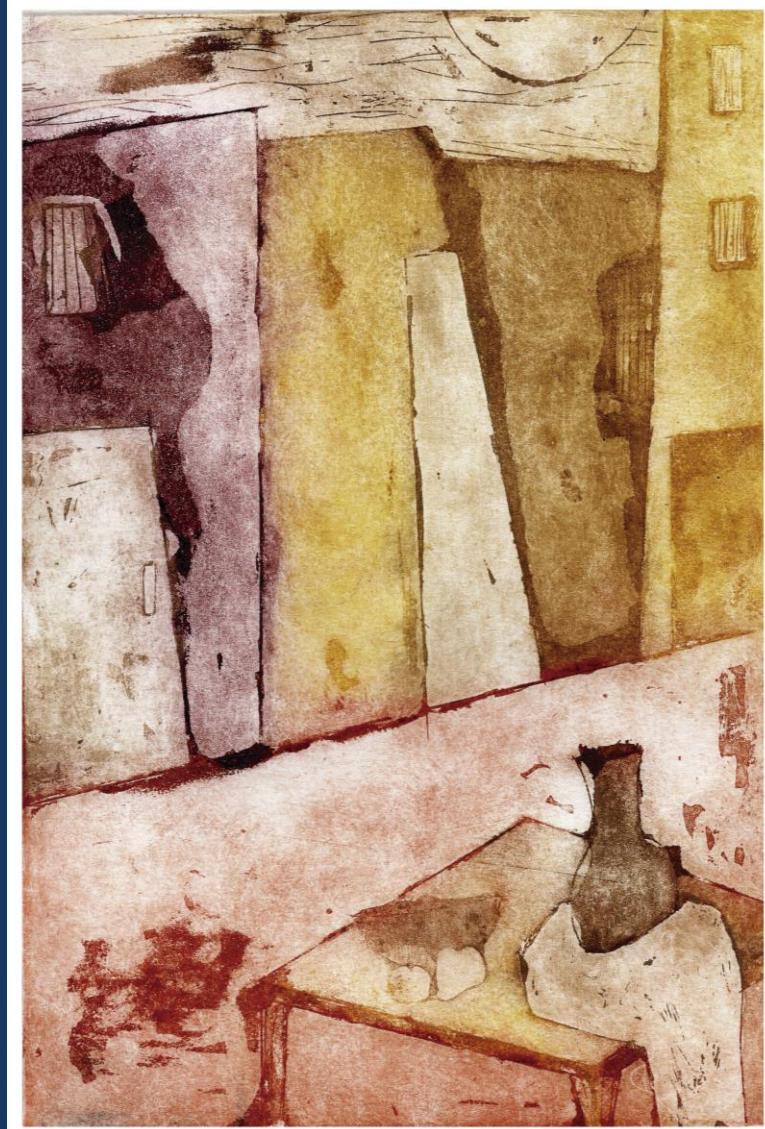


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La Tapa (Ver pág. 4)

Atardecer en la tarde

Antonella Ricagni

MEDICINA (Buenos Aires) – Revista bimestral – ISSN 0025-7680 (Impresa) – ISSN 1669-9106 (En línea)

REVISTA BIMESTRAL

Registro de la Propiedad Intelectual N° 02683675

Personería Jurídica N° C-7497

Publicación de la Fundación Revista Medicina (Buenos Aires)

Propietario de la publicación: Fundación Revista Medicina

Queda hecho el depósito que establece la Ley 11723

Publicada con el apoyo del Ministerio de Ciencia, Tecnología e Innovación Productiva.

MEDICINA no tiene propósitos comerciales. El objeto de su creación ha sido propender al adelanto de la medicina argentina.

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Aparece en MEDLINE (PubMed), ISI-THOMSON REUTERS (Journal Citation Report, Current Contents, Biological Abstracts, Biosis, Life Sciences), CABI (Global Health), ELSEVIER (Scopus, Embase, Excerpta Médica), SciELO, LATINDEX, BVS (Biblioteca Virtual en Salud), DOAJ, Google Scholar y Google Books.

Incluida en el Núcleo Básico de Revistas Científicas Argentinas del CONICET.

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Tel. 5287-3827 Int. 73919 y 4523-6619

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Vol. 79, Supl. IV, Noviembre 2019

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**con la participación de
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Hotel 13 de Julio - Mar del Plata**

EDITORES RESPONSABLES

**Dra. Mónica Costas
Dra. Gabriela Marino
Dr. Pablo Azurmendi**

structure, chromatin binding and transcriptional output. This has important implications for the therapeutic uses of steroid hormones and the goal of finding selective

anti-inflammatory drugs that do not create unwanted side-effects.

AWARDS

SAIC AWARD I

'LUCIO CHERNY FOUNDATION'- MULTIDISCIPLINARY

Juries - Alicia Belgorosky | Graciela Cremaschi | Alejandro De Nicola | Mirta Schattner | Elba Vázquez

0296 - INTEGRIN-MECHANOSIGNALING ROLE IN SMALL GTPASES ACTIVATION AND CANCER

Georgina COLÓ (1) | Lucía FERNÁNDEZ CHÁVEZ(1) | Karen SCHWEITZER(1) | Nazarena BARREIRA-LAMAS(2) | Norberto GANDINI(1) | Ezequiel ALONSO(1) | Marilina MASCARÓ(2) | Pamela PICHEL(1) | Sergio RECIO(3) | Reinhard FÄSSLER(4) | María Marta FACCHINETTI(1) | Alejandro CURINO(1)

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The ability of cells to adhere and simultaneously probe their mechanical environment is central to many physiological and pathological processes. Extracellular matrix sensing and mechanotransduction are mediated by the integrin family of cell adhesion receptors. Using genetically engineered cells, we studied the specific fibronectin integrin binding signaling and its role in tumor development. We observed that α5β1-integrins promoted the formation of small adhesions, low RhoA activation and high force, while αβ3-expressing cells showed large adhesions, thick stress fibers, high RhoA activation and low force. To further analyse these cellular phenotypes, we looked for specific RhoA activators (GEFs). For this purpose, we performed Mass Spectrometry (MS) analysis follow by biochemical assays and observed that GEF-H1 activation is αVβ3-integrin dependent. Furthermore, using integrin-tail pull-down and MS assay, we observed that GEF-H1 binds to β3-tail, suggesting that specific integrins may activate different Rho-GEFs during tumor progression. In order to study the role of GEF-H1 in cancer, we analysed by immunohistochemistry GEF-H1 expression in human biopsies. We observed overexpression of GEF-H1 in breast ($p=0.0053$, $n=61$) and thyroid ($p=0.0006$, $n=32$) tumor biopsies compared with normal tissue. Similar results were obtained in cancer cell lines (CCL). To further study the role of GEF-H1 in tumor development using CRISPR/Cas9 technology, we generated GEF-H1-knock out (KO) clones in a murine invasive breast CCL. We observed a decrease in the proliferation, migration and invasion rates ($p<0.001$) in GEF-H1-KO cells. These results showed that GEF-H1-RhoA activation is αVβ3-integrin dependent and that may mediate the signaling involved in controlling cell structure, force generation, proliferation, migration and invasion of breast cancer cells. In addition, the studies in human tumor samples suggest that GEF-H1 might be a molecular biomarker in cancer.

0331 - REGULATORY MECHANISMS UNDERLYING FUNCTIONAL MATURATION OF SERTOLI CELLS IN RESPONSE TO ANDROGENS DURING POSTNATAL DEVELOPMENT

Nadia Yasmín EDELSZTEIN | Helena Fedora SCHTEINGART | Rodolfo Alberto REY

CENTRO DE INVESTIGACIONES ENDOCRINOLÓGICAS "DR. CÉSAR BERGADÁ" (CEDIE) - CONICET - FEI

Androgen-dependent maturation of Sertoli cells during postnatal testicular development is key for the establishment of spermatogenesis. Meiosis in the male begins at puberty and relies on androgens and retinoic acid. Immature Sertoli cells produce high levels of AMH, which is inhibited by androgens at puberty. The molecular mechanisms underlying androgen-mediated AMH decline are unknown. CYP26B1 degrades retinoic acid in the prenatal testis preventing meiosis initiation. The concurrence of meiotic entry and Sertoli cell maturation in response to androgens led us to propose that CYP26B1 —like AMH— is downregulated by androgens in the Sertoli cell during puberty, thus enabling meiosis initiation. By immunohistochemistry, we saw that CYP26B1 expression declines in the postnatal mouse Sertoli cell as androgen receptor (AR) expression increases, closely before meiotic spermatocytes appear. Luciferase reporter assays in the SMAT1 Sertoli cell line showed a direct negative effect on Amh promoter activity in the presence of dihydrotestosterone (DHT, $p<0.001$). Site-directed mutagenesis and ChIP-qPCR assays showed that androgen-mediated inhibition requires the SF1 sites in the Amh promoter. Regarding Cyp26b1, we saw no changes in promoter activity in response to androgens ($p=0.34$). This lack of response was further supported by invariant levels of endogenous Cyp26b1 expression in SMAT1 cells transfected with the AR ($p=1.0$) and in primary Sertoli cells of 10-day-old mice in culture ($p=0.7$), after DHT treatment. ChIP-qPCR showed no enrichment in AR sequences analyzed, indicating a lack of functional binding of the AR. In sum, we confirmed a negative correlation between the immature Sertoli cell markers AMH and CYP26B1 and AR expression and meiotic initiation in postnatal development. We identified the molecular mechanism underlying AMH inhibition by androgens but found that the decline in CYP26B1 expression is not caused by a direct inhibitory androgen effect on Sertoli cells.

0359 - CDC42 ACTIVITY IS NECESSARY FOR THE INTERPLAY BETWEEN CAMP/PKA PATHWAY AND CATSPER FUNCTION

Guillermina LUQUE (1) | Ana ROMAROWSKI(1) | Cintia STIVAL(2) | Nicolas GILIO(1) | Tomas DALOTTO-MORENO(1) | Paula BALESTRINI(1) | Martina JABŁOŃSKI(1) | Jamaica SCHIAVI-EHRENHAUS(1) | Diego KRAPF(3) | Dario KRAPF(2) | Mariano BUFFONE(1)

INSTITUTO DE BIOLOGÍA Y MEDICINA EXPERIMENTAL (IBYME-CONICET) (1); IBR-CONICET, UNR (2); COLORADO STATE UNIVERSITY (3)

Sperm acquire the ability to fertilize in the female genital tract in a process called capacitation. During capacitation, sperm undergo changes in the motility pattern called hyperactivation, which depends on Ca^{2+} transport by the sperm-specific Ca^{2+} channel CatSper. CatSper is essential for fertilization and therefore, it is subjected to a complex regulation that is not fully understood. Recent reports found that mouse CatSper is upregulated by cAMP-dependent activation of protein kinase A (PKA). From a molecular point of view, bicarbonate stimulation of the soluble adenylyl cyclase (sAC) leads to an increase in cAMP, PKA activity and tyrosine phosphorylation of sperm proteins. It remains incompletely understood if PKA itself phosphorylates CatSper or if its activation relays on other intermediary events. By using super-resolution microscopy, we report that similar to CatSper, the small