2018

# medicina

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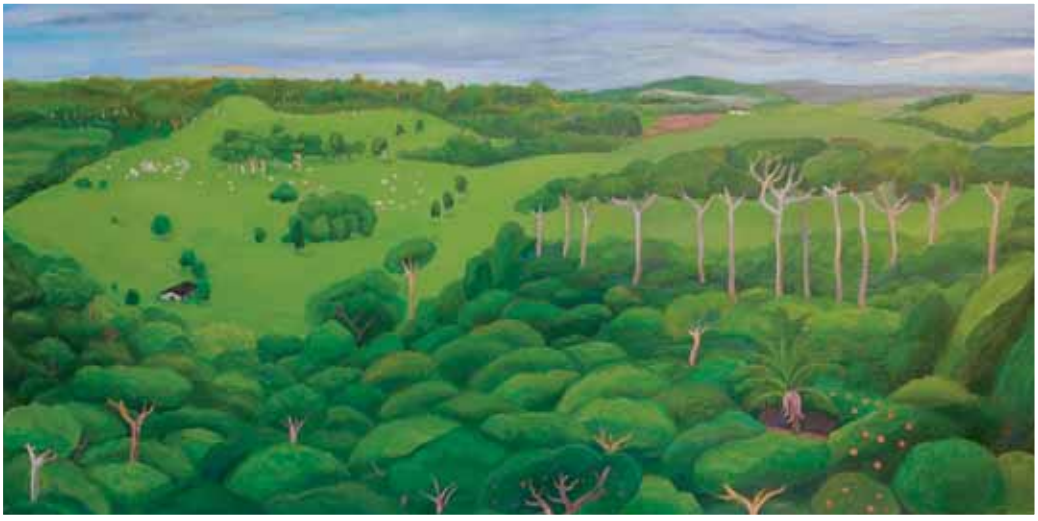

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## COMITÉ DE REDACCIÓN



La Tapa (Ver p xx)
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# REUNIÓN CONJUNTA SAIC SAI SAFIS 2018

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## LXVI REUNIÓN ANUAL DE LA

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**14-17 de noviembre de 2018**
**Hotel 13 de Julio – Mar del Plata**

EDITORES RESPONSABLES

Claudia Pérez Leirós
Pablo Baldi
Alberto Crottogini

also showed that the inhibition of caspase-1 and neutralization of IL-18 did not affect neutrophil-mediated modulation. By contrast, the treatment with serine proteases inhibitors prevented the potentiation of γδ T cell activation induced by neutrophils. Moreover, the addition of elastase to γδ T cell culture increased their stimulation, and the treatment of neutrophils with elastase inhibitor prevented the effect of neutrophils on γδ T cell activation. Furthermore, we demonstrated that the effect of elastase on γδ T cells was mediated through the proteases-activated receptor, PAR1, since the inhibition of this receptor with a specific antagonist, RWJ56110, abrogated the effect of neutrophils on γδ T cell activation.

## YOUNG RESEARCHER PRESENTATION

### CRITICAL ROLE FOR SEC22B-DEPENDENT ANTIGEN CROSS-PRESENTATION IN ANTI-TUMOR IMMUNITY

**Andrés Alloatti**
*IDICER-CONICET/UNR, Rosario, Argentina*

$CD8^+$ T cells mediate antigen-specific immune responses that can induce rejection of solid tumors. In this process, dendriticcells (DCs) are thought to take up tumor antigens, which are processed into peptides and loaded onto MHC-I molecules, aprocess called “cross-presentation”. Neither the actual contribution of cross-presentation to anti-tumor immune responses northe intracellular pathways involved *in vivo* are clearly established because of the lack of experimental tools to manipulate thisprocess. To develop such tools, we generated mice bearing a conditional DC-specific mutation in the sec22b gene, a criticalregulator of endoplasmic reticulum–phagosome traffic required for cross-presentation. DCs from these mice show impairedcross-presentation *ex vivo* and defective cross-priming of $CD8^+$ T cell responses *in vivo*. These mice are also defective for anti-tumorimmune responses and are resistant to treatment with anti–PD-1. We conclude that Sec22b-dependent cross-presentation in DCs is required to initiate $CD8^+$ T cell responses to dead cells and to induce effective anti-tumor immune responsesduring anti–PD-1 treatment in mice.

## SAFIS SYMPOSIUM: ADVANCES IN RENAL PHYSIOLOGY AND PATHOPHYSIOLOGY

### NEW BIOMARKERS OF ACUTE KIDNEY INJURY

**Adriana M. Torres**
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Acute kidney injury (AKI) is common in intensive care units. The mortality rate for hospitalized patients who develop AKI is approximately five times higher than without AKI. Despite the introduction of new therapies, the mortality associated with this pathology has improved little over the last years. The routinely available clinical parameters of kidney disease (plasma creatinine and urea) do not provide in practice either a sensitive or specific indication of renal function, and show AKI well after the injury has occurred. Early detection of this pathology could permit implementation of salvage therapies and improve patient outcomes. Over the last years, tubular proteins released during tubular insult have garnered much attention as promising AKI urine biomarkers. Neutrophil gelatinase associated lipocalin (NGAL), kidney injury molecule-1 (Kim-1) and N-acetyl-D-glucosaminidase (NAG) have demonstrated to be early predictors for diagnosis or outcome of AKI in human as well as in animal models. Related to this issue, in the last years we have been trying to validate different proteins excreted in urine as noninvasive biomarkers of AKI of different etiologies. Our group was pioneering in detecting the Organic Anion Transporter 5 (Oat5), the Sodium-Dicarboxylate Cotransporter 1 (NaDC1) and Caveolin-2 (Cav-2) in urine. The urinary excretion of these proteins has been evaluated in different experimental models of renal diseases in rats and compared with traditional parameters of renal function (plasma urea and creatinine, creatinine clearance, urinary alkaline phosphatase activity, histological lesions). The results obtained in experimental models of AKI (ischemic, obstructive and nephrotoxic-induced by methotrexate, cisplatin and mercuric chloride) allow us to propose Oat5, NaDC1 and Cav-2 as potential biomarkers of different stages of this disease. The next step would be passing from preclinical animal research to clinical trials in order to evaluate the utility of these proteins as biomarkers of AKI in humans.