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
de Hebe Iriarte

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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88 (201) PROTEOME RADIOLABELING ASSAYS REVEAL THAT INFECTION OF THP1 MACROPHAGES WITH DIFFERENT BACTERIA UP-REGULATES A NOVEL CYTOSOLIC VARIANT OF AN ER CHAPERONE, SHOWING A pI-REDUCING PTM OF UNKNOWN STRUCTURE. DEPENDENCY ON TLR2-LIGATION AND MICROTUBULES.

Cristian J. A. Asensio [1, 2] , Michael P. Myers [2] , Francisco E. Baralle [2], Rodolfo C. García [2]
[1]CONICET, Argentina. [2]ICGEB, International Centre For Genetic Engineering and Biotechnology, Trieste, Italy.

Finding proteins altered in level/PTMs during bacterial infection in macrophages requires sensitive screening protocols in gels, outperforming dyes. We searched cytosolic proteins altered in a time-dependent, sustained manner, at days 1-4 post-infection (pi). Thus, we optimized cell-free, *in vitro* radiolabeling (IVR) assays of cytosolic proteomes of THP1 cells to covalently label some proteins with P-32. Proteomes were resolved by 1D/2D gels to detect bands/spots with altered IVR. 3-4 experiments were performed per condition. Interesting proteins were identified by MS. Bibliometric and informatic studies were initiated to interpret the findings. RESULTS: After infection with *Mycobacterium avium*, a labeled 78 KDa band, p78, was always upregulated at day 1 pi, equally by live or heat-killed bacteria. Time-course infection experiments revealed that live bacteria sustained p78 longer, with a 2-fold difference with killed, at day 4. So, p78 was sustained by live intracellular bacteria. The p78 spot had pI=3.9 in 2D gels. A stained spot was identified by MS as HSPA5, an ER chaperone (normal pI=5.3). So, p78 was an ultra-acidic, cytosolic, charge variant of HSPA5, but with similar mobility in gels. p78 was upregulated at day 1 also by 2 gram-negative bacteria (live or killed). Thus, p78 was likely a conserved cell response to heat-resistant molecules of different bacteria. Since it did suggest participation of TLRs, we treated cells with pure TLR2 ligands like lipopeptides, LTA, etc., upregulating p78 again. Contrary to the normal HSPA5 precursor, p78 was ER-stress independent. So, p78 might arise from a minor precursor pool after addition of a PTM. p78 was undetectable in WB and IVR outperformed silver and dyes in sensitivity. We improved the IVR to monitor p78 only in 1D gels and identified a kinase labeling p78 by IVR (did not label HSPA5). Taxol treatment revealed that microtubules were involved with p78. LPS as TLR4 ligand did not generate p78. TLR2 negative HeLa cells had no upregulation. The 1.4 pH units shift, without intermediate spots, would imply a single-step polyanionic PTM, incompatible with multisite phosphorylation. Besides, a phosphate-binding dye did not detect p78. So far, the PTM escaped MS identification. We conclude that any candidate PTM should comply these characteristics: a) be poly-anionic and/or neutralize many basic HSPA5 residues, b) generate a reproducible pI reduction, maintaining equal mobility, c) compatibility with the spatiotemporal aspects of the upregulation plus association to microtubules, d) TLR2 ligation dependency, e) ER-stress and ligand-structure independency, f) if more than 1 enzyme is involved, they should be spatiotemporally coordinated. We discuss the literature about candidate PTMs, together with reports of other HSPA5 forms. p78 in THP1 is a reproducible biomarker useful to compare different TLR2 ligands and bacteria and deserves more studies about its roles in innate immune responses and its PTM.