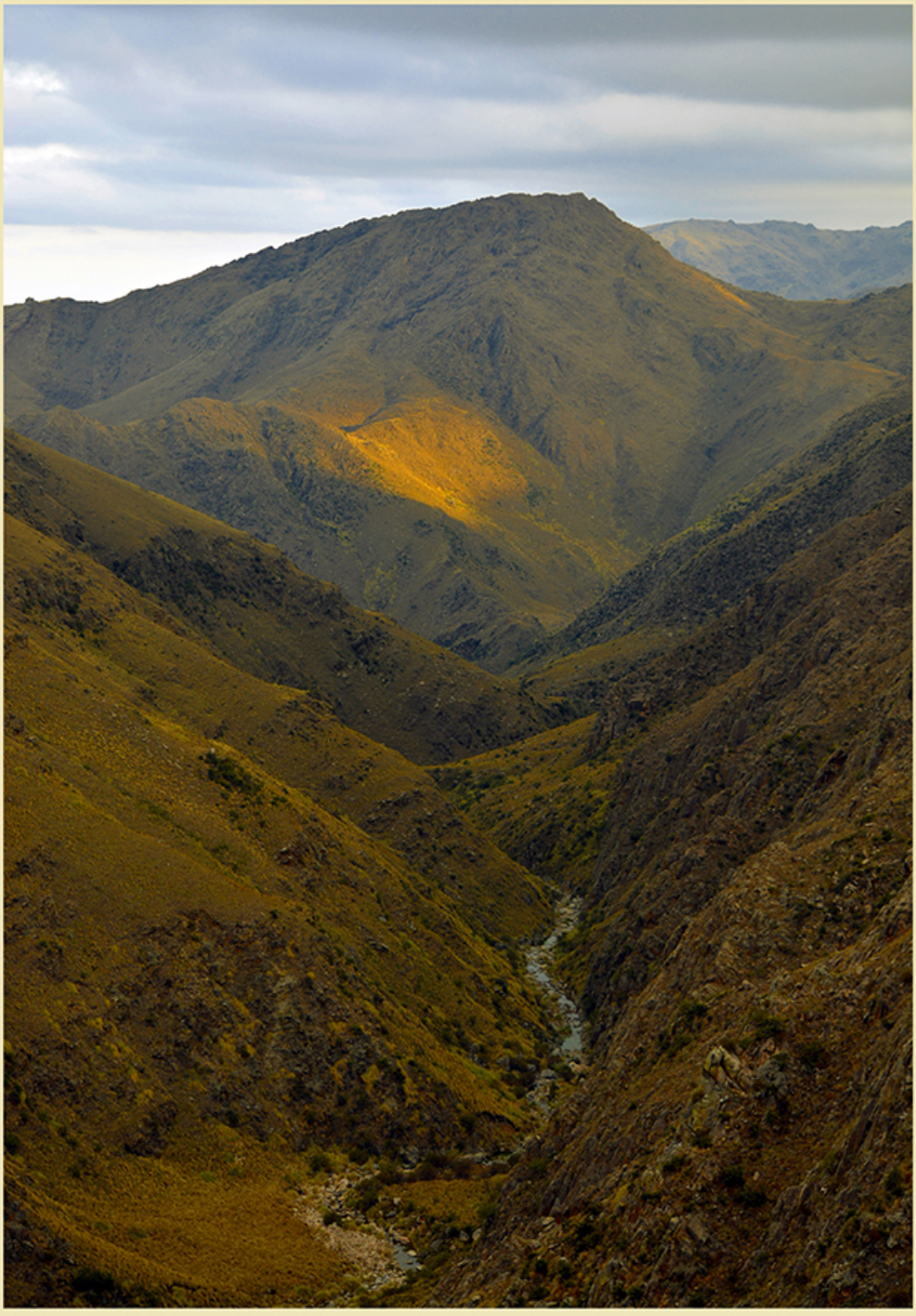
LXXI REUNIÓN ANUAL
DE LA SOCIEDAD ARGENTINA DE INMUNOLOGÍA

9 al 11 de noviembre de 2023 / San Luis



Sociedad Argentina de Inmunología

FQBF UNSL

neu
nueva editorial universitaria

50 años
Universidad Nacional de San Luis

LXXI REUNIÓN CIENTÍFICA ANUAL DE LA SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

9 -11 de noviembre de 2023

Universidad Nacional de San Luis-San Luis

LXXI ANNUAL MEETING OF THE ARGENTINEAN SOCIETY OF IMMUNOLOGY (SAI)

November 9 - 11, 2023

Universidad Nacional de San Luis-San Luis

EDITORES RESPONSABLES

Comisión Directiva SAI
María Silvia Di Genaro

LXXI REUNIÓN CIENTÍFICA ANUAL DE LA SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

9 -11 de noviembre de 2023
Universidad Nacional de San Luis-San Luis

LXXI ANNUAL MEETING OF THE ARGENTINEAN SOCIETY OF IMMUNOLOGY (SAI)

November 9 - 11, 2023
Universidad Nacional de San Luis-San Luis

RESPONSIBLE EDITOR
Comisión Directiva SAI
María Silvia Di Genaro

50 años
Universidad
Nacional
de San Luis

LXXI Reunión Científica Anual de la Sociedad Argentina de Inmunología SAI / María Silvia Di Genaro... [et al.]; compilación de María Silvia Di Genaro - 1a ed. - San Luis: Nueva Editorial Universitaria - UNSL, 2023. Libro digital, PDF

Archivo Digital: descarga y online
ISBN 978-987-733-386-2

1. Inmunología. I. Di Genaro, María Silvia, comp.
CDD 616.979

Universidad Nacional de San Luis
Rector: CPN Víctor A. Moriñigo
Vicerrector: Mg. Héctor Flores

Nueva Editorial Universitaria
Avda. Ejército de los Andes 950
Tel. (+54) 0266-4424027 Int. 5197 / 5110
www.neu.unsl.edu.ar
E mail: unslneu@gmail.com

Directora: Lic. Jaquelina Nanclares

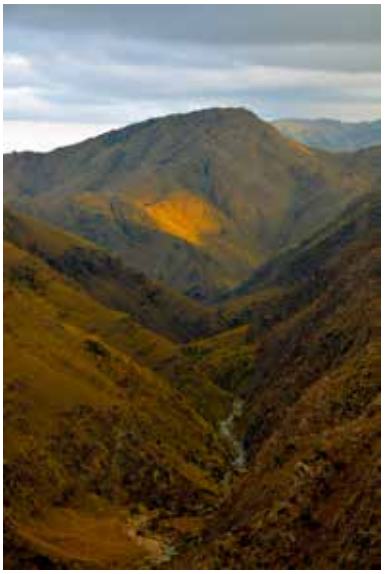
Director Administrativo: Sr. Omar Quinteros

Administración: Esp. Daniel Becerra

Dpto de Imprenta: Sr. Sandro Gil

Dpto. de Diseño: Tec. Enrique Silvage - DG Nora Aguirre

Foto de tapa:
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.

ISBN 978-987-733-386-2
Queda hecho el depósito que marca la ley 11.723
© 2023 Nueva Editorial Universitaria
Avda. Ejército de los Andes 950 - 5700 San Luis

85 (86) NOVEL, SENSITIVE, *IN VITRO* RADIOLABELING ASSAYS ALLOW THE MONITORING OF CYTOSOLIC VIMENTIN PROTEOFORMS BY SDS-PAGE IN NON-INFECTED, MYCOBACTERIAL-INFECTED AND TLR2-LIGATED THP-1 CELLS. POSSIBLE VIMENTIN ROLES IN MONOCYTE TO MACROPHAGE DIFFERENTIATION, INFECTION AND INFLAMMATION.

Cristian J. A. Asensio[1,2,3], Stefania Zampieri[2], Karim Zuppan[2], Corrado Guarnaccia[2], Alessandro Vindigni[2], Renato A. S. Oliveira[2], Jerry Saklatvala[3], Francisco E. Baralle[2], Rodolfo C. García[2]

[1]CONICET, Argentina. [2]ICGEB, International Centre For Genetic Engineering and Biotechnology, Trieste, Italy. [3]Kennedy Institute of Rheumatology, UK.

Discovering low-abundant macrophage proteins/proteoforms, altered in level/PTMs during intracellular bacterial infection and innate immune responses, needs sensitive proteome screening tools in electrophoretic gels, but outperforming dyes. AIMS: to search for, in human THP1 macrophage-like cells, cytosolic proteins reproducibly altered in a time-dependent and sustained manner, at days 1-4 post-infection with mycobacteria (live or killed). METHODS: The cytosolic fraction was obtained and used in novel, post-cell harvest, cell-free, *in vitro* radiolabeling (IVR) assays, allowing the covalent labeling of cytosolic proteomes with P-32. Labeled proteomes were separated in 1D/2D gels to detect bands/spots with altered labeling, normalizing them against total stained and total labeled proteomes. Proteins of interest were identified by MS and characterized. Bibliometric and bioinformatic studies were initiated to interpret findings in terms of PTMs, protein-protein interactions and possible roles of altered proteins and to plan how IVR might help future studies. RESULTS: in all 12 time-course infection experiments, cytosolic vimentin (VIM) was upregulated by infection in a time-dependent manner. In 3 monocytic- to-macrophage differentiation experiments (PMA-treated, non-infected), the VIM IVR increased during 4 days. We identified cytosolic kinases allowing detection of VIM with cleaved forms. Metabolic labeling in cell culture detected VIM profiles different to IVR. In WB, different antibodies and sera against other proteins often did bind non-specifically to VIM. So, to monitor minor cleavage/expression changes in VIM, IVR was more sensitive, quantitative and robust than WB. The literature indicated that VIM: **a)** is emerging as a multifunctional protein located in the perinuclear area, cytosol, endosomes, viral factories, cell surface, extracellular space and blood; **b)** has roles in auto-/xeno-/aggre-phagy, apoptosis, scaffolding of signaling complexes and in binding to DNA, RNA, phospholipids, O-GlcNAc, Rab7a, p62, HDAC6, MTOC, NFκB, NOD2, NLRP3, ERK; **c)** is a modulator of infectious, immune, autoimmune, inflammatory, cell stress, and fibrotic responses and is a target of toxins from many bacteria; **d)** has roles other than the cytoskeletal/mechanical by using different PTMs and by assembling as 1-, 2- and 4-mers, cages, and filamentous networks; **e)** Surface VIM binds many bacteria and viruses including SARS-Cov-2; **f)** Non-specific WB signals might depend on VIM-Fc and/or on citrullinated VIM-Ab interactions; **g)** VIM can be cleaved in cells. CONCLUSIONS: IVR helped detecting dynamic changes in cytosolic VIM levels, complementing WB. IVR would help to study VIM functional diversity, to correlate VIM alterations with those in binding partners, and to study VIM as biomarker or drug-target in cell infection and/or differentiation. Dissecting pro-infection and infection-restricting VIM roles will improve our knowledge of host–pathogen interaction complexity.