LXXI REUNIÓN ANUAL
DE LA SOCIEDAD ARGENTINA DE INMUNOLOGÍA

9 al 11 de noviembre de 2023 / San Luis

Sociedad Argentina de Inmunología

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Camino del Macizo Central de San Luis
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Macizo de San Luis: cima de las Sierras de San Luis, a 2088m sobre el nivel del mar, con vistas inolvidables de cerros, quebradas, valles y pequeñas mesetas de altura. (Extraído de Ser Argentino.com).

Hebe Iriarte: Microbióloga, docente en el Área Microbiología e Inmunología de la Universidad Nacional de San Luis, personal técnica de apoyo de CONICET. Fotógrafa profesional, realiza fotografías de flora y fauna.

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39 (50) THE DOWN-MODULATION OF IFN-γ-INDUCED MHC-I EXPRESSION BY *BRUCELLA ABORTUS* RNA IN CALU-6, HMEC AND A-549 CELLS SHARES FEATURES WITH MONOCYTES/MACROPHAGES'

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Brucella abortus (*Ba*) is an intracellular pathogen capable of surviving inside macrophages. Since the disease is presented in multiple forms, many different cells are susceptible to be infected by *Ba*. We previously demonstrated that *Ba* RNA is a *vita*-PAMP involved in the immune evasion mediated by this pathogen. One of the mechanisms displayed by *Ba* is the down-modulation of MHC-I on monocytes/macrophages when Th1 response is being held, *i.e*., in the presence of IFN-γ. Moreover, MHC-I total expression is not altered, instead these proteins are retained within the Golgi Apparatus (GA). More recently, we demonstrated that *Ba* RNA diminishes the IFN-γ-induced MHC-I surface expression in other cells able to be infected with *Ba.* However, we do not know if this phenomenon is due to the retention of MHC-I within the GA by *Ba* RNA, as occurs in human macrophages, their preferential niche. To evaluate this, we stimulated the human bronchial epithelium cell line (Calu-6), the human alveolar epithelium cell line (A-549) and the endothelial microvasculature cell line (HMEC) with 10 µg/ml of *Ba* RNA in the presence of IFN-γ. After 48 h, MHC-I expression and GA marker GM130 were detected by confocal microscopy. We observed that *Ba* RNA induces colocalization of MHC-I and GM130 in Calu-6 and HMEC cells. However, no colocalization was detected in A-549 cells. Then, we evaluated the effect of *Ba* RNA on the secretion of IL-8, IL-6 and MCP-1. For this, Calu-6, A-549 and HMEC cells were stimulated with *Ba* RNA (1, 5 and 10 µg/ml) in the presence of IFN-γ for 48 h. Afterwards, supernatants were collected and the secretion of IL-8, IL-6 and MCP-1 was quantified by sandwich ELISA. We did not observe any changes in MCP-1 in *Ba* RNA-treated cells. Conversely to what we expected, Calu-6, HMEC and A-549-*Ba* RNA-treated cells had higher IL-8 and IL-6 levels compared to those from untreated cells ($p<0.05$). In addition, our previous results indicate that *Ba* RNA inhibits the IFN-γ-induced MHC-I surface expression on human monocytes/macrophages by a TLR8-dependent mechanism and through the Epidermal Growth Factor Receptor (EGFR) pathway. In order to extend this finding Calu-6, A-549 and HMEC cells were stimulated with 10 µg/ml of *Ba* RNA in the presence of IFN-γ for 48 h. TLR-8 expression was confirmed by flow cytometry in all cell lines. Next, cells were stimulated with 10 µg/ml of *Ba* RNA in the presence of an EGFR ligand-blocking antibody (Cetuximab). Neutralization of the EGFR partially reversed the inhibition of MHC-I surface expression mediated by *Ba* RNA in HMEC and A-549 cells. Overall, these results show that the down-modulation of MHC-I expression by *Ba* RNA in different cells susceptible to be infected by *Ba* would allow the bacteria to persist successfully within the host, remaining unnoticed and evading $CD8^+$ T cell surveillance.