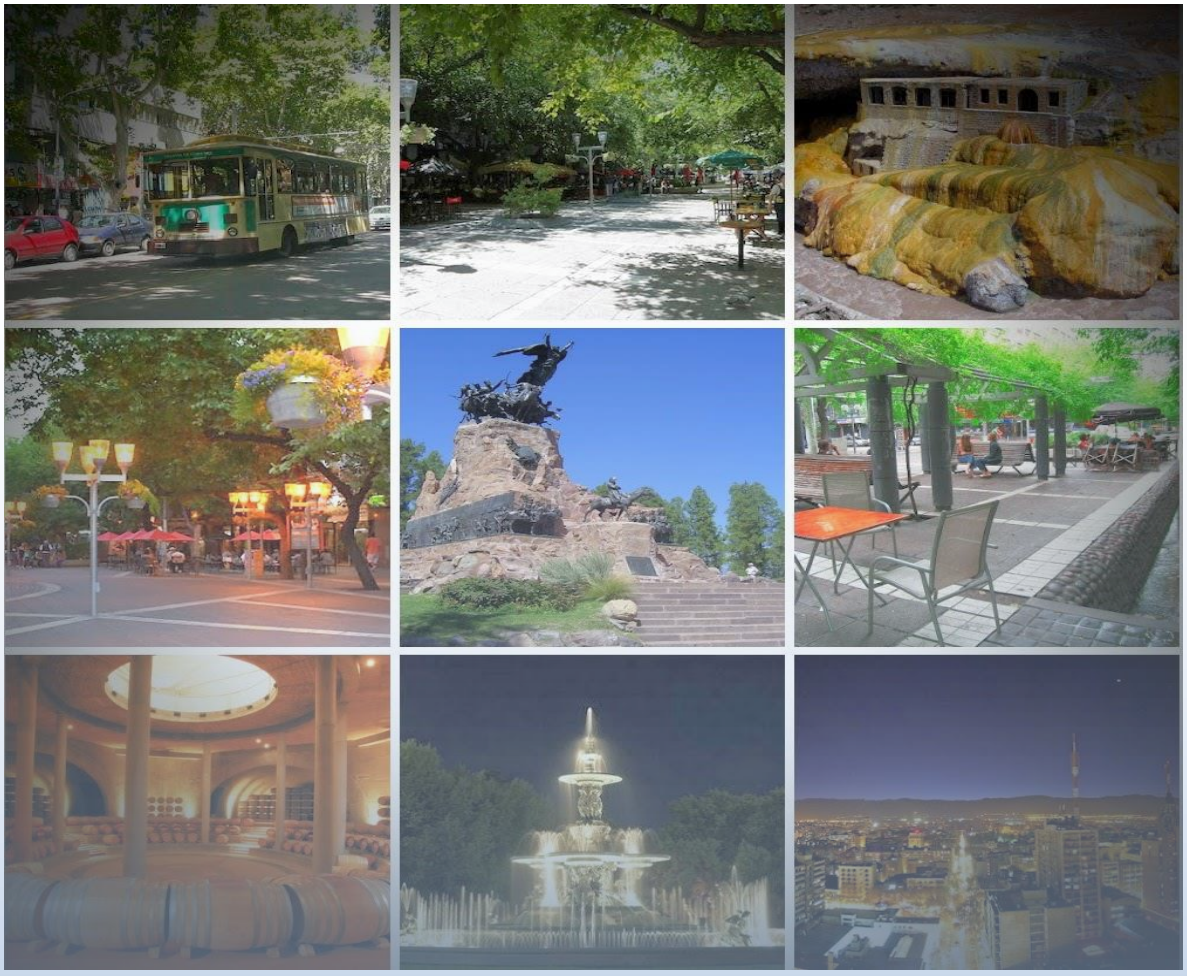
XXXIV Reunión Científica Anual De La Sociedad De Biología De Cuyo



01 al 03 de Diciembre – Mendoza- Argentina

carbohydrate-binding protein, participates in the recognition and attachment of CT to a human cervical epithelial cell line (HeLa). By western blot, we showed that Gal-1 binds to CT in vitro. In addition, Gal-1 increased CT attachment to HeLa cells in a doses dependent manner assessed by flow cytometry and confocal microscopy. Likely, Gal-1 promotes chlamydial infection by bridging bacterial N-glycans to eukaryotic membrane glycoproteins. Furthermore, Gal-1 could tie CT together, helping bacteria to enter into the eukaryotic cell in groups, rising even more chlamydial infection. Unveiling the mechanisms used by CT to invade host cells could help in finding new therapeutic targets for controlling this highly frequent sexually transmitted disease.

A204

THREE-DIMENSION SHAPE RECONSTRUCTION OF PERITUBULAR MYOID CELLS.

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Laboratory cytoskeleton and cell cycle (IHEM-Universidad Nacional de Cuyo-CONICET-FCM, Mendoza).

Peritubular myoid cells (MP cells), are part of seminiferous tubule (TS) wall. These cells are similar to smooth muscle cells but myophilaments are organized in two perpendicular layers. Were constructed the three dimension (TD) shape of individual MP cells using the density of actin filaments (AF). TS were isolated from adult Wistar rat testes, fixed with 4% paraformaldehyde and AF stained with anti-alpha actin antibody conjugated with Cy3. AF distribution were analyzed by confocal microscopy in 40 transverse sections of 0.4 µm. Using Image J program, the TD shape of individual MP cells were reconstructed. MP cells looked like a hexagon of 6 µm high containing a groove of 16 µm depth in the center of the body. We interpreted that the shape of PM is directly related to the way of TS contraction.

A205

IN VIVO AND IN VITRO ANTIPROLIFERATIVE EFFECT OF 4-HYDROXY-3-(3-METHYL-2-BUTENYL)-ACETOPHENONE (4-HMBA) IN B16F0 TUMOR CELLS MODEL

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The basic physiopathology of vancer includes aberrations in different parts of the molecular mechanisms that control the cell cycle. Due to the increasing incidence of cancer worldwide, there is an intensive search for new therapeutic strategies to treat this disease. In this area, the research has focused on exploring the action of compounds of plant origin. 4-HMBA is the main secondary metabolite from *Senecio nutans*, commonly known as chachacoma, a medicinal plant widely used in Andean traditional medicine; this compound exhibits antifungal and tripanocidal activities. We analyzed, *in vitro and in vivo*, the effect of 4-HMBA. B16F0 cells were cultured in presence of ethanol (vehicle) or 5.0-17.5 µg/mL of 4-HMBA dissolved in ethanol. The growing index (GI) ± SE in 3 independent experiments was assayed from 0 to 72 h. At 72 h of culture, GI of vehicle treatment, was 6.3 ± 0.7 and of 4-HMBA treatments were: 4.9 ± 0.7 (A);3.2 ± 0.8 (B) and 1.0 ± 0.3 (C) for 7.5; 15 and 17.5 µg/mL respectively. Both (B) and (C) GIwere $p \leq 0.001$ vs. vehicle.In*i*n *vivo* assays we inoculated B16F0 cells in C57 mice to generate a melanoma. When the tumor reached a volume of 200mm^2, the animals were treated with 25mg4-HMBA/day/Kg in ethanol or vehicle (ethanol, as a control). After 13 days we observedthatcontrol animal melanomas reached a volume of 2198 mm^2± 970, while treated animal melanomas reached a volume of 460 mm^2± 229. These results show that 4-HMBA generated a significant inhibition of proliferation of B16 F0 cell and an important reduction of tumor volume by therapeutic treatment.

A206

DESMOGLEIN-4 DEFICIENCY INCREASES CD45$^+$ LEUKOCYTES POPULATION IN SKIN

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Desmogleins are involved in cell to cell adhesion mechanisms and are crucial in keeping structural integrity of different tissues including skin and heart. These family of molecules, e.g. desmoglein-3, modulate keratinocyte activation and may control key molecules such as actin and p38 MAPK. However, whether desmoglein-4 may drive inhibitory or activating signals to skin-resident immune cells is unknown. The aim of our work was to assess the impact of desmoglein-4 deficiency in the amount of skin leukocyte populations. To this end, OFA $^{hr/hr}$ rats (n=3) which are mutant for the desmoglein-4 gene and their strain of origin, Sprague-Dawley (SD) (n=3) rats were used in this study. Skin biopsies from OFA and SD rats were weighed, minced to obtain cell suspensions and stained with monoclonal antibodies against CD45 (pan-leukocyte marker) and CD3 (T cell marker), conjugated with APC and FITC respectively. Beads were added to stained cell suspensions from skin biopsies derived from OFA and SD rats and acquired by FACS to measure total cell counts per milligram of tissue. OFA rats showed an expansion of CD45$^+$ cells compared to SD control rats (SD 1,2±0,1 vs OFA 3,9±0,5; cells/mg) (*t* test; $p<0,05$). In addition, we found that desmoglein-4 deficiency increased the percentage of CD3$^+$ T cells compared to SD control rats (SD 17,4±9,3 vs OFA 41,6±14,3) but without reaching statistical significance by the *t* test. In conclusion, these results suggest that desmoglein-4 deficiency promotes an