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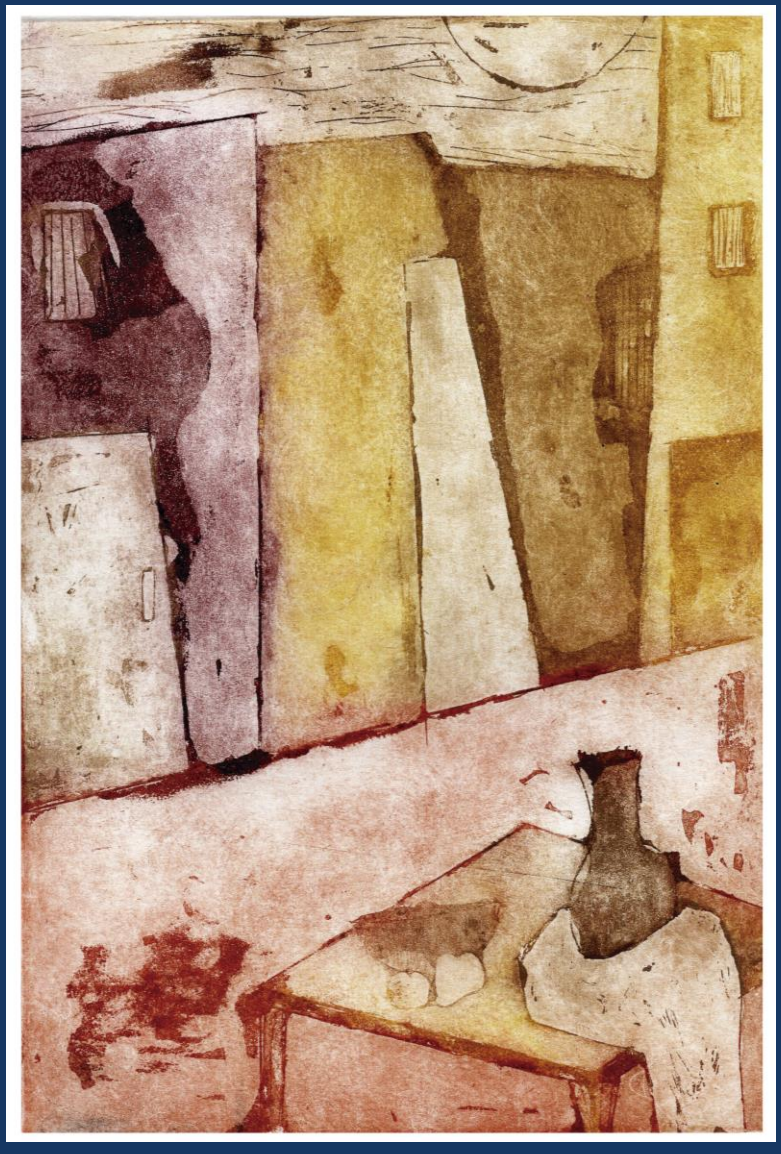

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MIEMBROS EMÉRITOS

La Tapa (*Ver* pág. 4)
Atardecer en la tarde
Antonella Ricagni

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Sociedad Argentina de Protozoología (SAP)

IX Reunión Anual de la
Asociación Argentina de Nanomedicinas
(NANOMED-ar)

VI Reunión Científica Regional de la Asociación Argentina
de Ciencia y Tecnología de Animales de Laboratorio
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con la participación de
The Histochemical Society

13 - 16 de noviembre de 2019
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tumor apoptotic rate, accelerated tumor progression and impaired the antitumor and antimetastatic effect of chemotherapy suggesting that HN inhibits the response of TNBC cells to cytotoxic stimuli, facilitating tumor progression and chemoresistance. Inhibition of endogenous HN increased apoptosis and chemosensibility of TNBC and glioblastoma cells. In addition, chemotherapeutic drugs increased HN expression. Our results put into question the safety of systemic administration of HN or its analogs for chronic diseases and suggest that local administration of gene therapy vectors that silence endogenous HN could hold therapeutic potential in cancer.

IMPLICATION OF THE PROLIFERATION / APOPTOSIS BALANCE IN OVARIAN ALTERATIONS IN BOVINES

NATALIA SALVETTI

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Follicular persistence is caused by the failure of ovulation and the consequent permanence of the follicular structure in the ovary, which alters the cyclicity of the female and causes infertility. This process is one of the main components of cystic ovarian disease (COD) and other diseases of ovarian origin that causes great economic losses in the dairy industry because of unsuccessful artificial inseminations, veterinary treatment and decrease in milk production related to the increase in the interval from calving to conception. Along folliculogenesis, the cells that compose the ovarian follicles normally proliferate and then differentiate. Finally, the follicle can take one of two pathways: ovulation, if the follicle is dominant in an ovulatory wave, or atresia, which occurs with most of the non-dominant follicles. Successful follicle development depends on the presence of survival factors that promote follicle growth and also protect cells from apoptosis. These include factors produced within the ovary like 17-β estradiol, progesterone, insulin-like growth factor (IGF)-1, as well as gonadotropins. In the absence of survival factors, endogenous apoptotic pathways within the follicle become activated and lead to follicular atresia. Many studies in different species have shown that the processes of proliferation and apoptosis are altered in the cysts already formed, where there is a decrease in cellular proliferation and an increase in the survival of the cells, which contribute to the persistence of these follicles, preventing their ovulation or regression. However, the study of initial events in the development of persistence indicates that, initially, the proliferation rate is maintained in the absence of ovulation, with low levels of apoptosis and an increase in cell survival due to the increase in anti-apoptotic proteins. It is probable that the hormonal changes that occur later, both at endocrine and paracrine level, are responsible for the alterations observed in the parameters of cell proliferation and differentiation of already developed cysts.

SAB SYMPOSIUM III

YOUNG RESEARCHERS

Chairs: Débora Cohen / Clara Marín Briggiler

INTEGRATING IMMUNOREGULATORY AND VASCULAR SIGNALING PROGRAMS THROUGH LECTIN-GLYCAN INTERACTIONS

DIEGO CROCI

Institute of Histology and Embryology of Mendoza Dr. Mario Burgos (CCT-CONICET). Mendoza. Mendoza, Argentina.

Recent efforts toward decoding the glycosylation signature of immune cells have revealed dramatic changes in N- and O-glycan structures during immune cell maturation, activation and differentiation. The responsibility of deciphering these glycosylation changes is assigned to endogenous lectins which expression is dynamically regulated during chronic inflammatory responses. We will discuss recent findings from our laboratory demonstrating the contribution of glycosylation-dependent mechanisms and lectin-glycan interactions to the regulation of a broad range of immunological programs including T cell survival, dendritic cell fate, microglia activation and endothelial cell signaling. These mechanisms, which could be usurped by tumors to evade and thwart immune responses, have been proposed to shift the balance of immune responses and control immune cell tolerance, inflammation and angiogenesis.

THE YIN AND YANG OF HISTAMINE IN THE REGULATION OF TESTICULAR LEYDIG CELL PROLIFERATION

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Histamine (HA) is a biogenic amine with indisputable significance for medicine and biology. It is synthesized exclusively by histidine decarboxylase (HDC) in all mammalian tissues, albeit tissue-specific mechanisms operate to keep its concentration within strict limits: both a deficit of HA or a slight excess can lead to health

loss. With regard to the male gonad, previous studies have linked increased mast cell numbers and mast-cell related HA with the pathogenesis of infertility. In contrast, HA levels are normally higher in the neonatal testis, and in a former report we described that HDC gene knockout mice show reduced testis weight already at 7 days of age, implying that important HA-dependent events may occur during testicular development. To assess this apparent conflict and considering the ubiquitous role of HA as modulator of cell proliferation, we speculated that testicular HA might contribute to the regulation of LC number. Firstly, we studied the effect of intratesticular HA treatment in adult male rats injected with the specific LC cytotoxic agent ethane dimethanesulphonate (EDS), a convenient model to study normal LC proliferation and development from LC precursors in vivo. As expected, LC re-population was faster in HA-treated rats. Thus, to better identify the stage/s of testicular development in which HA would play a role, we conducted in vitro experiments to evaluate its effect on the proliferation of progenitor and immature LC, isolated from 21- and 35-day old male rats, respectively. However, none exhibited a proliferative response upon stimulation with HA. We then studied HDC immunoexpression in testes of rats aged 7 to 240 days, and found that it was highest in 7-day old rats but then decreased abruptly, with remnants of the fetal LC population being the most intensively stained. In line with previous observations in HDC KO mice, these results suggest that local HA synthesis may be important to influence LC numbers in the early stages of normal testicular development, while it would be negatively regulated with age. Interestingly, we have recently detected HDC overexpression in 2 murine Leydig tumor cell lines, as well as in human LC hyperplasia and prepubertal LC tumors. Moreover, HA promoted the proliferation of Leydig tumor cells in vitro, while specific HDC inhibitors had the opposite effect. Importantly, these findings indicate that autocrine overproduction of HA might be related to abnormally increased proliferation in LC.

UNDERSTANDING THE CRITICAL ROLE OF MONOCYTE-MACROPHAGE LINEAGE IN INFLAMMATORY DISEASES AND HOW THE COOPERATION WITH OTHER CELL TYPES DEFINE THE EFFECTOR RESPONSE

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Monocyte-macrophage lineage cells are multifunctional and found in nearly all tissues throughout the body. They orchestrate the initiation and resolution phases of both innate and adaptive immunity, significantly impacting protective immunity and immune-mediated pathological damage through their ability to adopt distinct functional capacities in different microenvironment. A major goal of the macrophage field is to link specific functions with specific cellular and molecular pathways associated with different macrophage activation profiles. Negative regulatory feedback is a critical aspect of the homeostatic immune response and disruption on this point could lead to inflammatory-based disease. The tyrosine kinase receptors TYRO3, AXL and MERTK (TAM) and their ligands Protein S (PROS1) and growth arrest-specific 6 (GAS6) are critical players in maintaining immune homeostasis by dampening inflammatory response, mediate efferocytosis and to contribute to tissue repair process. Our research is focus in understanding the participation of monocyte-macrophage compartment and TAM axis in the protection or development of some chronic inflammatory human diseased such as Inflammatory Bowel disease, Langerhans Cell Histiocytosis or Multiple Sclerosis as well as in acute infection and sepsis. We have found that monocyte-macrophage linage play central role not only in immune-mediated diseases but also because regulatory T cell or platelets can orchestrate macrophage effector responses improving clinical outcome.

DECONSTRUCTING THE GLUCOCORTICOID RECEPTOR: PHARMACOLOGICAL IMPLICATIONS

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Synthetic glucocorticoids (GCs) are one of the most prescribed pharmaceuticals world-wide due to their powerful anti-inflammatory and immunosuppressive activities. The action of GCs is mediated by the glucocorticoid receptor (GR), a member of the nuclear receptor superfamily of transcription factors. The GR is involved in many physiological processes, including the regulation of cell death and proliferation. Historically, GR transcriptional activity and clinical outcomes have been linked to its dimeric/monomeric state. A widely discussed model suggests that dimeric GR regulates unfavorable metabolic pathways, while monomeric GR is responsible for anti-inflammatory activities. Hence, GR ligands that preferentially induce the monomeric rather than the dimeric pathway should retain the desired pharmacological effects but will lack the undesired adverse reactions. However, the search for improved ligands under this paradigm has produced no significant results for the last 25 years. During this talk, I will present evidence against this predominant "dissociated model". By combining advance quantitative fluorescence microscopy techniques with genomic analysis on oligomeric mutant receptors, I will propose a new relationship between GR's quaternary