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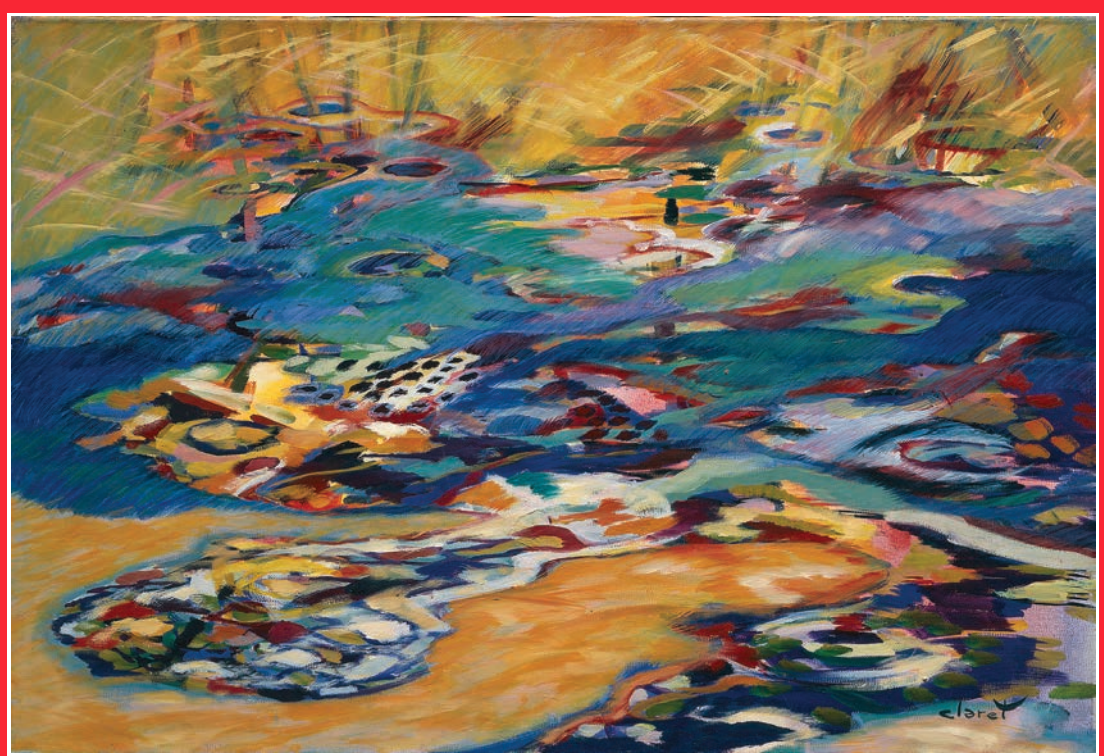

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LXI REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA (SAIC)

LXIV REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

XLVIII REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE FARMACOLOGÍA EXPERIMENTAL (SAFE)

VII REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE NANOMEDICINA (NANOMEDAR)

V CONGRESO NACIONAL DE LA ASOCIACIÓN ARGENTINA DE CIENCIA Y TECNOLOGÍA DE ANIMALES DE LABORATORIO (AACYTAL)

15-19 de noviembre de 2016
Hotel 13 de Julio – Mar del Plata

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LXI ANNUAL MEETING
ARGENTINE SOCIETY FOR CLINICAL INVESTIGATION
(SAIC)

LXIV ANNUAL MEETING
ARGENTINE SOCIETY OF IMMUNOLOGY
(SAI)

XLVIII ANNUAL MEETING
ARGENTINE SOCIETY OF EXPERIMENTAL PHARMACOLOGY
(SAFE)

VII ANNUAL MEETING
ARGENTINE SOCIETY OF NANOMEDICINE
(NANOMEDAR)

V NATIONAL CONGRESS
ARGENTINE ASSOCIATION FOR SCIENCE AND TECHNOLOGY OF LABORATORY ANIMALS
(AACYTAL)

November 15-19, 2016
13 de Julio Hotel – Mar del Plata

an experimental model of mammary tumors. Female Balb-c mice, 8 weeks of age, were subjected to tumor induction. Briefly, five 1mg/kg consecutive doses of 7,12-Dimethyl Benzanthracene (DMBA) was administered intragastrically (i.g). Subsequently, a single dose of 10 mg/kg of medroxyprogesterone acetate (MPA) was given. Additionally, weekly doses of 2000 IU of rhEPO were subcutaneously administered. Animals from each experimental group were monitored throughout the experience and hematological parameters were determined at each time of the experimental protocol. Animals were sacrificed and mammary fat pads were removed for histological and immunohistochemical analysis. The control group presented normal hematologic and histological values. Interestingly, the erythropoietin treated groups showed and increased EPOR immunohistochemical staining in most mammary tumors related to low or none EPOR expression of the normal control group. The results lead us to conclude that there are controversies regarding the effects that rhEPO can produce in a tumor environment, providing an impetus to the review of the EPO-EPOR biology.

FARMACOLOGÍA / PHARMACOLOGY

408 (1068) **COMBINED THERAPY WITH BENZNIDAZOLE AND MILTEFOSINE IN AN ACUTE MURINE MODEL OF TRYPANOSOMA CRUZI INFECTION**

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Treatment options for Chagas disease are limited to benznidazole (BZ) and nifurtimox (NFX). New treatments are not available due to high cost of developing active molecules. Drug repurposing is a cost-effective solution to fulfill current needs of better and safer therapy and drug combination is astrategy to improve efficacy reducing treatment time and doses, with lower side effects rates.

We have previously assessed anti-*T. cruzi* activity of miltefosine (MLT) both *in vitro* and *in vivo* withpromissory results. The aim of this work was to determine the *in vivo* efficacy of MLT and the synergism between MLT and BZ.

Six weeks old BALB/c female mice were infected with 500 *T. cruzi* trypomastigotes (VD strain; DTUTcVI) by intraperitoneal (ip) route. At parasitemia onset, 5 mice/group were randomly assigned to the following treatments: Non-treated (NT); BZ 5 mg/kg; MLT 25 mg/kg; MLT 25 mg/kg+BZ 5 mg/kg; MLT 50 mg/kg+BZ 50 mg/kg; BZ 100 mg/kg. Treatment was administered orally for 20 consecutive days. Effects on parasitemia, mortality and parasitic load in blood were recorded.

Parasitemia reached $3.8x10^5$ trypomastigotes/mL in NT group, but decreased significantly (p=0.002)in all treatment groups, with 76-94% parasite reduction. The effect on parasitemia was similar to anycombined therapy and any treatment prevented mortality. At the end of therapy, animals treated with BZ 5 mg/kg or MLT 25 mg/kg alone or in combination still exhibited parasitemia. However, animals treated with BZ 100 mg/kg or BZ 50 mg/kg+MLT 50 mg/kg remained negative. These groups were immunosuppressed with cyclophosphamide (CYP). At the end of the CYP cycle, 40% of BZ 100 mg/kg group had parasite reactivation but none from BZ 50 mg/kg+MLT 50 mg/kg. qPCR revealedthat 60% of animals treated with BZ alone and 20% of mice treated with BZ and MLT had parasite in circulation.

These results suggest an additive effect of BZ 50 mg/kg combined with MLT 50 mg/kg in an acutemurine model of *T. cruzi* infection.

409 (1074) **EVALUATION OF SDF-1/CXCR4 EXPRESSION IN HUMAN HER2/NEU POSITIVE BREAST CANCER SAMPLES**

María de los Ángeles Martínez[1], Silvana Beatriz Larroza[1], María del Rosario Mariana Gómez-Pescié[1], Lorena Dos Santos Antola[2], María Carla Zimmermann[1,2].
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Stromal Derived Factor-1 (SDF-1) is a chemokine whose membrane receptor CXCR4 can be found in breast tumor and the metastatic sites of the primary tumor. The signaling pathway of this chemokine and its receptor is involved and is capable to induce directional migration of cells due to a chemokine gradient. Recently, it has been observed that CXCR4 works as a mediator in the migration of tumor cells to specific organs and t it could be correlated with expression of the Human Epidermal Growth Factor Receptor-2 (HER2/neu) in breast tumors. HER2/neu is a transmembrane protein, also known as ErbB-2, which has been implicated in many types of human cancers, specifically breast cancer, in which it is used as a prognostic factor. We hypothesized that the overexpression of HER2/neu activates the SDF-1/CXCR4 axis in HER2/neu positive breast tumors, and that transactivation enhances tumor progression, malignancy and metastasis capacity. The aim of this work was to investigate the association between SDF-1/CXCR4 and HER2/neu expression in breast cancer paraffin sections. We carried out an RT-qPCR of human breast tumor paraffin samples to determine the SDF-1 and CXCR4 expression. We, additionally, performed the same SDF-1/CXCR4 RT-qPCR in some of the metastatic sites of the primary tumor. Also, analysis of HER2/neu expression in these samples were determined by immunohistochemistry. Most HER2/neu positive breast tumor paraffin samples were found to express high levels of either SDF-1 of CXCR4. Some of the metastatic sites presented higher levels of this chemokine and its receptor expression, while others presented no expression at all. Further investigation is needed, particularly in HER2/neu negative samples, in order to achieve acceptable information to validate our hypothesis.

410 (1082) **STEVENS JOHNSON SYNDROME SECONDARY TO ADMINISTRATION OF ANTIEPILEPTICS DRUGS**

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[1]Facultad de Medicina Universidad Nacional del Nordeste.

Stevens Johnson Syndrome (SJS) is idiopathic in 25.5% of the patients but can also becaused by certain drugs (phenobarbital, carbamazepine, valproic acid). The Antiepileptic drugs hypersensitivity syndrome is a serious adverse drug reaction (ADR), initially described with aromatic antiepileptics such as phenytoin, carbamazepine, phenobarbital and primidone. Objective: To identifySJS as an adverse reaction of antiepileptic drugs notified to the Regional Pharmacovigilance Centre (CRF-UNNE). We performed an observational descriptive, cross-sectional study. All notifications of the CRF-UNNE database were included. The ADRs were classified considering: severity (mild, moderate, severe) and the mechanism of appearance (type A and B Rawlins and Thompson classification). From the 200 notifications of ADR by antiepileptic drugs, 2% (4) were SJS. Average age: 35 years old, female / male ratio: 1/1. Antiepileptic drugs were involved in SSJ according to the Anatomical-Therapeutical-Chemical classification (ATC-2010) N03 code: N03AA barbiturates and derivatives (phenobarbital N03AA02), N03AB hydantoin derivatives (N03AB02 phenytoin), N03AX other antiepileptics (lamotrigine N03AX09). According to severity: 10% were severe (1 fatal case of SJS). According to Rawlins and Thompson classification all cases of Stevens Johnson were type B. They were other SJS cases not related to antiepileptic drugs. They may occur with drugs other than antiepileptics that also act at the Central Nervous System.

411 (418) **IN VIVO EFFECT OF 5-FLUOROURACIL ALONE OR IN COMBINATION WITH ALBENDAZOLE AGAINST ECHINOCOCCUS GRANULOSUS**

Patricia Eugenia Pensel[1,3], Gabriela Ullio Gamboa[2, 3], Jean Pierre Benoit[4], María Celina Elissondo[1, 3].
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