Lysozymes constitute an ancient family of proteins that are important components of the innate immune system of animáis. They hydrolyze the 1,4-P-linkages between N -acetyl-d-glucosamine and N-acetylmuramic acid in the peptidoglycan of bacterial cell walls, and are part of a nonimmunological ancestral bactericidal system in vertebrates. Hydrolysis of peptidoglycan compromises cell wall integrity, causing cell lysis and bacterial death. Most bacteria engaged in commensal or pathogenic interactions with an animal host have evolved various strategies to evade lysozyme action. These uiclude shielding by the outer membrane, in the case of most gram-negative bacteria, peptidoglycan modification, or the more recently emerged production of lysozyme inhibitors. In the case of Brucella, añer invasión of the host, Brucella is phagoc5^osed by neutrophils (PMNs), macrophages, and dendritic cells. Brucella spp. resists a wide range of bactericidal cationic peptides and lysozyme, and is able to prevent lysosome fiision - and to lócate in autophagosome-like compartments. Moreover, B. abortus cells elicit little respiratory burst and only reduced levéis of degranulation in PMNs, thus contributing to a limited exposure of Brucella to lysozyme. We have identified a gene that encodes for a putative lysozyme inhibitor of the MhC family in B. abortus 2308. The gene was overexpressed and purifíed as an Histagged protein, and the protein activity was determined in vitro against suspensions of Microccocus fysodeitucus, confírming that it is a real lysozyme inhibitor. The role of this inhibitor in resistance of Brucella to lysoz3ane has been studied in a knock-out mutant, both in vitro as in vivo, using purifíed lysoz3Tne, macrophages and human neutrophils.