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Complement Receptor 1 Dynamics and Erythrocyte Immunological Function: Mechanisms, Modulation, and Clinical Implications

[1]Dr. Elena V. Korsakova
[1] Department of Immunology and Molecular Medicine, University of Geneva, Switzerland

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Abstract

Complement receptor 1 (CR1, CD35) serves as a pivotal mediator of erythrocyte immune function, orchestrating processes that range from immune complex clearance to modulation of host-pathogen interactions. CR1's structural heterogeneity, including the F and S allelic forms, contributes to variable receptor expression and clustering, thereby influencing erythrocyte deformability, immune adherence, and susceptibility to pathogen-mediated rosetting (Vik & Wong, 1993; Rowe et al., 1997). The molecular mechanisms underlying CR1 clustering involve complex interactions with cytoskeletal scaffolding proteins such as FAP-1 and ATP-mediated signaling pathways, which enhance receptor aggregation and facilitate efficient immune complex transport (Ghiran et al., 2008; Melhorn et al., 2013). CR1's role extends beyond erythrocyte-mediated clearance, influencing complement-mediated tissue injury in ischemia-reperfusion scenarios, where soluble CR1 demonstrates protective effects against microvascular and myocardial damage (Lindsay et al., 1992; Shandelya et al., 1993).

This review integrates molecular, cellular, and translational perspectives on CR1 function, emphasizing receptor clustering dynamics, immune adherence, and erythrocyte deformability. Theoretical frameworks and historical studies contextualize CR1's evolution as a critical immune modulator, while contemporary research elucidates the interplay between receptor polymorphisms, ligand binding, and immune signaling pathways. Mechanistic insights from human and animal models reveal the receptor's dual role in pathogen defense and modulation of complement-mediated injury (Pringle et al., 2012; Sun et al., 2012). Additionally, CR1-targeted interventions, including soluble recombinant constructs, offer therapeutic potential in ischemia-reperfusion injury and infectious disease contexts, underscoring the clinical relevance of receptor modulation (Smith et al., 1993; Chavez-Cartaya et al., 1995).

This article provides a comprehensive synthesis of the CR1 literature, critically evaluating empirical findings, methodological approaches, and theoretical constructs. Emphasis is placed on the implications of receptor clustering for immunological homeostasis, the influence of genetic polymorphisms on receptor functionality, and the translational significance of manipulating CR1 in disease contexts. By integrating cellular, molecular, and clinical perspectives, this review delineates current knowledge gaps and proposes directions for future research, including high-resolution mapping of receptor-ligand interactions, longitudinal assessment of erythrocyte-mediated immune clearance, and therapeutic modulation of complement activity.

Keywords: Complement receptor 1, erythrocyte immune adherence, receptor clustering, ischemia-reperfusion injury, polymorphism, immune complex clearance, cytoskeletal interactions.

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1. Introduction

The complement system, a cornerstone of innate immunity, operates through a finely regulated network of proteins designed to identify, opsonize, and clear pathogens, apoptotic cells, and immune complexes. Among its regulatory components, complement receptor 1 (CR1, CD35) assumes a central role in mediating erythrocyte-driven immune clearance, bridging innate and adaptive immunological functions (Chevalier & Kazatchkine, 1989). CR1 is a type I membrane glycoprotein expressed on erythrocytes, leukocytes, and various other cell types, whose functionality is critically dependent on receptor density, allelic variation, and spatial organization within the membrane (Vik & Wong, 1993). The receptor engages with C3b and C4b fragments, enabling erythrocytes to bind immune complexes and transport them to the liver and spleen for clearance, thereby preventing immune complex deposition and systemic inflammation (Deng et al., 2018).

Historically, the discovery of CR1's role in immune adherence emerged from studies of erythrocyte-mediated clearance of opsonized particles, with Chevalier and Kazatchkine (1989) elucidating the clustered distribution of CR1 on human erythrocytes. This clustering is not a passive attribute; rather, it is a dynamic process influenced by receptor-ligand interactions, intracellular signaling cascades, and cytoskeletal scaffolding. The involvement of FAP-1, a scaffolding protein, in CR1 clustering illustrates the receptor's integration into broader cellular machinery, linking extracellular immune signals to intracellular structural modulation (Ghiran et al., 2008). Furthermore, CR1 ligation induces ATP release, which acts in an autocrine and paracrine manner to facilitate receptor aggregation and optimize immune complex handling (Melhorn et al., 2013).

Polymorphisms in the CR1 gene, particularly the F and S alleles, introduce additional complexity to receptor function. These variants confer differential binding affinities, receptor densities, and susceptibility to pathogen-mediated rosetting, as observed in Plasmodium falciparum infections (Rowe et al., 1997; Vik & Wong, 1993). The interplay between CR1 polymorphisms and pathogen virulence underscores the receptor's dual role in host defense and disease susceptibility. Such variation not only impacts infectious disease outcomes but also influences the erythrocyte's mechanical properties, with implications for circulation dynamics and tissue perfusion (Glodek et al., 2010).

Complement-mediated ischemia-reperfusion (I/R) injury represents a critical context in which CR1 function exerts therapeutic relevance. Animal and clinical studies demonstrate that complement activation drives microvascular permeability, neutrophil retention, and subsequent tissue damage following ischemic insult (Lindsay et al., 1992; Xiao et al., 1997). Intervention with soluble CR1 effectively inhibits complement pathways, mitigates endothelial injury, and preserves myocardial and skeletal muscle contractility (Shandelya et al., 1993; Smith et al., 1993; Chavez-Cartaya et al., 1995). These findings illuminate the translational potential of modulating CR1-mediated mechanisms, bridging molecular immunology and clinical therapeutics.

Despite extensive investigation, several knowledge gaps persist. The precise molecular determinants of CR1 clustering, the signaling pathways governing erythrocyte deformability post-ligation, and the long-term impact of receptor modulation on systemic immune function remain incompletely understood (Ghiran et al., 2008; Glodek et al., 2010). Moreover, comparative studies across species—including bovine, porcine, and avian models—highlight the evolutionary conservation and divergence of CR1 function, suggesting both shared and unique mechanisms of immune adherence (Pringle et al., 2012; Sun et al., 2012; Li et al., 2007). Understanding these nuances is critical for designing effective therapeutics and predicting outcomes in both infectious and non-infectious disease contexts.

In this review, we provide a detailed examination of CR1 structure, function, and clinical implications. By synthesizing molecular insights, experimental evidence, and translational applications, we aim to construct a comprehensive understanding of how CR1 dynamics regulate erythrocyte-mediated immunity and complement-related pathophysiology. This approach underscores the importance of integrating historical perspectives, mechanistic studies, and clinical observations to generate a coherent model of CR1

biology that can guide future research and therapeutic innovation.

2. Methodology

This article adopts a systematic, literature-driven methodology aimed at synthesizing existing knowledge on complement receptor 1 and its functional implications for erythrocyte-mediated immunity and tissue protection. The methodological framework comprises four primary dimensions: literature selection, data extraction, critical analysis, and theoretical integration. Literature selection was guided by the inclusion criteria of peer-reviewed primary research articles, comprehensive reviews, and translational studies that specifically address CR1 function, erythrocyte deformability, receptor clustering, immune adherence, and complement-mediated tissue injury. Sources were identified from multiple biomedical databases, including PubMed, Web of Science, and Scopus, and included studies from human, murine, bovine, porcine, and avian models to ensure evolutionary and mechanistic breadth.

Data extraction focused on detailed parameters including receptor density, allelic variation (F vs. S alleles), binding affinities to C3b/C4b fragments, cytoskeletal interactions, ATP-mediated signaling, and erythrocyte mechanical properties. Additionally, studies examining soluble CR1 interventions in ischemia-reperfusion injury were analyzed for outcome measures such as microvascular permeability, neutrophil retention, myocardial contractility, and tissue necrosis. Emphasis was placed on experimental design, sample size, species specificity, and methodological rigor to ensure analytical validity and relevance.

Critical analysis employed a multi-layered approach, integrating mechanistic, molecular, and translational perspectives. Each study was evaluated for its contribution to understanding CR1 dynamics, including ligand-receptor interactions, clustering phenomena, signaling cascades, and functional outcomes in immune clearance and tissue protection. Comparative analyses were performed to identify species-specific variations, mechanistic conservation, and divergence in receptor function, thereby contextualizing human CR1 within broader immunological frameworks (Pringle et al., 2012; Sun et al., 2012).

Theoretical integration extended beyond empirical findings to synthesize historical perspectives, molecular models, and clinical implications. The analysis included an evaluation of receptor clustering mechanisms, cytoskeletal modulation, ATP-mediated signaling, and the functional consequences of CR1 polymorphisms. Additionally, the interplay between erythrocyte-mediated immune clearance and complement-mediated tissue injury was examined, with particular attention to the mechanistic underpinnings of ischemia-reperfusion injury and the protective effects of soluble CR1 (Lindsay et al., 1992; Shandelya et al., 1993).

Limitations of the methodology include reliance on existing literature, which may underrepresent unpublished negative findings or emerging preclinical models. Additionally, cross-species extrapolation, while informative, may not fully capture human-specific receptor dynamics. Nonetheless, this integrative approach provides a comprehensive framework for understanding CR1-mediated mechanisms, bridging molecular insights with translational applications, and offering a foundation for hypothesis-driven future research.

3. Results

Analysis of the existing literature reveals several key findings regarding CR1 function and erythrocyte-mediated immunity. First, CR1 clustering is a central determinant of receptor functionality. Ghiran et al. (2008) demonstrated that ligation of erythrocyte CR1 induces aggregation in concert with the scaffolding protein FAP-1, forming multimeric complexes that enhance immune complex adherence. Melhorn et al. (2013) further elucidated the role of ATP release in promoting clustering, suggesting a feedback mechanism whereby receptor engagement modulates subsequent binding efficiency and immune transfer.

Second, polymorphisms in the CR1 gene significantly influence receptor density and immune adherence capacity. Vik and Wong (1993) characterized the structural distinctions between the F and S alleles, revealing differential binding affinities and functional consequences for pathogen-mediated rosetting. Rowe et al. (1997) highlighted the clinical relevance of these polymorphisms in P. falciparum infections, where CR1-mediated rosetting contributes to severe malaria pathophysiology.

Third, receptor clustering directly impacts erythrocyte deformability, with ligated CR1 enhancing membrane flexibility and facilitating microvascular passage (Glodek et al., 2010). This property not only optimizes

immune complex transport but also mitigates mechanical stress on erythrocytes, reducing hemolysis and preserving circulatory integrity.

Fourth, soluble CR1 exhibits protective effects in ischemia-reperfusion injury. Studies in skeletal muscle, myocardium, and liver demonstrate that complement blockade via recombinant CR1 reduces microvascular leakage, neutrophil infiltration, and contractile dysfunction (Lindsay et al., 1992; Pemberton et al., 1993; Shandelya et al., 1993; Smith et al., 1993; Chavez-Cartaya et al., 1995). These findings underscore the therapeutic potential of CR1 modulation in complement-driven pathologies.

Fifth, comparative studies across species indicate both conserved and divergent CR1-mediated mechanisms. Pringle et al. (2012) observed co-expression of CR1 and CR2 in cattle, suggesting overlapping and potentially compensatory immune functions, while Sun et al. (2012) and Li et al. (2007) documented species-specific variations in erythrocyte immune adherence, highlighting the evolutionary adaptation of CR1 in response to pathogen pressures.

4. Discussion

The integrative analysis of CR1 function illuminates its multifaceted role in immune regulation and clinical pathology. Mechanistically, receptor clustering emerges as a dynamic and regulated process. The interaction with FAP-1 scaffolding proteins not only stabilizes clusters but also facilitates signal transduction and cytoskeletal reorganization, enhancing erythrocyte resilience and immune complex processing (Ghiran et al., 2008). ATP-mediated feedback mechanisms further optimize clustering, indicating that CR1 functions as both a structural and signaling hub.

Allelic variation introduces a nuanced layer of functional heterogeneity. The F and S alleles, while structurally similar, demonstrate distinct binding affinities, receptor densities, and susceptibility to pathogen interactions (Vik & Wong, 1993). This variation has been implicated in disease outcomes, particularly in malaria, where CR1-mediated rosetting contributes to microvascular obstruction and severe clinical manifestations (Rowe et al., 1997). These findings prompt consideration of CR1 polymorphisms in therapeutic strategies and epidemiological risk assessments.

Erythrocyte deformability, modulated by CR1 ligation, represents an intersection of immune function and biomechanical integrity. Enhanced flexibility facilitates immune complex transport while preserving circulatory dynamics, suggesting an evolutionary advantage in maintaining CR1 functionality (Glodek et al., 2010). Disruption of this balance, either through genetic variation, receptor blockade, or pathogen-mediated interference, can precipitate systemic inflammation and circulatory compromise.

The therapeutic application of soluble CR1 in ischemia-reperfusion injury exemplifies the translational significance of receptor modulation. By inhibiting complement activation, soluble CR1 reduces endothelial damage, neutrophil retention, and tissue necrosis, offering a targeted approach to mitigate reperfusion-mediated pathology (Shandelya et al., 1993; Chavez-Cartaya et al., 1995). These studies support a broader framework in which CR1 serves as a pharmacological target in both acute and chronic complement-mediated diseases.

Comparative analyses underscore evolutionary conservation and divergence in CR1 function. While core mechanisms—ligand binding, clustering, and immune adherence—are preserved, species-specific adaptations reflect differential pathogen pressures and immune system architecture (Pringle et al., 2012; Sun et al., 2012; Li et al., 2007). Such insights provide a foundation for translational research, enabling the design of cross-species therapeutic strategies and predictive modeling of immune responses.

Future research should focus on high-resolution mapping of CR1-ligand interactions, detailed mechanistic elucidation of ATP-mediated signaling pathways, and longitudinal studies assessing erythrocyte-mediated immune clearance under physiological and pathological conditions. Integration of structural biology, computational modeling, and in vivo experimentation will be critical to advancing our understanding of CR1's role in immunity and disease.

4.1. Translational Implications and Therapeutic Perspectives

The translational potential of complement receptor 1 (CR1) extends well beyond its canonical role in erythrocyte-mediated immune clearance, presenting significant opportunities for therapeutic innovation in both infectious and non-infectious disease contexts. CR1's capacity to modulate immune complex handling, complement activation, and erythrocyte deformability

establishes it as a critical molecular target for the mitigation of systemic inflammation, tissue injury, and pathogen-mediated pathology. The development of soluble CR1 and recombinant receptor constructs represents one of the most direct applications of mechanistic insights into clinical therapeutics, translating molecular understanding into disease-modifying interventions (Shandelya et al., 1993; Chavez-Cartaya et al., 1995).

Soluble CR1 (sCR1) has been investigated extensively in preclinical models of ischemia-reperfusion (I/R) injury, demonstrating significant protective effects across multiple organ systems. In skeletal muscle ischemia-reperfusion, blockade of complement activation via sCR1 reduces microvascular leakage, diminishes leukocyte adhesion, and preserves structural integrity of endothelial networks, resulting in improved tissue perfusion and functional recovery (Pemberton et al., 1993). The mechanistic underpinnings of these observations are rooted in the receptor's ability to inhibit C3b and C4b-mediated complement cascade propagation, thereby preventing the formation of the membrane attack complex and limiting secondary tissue damage (Lindsay et al., 1992). These findings underscore CR1's potential as a modulator of vascular and microcirculatory homeostasis, providing an immunological lever to attenuate reperfusion-mediated injury.

In myocardial reperfusion contexts, sCR1 administration has similarly demonstrated protective outcomes, mitigating contractile dysfunction and reducing infarct size in experimental models (Smith et al., 1993). The mechanistic rationale involves suppression of neutrophil-mediated cytotoxicity, as complement activation promotes chemotaxis, adhesion, and reactive oxygen species generation within reperfused myocardial tissue. By attenuating these complement-dependent processes, sCR1 facilitates cardiomyocyte survival and enhances post-ischemic functional recovery. These preclinical observations have catalyzed discussions regarding sCR1 as a therapeutic adjunct in cardiac surgery, percutaneous coronary interventions, and acute myocardial infarction management, highlighting the receptor's translational relevance in cardiovascular medicine (Shandelya et al., 1993).

Beyond I/R injury, CR1 modulation holds promise in infectious disease management, particularly in contexts where erythrocyte-mediated immune adherence is central to pathogen clearance. The polymorphic nature of CR1, particularly the F and S allelic variants, has been shown to influence susceptibility to Plasmodium falciparum-mediated rosetting, a key pathogenic mechanism underlying severe malaria (Rowe et al., 1997; Vik & Wong, 1993). Therapeutic strategies leveraging CR1 functionality could, therefore, target the modulation of receptor clustering or expression density on erythrocytes to reduce parasite adhesion, interrupt microvascular obstruction, and improve clinical outcomes. The potential for small-molecule modulators, monoclonal antibodies, or receptor mimetics to alter CR1-mediated rosetting represents a frontier in host-directed antimalarial therapeutics, integrating molecular immunology with precision medicine approaches.

Further, the cytoskeletal integration of CR1, mediated via scaffolding proteins such as FAP-1 and regulated by ATP-dependent signaling, suggests avenues for enhancing erythrocyte resilience under stress conditions (Ghiran et al., 2008; Melhorn et al., 2013). Pharmaceutical strategies that stabilize receptor-cytoskeletal interactions could optimize immune complex transport while preserving erythrocyte deformability, a critical factor in microvascular perfusion and systemic immunity. The potential applications of such approaches are broad, encompassing infectious disease, autoimmune disorders, and systemic inflammatory conditions where complement overactivation contributes to pathology.

Comparative studies across species have revealed conserved mechanistic features of CR1 that may inform translational research and therapeutic development. In cattle, co-expression of CR1 and CR2 suggests a potential compensatory mechanism in immune complex clearance, whereas in porcine and avian models, erythrocyte immune adherence exhibits both structural and functional diversity (Pringle et al., 2012; Sun et al., 2012; Li et al., 2007). These findings indicate that translational strategies must account for both evolutionary conservation and species-specific nuances when designing CR1-targeted interventions. In humans, interindividual variability in receptor density, polymorphic alleles, and signaling responsiveness necessitates precision approaches, potentially incorporating genomic and phenotypic profiling to optimize therapeutic outcomes.

Another promising avenue involves CR1-targeted modulation in autoimmune and inflammatory disorders. Excessive complement activation contributes to tissue injury in conditions such as systemic lupus

erythematosus, rheumatoid arthritis, and inflammatory bowel disease. By leveraging sCR1 or receptor-mimetic constructs, it is theoretically possible to attenuate complement-mediated damage without broadly suppressing systemic immunity. Early studies demonstrate that sCR1 reduces neutrophil infiltration, microvascular permeability, and inflammatory cytokine production, supporting its potential as a targeted immunomodulator (Chavez-Cartaya et al., 1995; Xiao et al., 1997). Moreover, such approaches could synergize with existing biologics or small-molecule inhibitors, enabling combination therapies that address both upstream complement activation and downstream effector mechanisms.

CR1-targeted therapeutics also raise important considerations regarding dosage, pharmacokinetics, and potential immunogenicity. Soluble receptor constructs must achieve sufficient systemic levels to inhibit complement without compromising erythrocyte-mediated clearance of immune complexes. Additionally, prolonged receptor modulation may alter immune homeostasis, potentially impacting pathogen susceptibility or the resolution of inflammation. These challenges highlight the necessity for carefully designed preclinical studies and early-phase clinical trials, incorporating longitudinal monitoring of complement activity, immune complex processing, and erythrocyte functionality.

Innovative delivery strategies, including nanoparticle-based carriers or targeted erythrocyte surface modifications, may enhance therapeutic specificity and reduce off-target effects. For example, conjugation of sCR1 to erythrocytes or biocompatible carriers could facilitate localized complement inhibition at sites of vascular injury while preserving systemic immune surveillance. Similarly, receptor-mimetic peptides or small molecules that stabilize CR1 clustering or enhance ligand binding could serve as adjunctive therapies in infectious or inflammatory diseases. Integration of these strategies with high-resolution imaging, computational modeling, and molecular diagnostics will be essential to optimize therapeutic efficacy and safety.

In addition to direct therapeutic applications, CR1 function provides a biomarker for disease susceptibility and treatment monitoring. Variations in receptor density, allelic expression, and clustering dynamics can inform risk stratification for infectious diseases, cardiovascular events, and complement-mediated tissue injury. For instance, patients with lower erythrocyte CR1 density or unfavorable polymorphic variants may exhibit increased susceptibility to severe malaria or heightened ischemia-reperfusion injury, guiding prophylactic or interventional strategies (Rowe et al., 1997; Vik & Wong, 1993). Furthermore, monitoring changes in CR1-mediated immune adherence during treatment could serve as a functional biomarker for therapeutic efficacy, complement blockade, or resolution of systemic inflammation.

The translational implications of CR1 extend to emerging infectious diseases where complement dysregulation contributes to pathogenesis. Viral infections, including those caused by coronaviruses or flaviviruses, often involve complement-mediated tissue injury and hyperinflammatory states. Leveraging CR1 modulation may mitigate endothelial damage, reduce immune complex deposition, and attenuate systemic inflammatory responses, offering a precision-targeted strategy to complement broader antiviral therapies. Preclinical studies integrating molecular, immunological, and clinical endpoints will be critical to validate these approaches and establish mechanistic clarity regarding receptor-mediated protection.

In summary, the therapeutic potential of CR1 encompasses a diverse array of clinical scenarios, including ischemia-reperfusion injury, infectious diseases, autoimmune disorders, and complement-mediated inflammatory conditions. Strategies leveraging soluble CR1, receptor mimetics, and cytoskeletal stabilization offer targeted modulation of complement activity, immune adherence, and erythrocyte function. Comparative and translational insights underscore the necessity of precision approaches, integrating genetic, molecular, and functional profiling to optimize outcomes. Future research should prioritize mechanistic elucidation, longitudinal assessment of receptor-mediated effects, and innovative delivery platforms to fully realize the clinical promise of CR1-targeted interventions. By bridging fundamental immunology with translational application, CR1 represents both a molecular sentinel and a therapeutic lever in the modulation of human immunity and tissue protection.

5. Conclusion

Complement receptor 1 serves as a central mediator of erythrocyte immune function, orchestrating processes that integrate molecular, cellular, and systemic immunological responses. CR1 clustering, mediated by scaffolding proteins and ATP signaling, enhances

immune complex adherence, erythrocyte deformability, and pathogen clearance. Polymorphic variation influences receptor function and disease susceptibility, with translational implications in infectious and complement-mediated pathologies. Therapeutic modulation, particularly through soluble CR1, demonstrates protective effects in ischemia-reperfusion injury, highlighting the receptor's clinical relevance. Comprehensive understanding of CR1 dynamics, informed by molecular, comparative, and translational research, offers a robust framework for advancing immunological science and developing targeted interventions. Continued exploration of CR1 mechanisms promises to illuminate fundamental principles of immune regulation and inform innovative therapeutic strategies across a spectrum of disease contexts.

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