol and kahweol), present mainly in unfiltered coffee, influence lipid metabolism and may affect coagulation indirectly by altering plasma viscosity and fibrinogen levels [4].

Effects on Platelet Function

Several in vitro studies demonstrate that caffeine inhibits platelet aggregation induced by ADP and collagen. This inhibition is associated with increased intracellular cyclic AMP, leading to reduced calcium mobilization and decreased thromboxane A₂ synthesis. Polyphenols in coffee further contribute to platelet stabilization by scavenging reactive oxygen species that promote platelet activation.

Conversely, high caffeine doses (above 400 mg/day) can increase catecholamine release (epinephrine and norepinephrine), which enhances platelet reactivity and vascular constriction. Thus, moderate consumption (1-3 cups per day) may have anti-thrombotic effects, whereas excessive intake could shift the balance toward hypercoagulability [5].

Impact on Coagulation Factors.

Human studies suggest that regular coffee consumption slightly reduces plasma fibrinogen levels and prolongs prothrombin time, indicating a mild anticoagulant effect. The underlying mechanism may involve suppression of hepatic synthesis of fibrinogen through modulation of inflammatory cytokines (e.g., IL-6). Moreover, coffee polyphenols can chelate transition metals, reducing oxidative modification of coagulation proteins.

In contrast, unfiltered coffee, which retains diterpenes, has been reported to increase serum cholesterol and factor VII activity – both associated with enhanced clotting potential. Therefore, brewing method significantly influences coffee's coagulative effects [6].

Endothelial and Fibrinolytic Effects.

The vascular endothelium plays a central role in regulating coagulation through the secretion of nitric oxide and prostacyclin, both of which inhibit platelet aggregation. Coffee intake has been shown to improve endothelial-dependent vasodilation due to increased bioavailability of nitric oxide and reduced oxidative stress. Chlorogenic acids contribute by enhancing endothelial nitric oxide synthase (eNOS) activity.

Regarding fibrinolysis, caffeine may enhance tissue plasminogen activator (t-PA) activity, promoting clot breakdown. However, this effect appears transient and dependent on individual caffeine metabolism rates determined by CYP1A2 enzyme polymorphisms [7].

Oxidative Stress and Inflammation.

Oxidative stress is a key modulator of coagulation, promoting platelet activation and fibrin cross-linking. Coffee's high antioxidant content – mainly from polyphenols – reduces lipid peroxidation and stabilizes redox-sensitive pathways in endothelial and platelet cells. Regular consumption of coffee has been linked with decreased C-reactive

protein (CRP) levels, lower oxidative DNA damage, and improved antioxidant enzyme activity (superoxide dismutase, glutathione peroxidase).

These anti-inflammatory and antioxidant effects collectively support an antithrombotic profile, particularly when coffee is consumed in moderation without added sugars or high-fat creamers [8].

Dose-Dependent and Individual Variability.

The influence of coffee on hemostasis exhibits significant inter-individual variability depending on genetic, metabolic, and lifestyle factors. Slow caffeine metabolizers may experience prolonged stimulant and pressor effects, potentially promoting thrombogenesis under stress conditions. Additionally, dehydration from excessive caffeine intake may transiently increase blood viscosity, influencing clot formation dynamics.

Epidemiological data suggest a U-shaped relationship between coffee consumption and thrombotic risk, with lowest risk observed at moderate intake (2-4 cups daily). Both abstinence and excessive consumption correlate with higher cardiovascular event rates, emphasizing the importance of balance [9].

Clinical Implications.

From a clinical standpoint, understanding coffee's biochemical influence on coagulation is relevant for individuals at risk of thromboembolic events, patients on anticoagulant therapy, and those with bleeding disorders. Moderate coffee intake appears safe and potentially beneficial for cardiovascular health. However, high consumption or combination with other stimulants may pose risks. Monitoring caffeine intake and considering brewing methods (filtered vs. unfiltered) should be part of dietary recommendations in patients with coagulation abnormalities [10].

Conclusions. Coffee contains multiple bioactive compounds that modulate blood coagulation through complex biochemical mechanisms involving platelet function, coagulation factors, and endothelial activity. Moderate coffee consumption (up to 3 cups per day) generally exerts antithrombotic effects by inhibiting platelet aggregation, enhancing endothelial nitric oxide production, and reducing oxidative stress. Excessive caffeine intake or unfiltered coffee consumption may promote pro-coagulant activity through catecholamine release, dehydration, and elevated factor VII levels. The overall impact of coffee on coagulation is dose-dependent and influenced by genetic factors, metabolic rate, and brewing methods. Coffee can be included as part of a balanced diet, but individuals with cardiovascular or hematological disorders should monitor intake carefully. Further controlled studies are required to elucidate molecular pathways and establish precise consumption guidelines for optimal vascular health.

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КЛІНІКО-МОЛЕКУЛЯРНІ ОСОБЛИВОСТІ РАКУ МОЛОЧНОЇ ЗАЛОЗИ

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Анотація. Рак молочної залози характеризується значною біологічною та клінічною гетерогенністю, що зумовлює потребу у використанні великих масивів даних для його вивчення. Відкриті бази даних, такі як СРТАС/OncoKB та The Cancer Genome Atlas (TCGA), містять детальну інформацію про геномні та транскриптомні зміни, а також клінічні характеристики пацієнтів, і можуть бути використані для навчальних і дослідницьких цілей без додаткового збору матеріалу.

Метою роботи було узагальнити клінічні особливості когорти пацієнтів з раком молочної залози TCGA-BRCA та описати загальні тенденції мутаційних і