

ImmunoTools *special* Award 2014



Pablo Baldi, PhD

IDEHU, Immunology Department, School of Pharmacy and Biochemistry, University of Buenos Aires, Argentina

Characterization of *Brucella* interaction with the respiratory system

Brucella spp. are gram negative bacteria that infect several domestic and wild animals, and can be transmitted to humans through different ways, thus constituting a zoonosis. Brucellosis is worldwide distributed, affecting over 500,000 people annually. *Brucella melitensis*, *B. suis* and *B. abortus* are the most pathogenic species for humans and are responsible for the vast majority of human cases. The infection is usually transmitted to humans by direct contact (usually through mucosae) with infected animal tissues, including dairy products. Inhalation is one of the most important ways of contagion and, probably due to this fact, brucellosis is the leading laboratory-acquired bacterial infection.

In spite of the importance of the airways for *Brucella* entry to the organism, the respiratory route of infection has been explored in only a few animal studies. The interaction of *Brucella* with the pulmonary tissues has been scarcely studied. The aim of our studies is to characterize the cell populations that interact with *Brucella* spp. after the inhalatory infection, and to establish the kinetics of the inflammatory response mounted in the lung after *Brucella* entry. To accomplish these goals it will be necessary to label the cells in lung and bronchoalveolar lavage (BAL) samples using antibodies against well characterized cellular markers including CD3, CD4, CD8, CD11b, and CD25. In addition, it would be of interest to study in vitro the effect of recombinant proinflammatory cytokines and chemokines such as IL-1beta, TNF-alpha, IFN-gamma and CCL20, on the ability of different primary pulmonary cells to contribute to the control of *Brucella* infection.

While the use of the mouse model is a necessary tool to study the kinetics of *Brucella* respiratory infection in an intact animal, the study of the interaction of *Brucella* with human respiratory cells is very important taking into account the importance of the inhalatory route for the contagion of brucellosis in humans. In this sense, it would be interesting to determine whether the different cell types elicit an inflammatory response to the in vitro infection with *Brucella*, including the production of IL-6, IL-8

IL-12 and TNF-alpha. In the innate branch of immunity, the production of IL-6 and TNF-alpha may be important for activating the endothelium close to the infection site, thus promoting the migration of phagocytes. In a similar way, the neutrophil-attracting chemokine IL-8 may be produced and may contribute to recruit these phagocytes to the site of infection. Regarding the adaptive branch of immunity, the Th1 response is of utmost importance for controlling *Brucella* infection due to the intracellular nature of this bacterium. It is widely known that IL-12 promotes the differentiation of Th0 cells to the Th1 phenotype. In agreement with this concept, several studies using mouse models have shown that IL-12 is essential for controlling *Brucella* infection. These studies may help to establish whether the early immune response in the lung contributes to the elicitation of a potentially protective Th1 response to *Brucella*. As in the case of the mouse model, it would be of interest to study in vitro the effect of recombinant proinflammatory cytokines and chemokines such as IL-1beta, TNF-alpha, IFN-gamma, on the ability of different primary pulmonary cells to contribute to the control of *Brucella* infection

In summary, **ImmunoTools** reagents could help us to characterize the innate and adaptive immune response of murine and human lung cells to *Brucella* infection.

ImmunoTools special AWARD for **Pablo Baldi** includes 24 reagents
recombinant human cytokines: rh IFNgamma, rh IL-1beta, rh TNFalpha,
human IL-8 ELISA-set for 96 wells, human IL-12p40 ELISA-set for 96 wells,
human TNFa ELISA-set for 96 wells (each 3 reagents),
FITC - conjugated anti-mouse CD62L, Gr-1, isotype control IgG2b,
PE - conjugated anti-mouse CD3e, CD11b, isotype control IgG2b,
APC- conjugated anti-mouse CD25, NK, isotype control 2b
recombinant mouse cytokines: rm IFNgamma, rm IL-1beta, rm MIP3a / CCL20,

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