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Investigation of Blood Clotting Influential Parameters for Hemostatic Powder Based on Blood Coagulation Mechanisms

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Abstract

Hemorrhage is a major cause of preventable mortality in trauma, emergency medicine, and surgical procedures. Rapid and effective bleeding control is essential for improving patient survival. Hemostatic powders have emerged as promising biomaterials capable of accelerating blood coagulation through physical, chemical, and biological mechanisms. Their performance depends not only on physicochemical properties but also on physiological parameters regulating the coagulation process. Blood coagulation is a complex mechanism involving platelet activation, coagulation cascade reactions, fibrin formation, and clot stabilization. Various hematological and biochemical factors, including platelet count, fibrinogen concentration, thrombin activity, calcium ion levels, hematocrit, blood viscosity, pH, temperature, and coagulation factor function, significantly influence clot formation and stability. These parameters affect the interaction between blood components and hemostatic powders, thereby determining their hemostatic efficiency. This review discusses the key physiological parameters affecting blood coagulation and their impact on powder-based hemostatic agents. Recent developments in mineral, polysaccharide-based, clay, zeolite, chitosan-derived, and composite hemostatic powders are reviewed, with emphasis on their coagulation mechanisms, current challenges, and future perspectives for developing advanced hemostatic materials.

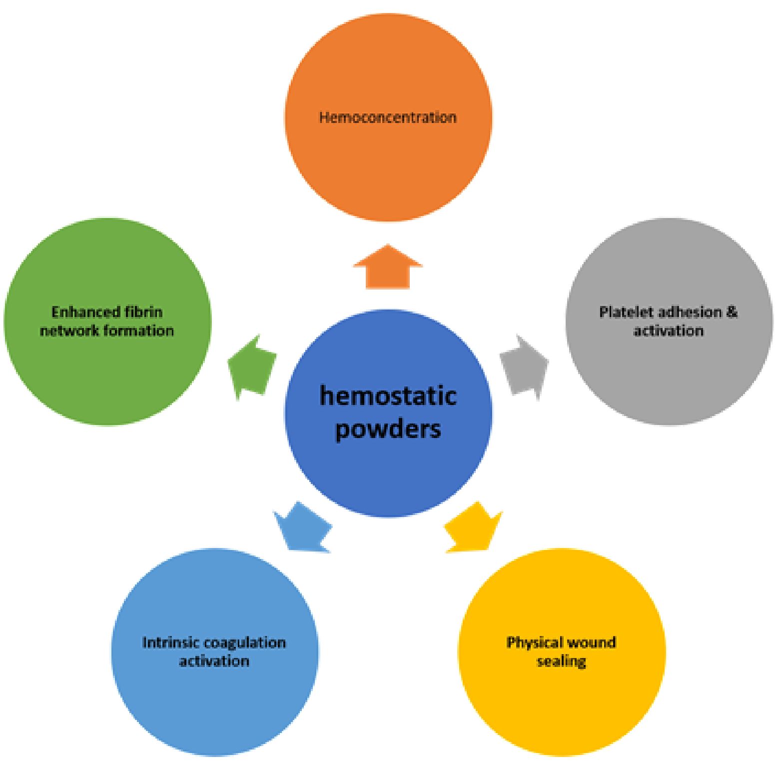
Keywords: Blood coagulation, hemostatic powder, blood clotting, platelets, fibrinogen, coagulation factors, biological materials, hemostasis.

1. Introduction

Uncontrolled hemorrhage continues to represent one of the most significant causes of death following traumatic injury worldwide. According to the World Health Organization (WHO), severe bleeding contributes substantially to trauma-related mortality, particularly during the first few hours after injury. Despite considerable advances in surgical techniques and emergency medicine, rapid hemorrhage control remains a major clinical challenge, especially in prehospital environments where immediate surgical intervention is unavailable [1]. Hemostasis is the natural physiological mechanism responsible for preventing excessive blood loss following vascular injury. It consists of a coordinated sequence of vascular constriction, platelet adhesion, platelet aggregation, activation of coagulation pathways, fibrin formation, and clot remodeling. Any disruption in these mechanisms may result in prolonged bleeding or pathological thrombosis [2].

Conventional approaches for hemorrhage control include direct compression, tourniquets, surgical ligation, electrocautery, suturing, and systemic administration of coagulation-promoting drugs. However, these methods may not always be feasible under emergency conditions or in deep, irregular wounds. Consequently, topical hemostatic agents have become an important alternative for rapid bleeding control [3]. Among topical hemostatic materials, powder-based hemostatic agents have attracted increasing scientific and clinical attention due to several advantages including rapid application, high surface area, ease of storage, portability, and compatibility with irregular wound geometries.

Figure 1: Key Mechanisms Underlying the Hemostatic Effects of Commercial Hemostatic Powders



Commercially available hemostatic powders achieve hemostasis through multiple complementary mechanisms, as illustrated in Figure 1. These mechanisms include rapid absorption of plasma water, resulting in the concentration of coagulation proteins and local enhancement of clot formation; promotion of platelet adhesion and activation; activation of the intrinsic coagulation pathway; physical sealing of the wound surface; enhancement of fibrin network formation; and local accumulation of calcium ions and clotting factors. Collectively, these processes contribute to accelerated clot formation and effective bleeding control. The effectiveness of these mechanisms depends strongly on the physiological status of circulating blood. Numerous biological parameters determine whether clot formation occurs rapidly or slowly. These include platelet count, fibrinogen concentration, coagulation factor activity, calcium availability, blood pH, temperature, hematocrit, erythrocyte deformability, blood viscosity, inflammatory mediators, and endothelial function [4].

Recent advances in biomaterials engineering have enabled the development of multifunctional hemostatic powders capable of simultaneously promoting coagulation, preventing infection, accelerating wound healing, and reducing inflammatory responses. Natural polymers such as chitosan, cellulose, alginate, collagen, gelatin, starch, and silk fibroin have been extensively investigated due to their excellent biocompatibility and biodegradability [5]. In parallel, inorganic materials including kaolin, zeolite, montmorillonite, bentonite, mesoporous silica, bioactive glass, and calcium phosphate have demonstrated remarkable hemostatic efficiency through activation of coagulation pathways and rapid plasma absorption [6].

Although significant progress has been achieved, the performance of hemostatic powders varies considerably under different physiological conditions. Patients suffering from hypothermia, acidosis, thrombocytopenia, anticoagulant therapy, liver dysfunction, or coagulopathies often exhibit delayed clot formation despite the application of advanced hemostatic materials. Therefore, understanding the interaction between blood physiology and material properties remains essential for the rational design of next-generation hemostatic powders [7].

This review aims to comprehensively analyze the physiological parameters influencing blood clotting and their relationship with powder-based hemostatic agents. Particular attention is given to platelet biology, coagulation factors, fibrin formation, biochemical environment, and recent advances in biomaterial engineering. The findings presented in this review may contribute to the development of more efficient hemostatic powders for trauma care, military medicine, surgical applications, and emergency treatment.

2. Blood Coagulation Mechanism

Blood coagulation, also referred to as hemostasis, is a highly regulated physiological process that prevents excessive blood loss following vascular injury while maintaining normal blood flow within intact vessels. This process involves a coordinated interaction among vascular endothelial cells, circulating platelets, plasma coagulation factors, fibrinolytic proteins, inflammatory mediators, and extracellular matrix components. The balance between procoagulant and anticoagulant mechanisms is essential for maintaining vascular homeostasis. Dysregulation of this balance may lead to severe hemorrhage or pathological thrombosis [8].

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Hemostasis is traditionally divided into primary hemostasis, secondary hemostasis, and fibrinolysis, each of which contributes to clot formation and stabilization.

2.1 Primary Hemostasis

Primary hemostasis begins immediately after vascular injury. Damage to the endothelial layer exposes subendothelial collagen, fibronectin, and tissue matrix proteins that initiate platelet adhesion. Endothelial disruption also results in the release of von Willebrand factor (vWF), a multimeric glycoprotein that mediates platelet attachment to exposed collagen through the platelet glycoprotein Ib (GPIb) receptor [9]. Following adhesion, platelets undergo activation characterized by dramatic morphological changes from a discoid to a spherical shape with numerous pseudopodia. Activated platelets release bioactive molecules stored in α-granules and dense granules, including adenosine diphosphate (ADP), serotonin, calcium ions (Ca^{2+}), platelet factor 4 (PF4), fibrinogen, and thromboxane A_2 (TXA_2). These mediators amplify platelet recruitment and aggregation at the injury site [10]. Platelet aggregation occurs through fibrinogen bridges linking activated glycoprotein IIb/IIIa (GPIIb/IIIa) receptors on adjacent platelets, leading to the formation of an unstable platelet plug that temporarily seals the damaged vessel.

2.2 Secondary Hemostasis

Secondary hemostasis reinforces the initial platelet plug through activation of the coagulation cascade, ultimately resulting in fibrin formation and stabilization of the developing clot. Coagulation factors circulate in the blood as inactive zymogens and are sequentially activated through proteolytic cleavage in response to vascular injury. Traditionally, the coagulation cascade has been described as three interconnected pathways: the intrinsic pathway, the extrinsic pathway, and the common pathway.

The intrinsic pathway is activated when blood comes into contact with negatively charged surfaces, including exposed collagen, glass, kaolin, silica, and certain mineral-based hemostatic powders. This process begins with activation of Factor XII (Hageman factor), followed by sequential activation of Factors XI, IX, and VIII. Activated Factor IX (FIXa), together with activated Factor VIII (FVIIIa), calcium ions, and phospholipid membranes, forms the intrinsic tenase complex, which efficiently activates Factor X (FX) [11]. Several commercially available mineral-based hemostatic powders, particularly kaolin-containing formulations, enhance coagulation by promoting Factor XII activation and accelerating the intrinsic pathway.

The extrinsic pathway is initiated following vascular injury through exposure of tissue factor (Factor III), which rapidly binds circulating activated Factor VII (FVIIa) to form the tissue factor–Factor VIIa (TF–FVIIa) complex. This complex directly activates Factor X and, to a lesser extent, Factor IX, providing a rapid mechanism for coagulation initiation. Under physiological conditions, the extrinsic pathway serves as the primary trigger of coagulation after tissue injury because tissue factor is highly expressed by extravascular cells, including fibroblasts and smooth muscle cells [12].

The intrinsic and extrinsic pathways converge at the activation of Factor X, leading to the common pathway of coagulation. Activated Factor X (FXa), in combination with activated Factor V (FVa), calcium ions, and phospholipid surfaces, forms the prothrombinase complex, which converts prothrombin (Factor II) into thrombin (Factor IIa). Thrombin acts as the central enzyme of coagulation due to its multiple regulatory functions, including conversion of soluble fibrinogen into insoluble fibrin, activation of Factors V, VIII, XI, and XIII, amplification of platelet activation, and stabilization of the fibrin clot through Factor XIII activation. Activated Factor XIII promotes covalent cross-linking of fibrin monomers, generating a mechanically stable fibrin network capable of withstanding blood flow and mechanical stress [13]. Understanding these coagulation mechanisms is essential for evaluating how hemostatic powders interact with and enhance different stages of the clotting process.

2.3 Fibrinolysis

Following tissue repair, fibrin clots are removed through the fibrinolytic system. Plasminogen is converted into plasmin primarily by tissue plasminogen activator (tPA). Plasmin enzymatically degrades fibrin into soluble fibrin degradation products, including D-dimer, which serves as an important clinical biomarker of coagulation and thrombus degradation [14]. The fibrinolytic system is tightly regulated by plasminogen activator inhibitors (PAI-1 and PAI-2) and α_2-antiplasmin to prevent excessive clot dissolution.

2.4 Cellular Model of Coagulation

Although the classical intrinsic–extrinsic coagulation cascade remains valuable for educational purposes, recent advances in hemostasis research have highlighted the cell-based model of coagulation as a more accurate representation of in vivo clot formation. This model describes coagulation as a dynamic process consisting of three overlapping phases: initiation, amplification, and propagation. During the initiation phase, small amounts of thrombin are generated on tissue factor-bearing cells immediately following vascular injury. In the amplification phase, the generated thrombin activates platelets and multiple coagulation cofactors, thereby enhancing the efficiency of subsequent coagulation reactions. Finally, during the propagation phase, extensive thrombin production occurs on activated platelet surfaces, leading to rapid fibrin generation and the formation of a stable clot [15]. This cellular model emphasizes the essential role of platelet function in effective coagulation and highlights the importance of platelet–material interactions in determining the performance of hemostatic powders.

Modern hemostatic powders are specifically designed to interact with one or more components of the coagulation process to enhance bleeding control. Proposed mechanisms of action include rapid absorption of water from plasma, resulting in local concentration of coagulation proteins; activation of Factor XII by negatively charged mineral surfaces; enhancement of platelet adhesion through surface roughness and porosity; localized accumulation of calcium ions; promotion of fibrin polymerization; and formation of a physical barrier that limits blood loss while endogenous coagulation progresses. Therefore, the effectiveness of a hemostatic powder depends not only on its physicochemical properties but also on the physiological status of the patient's coagulation system. Clinical conditions such as thrombocytopenia, hypofibrinogenemia, anticoagulant *therapy, hypothermia, acidosis, and impaired platelet function can substantially reduce hemostatic performance, even when advanced biomaterials are applied [16]. A comprehensive understanding of coagulation biology is consequently essential for the rational design of next-generation hemostatic powders with improved efficacy and broader clinical applicability.*

3. Parameters Affecting Blood Clotting And Their Role In Hemostatic Powder Performance

Blood coagulation is influenced by a wide range of physiological, biochemical, and rheological parameters. The effectiveness of hemostatic powders is directly dependent on these variables because topical hemostatic materials interact with circulating blood components during clot formation. Variations in blood composition or coagulation status may significantly alter the performance of hemostatic agents. Therefore, understanding these parameters is fundamental for the development of advanced biomaterials capable of achieving rapid and stable hemostasis under different clinical conditions [17].

3.1 Platelet Count and Platelet Function

Platelets (thrombocytes) are small anucleate blood cells derived from megakaryocytes and play a central role in primary hemostasis. Under physiological conditions, the normal platelet count ranges from $150 \times 10^9/L$ to $450 \times 10^9/L$. Following vascular injury, platelets adhere to exposed collagen through interactions mediated by von Willebrand factor (vWF), undergo activation, and aggregate to form the initial platelet plug [18]. The efficiency of most powder-based hemostatic agents depends on their ability to promote platelet adhesion and activation. Materials such as chitosan, kaolin, mesoporous silica, and collagen powders possess surface characteristics that enhance platelet attachment and stimulate the release of procoagulant mediators, including ADP, thromboxane A_2, and platelet factor 4 [19].

However, patients with thrombocytopenia or platelet dysfunction exhibit delayed platelet plug formation, reducing the effectiveness of many conventional hemostatic powders. Consequently, recent studies have focused on developing platelet-independent hemostatic materials capable of inducing coagulation even under conditions of impaired platelet activity. Surface roughness, porosity, surface charge density, and hydrophilicity of powder particles strongly influence platelet-material interactions. Nanostructured materials with high specific surface area have demonstrated superior platelet activation compared with conventional micron-sized particles.

3.2 Fibrinogen Concentration

Fibrinogen (Factor I) is a soluble plasma glycoprotein synthesized by the liver and represents the precursor of fibrin. During coagulation, thrombin cleaves fibrinopeptides A and B from fibrinogen, producing fibrin monomers that polymerize into an insoluble fibrin network. Normal plasma fibrinogen concentration ranges from 2.0 to 4.0 g/L. Reduced fibrinogen levels, commonly observed during massive hemorrhage, liver disease, trauma-induced coagulopathy, and disseminated intravascular coagulation (DIC), significantly prolong clot formation time [20]. Hemostatic powders primarily accelerate coagulation by concentrating plasma proteins through rapid water absorption. Consequently, adequate fibrinogen concentration is essential for optimal *clot stabilization. Recent composite powders incorporating fibrinogen-mimetic peptides or fibrin-binding polymers have shown enhanced performance under hypofibrinogenemic conditions. Furthermore, fibrin architecture influences clot mechanical strength. Dense fibrin networks exhibit greater resistance to enzymatic degradation and mechanical disruption, improving wound stability.*

3.3 Calcium Ion Concentration (Ca^{2+})

Calcium ions are essential cofactors in multiple stages of the coagulation cascade, contributing to the activation of coagulation Factors II, VII, IX, and X and facilitating the assembly of enzyme–cofactor complexes on phospholipid membranes. Under physiological conditions, the concentration of ionized calcium is maintained within a range of approximately 1.1–1.3 mmol/L. However, hypocalcemia is frequently observed following massive blood transfusion, as citrate-based anticoagulants used in stored blood products chelate circulating calcium ions. Reduced calcium availability can significantly impair thrombin generation and fibrin formation, resulting in delayed clot development and compromised hemostasis. To address this limitation, several newly developed hemostatic powders incorporate calcium-containing minerals, including calcium alginate, calcium phosphate, bioactive glass, calcium silicate, and calcium carbonate nanoparticles. These materials provide localized calcium ion release at the wound site, thereby enhancing coagulation cascade activation, accelerating clot formation, and improving clot stability, particularly under conditions of severe hemorrhage [21].

3.4 Coagulation Factors

Blood coagulation requires coordinated activation of more than twelve plasma coagulation factors. Inherited deficiencies (e.g., hemophilia A and B) or acquired deficiencies caused by liver disease, trauma, anticoagulant therapy, or vitamin K deficiency substantially impair coagulation efficiency. Mineral-based hemostatic powders, particularly kaolin-containing formulations, activate Factor XII, initiating the intrinsic coagulation pathway. In contrast, bioactive polymers primarily enhance platelet aggregation rather than directly activating coagulation factors. Recent investigations suggest that engineered biomaterials capable of selectively activating Factors XI and XII may significantly reduce bleeding time without increasing systemic thrombotic risk.

3.5 Blood pH

Normal arterial blood pH ranges between 7.35 and 7.45. Trauma patients frequently develop metabolic acidosis due to hypoperfusion and anaerobic metabolism. Decreased pH profoundly inhibits coagulation enzyme activity because most serine proteases exhibit maximal catalytic activity under physiological pH conditions. Experimental studies have demonstrated that reducing blood pH below 7.2 significantly decreases thrombin generation, platelet activation, and fibrin polymerization.

Therefore, although hemostatic powders can accelerate local coagulation, their efficacy may be compromised in severely acidotic patients. Current research is investigating pH-buffering biomaterials capable of partially correcting local acidosis within wound environments [22].

3.6 Temperature

Body temperature plays a critical role in regulating coagulation kinetics and overall hemostatic function. Hypothermia, commonly defined as a core body temperature below 35°C, is frequently observed in patients with severe trauma and represents one component of the well-established "lethal triad," which also includes acidosis and coagulopathy. Reduced temperatures significantly impair enzymatic reactions involved in the coagulation cascade, leading to decreased platelet activation, reduced thrombin generation, and impaired fibrin cross-linking. These alterations contribute to delayed clot formation and compromised hemostatic efficiency. Therefore, the evaluation of hemostatic powders under hypothermic conditions has become an important area of research, particularly in the fields of military medicine and emergency trauma management, where effective bleeding control under adverse physiological conditions is critical.

3.7 Hematocrit

Hematocrit represents the percentage of blood volume occupied by red blood cells. Although erythrocytes are not directly involved in coagulation, they strongly influence blood rheology and platelet transport. Higher hematocrit values promote platelet margination toward vessel walls, increasing platelet-endothelium interactions and accelerating primary hemostasis. Conversely, severe anemia reduces platelet-wall interactions, thereby delaying clot initiation. Several investigations have demonstrated that hemostatic powders exhibit different coagulation efficiencies depending on hematocrit levels, emphasizing the importance of evaluating biomaterials under clinically relevant blood conditions.

3.8 Blood Viscosity

Blood viscosity determines resistance to flow and directly influences transport of coagulation proteins and blood cells. Hyperviscosity promotes localized coagulation but may increase thrombotic risk. Hypoviscosity, often observed after extensive fluid resuscitation, dilutes coagulation factors and delays clot formation. Highly absorbent hemostatic powders partially compensate for hemodilution by concentrating plasma proteins near the wound surface.

3.9 Anticoagulant Medications

The widespread use of anticoagulant medications represents a significant challenge for the development and clinical application of topical hemostatic materials. Commonly used anticoagulants, including heparin, warfarin, dabigatran, rivaroxaban, and apixaban, interfere with key components of the coagulation cascade by inhibiting thrombin generation or Factor Xa activity, thereby prolonging bleeding time and impairing effective clot formation. Recent studies have demonstrated that advanced mineral–polymer composite hemostatic powders can retain partial hemostatic efficacy even under anticoagulated conditions. These materials achieve improved bleeding control through mechanisms that are less dependent on the conventional coagulation pathway, including rapid plasma absorption, enhanced platelet activation, and mechanical sealing of the bleeding site [23].

3.10 Inflammatory Response

Inflammation and coagulation are closely interconnected biological processes. Inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) stimulate tissue factor *expression, thereby enhancing coagulation activation. Conversely, excessive inflammation may lead to endothelial dysfunction and disseminated intravascular coagulation. Modern multifunctional hemostatic powders increasingly incorporate anti-inflammatory agents, antioxidants, or antimicrobial compounds to simultaneously control bleeding and promote wound healing.*

4. Hemostatic Powders And Their Mechanisms Of Action

Hemostatic powders are among the most widely used topical hemostatic agents because of their ease of application, portability, and rapid ability to control bleeding. Unlike conventional dressings, these powders rapidly interact with blood components and accelerate clot formation through physical, chemical, and biological mechanisms. Their performance depends on particle size, porosity, surface chemistry, hydrophilicity, ion release, and interaction with blood cells and plasma proteins [24].

4.1 Mineral-Based Hemostatic Powders

Mineral-based hemostatic powders are widely utilized due to their high fluid absorption capacity and their ability to enhance coagulation through activation of the intrinsic pathway. Among these materials, kaolin is one of the most extensively studied inorganic hemostatic agents. Kaolin promotes Factor XII activation, thereby initiating the intrinsic coagulation cascade without the need for exogenous coagulation factors. Owing to its rapid hemostatic activity and thermal stability, kaolin-based products have been broadly adopted in both military and civilian trauma management settings [25]. Zeolite powders represent another important class of mineral-based hemostats, characterized by their high surface area and efficient plasma absorption capacity. By removing plasma water, zeolites increase the local concentration of platelets and coagulation proteins at the bleeding site, thereby accelerating clot formation. However, early zeolite formulations were associated with heat generation during hydration, which raised concerns regarding potential tissue damage; subsequent material modifications have substantially reduced this limitation. Other investigated mineral-based hemostatic materials include bentonite, montmorillonite, mesoporous silica, bioactive glass, calcium phosphate, and calcium silicate. These materials contribute to hemostasis through multiple mechanisms, including concentration of coagulation proteins, activation of clotting factors, and release of calcium ions to support coagulation cascade progression.

4.2 Polymer-Based Hemostatic Powders

Natural polymers have attracted considerable attention in the development of advanced hemostatic materials due to their excellent biocompatibility, biodegradability, and low toxicity. Among these biomaterials, chitosan, a derivative of chitin, exhibits intrinsic hemostatic activity through mechanisms that are largely independent of the classical coagulation cascade. The positively charged amino groups of chitosan interact electrostatically with negatively charged erythrocyte and platelet membranes, promoting cellular aggregation and facilitating rapid clot formation. In addition to its hemostatic properties, chitosan possesses antimicrobial activity and contributes to improved wound healing processes [26]. Other naturally derived polymeric materials, including alginate, gelatin, collagen, oxidized cellulose, starch, and silk fibroin, have *also been investigated for hemostatic applications. These biomaterials primarily enhance coagulation by promoting plasma absorption, increasing local concentration of clotting components, and facilitating platelet adhesion, thereby improving overall clot formation and wound stabilization.*

4.3 Composite Hemostatic Powders

Recent research has increasingly focused on the development of hybrid composite hemostatic powders that combine inorganic minerals with natural or synthetic polymers to achieve enhanced multifunctional performance. Examples of these advanced composites include kaolin–chitosan, silica–alginate, bioactive glass–gelatin, and calcium phosphate–collagen systems. By integrating the complementary properties of their individual components, composite powders can simultaneously promote multiple hemostatic mechanisms, including rapid plasma absorption, platelet activation, Factor XII activation, calcium ion release, antimicrobial activity, and stimulation of tissue regeneration. Compared with single-component hemostatic powders, these multifunctional materials have demonstrated improved hemostatic performance, characterized by shorter clotting times, enhanced clot stability, and accelerated wound healing outcomes [27].

5. Comparison Of Commercial Hemostatic Powders

Several commercial hemostatic products have been developed for emergency medicine, trauma care, military applications, and surgery. Their mechanisms differ depending on material composition.

Table 1: Commercially available hemostatic products, their primary components, and mechanisms of action.

Product	*Main component*	*Primary Mechanism*
QuikClot®	*Kaolin*	*Factor XII activation*
Celox™	*Chitosan*	*Platelet-independent aggregation*
WoundSeal®	*Hydrophilic polymers*	*Mechanical barrier formation*
Traumastem®	*Oxidized cellulose*	*Plasma absorption and clot stabilization*
Arista™ AH	*Microporous polysaccharide*	*Plasma absorption and clot stabilization*

Clinical studies have shown that no single product is universally superior. Selection depends on wound type, bleeding severity, patient coagulation status, and clinical setting [28].

6. Current Challenges And Future Perspectives

Despite remarkable advances, several challenges remain in the development of ideal hemostatic powders.

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First, many existing hemostatic products rely primarily on the patient's endogenous coagulation mechanisms to achieve effective bleeding control, which may limit their performance under conditions associated with impaired hemostasis. Their efficacy can be significantly reduced in patients with severe thrombocytopenia, hypothermia, metabolic acidosis, liver dysfunction, disseminated intravascular coagulation, or those receiving anticoagulant therapy. These clinical conditions disrupt various components of the coagulation cascade, platelet function, or clot stability, thereby limiting the effectiveness of conventional hemostatic approaches.

Future biomaterials should maintain hemostatic performance under these compromised physiological conditions. Second, the risk of thrombosis associated with highly procoagulant materials requires careful evaluation. An ideal hemostatic powder should accelerate local clot formation without inducing systemic coagulation. Third, multifunctional biomaterials capable of combining hemostasis with antibacterial activity, anti-inflammatory effects, angiogenesis, and tissue regeneration are becoming increasingly attractive.

Emerging technologies such as nanotechnology, bioactive nanoparticles, peptide engineering, and smart biomaterials responsive to pH or temperature changes are expected to revolutionize next-generation hemostatic agents [29].

7. Discussion

The effectiveness of hemostatic powders depends on a complex interaction between biomaterial properties and physiological characteristics of blood. Although particle morphology, porosity, chemical composition, and surface charge significantly influence coagulation, patient-related variables remain equally important.

Among all investigated parameters, platelet function, fibrinogen concentration, calcium availability, coagulation factor activity, blood pH, and temperature appear to exert the greatest influence on hemostatic performance. Current evidence suggests that multifunctional composite powders outperform conventional inorganic materials because they combine several complementary mechanisms rather than relying on a single coagulation pathway.

Furthermore, recent advances in biomaterial engineering indicate that future hemostatic powders will likely incorporate controlled drug delivery, antimicrobial agents, growth factors, and regenerative biomolecules while maintaining rapid coagulation capability. Standardized in vitro coagulation assays together with clinically relevant animal models will remain essential for evaluating newly developed materials before clinical application.

8. Conclusion

Blood coagulation is a highly coordinated physiological process influenced by numerous hematological, biochemical, and rheological parameters. Platelet count, fibrinogen concentration, calcium ion availability, coagulation factor activity, blood pH, body temperature, hematocrit, blood viscosity, inflammatory status, and anticoagulant medications all significantly affect clot formation and consequently influence the performance of topical hemostatic powders.

Modern hemostatic powders accelerate coagulation through multiple mechanisms including plasma absorption, platelet activation, coagulation factor activation, calcium release, and physical sealing of bleeding wounds. Composite biomaterials integrating inorganic minerals with natural polymers have demonstrated superior performance compared with conventional single-component formulations.

Future research should focus on designing intelligent multifunctional hemostatic powders capable of maintaining high efficacy under coagulopathic conditions while simultaneously promoting tissue regeneration, reducing inflammation, and preventing microbial infection. A comprehensive understanding of blood coagulation physiology together with advances in biomaterials engineering will facilitate the development of safer and more effective hemostatic technologies for trauma care, surgery, and emergency medicine.

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