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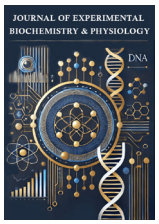

Brucella Bp26: A Multifunctional Protein Bridging Pathogenesis and Immunodiagnostics

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Article Info

Articlehistory:

Received: 10November 2014
Editor: 12November2014
Reviewed: 02December2014
Revised: 11December2014
Published: 22December 2014

Abstract

Strength training is a potent physiological stimulus that induces numerous adaptations across multiple biological systems, including the immune system. The immune response to strength training is highly dynamic and depends on various factors such as training intensity, duration, frequency and the individual's baseline fitness level. Understanding the interplay between strength training and immune function is crucial for optimizing athletic performance, promoting general health and preventing adverse immune-related consequences. One of the primary immune adaptations observed during strength training is the modulation of leukocyte subpopulations. Acute bouts of resistance exercise typically led to transient increases in circulating neutrophils, monocytes and lymphocytes due to catecholamine release and mechanical stress on the muscle. However, chronic strength training induces more stable immunomodulatory effects, often enhancing the functional capacity of immune cells and improving overall immune surveillance. These adaptations may contribute to reduced infection risk and enhanced recovery from training-induced muscle damage.The implications of immune adaptation in strength-trained individuals extend beyond athletic performance. Regular strength training has been linked to improved immune function in older adults, contributing to enhanced resistance against infections and age-related immune decline. Additionally, individuals with metabolic disorders or chronic inflammatory conditions may benefit from the immunomodulatory effects of resistance exercise, supporting its role as a non-pharmacological intervention for overall health maintenance. In conclusion, strength training exerts profound effects on immune function, promoting both acute and chronic adaptations that influence leukocyte activity, cytokine balance and humoral immunity. Understanding these immune responses is essential for athletes, healthcare professionals and the general population to optimize training benefits while minimizing immune-related risks. Future research should focus on individual variability in immune responses and the long-term consequences of strength training on immune resilience and disease prevention.

Description

The *Brucella* genome encodes a variety of surface-associated proteins that are likely involved in host-pathogen interactions. Among these, the 26-kDa protein Bp26 has been extensively studied for its strong immunogenicity, making it a valuable diagnostic marker for brucellosis. While its diagnostic utility is well-established, the functional role of Bp26 in *Brucella* pathogenesis, particularly its potential to act as an adhesin, has been less understood. The *in vitro*interaction of Bp26 with selected ECM molecules, including type I collagen, fibronectin, vitronectin and laminin [1]. Their objective was to determine if Bp26 could contribute to *Brucella* adhesion to host tissues. Several key findings regarding the interaction of Bp26 with ECM molecules. Using Enzyme-Linked Immunosorbent Assays (ELISA), the researchers demonstrated that Bp26 bound in a dose-dependent manner to both immobilized type I collagen and vitronectin [2]. Interestingly, Bp26 exhibited weak binding to soluble fibronectin but did not bind to immobilized fibronectin, and no binding to laminin was detected in either immobilized or soluble form [3]. These findings were further corroborated by Biolayer Interferometry (BLI) analysis, which showed a high binding affinity between Bp26 and immobilized type I collagen and significant binding to soluble vitronectin, while confirming the lack of binding to fibronectin or laminin. To pinpoint the specific regions on Bp26 responsible for these interactions, the researchers performed epitope mapping using biotinylated overlapping peptides [4]. This analysis identified five linear epitopes on Bp26. Collagen and vitronectin bound to peptides from several regions, with the strongest binding observed at the C-terminus of Bp26, where the binding sites for these two ECM components overlapped [5]. Notably, fibronectin did not bind to any of the tested peptides, despite its weak interaction with the whole Bp26 protein. A comprehensive assessment of Bp26-ECM

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Citation: Webb E (2014). *Brucella* Bp26: A Multifunctional Protein Bridging Pathogenesis and Immunodiagnostics. J Exp Bio Physiol; 1:005.

interactions. ELISA, a sensitive assay for detecting protein-protein interactions, allowed for the quantitative measurement of Bp26 binding to both immobilized and soluble ECM molecules under various concentration conditions [6]. The dose-dependent binding observed for collagen and vitronectin provides strong evidence for specific interactions. Biolayer interferometry, a label-free technique that monitors biomolecular interactions in real-time, served as a valuable confirmatory method, providing kinetic parameters such as binding affinity. The high affinity of Bp26 for collagen determined by BLI underscores the stability and potential biological significance of this interaction. Epitope mapping using overlapping synthetic peptides enabled the identification of linear binding sites on Bp26 for the ECM molecules and anti-Bp26 antibodies. This technique is particularly useful for identifying specific amino acid sequences involved in binding. The subsequent mapping of these binding sites onto the known three-dimensional structure of Bp26 offered crucial insights into the accessibility and potential mechanism of these interactions [7]. The observation that the strongest binding region, located at the C-terminus, is partially exposed on the hexadecameric Bp26 complex suggests its accessibility for interaction with ECM components. A specific binding profile of Bp26 to ECM molecules. The robust and consistent binding to both immobilized and soluble type I collagen and vitronectin, as demonstrated by ELISA and BLI, indicates a significant affinity for these two ECM components [8]. Type I collagen is a major structural protein found in various connective tissues, while vitronectin is present in blood and the ECM and is involved in cell adhesion and migration.

The capacity of Bp26 to interact with these molecules suggests its potential involvement in *Brucella*'s attachment to a wide range of host tissues and potentially in its dissemination through the bloodstream [9]. The differential binding to fibronectin, with weak interaction observed only in its soluble form, implies that the conformation or presentation of fibronectin might be crucial for this interaction. Immobilization might alter the structure of fibronectin, rendering the Bp26 binding site inaccessible. The absence of binding to laminin, a key component of the basement membrane, suggests that Bp26's role in adhesion might be selective for specific tissue types or stages of infection. The study's comparison with previous research on B. abortus highlights the complexity of *Brucella*'sinteraction with the ECM, suggesting that multiple bacterial surface proteins likely contribute to adhesion, with varying specificities for different ECM components [10]. The ability of Bp26 to bind to collagen and vitronectin *in vitro* supports its potential role as an adhesin in *Brucella*pathogenesis. Adhesion to these ECM components could facilitate the initial attachment of *Brucella* to host tissues, a crucial step for subsequent colonization and invasion of host cells. However, the precise subcellular localization of Bp26 remains a subject of debate, with some studies suggesting it's an outer membrane protein while others indicate a periplasmic location. If Bp26 is indeed periplasmic, the mechanism by which it interacts with extracellular ECM molecules is not fully understood. Possible explanations include secretion of Bp26 or its release upon bacterial lysis, allowing it to mediate adhesion. *Brucella*expresses a range of other adhesins that contribute to its interaction with host cells and the ECM, including proteins like SP29, SP41, BigA, BigB, BmaA, BmaB, BmaC, BtaE and BtaF, which exhibit diverse binding specificities. Bp26 likely functions in concert with these other adhesins to facilitate the complex process of infection. While some studies suggest that Bp26 contributes to *Brucella* virulence, further research is needed to fully elucidate its role *in vivo*. Beyond its potential role in pathogenesis, Bp26 is a well-established diagnostic marker for brucellosis due to its high immunogenicity. Its specificity makes it particularly useful for serodiagnosis and for differentiating infected from vaccinated animals when using Bp26-deleted vaccines. The continued development and application of Bp26-based diagnostic assays, such as ELISAs, are crucial for effective brucellosis surveillance and control. Furthermore, if Bp26 plays a significant role in adhesion, it could potentially serve as a target for novel therapeutic interventions aimed at blocking *Brucella*'s interaction with host tissues. *In vivo*studies using bp26 mutant strains in animal models are essential to confirm the biological relevance of the observed ECM binding and to assess the impact of Bp26 on *Brucella*pathogenesis. Clarifying the precise subcellular localization of Bp26 during infection will also be critical for understanding its mechanism of action. Investigating the functional significance of the hexadecameric structure of Bp26 and the reasons behind its differential binding to fibronectin could reveal additional roles for this protein. Finally, exploring the potential for Bp26 to directly interact with host cell surface receptors could provide a more complete understanding of its involvement in the early stages of *Brucella*-host interaction.

Conclusion

The ability of *Brucella* protein Bp26 to bind to specific extracellular matrix molecules, particularly type I collagen and vitronectin. These findings suggest that Bp26 may function as an adhesin, contributing to *Brucella*'s interaction with host tissues during infection. While Bp26's role as a diagnostic marker is well-established, this research highlights its potential involvement in pathogenesis, underscoring the multifaceted nature of this protein in the context of brucellosis. Further research, especially *in vivo*, is warranted to fully understand the complex role of Bp26 and its potential as a target for future therapeutic strategies against this significant zoonotic disease.

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