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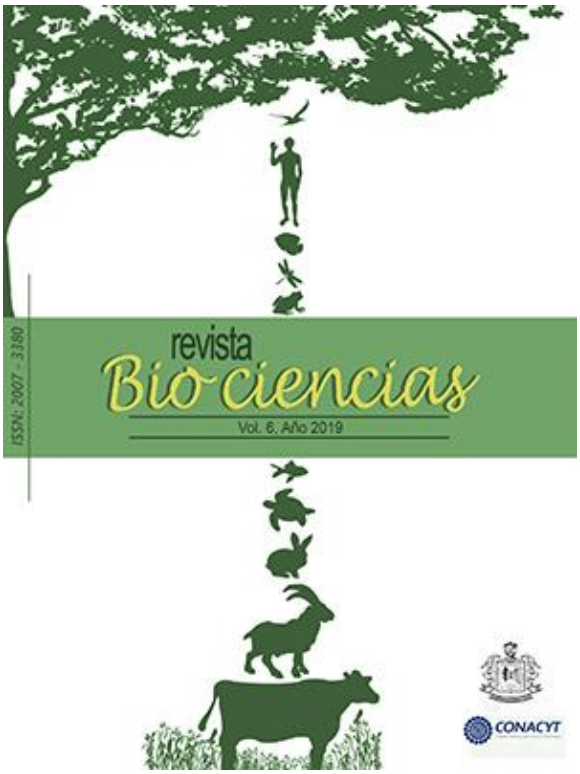

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Psychoimmune profile of mexican women with breast cancer.

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Worldwide, breast cancer (BC) is the most common neoplasia in women. Diagnostic is made considering genetics and reproductive history, explaining only 40% of cases. A cancer prone personality has been recognized, but not considered as a tool for diagnosis. So we analize the presence of this personality in mexican women histologically diagnosed with BC and benign breast pathology (BBP), and also identify the presence of autoantibodies for TD47 antigens in sera. Our result showed that women with BC or BBP have five distinctive features: low restraint, low global stress symptoms, low physical stress symptoms, low restraint-defensiveness composite and high distress, before realize diagnosis. This psychological profile was also associated with an increased number of autoantibodies and a low variability of them. The results suggest psychological profiling might be used as a tool to identify women at risk of developing cancer or bening breast pathology, o even reinforce diagnosis.

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Asociación de polimorfismos, cuantificación de citocinas y receptores en placentas humanas con y sin preeclampsia en Culiacán, Sinaloa.

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La mortalidad materno-fetal es un problema de salud pública. Según la OMS diariamente mueren 830 mujeres por causas prevenibles relacionadas con el embarazo y el parto. Respecto a la mortalidad fetal, más de 2 millones de decesos ocurren anualmente. Una de las principales complicaciones de las muertes materno-fetales es la preeclampsia (PE), la cual se asocia fisiopatológicamente a diversos factores entre los que destaca el sistema inmunológico y los factores genéticos. Desde el punto de vista inmunológico, un embarazo normal se caracteriza por la presencia de linfocitos Th2 y Treg sistémicos, mientras que un embarazo preeclámptico se caracteriza por la presencia de los linfocitos Th1 y Th17. A nivel genético se sabe que en la PE más de 70 genes candidatos han sido analizados y más de 10,000 polimorfismos de un solo nucleótido (SNP) disponibles para su estudio. A la fecha sólo existe como prueba molecular diagnóstica tardía, la proporción sFlt-1/PlGF que se encuentra aumentada en las pacientes preeclámpticas y en México la NOM-007-SSA2-2016 para la atención de la mujer durante el embarazo, el parto y puerperio, así como del recién nacido, carece de estudios diagnósticos preclínicos para identificar la PE mediante el empleo de marcadores más precisos, efectivos y tempranos del síndrome. El objetivo del presente trabajo fue determinar la relación entre los SNPs en citocinas proinflamatorias, sus niveles séricos y la expresión de sus receptores en placenta, así como la desregulación de genes relacionados con inflamación con el desarrollo de PE en embarazadas. El presente estudio fue multicéntrico y realizado en Culiacán, Sinaloa, de enfoque cuantitativo, no experimental, longitudinal de tipo casos y controles anidado a diseños panel (n = 10 para c/grupo). Se realiza toma muestra de sangre periférica al 1er, 2do y 3er trimestre del embarazo para la cuantificación de citocinas Th1/Th2/Th17 mediante citometría de flujo y para el análisis genético será mediante Sondas Taqman (n = 100 casos vs 200 controles). De modo alterno se toman explantes placentarios para inmunofluorescencia (IF) de los receptores IL-17A y AGT1 en cotiledón. A la fecha se han reclutado 170 gestantes, de las cuales 67% (115 Px) llegaron a término y de éstas, el 36% (41) cuentan con explante placentario, lo anterior ha permitido captar hasta el momento 4 casos y 37 controles. Se obtuvo el DNA de los 100 casos y 200 controles a término para el análisis genético. De modo alterno, ya se tiene estandarizada la tinción de H&E para el análisis histopatológico de cotiledón, así como también se tiene estandarizada la técnica de IF para AGTR1. El proceso de reclutamiento de gestantes en las clínicas y hospitales participantes sigue activo se cuenta con banco de muestras de suero sanguíneo, DNA, RNA cDNA y explantes placentarios en ultracongelación.

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Effect of aromatherapy with lavender (*Lavandula angustifolia*) in school children anxiety.

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The objective was to determine the effect of aromatherapy with lavender (*Lavandula angustifolia*) on the anxiety of school children. Quantitative, explanatory, quasi-experimental study, 154 students from 6th grade were surveyed, degree, distributed in two groups (n = 77 each). Anxiety was measured with the CMAS-R2 pre and post-intervention scale, which was done by saturating the atmosphere of lavender essential oil for the intervention group (GI) and water vapor for the comparison group (GC). The analysis of anxiety values showed statistically significant differences when comparing the post-intervention GI with respect to the pre-intervention GC (P = 0.0242) and between the same pre and post-intervention GI (P = 0.0046). In conclusion, the intervention with lavender essential oil managed to reduce anxiety in the GI. However, the GI and GC are not different after the intervention, which could be due to the type of population studied, where the behavior of the school is determined by several factors, which increases the variability of the group.

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Blood Brain Barrier alteration by extracellular vesicles released by Zika virus-infected monocytes.

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The blood brain barrier (BBB) is a cellular complex located at brain microvasculature, that regulates the exchange of different molecules between the cerebral parenchyma and the bloodstream, which can be disrupted during the infection with neurotropic viruses such as Zika virus (ZIKV), producing encephalitis, microcephaly and autoimmunity (Guillain-Barré syndrome).The main ZIKV target cell is the monocyte, when itwas activated released different types of extracellular vesicles (EVs). The EVs are a heterogeneous group of particles released by activated or apoptotic cells delimited by a lipid bilayer and do not be able to replicate. EVs are classified by their biogenesis in: vesicles derived from the plasma membrane (microparticles) and vesicles derived from endosomal maturation (exosomes), its main function is the intercellular communication. We evaluate the participation of the EVs released by ZIKV-infected monocytes in the BBB permeability by means of an in vivo model. Male Balb/c mice were intra-peritoneal injected with exosomes of ZIKV infected human monocytes (exZIKV). For the BBB permeability assays we use Evans blue and sodium fluorescein as tracers, and cerebellum, cortex and subcortical regions were evaluated.We founded that the BBB permeability was altered, probably by the pro-inflammatory process induced by the exZIKV. The BBB alteration was mainly observed in the cerebral cortex region at 48 hours after stimulation.By Western-blot assay, we founded the change of claudin (endothelial tight-junction protein) presence.

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Inmmunophenotypic classification of medulloblastoma and its correlation with clinical and histopathological variables of prognostic value.

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Medulloblastomas (MB) are aggressive neoplasms of the central nervous system of embryonic origin. In 2016, the WHO proposed a new categorization that complements the previous histopathological classification and divides them into four molecular subgroups: WNT activated, SHH activated, group 3 and 4. Due to the complexity and cost of molecular methods, alternative methodologies have been sought to approach molecular diagnosis in a more affordable way. A panel of biomarkers identifiable by immunohistochemistry (IHC) has been proposed that would allow inferring the molecular group and thus predict the behavior of the neoplasm. The objective wasTo classify cases of MB according to the immunophenotype proposed for each molecular group, and to determine its association with clinical and histopathological variables with prognostic value. Observational, retrospective cohort follow-up study. Cases of patients with diagnosis of MB were selected in a period of 10 years from a public hospital. Relevant clinical and histopathological data were recorded. A IHC panel of six biomarkers was performed: ß-catenin, GAB1, YAP1, p75NTR, p53 and Ki-67. Twenty-six cases of MB were included for analysis: 1 case (3.8%) WNT, 15 cases (57.7%) SHH, 6 cases (23.1%) No WNT/SHH, 4 cases (15.4%) NOS. A significant association was found between p53 expression and the variables recurrence/progression (p=0.014) and disease-free survival (p=0.008). The remaining variables did not show significant differences between the groups. No association was found between immunophenotype classification and the other variables. The percentage of p53 expression is associated with events related to poor prognosis (recurrence/progression and shorter disease-free survival time).

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Sex Susceptibility in the induction of colorectal tumors evaluated in a murine model.

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Sex susceptibility in a particular disease it is reflected in different parameters like incidence, for example, men have a higher incidence of colorectal cancer than women. on the other hand, epidemiology and experimental studies suggest that sex steroids have an important role in this phenomenon of sex susceptibility. In general, estrogens are considered as antitumor while androgens protumoral. nevertheless, most experimental studies are done in APC min +/- mouse model, and these do not reflect the involvement of sex hormones in the development of colorectal tumors. The gold standard in the study of human colorectal cancer in mouse model is the chemical methods of induction of tumors by azoxymethane and dextran sulfate sodium (AOM-DSS). In this work it was evaluated the role of sex steroids in the induction of colorectal tumors in male and female BALB/c mice by AOM-DSS treatment. Mice were gonadectomized at four weeks and two weeks later the tumors were induced. Unexpectedly, female mice had more and big tumors than male; interestingly, the orchiectomy had a protective role and the ovariectomy didn't have a significant effect. This suggest that androgens have a preponderant role in sex susceptibility in the development of colorectal tumors.

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Relationship of depression with serum levels of TNF-α and IL-6 in skin cancer patients.

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Cancer patients frequently manifest alterations such as depression and anxiety, these conditions can modify and trigger inflammatory responses that influence the evolution of the disease. The aim was to identify the prevalence of depressive syndrome and its relationship with serum levels of TNF-α and IL-6 in patients with skin cancer. This study included 35 patients from the Altos of Jalisco region with histopathological diagnosis of skin cancer with an age range of 35-90 years. Depressive syndrome was evaluated by the Calderón questionnaire and the TNF-α and IL-6 cytokines using the ELISA method (DuoSet, R&D Systems). The combined prevalence of anxiety and moderate depression was 45.7%. Serum levels of TNF-α they tend to be increased in the anxiety/depression group (36.3± 17.30 vs 14.40± 4.26 pg/ml) ($p>.05$); While there was no difference in the values of IL-6 (97.07± 37.8 vs 102.84± 32.79 pg/ml) ($p>.05$). In addition, a positive correlation was observed between the cytokines ($r = 0.783$, $p<0.001$). The results obtained suggest an inflammatory role for depression in skin cancer.

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Effect of oestradiol on the cytokine levels and oxidative stress in an experimental model of malaria.

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Malaria is the parasitic disease responsible of the higher mortality worldwide, it displays sexual dimorphism; men have higher mortality than women. Sex hormones are responsible of main differences between sexes and the principal female hormone is oestradiol which has deep effects on the immune system. In addition, the most recognized immune mechanism to eliminate the malaria parasite is generation of oxidative stress. However, this system is not specific, consequently oxidative stress regulation is very important through the antioxidant enzymes such as catalase (CAT), glutathione peroxidase (GPx) and superoxide dismutase (SOD). Thus, measuring the activity of those enzymes is an indirect way to measure oxidative stress. In addition, malondialdehyde (MDA) detection is used to evidence the damage induced by reactive oxygen species. Despite all the knowledge generated in this regard, it is unknown whether 17β-oestradiol is involved in this process. For this purpose, four weeks old males and female CBA/CA mice were gonadectomized and after one month were treated with 545 µg of 17β-oestradiol/Kg during 3 weeks. After the last day of treatment, mice were infected with $1x10^3$ *P. berghei* ANKA parasitized red blood cells. Parasitaemia was measured using blood smears stained with Giemsa. The eighth day post-infection, mice were sacrificed, and their blood, spleen, liver and brain were removed; and the activity of SOD, GPx, CAT and MDA levels were spectrophotometrically measured. Groups of intact and gonadectomized mice treated with vehicle or 17β-oestradiol uninfected were used as controls. Infection increased the antioxidant activity, mainly of females and decreased the concentration of NO in spleen, liver and brain. On the other hand, gonadectomy, increased antioxidant activity and oxidative stress in the brain and the concentration of NO mainly in males, therefore; parasitaemia decreased in males and increased in females. We conclude that estradiol modulates oxidative stress in malaria, however it is important to analyze other hormones such as testosterone to better understand the role of sexual hormones in the malaria sexual dimorphism.

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Prolactin is associated with higher parasitaemia, and cerebral malaria.

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Malaria is the parasitic disease responsible of the highest mortality worldwide. The most deadly complication is cerebral malaria (CM). The precise mechanisms involved in this inflammatory process is not well known, CM has been associated with pro-inflammatory cytokines such as TNF-α. This cytokine promote the parasite adherence on endothelial cells, cytotoxicity, apoptosis and necrosis in the brain particularly in hippocampus. Malaria exhibits sex dimorphism, male develop higher symptomatology and mortality than females, which suggest the participation of hormones in the immunoregulation of this disease. Prolactin (PRL) is a hormone involved in immunomodulation during inflammatory process. In this work, we studied whether PRL is involved in CM. We used knockout mice for the PRL receptor (KO) infected with *Plasmodium berghei* ANKA, as control we used wild type mice infected with the same parasite and measured parasitemia and the expression of TNF-α and IL-10 in brain and hypocampus. KO mice developed lower parasitaemia than wild type mice. Interestingly, infection decreased the mRNA expression of TNF-α in brain of both KO and wild type mice. This cytokine was expressed in a dimorphic way. We did not detect the expression of IL-10 in brain or hypocampus in KO or wild type mice infected with *P. berghei* ANKA. The results suggest that PRL decreased the immune response which was associated with higher parasitaemia. This work was supported by DGAPA IN220417.

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Effect of Acute Ingestion of Sucralose on Glucose Tolerance and Monocyte Subpopulations in Healthy Young Adults.

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The objective of this study was to evaluate the effect of acute exposure to 48 mg of sucralose in a single dose on mononocit subpopulations as well as its inflammatory and migratory capacity, studying its association with glucose, insulin, glucagon, peptide concentrations C incretins (GIP and GLP-1) in healthy young adults, subjected to an oral glucose tolerance curve of 180 minutes with measurements every 15 minutes. It is a cross-sectional, double-blind, placebo-controlled clinical trial with two groups, each with 25 healthy volunteers.Our results show that in patients with sucralose consumption the concentrations of insulin, peptide C and the determination of HOMA-IR were greater in time 30, 90 to 180 min compared with the placebo group; with a tendency in the increase of GIP and GLP-1 in the times 30 and 60 min. Significantly in the exposed group, the percentage of classical monocytes increased along with the expression of CD11c and CX3CR1; visceversa the percentage of intermediate and non-classical monocytes decrease, with an increase in the expression of CD11c and CCR2 as well as a decrease in CD206 and CX3CR1. Overall, these data suggest that sucralose consumption is associated with an inflammatory profile in circulating human monocytes with greater migratory tissue capacity as well as with alterations in the insulin response, decreasing their sensitivity as their secretion is probably enhanced by incretins and being a factor of risk for the development of insulin resistance, thus being a harmful product for patients from an early age.

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Neonatal Bisphenol A Exposure Affects the IgM Humoral Immune Response to 4T1 Breast Carcinoma Cells in Mice.

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Bisphenol A (BPA) is an endocrine disruptor of estrogenic nature. During the early stages of development, any exposure to BPA can have long-term effects. In this work, we study the potential alterations to the humoral antitumor immune (IgM) response in adult life after a single neonatal exposure to BPA. Female syngeneic BALB/c mice were exposed to a single dose of BPA of 250 µg/kg. Once sexual maturity was reached, a breast tumor was induced. After 25 days, the serum was obtained, and the populations of B cells in the spleen and lymph nodes were analyzed by flow cytometry. The reactivity of IgM was evaluated by 2D immunoblots. No significant changes were found in the B cell populations in the peripheral lymph nodes and the spleen. The level of Erα expression was not significantly different. However, the IgM reactivity was affected. In individuals treated with BPA, a decrease in the number of IgMs that recognize tumor antigens was observed. The possibility that these antibodies are the high affinity products of the adaptive response is discussed. The recognition of IgG was also evaluated but a null recognition was found in the controls as in the individuals treated with the 4T1 cells.

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Differences in recognition of tumor antigens from MMTV-PyVT mice by postnatal and adult IgM measured by 2D immunoblot.

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Detection of new tumor antigens is a major goal in cancer research. Natural IgM is present since the early stages of development. However, functional kinetics of the recognition of tumor antigens by IgM has not been reported. PyVT mice are a model of breast cancer that well simulates its counterpart in humans. In this study we present the kinetics of recognition of tumor antigens by postnatal IgM until the adulthood of transgenic PyVT mice. 2D immunoblots were obtained by probing mammary tumor lysate blots with IgM from sera from PyVT mice from 5 to 65 days of age. Antigen/antibody spots were counted on 2D master images and plotted with respect to time. The antigenic recognition by IgM of two transgenic mice is presented with respect to the control (no transgenic mice). The kinetic of the antigenic identification was undulatory over time. This result may be due to the behavior of B1-a cells responsible for IgM synthesis. The fact that IgM recognize tumor antigens before the tumor completely manifests itself, and that some IgM could still remain throughout the entire tumor process makes these antibodies candidates for early immunodiagnostics of the breast cancer.

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When ecology matters: Testosterone and the expression of secondary sexual traits in men of two populations with contrasting socio-ecological contexts.

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Testosterone (T) in males act as a hormone that modulates the expression of sexually dimorphic characters. In human men, it has been seen that at the beginning of puberty, this hormone increases its values, wich is associated with important biochemical and physiological changes that will modulate subsequent reproductive success. The synthesis and maintenance of this hormone is associated with significant metabolic costs and immunosuppressive effects. For this reason, it has been proposed that the degree of expression of the secondary sexual traits is an honest signal in the individual regarding the physiological and genetic condition. The aim of this work was to investigate whether levels of T in saliva are associated with the expression of male characters in two populations with contrasting lifestyles, an indigenous population belonging to the Me´phaa ethnic group located in the high mountain of Guerrero and the other, a population in urban conditions in Mexico City. The results suggest that indigenous men show lower levels of T than the men in the city. In addition, it was found that these differences had an influence on the expression of T-dependent sexual traits; muscle tone, height and shoulder / hip ratio. We suggested that given the environmental pressures in the indigenous population, the associated costs with the expression of T levels are higher, therefore, their levels are lower. In addition to this, the expression of secondary sexual characteristics, show differences in the type of relationship with the T with respect to what was observed in the city.

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Estrogen regulation in the immune response in malaria.

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Malaria is a parasitic disease caused by the protozoan *Plasmodium*, in 2018 it generated 219 million new cases and 435 thousand deaths. Malaria exhibits sexual dimorphism, males develop more severe symptoms and higher mortality than females (2), which suggest the participation of sexual hormones in this disease. It is probable that 17β-estradiol and its interaction with estrogen receptors are involved in the immunoregulation of this disease, because 17β-oestradiol is responsible for the main differences between sexes. In our research group, we documented that the decrease in estrogen concentration by gonadectomy significantly increased parasitemia, the number of CD3+ T cells, CD19+ B cells in the spleen and the concentration of TNF-α and IL-6 in the sera. In addition, it is known that T lymphocytes, B lymphocytes, macrophages and NK cells have receptors for sex hormones (8-10). Therefore, estrogen is likely to participate in sexual dimorphism and in the regulation of the immune response in malaria. In this work, parasitemia, relative expression of estrogen receptor alpha (ER-α) in spleen and levels of 17β-estradiol concentration were quantified in mice infected with *Plasmodium berghei* ANKA. Groups of 7 female and male CBA/Ca mice were gonadectomized or left intact, half of each group were treated with 17β-oestradiol (12µg / 50µL) or vehicle and all the mice were infected with $1x10^3$ erythrocytes parasitized with *P. berghei* ANKA; an additional group of mice were left without infection as a control. All mice were sacrificed on day 8 post-infection, the spleen was removed to extract mRNA, which was retrotranscribed and the cDNA was amplified by real-time PCR. In addition, the serum concentration of 17β-estradiol was quantified by EIA. Treatment with 17β-17β-oestradiol significantly increased parasitemia on day 8 post-infection compared to females treated with vehicle or males treated with estradiol. Administration of 17β-oestradiol to both female and gonadectomized males decreased parasitemia significantly on day 8 post-infection compared to vehicle-treated gonadectomized mice. Interestingly, the concentration of 17β-oestradiol decreased in infected males relative to males treated with vehicle, without modifying the expression of ER-α, although females treated with estradiol without infection significantly increased the expression of ER-α compared to females treated with vehicle. Our results suggest that 17β-oestradiol promotes parasite elimination in gonadectomized female mice without modifying the expression of ER-α or the serum levels of 17β-oestradiol.

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Effect of low-density lipoproteins on interleukin-1 beta production in human monocyte subpopulations *in vitro.*

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Emerging evidence suggests that monocyte subpopulations and low-density lipoproteins (LDL) may interact to induce vascular endothelial injury in atherosclerosis. However, the effect of native non-oxidized LDL on human monocyte subpopulations has not been fully explored. Our main goal was to assess the effect of increased concentrations of LDL on the percentages of monocyte subsets and the *in vitro* production of the proinflammatory marker interleukin (IL) 1β in these immune cells. Primary human monocytes were isolated from whole blood (n=5) using the Pan Monocyte Isolation Kit. Isolated monocytes were placed in ultra-low attachment 24-well cell-culture plates and differentially exposed to 0, 50, and 100 µg/ml LDL for 9 hr. After 3 hr of *in vitro* culture, monocytes were stimulated with 10 ng/ml LPS for 6 hr. At the end of the culture (9 hr), monocyte subpopulations and IL-1β were measured by flow cytometry. LDL progressively increased the non-classical monocyte subset and decreased the classical monocyte subpopulation. LDL by itself did not modify IL-1β production at any concentration. In contrast, classical monocytes exposed to 50 or 100 µg/ml LDL plus LPS showed a significant 20% increase in IL-1β production as compared to classical monocytes only stimulated with LPS. These data demonstrate for the first time that native non-oxidized LDL acts as a non-prototypical proinflammatory stimulus for human monocytes by increasing the non-classical monocyte subpopulation and IL-1β production.

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Soluble uric acid induces CD11c and TNF-α production in human macrophages *in vitro.*

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Uric acid is the end-product of purine metabolism that is excreted in urine. The abnormal blood elevation of uric acid leads to hyperuricemia (HU), a major risk factor for atherosclerosis. Emerging evidence suggest that atherogenesis is associated with systemic inflammation, characterized by increased levels of proinflammatory macrophages with the ability to produce tumor necrosis factor alpha (TNF-α) and CD11c. However, the possible relationship of uric acid with human macrophages has not been fully elucidated. The main goal of this study was to investigate the *in vitro* effect of different concentrations of uric acid on the proinflammatory profile of human monocyte-derived macrophages (MDM) by measuring TNF-α and CD11c. Buffy-coat samples were obtained from healthy blood donors (n=8) and monocytes were isolated by Ficoll and Percoll density gradient. Isolated monocytes were differentiated to macrophages in the presence of 10 ng/ml M-CSF for 7 days. Then, differentiated macrophages were *in vitro* exposed to 0 (Control), 3.5, 7.5, and 15 mg/dl uric acid for 12 hours. Production of CD11c and TNF-α were measured by flow cytometry. Our results demonstrate that MDM exposed to 3.75 and 15 mg/dl uric acid have 15% higher TNF-α expression than control MDM. Similarly, MDM cultured in the presence of 3.75 and 7.5 mg/dl uric acid show 25% higher CD11c expression than control MDM. These results suggest that high uric acid concentrations are able to induce inflammatory polarization of human macrophages *in vitro* and may partially explain the relationship between hyperuricemia and systemic inflammation in the scenario of atherosclerosis. As far as we know, this is one of the first studies demonstrating that uric acid can act as a non-prototypical immune stimulus for human macrophages.

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High-density lipoprotein reduction differentially modulates to classical and non-classical monocyte subpopulations in metabolic syndrome patients and in LPS-stimulated primary human monocytes *in vitro.*

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The effect of metabolic syndrome on human monocyte subpopulations has not yet been studied. Our main goal was to examine monocyte subpopulations in metabolic syndrome patients, while also identifying the risk factors that could directly influence these cells. Eighty-six subjects were divided into metabolic syndrome patients and controls. Monocyte subpopulations were quantified by flow cytometry and interleukin (IL-) 1 beta secretion levels were measured by ELISA. Primary human monocytes were cultured in low or elevated concentrations of high-density lipoprotein (HDL) and stimulated with lipopolysaccharide (LPS). The non-classical monocyte (NCM) percentage was significantly increased in metabolic syndrome patients as compared to controls, whereas classical monocytes (CM) were reduced. Among all metabolic syndrome risk factors, HDL reduction exhibited the most important correlation with monocyte subpopulations and then was studied *in vitro*. Low HDL concentration reduced the CM percentage whereas also increased the NCM percentage and IL-1 beta secretion in LPS-treated monocytes. The LPS effect was abolished when monocytes were cultured in elevated HDL concentrations. Concurring with *in vitro* results, IL-1 beta serum values significantly increased in metabolic syndrome patients with low HDL levels as compared to metabolic syndrome patients without HDL reduction. Our data demonstrate for the first time that HDL directly modulates monocyte subpopulations in metabolic syndrome as a non-prototypical anti-inflammatory stimulus.

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Low serum interleukin-6 is a differential marker of obesity, hyperglycemia, dyslipidemia, and systemic inflammation in women and men.

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Emerging evidence in mice suggests that interleukin (IL-) 6 has a role in obesity, hyperglycemia, dyslipidemia, and systemic inflammation. However, there is scant information regarding the role of IL-6 in human obesity. Thus, we studied the serum levels of IL-6 in normal weight, overweight and obese subjects, and examined possible associations of IL-6 with hyperglycemia, insulin resistance, dyslipidemia, and systemic inflammation. One hundred three women and men were included in the study. Body mass index (BMI), waist circumference, body fat percentage, blood glucose, insulin, total cholesterol, and triglycerides were measured. Serum levels of tumor necrosis factor alpha (TNF-alpha), IL-10, and IL-6 were measured by ELISA. One-way ANOVA results showed a 2.5-fold significant decrease in the serum levels of IL-6 in overweight and obese individuals as compared to controls. Serum IL-6 levels exhibited significant inverse correlations with BMI (r=-0.39, P<0.0001), waist circumference (r=-0.42, P<0.001), blood glucose (r=-0.40, P<0.0001), triglycerides (r=-0.34, P<0.0001), and TNF-alpha (r=-0.48, P<0.0001), whereas a strongly positive correlation was found with IL-10 (r=0.77, P<0.0001). Multiple linear regression analyses revealed that behavior of IL-6 was mainly influenced by IL-10 (beta=0.28, P=1.95x10-6), TNF-alpha (beta=-0.67, P=0.0017), and body fat percentage (beta=-5.95, P=7.67x10-5) in women. In contrast, IL-10 (beta=0.37, P=1.34x10-9), TNF-alpha (beta=-0.85, P=0.0005), and triglycerides (beta=1.07, P=0.0007) were major influencing factors of IL-6 in men. This study demonstrates that IL-6 is a marker of hyperglycemia, hypertriglyceridemia, and systemic inflammation that is differentially regulated in women and men.

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Metabolic Alterations M2 macrophage adoptive transfer ameliorates glucose intolerance by increasing IL-10 expression and AKT activation in visceral adipose tissue of mice with high-fat diet-induced obesity.

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Glucose intolerance is an intermediate step in the development of type 2 diabetes (T2D). Glucose intolerance associates with M1/M2 macrophage unbalance in visceral adipose tissue (VAT). Thus, we speculate that restoration of the M2 macrophage population by adoptive transfer might result in improved glucose tolerance. Our main goal was to examine the effect of M2 macrophage administration on glucose intolerance of mice with high-fat diet (HFD)-induced obesity. C57BL/6 mice fed a HFD for 12 weeks and then received thrice 20 mg/kg streptozotocin (HFD-GI). Bone marrow-derived stem cells were collected from donor mice and differentiated/activated into M2 macrophages for intraperitoneal administration into HFD-GI mice. M2 macrophage treatment abolished glucose intolerance independently of obesity. M2 macrophage administration increased interleukin-10 in VAT and serum, but showed no effect on serum insulin. While nitric oxide synthase-2 and arginase-1 remained unaltered, M2 macrophage treatment restored AKT phosphorylation in VAT. These results show for the first time that M2 macrophage adoptive transfer ameliorates glucose intolerance by increasing IL-10 expression and AKT phosphorylation in VAT. Further studies in humans are needed to clarify whether adoptive transfer of M2 macrophages might be an alternative therapeutic option for prediabetic and T2D patients.

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Low serum levels of IL-10 and IFN-gamma in umbilical cord blood correlate with increased birth weight.

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Interleukin-10 (IL-10) and interferon-gamma (IFN-gamma) are associated with body weight alterations in adolescents and adults. However, little is known regarding IL-10 and IFN-gamma levels in the binomial mother-infant and their relationship with birth weight. Ninety two mother-infant pairs were enrolled in the study. Anthropometric parameters were registered in mothers and infants. IL-10 and IFN-gamma levels were measured in mothers (serum) and infants (umbilical cord blood) by ELISA. Correlation coefficients (*r*) were calculated by the Spearman's correlation model, and IL-10 and IFN-gamma levels were compared by performing the student's T-test. Fetal IL-10 and maternal IFN-gamma were inversely correlated with birth weight (*r*=-359/*P*=0.014 and *r*=-302/*P*=0.027, respectively). Infants with birth weights greater than the 90th percentile exhibited the lowest values of cord blood IL-10 with no significant changes in IFN-gamma. In mothers, serum IL-10 was correlated with both maternal and cord blood IFN-gamma (*r*=0.407/*P*=0.002 and *r*=0.380/*P*=0.014, respectively). Serum IFN-gamma was correlated with pBMI and gestational age (*r*=-0.277/*P*=0.039 and *r*=0.550/*P*=0.014, respectively). In infants, cord blood IFN-gamma was correlated with mother age, gestational age, and cord blood IL-10 (*r*=-0.324/*P*=0.028, *r*=0.418/*P*=0.013, and *r*=0.406/*P*=0.007, respectively). This work shows for the first time that fetal IL-10 and maternal IFN-gamma decrease as birth weight increases in infants born at term. To our knowledge, this is one of the first reports of the cytokine cross-talk between mother and infant that might potentially contribute to birth weight changes.

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Sexual dimorphism in the production of anti-GA antibodies and cytokines in patients with Multiple Sclerosis.

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Multiple Sclerosis (MS) is a chronic disease of the Central Nervous System (CNS) in which autoimmune processes against the myelin sheaths lead to neurodegeneration. MS patients show sexual dimorphism in prevalence, prognosis, progression, and age at disease onset. Glatiramer Acetate (GA) is one of the first-line Disease-Modifying Therapies (DMT) used to delay neurodegeneration. It has been reported that GA induces the production of anti-drug antibodies (ADA) with consequences not yet established. The objective was to determine if there is a differential pattern of correlations between anti-GA antibodies and cytokine levels in MS patients grouped by sex. Immunoglobulin IgG levels were measured in 43 MS patients treated with GA: total non-specific IgG by affinity chromatography, total IgG anti-GA, and IgG anti-GA subclasses (IgG1-4) by ELISA. A correlation analysis between 14 cytokine levels and anti-GA IgG antibodies was determined by Spearman's method. We observed a correlation triangle that connects IL-31, IFN-γ, and IL-25 only in men, while there is a strong positive correlation between IL-31 and sCD40L exclusively in women. About immunoglobulins, we found a correlation in women patients: sCD40L vs. IgG anti-GA ($r= 0.41$ $p<0.05$) and IL-31 vs. IgG3 anti-GA ($r= 0.498$ $p<0.01$). Men group showed no correlation of sCD40L and IL-31 with immunoglobulins. The evidence presented here suggests that there is a sexual dimorphism in the production of antibodies against GA that might be modulated by a different pattern of cytokines. Therefore, another different mechanism of action could be involved in MS pathophysiology in a sex-dependent manner.

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Proinflammatory effect of desmopressin in rats with adjuvant induced arthritis.

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Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory disease characterized by joint inflammation, bone reabsortion and disability. Several therapeutically approaches have been developed in order to decrease the activity of some proinflammatory interleukins. Previous evidence indicate that arginine vasopressin (AVP) play an important role in the regulation of immune and inflammatory responses, mainly associated to inflammatory cytokines. Previously we demonstrated that the deficiency of circulating AVP and oxytocin induced by the neurointermediate pituitary lobectomy in rats, prevent or significantly diminish the arthritic inflammation in the adjuvant induced arthritis (AIA) paradigm. Here we evaluate the immune response in the AIA rats treated with antagonists or agonists of AVP receptors (r). Groups of female Sprague-Dawley rats where divided in the following groups (n=5): 1) Intact control (CTRL), 2) AIA, 3) AIA+Conivaptan (CV) (1.5 mg/kg/12 hours(i.m), an antagonist of V1a-V2 AVPr, 4) AIA+C9 (2 mg/kg/12 hours/i.m.), an antagonist of V1a-V2 AVPr) and 5) AIA+Desmopressin (DP) (0.75 µg/kg/12/i.m.), an agonist of V2 AVPr). Drug treatments started 3 day before immunization and daily continued until the end of the experiment (21 days after immunization). Ankle joint Inflammation were evaluated by pletismometry before start the experiment and twice a week). Results showed that DP treatment to the AIA group induced a significant ankle inflammation ($p<0.001$ vs AIA), while the AIA+CV and AIA +C9 groups, treatments no changes in the inflammatory response where observed as compared with the AIA group. At the present doses of CV and C9 compounds, no any anti-inflammatory effect was observed, whereas desmopressin, the agonist of AVP-V2r, greatly stimulate the arthritic inflammation supporting the view that acting directly AVP is a strong immunostimulating hormone.

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Role of vasopressin in histamine-induced capillary permeability: physiological and ultrastructural study.

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Arginine vasopressin (AVP) deficiency induced by hypophysial neurointermedia lobectomy (NIP) and increased vascular permeability (swelling) in NIP animals has been described in the rat. Histamine participates in the edema of the inflammatory process. Here we hypothesize that, if the AVP participates in the regulation of the inflammatory process, this will be reflected in the capillary ultrastructure. Male rats were divided into the following groups: Intact Control (IQ), IQ+Histamine (HIST), LNI+HIST, LNI+DP+HIST and IQ+HIST+CONIVAPTAN (V1a/V2 AVP receptor antagonist). Except for the IQ group, all others underwent histamine-mediated Evans blue extravasation skin test. Skin fragments stimulated with the highest dose of histamine were prepared for transmission electron microscopy. The results confirmed the increase in capillary permeability in LNI animals and its decrease by PD. Compared to the IC group, the ultrastructural study of endothelial cells of the CI+HIST group showed an increase in the thickness of endothelial cells with an edematous appearance, the presence of vacuoles of different sizes, some with well-defined limits and density similar to that of the intravascular space, a greater number of vacuoles of the vesicular-vacuolar system that apparently converge to form larger vacuoles. In the LNI+HIST group, ultra-structural changes were more important; greater endothelium thickness, diffuse edema and presence of large vacuoles with poorly defined limits. In the LNI+DP group, endothelial morphology was restored to normal; normal endothelial thickness, disappearance of oedema and decrease in the number of vacuoles. Conclusions; The AVP plays an important role in the regulation of capillary permeability in histamine edema. The results suggest the presence of other mechanisms through which the AVP could participate in the regulation of vascular permeability.

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Interrelation of circulating levels of Neurokinin B with vasomotor symptoms and changes in mood in women at reproductive, perimenopausal and postmenopausal stages.

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Symptoms at menopause (vasomotor and mood alterations) strongly impact women's quality of life. In the absence of estrogens from the ovary, the hypothalamic pituitary axis release excess gonadotropins with the hypertrophy of KNDy neurons producing Kisspeptin (KISS), Neurokinin B (NKB) and Dynorphin (Dyn) involved in regulation of gonadotropin-releasing hormone (GnRH). There is evidence that vasomotor symptoms are strongly correlated with the secretion of the GnRH, suggesting the influence of NKB levels The aim was to evaluate the serum levels of NKB in women at the reproductive, perimenopausal and postmenopausal stages and to examine its association with vasomotor symptoms and changes in mood. In women aged 30-65 at reproductive stage (n=15), perimenopause (n=15) and early postmenopause (n=15) we collected scales of sleep alteration, hot flashes, perceived stress, anxiety and depression mood. A venous blood sample was obtained for the measurement of biochemical and hormonal parameters. Serum FSH, 17β-estradiol, and LH levels were measured by chemiluminescence. NKB was quantified by liquid chromatography coupled to mass spectrometry (LC-MS). We compared values between groups by one-way ANOVA and multiple regression was used to associate NKB levels with the symptoms. The average age of women at reproductive stage was 36.8±5.5 years, at perimenopause 44.0±6.0 years and at postmenopause 52.1±3.6 years. As expected, women at early postmenopausal stage had low estradiol levels (18.71±9.82 pg/ml *vs* 67.0±82.3 pg/ml), and high FSH (71.6±23.4 mUI/ml *vs* 41.7±40.3 mUI/ml) compared with the perimenopause group. The hot flashes were perceived at higher intensity in women at perimenopause and in a mild intensity at postmenopause. The peak of NKB was identified in a retention time of 4.2 minutes. NKB levels were lower at follicular phase compared to luteal (p<0.0001), as well as, NKB levels diminished at ovulatory in comparison with luteal phase. In addition, women at perimenopause had diminished NKB concentrations compared the follicular phase. Multivariate analysis showed that hot flashes intensity was negatively related with NKB concentrations. Surpringly, NKB concentrations inversely correlated with BMI. These data suggest that NKB levels vary at different reproductive stages and the variations depend on BMI and other factors. Further studies are needed for a better knowledge of factors implicated with hot flashes and other symptoms at menopause.

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Sleep extension increases the effect of caloric restriction over body weight and ameloriates the chronic low-grade inflammation in adolescents with obesity.

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Diminution of sleep may enhance the effect of energy consumption and diminished physical activity on obesity. Changes in the immune and metabolic responses have also been proposed. However, the evidence that extending sleep duration might favor weight loss is insufficient. The aim was to compare the effect of dietary restriction with or without prescribing sleeps extension, over weight loss and immune measurements in adolescents with obesity. In a prospective comparative, fifty-two adolescents with obesity (24 males and 28 females) received a diet with 500 calories restriction, randomly allocated to a group adding sleep extension (n=25). The control group (n=27) received only diet for 4 weeks. We collected data on anthropometry, caloric intake and self-reported sleep diaries. Fasting blood samples were obtained for metabolites and hormones. Serum IL-6, TNF-α, leptin and insulin were quantified by ELISA. MT6S and cortisol excretions were measured in first morning urine collection. Measurements were carried out at baseline and at the end of intervention. After diet, weight decreased in both groups. Sleep extension, improved weight loss (p<0.00001) and waist girth reduction (p=0.00003), diminishing insulin (p=0.002) and IL-6 levels (p=0.02), as well as sleep time increased 0.9 hrs with an increase in sleep efficiency. Caloric restriction was less effective in adolescent females. No differences in cortisol or MT6S excretion were found. Sleep extension favors weight loss in adolescents under caloric restriction, and improves metabolic conditions and chronic low-grade inflammation, thus supporting a possible additional benefit to diet treating obesity in adolescents.

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Growth hormone receptors in larvae of the gastrointestinal parasite of ruminants *Haemonchus contortus*.

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Recent studies demonstrated the presence of prolactin and progesterone receptors in larvae of *Haemonchus contortus*. The parasite infection is more severe in lambs than in adult animals, factors that could be involved in susceptibility could be the effect of growth hormone (GH) in the lambs. The objective of the study was to identify possible GH receptors (GHR) in larvae 3 of *H. contortus*. Larva cells were obtained and labeled with a primary anti-GHR antibody (Santa Cruz labs.) followed by a secondary anti-mouse IgG antibody (eBioscience labs.) coupled to Alexa 647. The counting of positive cells was performed with a FACScalibur cytometer. Immunolocation of possible GHRs was performed in permeabilized larvae treated with anti-GRH antibody, secondary mouse anti-IgG coupled to FITC (Molecular, Probes ®, USA) and confocal microscopy. Three cell populations were determined by according to granularity and size. Cell population with larger size and granularity have 3.34% of cells positive for GHR. The immunolocation of possible GHRs in the larval intestinal cells, which correspond to cells of large size and granularity was observed. These results indicate that *H. contortus* larvae express GHR in intestinal cells and possibly have the ability to respond to stimulation with GH as has been observed with other hormones. The identification of GHR in *H. contortus* larvae strongly suggests the existence of a biological effect of the hormone on the parasite in the host. Supported by PAPIIT-UNAM IN-218018.

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Effect of exposure to low doses of ozone on the expression of IL-17A and its receptor during the process of progressive neurodegeneration in the hippocampus of rats.

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In populated cities, air pollution is one of the main risk factors for the incidence of various health problems. The inhalation of tropospheric ozone of this gas pcauses a state of oxidative stress, which is considered a critical factor in the development of neurodegenerative diseases. Recently, our work group reported that during the initial stage of exposure to low doses of ozone an acute CNS damage occurs, which is accompanied by a Th17/IL-17A systemic response that can be self-regulated. However, if the exposure is prolonged, the damage caused is irreversible and the inflammatory response, characterized by an increase in the hippocampus of IL-17A is no longer self-limiting. However, we still do not know the cellular source and targets of the secreted IL-17A in the hippocampus of the animals of this experimental model. Thereby, the aim of this study was to evaluate the effect of chronic exposure to low doses of ozone on the expression of IL-17A and its receptor in neurons, microglia, astrocytes and T cells in rat hippocampus.For that purpose, we used 72 Wistar rats, divided into 6 groups (n = 12): control group (without ozone) and groups exposed to ozone (0.25 ppm, 4 h daily) for 7, 15, 30, 60 and 90 days, respectively. Six subjects from each group were processed to quantify IL-17A by ELISA, and the remaining 6 were processed for immunohistochemistry (against IL-17A or IL-17RA and GFAP, Iba-1, NeuN or CD3). The data obtained by ELISA test showed a significant increase in the concentrations of IL-17A in the groups of 7, 15, 30 and 60 days of exposure, compared with the control (P <0.05). Furthermore, they indicate that hippocampal neurons are the cells that showed the greatest immunoreactivity against IL-17A and IL-17RA between 60 and 90 days of exposure to ozone. With these data we conclude that exposure to ozone induces an increase in the expression of IL-17A in hippocampus. Also, data suggest an autocrine secretion of this cytokine by hippocampal neurons of rats under a state of oxidative stress induced by chronic exposure to low doses of ozone.

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Alliin lowers adipocyte hypertrophy and leptin levels in DIO mice.

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The energy imbalance and alterations in the metabolism observed in obesity causes adipocytes hypertrophy and high secretion of the adipocytokine leptin, which perpetuates a systemic dysmetabolism distinguished by dyslipidemia and insulin resistance. Alliin (S-allyl-cysteine sulfoxide), is the most abundant organosulfurized compound in garlic and it has been identified with antioxidant, antiinflammatory and antidiabetic properties. Previously, our group have described that alliin is able to immunomodulate the *in vitro* inflammatory response of 3T3-L1 adipocytes. The objective was to assess the *in vivo* effect of alliin on adipocyte hypertrophy and on leptin (serum levels and gene expression). Two groups of C57BL/6 mice were feed administering different diets: Standard diet, STD group (n=23) & High-Fat diet, HFD group (n=21), for 9 weeks. After that, the groups were subdivided: STD and HFD groups as controls, and STD+A and HFD+A as experimental groups traded with 15 mg/kg of alliin daily for 3.5 weeks. At 12.5 weeks the mice were sacrificed to obtain their epididymal adipose tissue and serum for H&E, ELISA and qRT-PCR performance. We observed significant differences in adipocyte size between HFD and HFD+A groups (p<0.0001). In addition, the serum levels of leptin (p<0.05) and the relative gene expression (p<0.01) was diminished in epididymal adipose tissue from HFD+A group. Alliin treatment decreases the size of adipocytes, also reduces the serum leptin levels and gene expression in mice with diet-induced obesity, this improvement should impact by estoring the energy balance and reducing the metabolic alterations that characterize obesity.

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Amelioration of Renal Fibrosis Development Mediated by Arginine Vasopressin (AVP) deficiency in a Model of Ureteral Obstruction.

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Arginine vasopressin is a pleiotropic hormone that has been recently described as a stress-inducible hormone that has several inflammatory and immune-regulatory functions. During renal fibrosis disease, there is a loss of organ functions because the inflammation, cell differentiation to fibroblasts and pathogenic (high) collagen deposition, that ends in the replacement of the parenchyma. Actually, there is not a specific treatment for this disease. We investigate if arginine vasopressin deficiency, by a neurointermediate pituitary lobectomy (NIL), could limit the development of inflammation, collagen deposition and fibroblast differentiation, in kidneys from rats with unilateral ureteral obstruction (UUO). Groups of rats were divided in: SHAM, renal fibrosis (RF), NIL and NIL+RF. Minimal fibrosis development and glomerular sclerosis were noted in the kidneys from NIL+RF compared to RF. No differences in inflammatory infiltration and parenchyma lost were identified in NIL+RF versus RF. Less damage percentage was significantly less in NIL+RF compared to RF. The presence of collagen type I in kidney samples was significantly increased compared to collagen type III in NIL+RF. No significant differences were noted in collagen type I and III in any other group. The results indicate that AVP deficiency delays the development of renal fibrosis by ureteral obstruction, limiting the kidney damage, glomerular sclerosis and reducing the collagen type I deposition.

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Relación entre el contaminante ambiental bisfenol A (BPA), la respuesta inmunitaria y la susceptibilidad a la infección por *T*oxocara canis.

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En la actualidad, la contaminación ambiental es un serio problema que afecta a todo el mundo. Dentro de los distintos compuestos contaminantes del ambiente podemos encontrar agentes químicos, como el BPA, y contaminantes biológicos como los huevos de distintos parásitos, tal es el caso de *Toxocara canis*. En el presente trabajo, se evaluó el efecto del BPA administrado durante la etapa perinatal en ratas, sobre la respuesta inmunitaria durante la vida adulta, y la susceptibilidad al parásito zoonótico *T. canis.* Se analizaron las cargas parasitarias, así como los porcentajes de las subpoblaciones celulares del sistema inmunitario, la expresión de citocinas, y los títulos de anticuerpos contra el parásito. Se observó un aumento en el número de parásitos a nivel hepático y pulmonar. No hubo cambios significativos en los porcentajes de las células de la respuesta inmunitaria analizadas, sin embargo, hubo un aumento en las citocinas Th1 (TNF-α e IFN-γ) y una disminución en las Th2 (Il-4, IL-5 e IL-13), además de una reducción en los títulos de anticuerpos contra el parásito en las ratas expuestas perinatalmente al BPA. En conclusión, la administración perinatal de BPA, afecta el desarrollo de la respuesta inmunitaria durante la vida adulta, modificando la producción de citocinas y anticuerpos por parte de las células de la respuesta inmune, lo cual ocasiona un aumento en la susceptibilidad a la infección por *T. canis*.

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Oral consumption of β-caryophyllene protects against body weight increasement and improves glucose homeostasis in high-fat diet consuming mice.

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Obesity is a non-communicable disease that impair the metabolic homeostasis, affects a considerable part of the world population and lead to a broad spectrum of comorbidities. In spite of the many therapeutic options available to treat this disease, none of them is completely effective. Prevention is an important approach against obesity, and in this field, the nutraceuticals have shown to be promising alternatives to prevent obesity. β-caryophyllene (BCP) is a sesquiterpene present in a variety of dietary products and has proven to generate regulatory effects on metabolism and immune system. The aim of the present study was to evaluate the protective effects of daily β-caryophyllene oral administration (50 mg/kg body weight) against body weight increase and glucose homeostasis in a high fat diet (HFD) fed protocol (16 weeks) on C57BL/6 mice, with standard diet as control. β-caryophyllene administration protected against body weight increase, reduced fasting glucose and improved the postprandial response to oral glucose consumption and insulin administration compared to HFD group without treatment.

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Relationship between lack of sleep and plasma levels of acylated Ghrelin in young adult with obesity.

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Lack of sleep (FS) refers to the inadequate amount of sleep hours to achieve rest, ideal functioning, performance and well-being; the time required varies throughout life, a pattern of 7-8 hours per night is established for the young adult. During the phases of sleep, I and IV (no -REM sleep, NREM), endocrine activation is generated, determined by the secretion of growth hormone, is ultimately related to the secretion of ghrelin that allows positive feedback, and increase energy expenditure translated into the release of regulatory hormones from food intake and satiety, associated with obesity. The goal of the study was to find the relationship between lack of sleep and plasma acylated ghrelin levels (GRp) in the young adult with obesity. Fifty-six young adults with obesity were studied, forming into 2 groups randomly (n = 28), a control group (GC) with normal sleep hours and experimental group (GE) with lack of sleep, participants diagnosed with chronic diseases, disorders were excluded from sleep, with hormonal treatment or to fall asleep, those who consume psychoactive substances. BMI determination, plasma acylated ghrelin and identification of sleep hours were performed. The analysis of GRp showed statistically significant differences between both groups and was correlated inversely with the hours of sleep.

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Neuroinflammation during experimental pulmonary Tuberculosis in association with behavioural abnormalities.

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Tuberculosis is an infectious disease caused by Mycobacterium tuberculosis that induces chronic inflammation. During the development of chronic inflammation there is neuronal activation and changes in the synthesis and production of neurotransmitters and cytokines, that affect functions such as learning, memory and mood. Patients with TB develop diverse mental disturbances like depression and anxiety. Thus, we investigated the effect of lung infection with M. tuberculosis on the immune response in the central nervous system (CNS) and its relationship with behavioural changes in absence of culturable mycobacteria in the brain in a murine model of pulmonary TB. The results showed an increase in TNFa, IFNg, IL-12, IL-4 and TGF-β, as well as the enzymes IDO and iNOS in the hypothalamus, hippocampus and cerebellum. There were significant changes in the production of the neurotransmitters noradrenaline, adrenaline, dopamine and serotonin. Neuronal damage occurred in the hippocampus and cortex at an early stage of the disease, and it led to neuronal death in the progressive phase of the disease, with an increase of p38 and JNK. BDNF levels decreased and an increase of blood-brain barrier permeability was seen. All these changes coexisted with depressive-like behaviour, sickness behaviour, anxiety-like behaviour, neurological damage and impairment of short and long-term memory.These results show for first time that chronic inflammation during pulmonary tuberculosis causes neuroinflammation and behavioral abnormalities in absence of brain infection.

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Expression of transcription factors on lymphocytes from Nile tilapia (*Oreochromis niloticus*) exposed *in vitro* to diazoxon.

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Organophosphorus pesticides (OPs) are a broad-spectrum insecticides widely used in agricultural activities, representing 50% of the world's use of pesticides. Diazoxon, a metabolite of diazinon, has as its main mechanism of toxicity the inhibition of the Acetylconlinesterase (AChE) enzyme, causing immunotoxic effects that can alter humoral and cellular parameters of vertebrate organisms, including fishes. Within the cellular mechanisms of adaptive immunity in fish, subpopulations of T helper lymphocytes CD4+ (Th1, Th2 and Th17) have been characterized, involved in the production of cytokines and that jointly collaborate in different phases of the immune response. The polarization towards the different phenotypes of these subpopulations of T lymphocytes is regulated by the expression of gene transcription factors such as T-bet, GATA-3, Foxp3 and RORγt. Various pathological processes of immunological origin have been described due to problems in the expression of these transcription factors generally associated with inflammatory diseases. Therefore, the present project intends to evaluate the *in vitro* effect of Diazoxon on the expression of these master transcription factors in lymphocytes, using the Nile tilapia fish (*Oreochromis niloticus*) as a study model.

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Molecular characterization of a prolactin receptor like in embryonary cells of the tick *Rhipicephalus microplus.*

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The tick *Rhipicephalus microplus* is an obligate hematophagous with a great impact in the cattle industry due to its expoliative and vectorial capacity to transmit diseases such as Babesiosis and Anaplasmosis. The control of this tick is based on the use of ixodicides, however, their inadequate use has selected highly resistant populations to these components. For this reason, the search for new forms of biological control turns out to be the most viable alternative. Currently, one of the questions unsolved in the biology of the tick, is the elucidation of some mechanisms involved in the host-parasite relationship. In this regard, during the engorging tick process, the host hormones in the blood may be regulating mechanisms related with establishment, stage differentiation, and reproduction, and even with the resistance or susceptibility to ixodicides. Several working groups worldwide have described the relationship between host hormones and parasites, and the course of the infection. Hormones exert their effects through the interaction with nuclear and membrane receptors, that results in genomic and non-genomic effects. Prolactin (PRL) is a peptide hormone produced and secreted mostly by specialized cells located in the adenohypophysis of mammals. In this work, we describe, for the first time, a PRL receptor-like gene (*r-prl*) encoded in the genome of *R. microplus*. Our results revealed the presence of the gene and the relative expression by PCR assays. The structural modeling and the molecular dynamics analysis showed a high similarity with the *Homo sapiens* PRL receptor, and interaction with the PRL molecule. The elucidation of the mechanisms involved in the host-parasite relationship mediated by hormones opens the possibility of new targets for the control of pests.

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Plaguicidas anti-colinesterásicos y su efecto sobre la funcionalidad de los linfocitos.

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Los linfocitos son células de la respuesta inmune y se caracterizado por poseer un sistema colinérgico extra-neural, es decir, presentan toda la maquinaria necesaria para generar de *novo* acetilcolina, molécula que podría jugar un papel importante en la regulación de la respuesta inmune. No obstante, la integridad de estas células, puede ser alterada por exposición a plaguicidas organofosforados (POFs), como diazinón, el cual durante su biotranformación genera un metabolito altamente tóxico, diazoxón (DZO), que se caracterizan por ser inhibidor la enzima AChE. De esta manera el sistema colinérgico linfocitario podría ser blanco de los POFs en el fenómeno de inmunotoxicidad. En este estudio se utilizó a tilapia nilótica (*O. niloticus*), un pez teleósteo con importancia económica y ecológica. El objetivo de este trabajo fue evaluar el efecto de DZO sobre Ca^{+2} intracelular, pERK, apoptosis, senescencia y potencial de membrana mitocondrial en linfocitos de pez. Células de pez fueron expuestas a DZO (0.001, 1.0 y 10 µM) con su respectivo grupo control durante una y dos horas, posteriormente cada parámetro fue determinado mediante citometría de flujo. Los resultados mostraron que DZO provoca disminución en el flujo de Ca^{+2} intracelular y pERK, además de inducción a apoptosis, senescencia y pérdida del potencial mitocondrial. Estos resultados indican que DZO causa un daño significativo en los parámetros funcionales de los linfocitos, lo que sugiere altaspropiedades inmunotóxicas de este plaguicida.

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Efecto de diazinón, un plaguicida anticolinesterásico, en la muerte celular de neutrófilos.

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Los neutrófilos son células del sistema inmune innato esenciales para la defensa de los organismos frente a patógenos y se caracterizan por ser la primera línea de defensa, para ello poseen diversos mecanismos microbicidas. Sin embargo, existen sustancias como diazinón (DZN), un plaguicida organofosforado, que se caracteriza por inhibir a la enzima acetilcolinesterasa, produciendo efectos inmunotóxicos y neurotóxicos en los organismos. Por lo que, el objetivo de este trabajo fue evaluar el efecto inmunotóxico de DZN sobre los diferentes tipos de muerte celular de neutrófilos de tilapia nilótica (*Oreochromis niloticus*). Para este propósito, los peces fueron expuestos *in vivo* a 3,91, 1,95 y 0,97 mg/L de DZN durante 6 y 24 h. La formación de trampas extracelulares de neutrófilos (NETs) se visualizó con microscopia de fluorescencia, mientras que la apoptosis y necrosis se analizaron mediante citometría de flujo. Los resultados mostraron que DZN no induce apoptosis, ni necrosis de neutrófilos; mientras que la formación de NETs se observó incrementada. Los resultados indican que DZN *per se* estimula a los neutrófilos, lo que puede tener repercusiones en procesos inflamatorios.

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Evaluación del sistema colinérgico en peces *Poecilia reticulata* en un periodo de recuperación posterior a la exposición a temefos y spinosad.

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El sistema colinérgico es fundamental en regulación de eventos como: proliferación, diferenciación, neurogénesis, gliogénesis, maduración neuronal, plasticidad y desarrollo axonal. Sin embargo, existen plaguicidas utilizados para el control de vectores, como temefos o spinosad, los cuales tienen potencial efecto neurotóxico, a través de la alteración del sistema colinérgico. Por lo que, el objetivo de este trabajo es determinar la expresión del receptor nAChR α7 y la actividad de la enzima acetilcolinesterasa (AChE) en peces guppy *(P. reticulata)* en un periodo de recuperación después de la exposición sub-aguda a spinosad y temefos.

Para lo cual, se expusieron peces a temefos (100 g/ 100 L) y spinosad (0.7 g/ 100 L) por un periodo de 21 días y después se transfirieron a un ambiente libre de plaguicida durante 65 días, durante ambos periodos se evaluó la actividad de AChE y la expresión de nAChR α7. Los resultados indican que ambos plaguicidas son capaces de alterar el sistema colinérgico, alteración que se recupera hasta 35 días después de la ausencia de plaguicidas en el medio. Sin embargo, el efecto inhibitorio de AChE ocasionado por temefos es más prolongado, lo que sugiere que este último plaguicida es más tóxico. No obstante, ambos plaguicidas representan un riesgo potencial para los organismos no blanco.

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Muerte celular inducida por plaguicidas anti-colinesterásicos utilizados en campañas de salud pública en México.

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Temefos es un plaguicida anti-colinesterásico frecuentemente utilizado en campañas de salud pública para el control de vectores trasmisores de enfermedades como dengue, chikungunya y zika. No obstante, existen reportes que indican que este plaguicida provoca efecto sobre la salud de organismos no blancos. Por lo que se ha propuesto, para este fin, el uso de sustancias de origen natural como spinosad, sin embargo, la toxicidad de estas sustancias no está completamente esclarecida. El objetivo del presente trabajo fue evaluar la inducción de muerte celular por exposición *in vivo* a temefos y spinosad en peces guppy (*Poecilia reticulata)*. Los organismos fueron expuestos a temefos y spinosad a concentraciones manejadas por la SSA en las campañas de fumigación para el control del mosquito (30 g/L de temefos y 0.21g/L de spinosad), por un periodo de 7, 14 y 21 días. Posteriormente se extrajo el bazo, y la muerte celular se determinó mediante citometría de flujo utilizando el kit de Anexina V-FITC. Los resultados indican que la exposición a temefos a 7, 14 y 21 días induce muerte por apoptosis (p<0.05) con respecto a los peces control. Además a 21 días de exposición el plaguicida provocó necrosis celular. Por otra parte los resultados indican que spinosad no provocó muerte celular en los tiempos de exposición probados con respecto a organismos control. Los resultados sugieren que spinosad es menos tóxico que temefos, sin embargo se necesitan más estudios para evaluar los efectos tóxicos de spinosad.

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Contaminación ambiental atmosférica, microambiente tumoral y cáncer de mama: un estudio traslacional.

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El cáncer de mama es una de las neoplasias malignas más comunes y la segunda causa de muerte en las mujeres**.** Pese a los esfuerzos para su detección temprana, su incidencia a nivel mundial sigue aumentando, por ello la identificación de factores de riesgo para su desarrollo es de vital importancia. En este sentido, diversos contaminantes derivados de la actividad humana, particularmente la industrial, se encuentran presentes en el ambiente y se han asociado con la predisposición al desarrollo de cáncer y de otras enfermedades en el ser humano. Entre dichos contaminantes, se encuentran los denominados Compuestos Disruptores Endócrinos (CDE), los cuales pueden unirse a receptores hormonales e interferir con su homeostasis a nivel sistémico. Un ejemplo de estos es el bisfenol A (BPA), el cual es ampliamente utilizado en la fabricación de policarbonatos y encontrado en contenedores de alimentos, o resinas epóxicas para recubrir latas, de los cuales, por cambios simples de temperatura o alcalinidad (pH), puede migrar hacia el agua o los alimentos. Otro tipo de contaminantes son los ftalatos, utilizados en la elaboración de productos de uso personal como cremas, desodorantes, maquillajes, entre otros. Por otro lado, se conoce que, dentro de la inmunopatogenia del cáncer de mama, ciertas estirpes celulares inmunes; como los linfocitos, T citotóxicos, T reguladores (Treg), células asesinas naturales (NK) y macrófagos asociados a tumores (TAMs), así como sus principales moléculas de señalización, juegan un papel importante en la iniciación, progresión o evasión de esta patología, así como en el pronóstico de sobrevida. Sin embargo, no existen trabajos de la interacción de los bisfenoles o ftalatos con el sistema inmune y la progresión tumoral del cáncer de mama en humanos. Por lo anterior, en este trabajo se propuso evaluar la concentración de bisfenoles y ftalatos en el suero de pacientes con cáncer de mama e identificar su relación con el porcentaje de células inmunitarias infiltradas, así como, indagar su posible correlación con el avance y la agresividad del cáncer de mama. Nuestros resultados muestran que tanto el dibutil y el 2-bishexiletil ftalato, fueron encontrados en concentraciones elevadas en el suero de pacientes con fenotipos agresivos de cáncer de mama y en etapas clínicas avanzadas y metastásicas, no siendo así en el caso del BPA. Adicionalmente se encontró un mayor porcentaje de células T regs con una disminución marcada en los linfocitos T citotóxicos en estos mismos fenotipos. Nuestros resultados también mostraron la presencia de macrófagos infiltrantes, principalmente en el fenotipo triple negativo, el cual en clínica es considerado uno de los más agresivos. De manera preliminar, nuestros datos indican que los niveles séricos de ftalatos, di-n-butil y el 2-bis hexiletil, podrían ser considerados como factores de riesgo y pronóstico en pacientes con cáncer de mama, además de participar en la modulación del microambiente inmune en pacientes con esta enfermedad.

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Alliin modulates expression of inflammatory markers in DIO brain regions.

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Obesity is defined as an increase in the number and size of the adipocytes. Nutrient overload causes the adipocyte to secrete its own cytokines and attract more macrophages, exacerbating the inflammatory process. Neuro-inflammation has been associated with many neurodegenerative diseases and is considered a critical promoter of cognitive impairment. Alliin, the main organosulfide compound in garlic, has demonstrated potential medicinal properties. However, its anti-inflammatory potential in the brain has not yet been studied. The aim was to evaluate the effect of alliin on the expression of inflammatory markers in different brain regions after diet-induced obesity. The mice were divided into 5 groups (n=5): normal weight with standard diet (GC), obese (GO), and three concentrations of alliin (GE1-3) fed with high fat diet (HFD) for 8 weeks to induce obesity, after this period, the GE1-3 groups were supplemented with alliin (1, 5 and 10 mg/kg weight) for 5 weeks. At week 13 the mice were stimulated with LPS, then sacrificed and the brains were dissected. The gene expression of pro-inflammatory cytokines in different brain regions was determined by RT-qPCR and analyzed by the $2^{-\Delta\Delta CT}$ method. The expression of the analyzed cytokines was higher in the hippocampus. In addition, the 1 mg dose of alliin induces a greater response, and higher doses induce a reduction in the expression of IL1-β, IL-6 and TNF-α, except in the case of MCP-1, which with higher doses increased its relative expression. In conclusion, Alliin can act as an immunomodulatory molecule on the expression of cytokines involved in brain inflammation.

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Pharmacological Properties of P2X Receptors in Human Macrophages.

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Several immune cell types respond to extracellular ATP as a danger signal under situations of cell death, stress or hypoxia. In monocytes and macrophages, ATP promotes the activation of the inflammasome and the release of interleukins through the activation of P2X and P2Y receptors. P2X receptor are ion channels involved in physiological and pathophysiological processes of immune system, such as multiple sclerosis, encephalomyelitis and lupus erythematosus. Understanding of the immunomodulator mechanisms of nucleotides in human macrophages could help to develop new therapeutic strategies for immune disease and inflammation. Expression analysis of the P2X1, P2X4 and P2X7 subunits was done using the single cell-PCR technique. The pharmacological characterization of P2X receptor was carried out analyzing the ATP-induced currents in the presence of suramin, a selective antagonist of P2X receptors. Of 24 macrophages obtained, 25% expressed mRNA of the P2X1 subunit, 20.8% P2X1*del*, 12.5% P2X4 and 45.8% P2X7. ATP currents mediated by native P2X receptors showed an EC50 = 3.1 µM and have a desensitization kinetics very similar to that observed for recombinant P2X1 and P2X1*del* receptors expressed in oocytes. In addition, it was observed that suramin has inhibitory and potentiator effects on native P2X receptor and recombinant P2X1 and P2X1*del*. These results suggest that the effects of extracellular ATP in human macrophages could be mediated by the receptors formed by the P2X1 and P2X1*del* subunits, or by heteromeric receptors.

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Effects of gestational exposure to cadmium on reproductive parameters in the male offspring of the Wistar rat.

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Cadmium (Cd) is a toxic, heavy, metal found in everyday use products. It has been of great interest because of the role it plays as an endocrine disruptor. Effects of Cd have been reported in males exposed *in utero*, resulting in a decrease of testosterone (T) blood, which directly effects on puberty onset, male sexual behavior (MSB) and sperm quality, compromising male reproductive success. However, the effects of Cd on preputial separation, (PS) and genital self-grooming have not been explored. Also, there is no data on MSB on progeny exposed to Cd. In this study we analyzed the effects of Cd on the index of puberty, MSB, T concentration, sperm quality and fertility of male rats gestationally exposed to Cd. Pregnant Wistar rats were used and were divided into two groups: a treated group (1 mg/Kg/$CdCl_2$) and a control group (saline) administered intraperitoneal from GD 11 to 20, once the individuals reached adulthood, (96 days) euthanasia was performed by decapitation. The results showed a delay in the PS, in the Cd-treated group, and a lower frequency and duration of the genital self-grooming *Vs* control group, regard to the performance of MSB from the experimental group, a low copulatory efficiency was observed, as well as low sperm quality, directly affecting the fertility of these males, along with lowered testosterone concentration. We concluded that prenatal exposure of Cd induced a negative effect on the reproductive parameters of the male's offspring of Wistar rats compromising male reproductive.

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Role of Glutathione S-transferase expression in women with breast cancer and cardiovascular disease risk factors.

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Obesity, low quality diet and age are common risk factors in breast cancer and cardiovascular disease (CVD). The enzyme Glutathione S-transferase (GST) has been also associated with the development of both diseases. Furthermore, GST enzyme activation could be a protective mechanism for the generation of these diseases, reducing oxidative stress. In a previous study, we identified different GST polymorphisms combinations in women with breast cancer and with overweight and obesity (>25kg/m^2, n=23), some of them were: null expression GSTT1, GSTM1 and GSTP1 (n=7), GSTM1/Val105 (n=6), GSTM1 (n=4), Ile105Val (n=1) and GSTM1/Ile105Val (n=5). Therefore, our current aim is to determine whether GST expression is associated with some diet components and alterations in lipids profile. A cross-sectional study of cases (n=38) and controls (n=41) was performed. Blood samples were obtained for the determination of lipid profile. Dietary data were recorded with a 24-hour recall. The preliminary results indicate that no significant difference in body mass index between groups. Women in both groups presented overweight and obesity 28.14- 50.13 kg/m^2, and had at least one alteration in the lipid profile (100%; n=73). The media of percentage of kcal from fat intake was 26.15-28.33%, (case/control respectively; n=66); but there was a maximum intake of up to 42.7-71.1% of total kcal corresponding to dietary fat per day**.** GST expression is currently being evaluated. In conclusion, the association between dietary patterns, body composition and lipids with GST expression could further provide information about the development of breast cancer and CVD and its impact during treatment.

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Evaluation of polycyclic aromatic hydrocarbons on human mast cells (HMC-1).

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Mast cells promote the release of cytokines that modify the functioning of various immune subpopulations. These are important in cancer because they have a dual effect in some neoplasms, since they act as protumoral or antitumor factors depending on their micolocalization. Recently, the role of hormonal receptors in these cells has been related to increased metastasis. With this project sought to evaluate the effect of two endocrine disruptors (Benzo [a] pyrene (BaP) and Benzo [a] anthracene (BaA)) on genotoxicity and apoptosis in a human mast cell line (HMC-1). The genotoxic effect was evaluated by the comet assay to observe DNA fragmentation at 1 hour of exposure. Cell death was evaluated in HMC-1 cells by the exposure to these PAH´s for 1 hour, 24 hours and 5 days. In the cell death assays, BaA was shown to have an effect on HMC-1 cells, while BaP showed no effect. In the genotoxicity assays with a higher concentration of BaA there is a higher frequency of comets and a higher percentage of flow intensity. The BaP at a higher concentration there is a lower frequency of comets and the percentage of flow intensity was the same in the four concentrations that we used. Therefore, BaP has no cell death effect on HMC-1 mast cells at 1 hour, 24 hours and 5 days at the concentrations used. On the other hand, BaA did not generate apoptosis at 1 hour and 24 hours, but after 5 days of treatment BaA generate apoptosis.

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Análisis comparativo de la expresión diferencial de proteínas de líneas celulares de cáncer de mama expuestas a disruptores endocrinos.

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El cáncer de mama se considera una de las principales neoplasias en población femenina a nivel mundial. Tradicionalmente, este se caracteriza en tres fenotipos distintos. Estudios previos han demostrado que el tratamiento con disruptores endocrinos como el bisfenol A (BPA) y el BPS alteran la proliferación de líneas celulares de cáncer de mama dependientes de hormonas, a través de su unión al receptor de estrógenos y la activación de vías mitogénicas. Sin embargo, los efectos sobre el patrón proteómico y el interactoma de estos compuestos, líneas celulares de cáncer de mama humanas con fenotipo triple negativo, no han sido explorados. El objetivo del presente trabajo, fue comparar el perfil proteómico de líneas celulares de cáncer de mama positivas y negativas al receptor de estrógenos posterior al tratamiento con bisfenol A (BPA) y bisfenol S (BPS). Las líneas celulares MCF-7 y HCC1937 fueron expuestas al tratamiento con BPA y BPS por 48 horas, posterior a este periodo las células fueron tripsinizadas y lavadas con PBS para su posterior lisis en buffer de muestra. Las células fueron sonicadas 1 min. 4 veces y las proteínas fueron precipitadas en acetona y metanol cloroformo. Para la separación por punto isoeléctrico se cargaron 100 microgramos de muestra en tiras de 7 cm de pH lineal de 7-10. La electroforesis para la separación por peso molecular se corrió en geles de poliacrilamida con un gradiente de 4-20%, los geles fueron teñidos con azul de Coomassie y analizados en el programa PDQuest. Los resultados indican que el tratamiento con los disruptores endocrinos mostró un perfil proteómico diferente con respecto al control y al ligando endógeno, estradiol, el cual también indujo cambios de expresión de proteínas con respecto al control. Los resultados muestran que grupos de proteínas fueron modulados positivamente y negativamente dependiendo del tratamiento. Para la línea celular HCC se modificó la expresión de 34 proteínas, cuando las células fueron expuestas a BPA y 50 proteínas cuando las células se expusieron a BPS; la mayoría de estas proteínas participan en vías de señalización, tienen actividad catalítica o son proteínas que interactúan con proteínas nucleares. Algunos ejemplos interesantes de las proteínas que disminuyen su expresión debido a la presencia de los disruptores endocrinos son: Deacitalasa sirtuin-2 dependiente de NAD, Inositol-3-fosfato sintasa 1, receptor de prolactina, Fosfatasa 1A.

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Evaluación del efecto de un plaguicida anticolinérgico en la expresión de IL- 1β e IL-6 en células mononucleares de O*reochromis niloticus.*

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Los plaguicidas organofosforados como diazinón, son sustancias químicas utilizadas para el control de plagas y vectores de transmisión de enfermedades. Sin embargo, se ha asociado la exposición a estas sustancias como un factor de susceptibilidad a infecciones y de riesgos para desarrollar enfermedades inflamatorias. No obstante, el mecanismo de inmunotoxicidad no está claro. Por lo que, el objetivo del presente estudio es evaluar el efecto de diazinón sobre la expresión de citocinas proinflamatorias y antiinflamatorias, utilizando como modelo de estudio la tilapia nilótica (*O. niloticus*). Para lo cual, se utilizaran peces machos adultos de la especie tilapia nilótica (*O. niloticus*) se expondrán a concentraciones subletales (0.97, 1.95 y 3.91 mg/mL) de diazinón durante 6 y 24 horas. A partir de células mononucleares de bazo (CMB) aisladas, se evaluará por RT-PCR en tiempo real, la expresión de interlucina 1(IL-1β) e interlucina 6 (IL-6). La información generada podrá evidenciar el efecto que tiene diazinón en la alteración de expresión de las citocinas, moléculas fundamentales para la señalización del sistema inmune. Las citocinas IL-1β e IL-6 actúan como potentes citocinas pro y antiinflamatorias, por lo que, la posible alteración en la expresión de estas moléculas por la exposición a diazinón provocará la desregulación de la respuesta del sistema inmunológico de los organismos expuestos.

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Análisis proteómico en pacientes con depresión: cuantificación relativa asistida con marcadores isobáricos.

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La depresión es un trastorno del estado del ánimo que se caracteriza por enlentecimiento psicomotor y en los casos más severos ideación suicida, acompañado de otros síntomas cognitivos, de comportamiento o neurovegetativos que afectan significativamente la habilidad del individuo para llevar una vida normal. Dada su alta prevalencia, recurrencia y enormes costos personales y sociales la depresión es la principal causa mundial de discapacidad. La etiología de la depresión es muy heterogénea así como su presentación, curso y respuesta al tratamiento. Varios mecanismos biológicos están relacionados con el establecimiento de la depresión, como la deficiencia de monoaminas, alteraciones neurotróficas, disfunción del eje hipotálamo-hipófisis-arenal y las alteraciones inflamatorias, pero actualmente se carece de una comprensión profunda y detallada de su fisiopatología. Actualmente, el diagnóstico de depresión se basa en la historia clínica, exploración física y la aplicación de escalas clinimétricas; pero, diversos autores coinciden en que el diagnóstico y el seguimiento clínico de los pacientes basado en el resultado de las escalas clinimétricas debe de ser reforzado, siendo los parámetros moleculares una opción potencial. El análisis de plasma, suero y leucocitos ha demostrado ser útil para valorar la función cerebral. En particular, las células mononucleares de sangre periférica (*PBMCs, peripheral blood mononuclear cells*) tienen un gran potencial a nivel de diagnóstico, ya que se han observado cambios transcriptómicos paralelos entre estas y el cerebro a través del analisis de microarreglos. Por lo anterior, el objetivo del presente trabajo fue identificar las alteraciones moleculares que nos permitan entender mejor la fisiopatología de la depresión y que en un futuro puedan servir como nuevos biomarcadores para este padecimiento, esto a través de un análisis proteómico de PBMCs de pacientes con depresión para una cuantificación relativa asistida con marcadores isobáricos.Se realizó un análisis proteómico comparativo entre un *pool* de proteínas de pacientes deprimidos (n=12, sin consumo de antidepresivos) y un *pool* de proteínas de voluntarios sanos (n=12) por medio de una cuantificación relativa asistida con marcadores isobáricos. La captación (pacientes y voluntarios sanos) se realizó en el Instituto Nacional de Psiquiatría Ramón de la Fuente Muñiz (protocolo NC150048SECITI). Todos los participantes firmaron el consentimiento informado y los formatos requeridos por el comité de ética institucional. Se obtuvo una muestra de sangre de 10 mL de los participantes, de la cual se llevó a cabo la separación de *PBMCs* por gradiente de densidad Histopaque, Sigma). Las *PBMCs* fueron lisadas para la extracción de proteínas con una solución de

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Urea 8M. Se utilizaron 100 µg de proteína de cada *pool*, las cuales fueron reducidas y alquiladas, y posteriormente digeridas con tripsina (1:30) toda la noche a 37°C. Los péptidos obtenidos fueron marcados con los reactivos TMT 6-plex (marcadores isobáricos) como lo indica el fabricante (Thermo Fisher Scientific). Se inyectaron 5 µL de muestra en una trampa nanoviper C18 trap a 3 µL/min, y luego a una columna EASY spray C-18 RSLC (2 µm, 75 µm x 25cm) a 300 nL/min. Las muestras se analizaron por espectrometría de masas en un equipo Orbitrap Fusion Tribid acoplado a "EASY Spray" (Thermo-Fisher Scientific) y a un cromatógrafo UltiMate 3000 (Dionex). Los datos fueron procesados con los programas ProteomeDiscoverer 2.1 (ThermoFisher), Mascot server (Matrix Scienc e), AMANDA(Dorfer et al., 2014) y SQUEST HT(Eng, McCormack, & Yates, 1994) contra la base de datos UniProt (humano). El interactoma se realizó con el programa *String,* en donde se describieron las abundancias relativas de las proteínas en pacientes con depresión respecto a voluntarios sanos (expresadas como $\log_2$). Se identificaron 253 proteínas por espectrometría de masas asistida con marcadores isobáricos, las cuales se procesaron con el programa *String* para construir un interactoma en donde se indica la abundancia relativa (*fold change* $\log_2$) de cada proteína en PBMCs de pacientes con depresión respecto a voluntarios sanos.

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Chronic infection with *Mycobacterium lepraemurium* induces alterations in the hippocampus associated with memory loss.

Becerril E.*[1], Ponce-Regalado M.D.[2], Pérez-Sánchez G.[1], Salazar-Juárez A.[3], Álvarez-Sánchez E.[4], Peña Aguirre R.[1], Hernández-Pando R.[6], Pavón L.[1], Arce Paredes P. [5], Rojas-Espinosa O.*[5]

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Murine leprosy is caused by Mycobacterium lepraemurium (MLM), which is used as a model of systemic-infection that resembles a chronic infection similar to human leprosy. Because this model does not directly involve the nervous system, we investigated a possible effect on working memory during chronic infection induced by MLM in Balb/c mice. We evaluated the dorsal region of the hippocampus and the peripheral levels of cytokines at 40, 80 and 120 days post-infection. To evaluate working memory we used the T-maze, meanwhile for morphological analysis we performed a morphometry analysis in the hippocampus (CA1, CA2, CA3, and Dentate Gyrus). In addition, neurochemical analysis was performed by HPLC. Our result show that at 40 days post-infection there was an increase in the bacillary load in the liver and spleen, concomitant to an increase IL-4 levels, working memory deterioration, and changes in hippocampal morphology including degeneration in the four subregions analysed. In the same time point of infection we found a decrease in neurotransmitters levels. Because MLM does not directly infect the nervous system, these findings provide a possible interaction on the link between the immune system activation and the central nervous system response.

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Evidencia de alteraciones neuroendocrinoinmunológicas causadas por la infección de *B. abortus* 2308 y los antibióticos empleados en un modelo murino.

Maldonado García Romero J.L., Pérez Sanchez G., Becerril Villanueva E., Alvarez Herrera S., Gamboa Sanchez C., Medina Rivero E., Hernández Colin A.K., López Santiago R., Moreno Lafont M., Pavon L.

La brucelosis genera 500 mil nuevos casos por año en el mundo, 20% de ellos se vuelven crónicos; los pacientes infectados presentan fiebre ondulante, dolor articular, cefaleas y debilidad. A pesar de la gravedad del cuadro los pacientes no reciben un manejo integral, lo cual limita su capacidad de recuperación. Un enfoque neuroendocrinoinmunologico permitirá evidenciar la importancia clínica de un manejo integral. Objetivos Analizar en animales sanos, sanos con antibiótico, infectados más antibiótico y animales infectados a 14 y 21 días, las interacciones neuro-inmuno-endócrinas en un modelo múrido de infección por *B. abortus*-2308. Método Ratones Balb/c de 8 semanas y 20 gr, fueron asignados aleatoriamente a los grupos de estudio, todos los grupos fueron almacenados en cajas de contención y manejados en campanas de bioseguridad. Los grupos infectados recibieron un millon de *B. abortus* 2308 por via intraperitoneal. Se generaron grupos que recibieron los siguientes tratamientos: control, rifampicina+doxiciclina (RIF+DOX), Infeccion por 10 días y tratamiento farmacologico (I-10+rif+dox), Infeccion por 14 días (I-14) e Infeccion por 21 días (I-21). Y se aplicaron pruebas conductuales modificadas de nado-forzado, suspensión-en-cola, campo-abierto, equilibrio y resistencia-muscular; los ratones recibieron eutanasia y se obtuvo suero para determinación de citocinas por Cytometric-Bead-Array (CBA) y corticosterona por ELISA; en corteza-frontal, corteza-temporoparietal, hipocampo y cerebelo se determinó la concentración de serotonina (5-HT), dopamina (D), epinefrina (E) y norepinefrina (NE) por HPLC. Todos los datos obtenidos fueron analizados estadísticamente para determinar significancia y correlación estadística. Resultados Se reportan sólo los cambios estadísticamente significativos (P< 0.05). La administración de RIF+DOX vs. Control indujo: aumento de IL-12, disminución de corticoesterona, aumento de NE en cerebelo y D en corteza-temporoparietal. Disminución de 5-HT en cerebelo e hipocampo. Se detectó una correlación entre la IL-12 y NE en cerebelo (r=0.74 P< 0.07). El grupo I-10+rif+dox vs RIF+DOX presentó una elevación de TNF-alfa, IL-6, IL-12 e IFN-gama, aumentó de 5-HT y D en cerebelo, disminución de NE en hipocampo, aumento de E en la corteza-temporoparietal. Se detectó una correlación entre los niveles de IL-12 y 5-HT en cerebelo (r=0.7; P< 0.005). El grupo I-14 vs control mostró: incremento de TNF-alfa, IL-6, IL-12 e IFN-gama, aumento de D y 5-HT en cerebelo, disminución de D, NE y 5-HT en hipocampo, disminución de D, E y 5-HT en corteza-frontal, aumento de E y disminución de 5-HT

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en la corteza-temporoparietal. Se detectaron múltiples correlaciones significativas entre IL-6 e IFN-gama vs neurotransmisores y pruebas conductuales. El grupo I-21 vs control mostró: elevación de IL-6 e IL-12, disminución de la concentración de E en la corteza-frontal, disminución en E, NE y 5-HT en corteza-temporoparietal, Se detectaron correlaciones entre la IL-12 y E en la corteza-frontal (r=0.5; P< 0.05) y corteza-temporoparietal (r=-0.064; P< 0.015), así como alteraciones en todas las ruelas conductuales. Discusión El tratamiento RIF+DOX en controles sanos e infectados alteró los parámetros neuro-inmunológicos sin cambios conductuales. El grupo I-14 tienen una respuesta inflamatoria exacerbada con actividad compensatoria disminuida en los ejes HPA y arco-antiinflamatorio-vagal, lo que induce alteraciones conductuales evidentes. El grupo I-21 presenta una autolimitación de la infección que disminuye la respuesta inflamatoria, lo que favorece una eficiente actividad compensatoria de los ejes HPA y arco-antiinflamatorio-vagal, lo que disminuye la severidad de las alteraciones conductuales. Este estudio evidencia la importancia de abordajes multidiciplinarios en infecciones crónicas, para mejorar la condición de vida del paciente y su familia.

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El 4(5)-metilimidazol impacta en la plasticidad sináptica de la rata.

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El 4(5)-metilimidazol (4(5)-MEI), utiliza en la fabricación de productos comerciales. Se reporta efecto neurotóxico en animales de experimentación, con alteraciones de la estructura celular del sistema nervioso central. Falta evidencia que demuestre alteraciones en la conectividad sináptica. El objetivo fue determinar el efecto neurotóxico del 4(5)-MEI, a nivel estructural de la conectividad sináptica del hipocampo dorsal de la rata. Dieciocho ratas *Sprague Dawley*, divididas en 3 grupos: control y 2 experimentales con ingesta oral de 4(5)-MEI (625 y 1250 ppm), durante 24 semanas. Se realizó la prueba de laberinto acuático de Morris. Se sacrificaron los animales, se determinan los niveles de malondialdehido (MDA) en plasma y el hipocampo dorsal para la cuantificación de espinas. La determinación de MDA, no mostró diferencias significativas entre los grupos. Los grupos tratados con 4(5)-MEI, presentaron un deterioro en la memoria espacial con respecto al grupo control y una disminución en la densidad de espinas dendríticas del hipocampo dorsal. El 4(5)-MEI genera disminución de la capacidad cognitiva dependiente del hipocampo, esto se puede correlacionar con la disminución en la densidad de espinas dendríticas.

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Alteraciones del sistema colinérgico inducidas por la exposición *in vitro* a diazoxón en cultivos primarios de leucocitos de Tilapia nilótica (*O. niloticus*).

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Los plaguicidas organofosforados como diazinón causan una disrupción de la comunicación neuroinmune, debido a los efectos neuro e inmunotóxicos, afectando la respuesta innata y adaptativa del sistema inmunológico de los organismos expuestos. Debido a que diazinón tiene como molécula blanco la enzima acetilcolinesterasa (AChE), la existencia de un sistema colinérgico no neuronal en leucocitos, los hace susceptibles a la perturbación por diazinón. Por lo que, el objetivo de este trabajo fue evaluar AChE, acetilcolina (ACh) y receptores de ACh nicotínicos (nAChR) y muscarínicos (mAChR) en linfocitos de tilapia nilótica (*O. niloticus*) expuestos *in vitro* a diazoxón, un metabolito de diazinón. Para lo cual, células mononucleares de bazo de *O. niloticus*, fueron expuestas *in vitro* a 1 nM, 1 y 10 uM de diazinón por 24 h. Posteriormente, se evaluó la actividad enzimática de AChE, se cuantificó ACh mediante espectrometría de masas y se evaluó la expresión de mAChR y nAChR por RT-qPCR. Los resultados indican que los niveles de AChE se inhiben significativamente a 1 y 10 uM de diazinón, la expresión relativa de mAChR y nAChR disminuye significativamente comparada con el control. Sin embargo, los niveles de ACh no muestran diferencia con respecto al control. Lo anterior evidencia la alteración del sistema colinérgico de linfocitos, lo que está directamente relacionado con las propiedades inmunotóxicas de diazinón.

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Cytokines and hormones involved in the host response to sheep hemonchosis.

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The study of the immune response against the nematode *Haemonchus contortus* is mandatory to understand the phenomena of resistance against hemoncosis and for the development of new strategies for its control in sheep. Resistance to hemoncosis is related to host genetics, which explains the existence of ovine genotypes with a relatively high resistance. The phenotypic expression of resistance can be attributed to nonspecific cells such as mast cells, globular leukocytes and eosinophils in the abomasal mucosa. The acquired response depends on other cells such as CD4 lymphocytes and gamma-delta lymphocytes. Frequently the protective response against hemoncosis has been related to a Th_2 cytokine profile (IL-4, IL-5 and IL-6), although there is evidence of Th_1 cytokines such as IFNγ and IL-2 that have also consistently been related to the response against *H. contortus*. In the induced protection against *H. contortus* achieved by the administration of a *Taenia hydatigena*-derived immunomodulator, the reduction of the parasitic load in lambs was related to the overexpression of Th_2 cytokines and the underexpression of Th_1 cytokines in the abomasal mucosa. *H. contortus* larvae respond to the *in vitro* stimulation with prolactin and progesterone. Prolactin promotes larval growth while progesterone inhibits larval molting. It was possible to identify the presence of receptors for both hormones mainly in the digestive tract of the larvae. The *in vitro* hormonal effects and receptors may be related to hypobiosis and larval reactivation phenomena in pregnant sheep. The effects of both hormones on the immune response to hemoncosis are unknown. Financed by PAPIIT-UNAM IN-218018.

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The natural resistance of mice to amoebic liver infection is due to a low chemotactic activity of complement which results in poor inflammatory response.

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Entamoeba histolytica is the parasite responsible for amoebiasis in humans, a disease causing ~one hundred thousand death year worldwide. Similarly, to the experimental model of amebic liver abscess (ALA) in hamsters (susceptible), the analysis of the natural resistance mechanisms of some rodents to ALA may result in new potential therapeutic alternatives. In this work, the natural resistance of the BALB/c mice to ALA was explored by performing: 1) in *vivo* chemotaxis analysis with a novelty chamber; 2) *in vitro* amoebic survival in fresh and decomplemented serum; 3) histological temporal course analysis of ALA development in mice with different treatments (hypocomplementemic, hyperimmune and treated with iNOS and NADPH oxidase inhibitors) intraportally injected with virulent *E. histolytica* ($1x10^6$/100 gr body weight); and 4) mouse liver amoebic infection by both *in situ* implantation of ALA from hamsters and inoculation of parasites into the peritoneal cavity. The results show that parasite clearance from mouse liver is related to a low chemotactic activity of complement, poor inflammatory response and parasite inability to cause tissue damage.

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Cuantificación de la expresión génica (mrna) de la proteína s-100a4 en células mononucleares de sangre periférica (pbmc) de pacientes con trastorno depresivo mayor (tdm).

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La depresión es una enfermedad mental frecuente en todo el mundo, y se calcula que afecta a más de 300 millones de personas. Puede convertirse en un problema de salud serio, y alterar las actividades laborales, escolares y familiares. El TDM es un trastorno mental frecuente, que se caracteriza por la presencia de tristeza, pérdida de interés o placer, sentimientos de culpa o falta de autoestima, trastornos del sueño o del apetito, sensación de cansancio y falta de concentración. En el peor de los casos puede llevar al suicidio. Cada año se suicidan cerca de 800 000 personas (Organización Mundial de la Salud, 2018). La fisiopatología de la depresión integra varios sistemas como son: el sistema nervioso, endocrino e inmune. El sistema nervioso participa en la regulación de neurotransmisores serotonina y noradrenalina etc. Así como, factores transcripción (BDNF) relacionados con plasticidad neuronal y neurogénesis. El sistema endocrino también se ve involucrado en el TDM, el cual se ve afectado por una desregulación hormonal, por estrés físico, social y oxidativo, generando la activación del eje HPA (Hipotálamo-Pituitario-Adrenal), el cual provoca la activación de la hormona liberadora de corticotropina (CRH), la cual genera niveles altos en cortisol (hipercortisolemia) debido a la activación de las glándulas suprarrenales. El sistema inmune también se ve afectado por trastorno depresivo mayor, ya que se activa el sistema inmune innato generando la producción de citosinas proinflamatorias, proteínas de fase aguda (S100), proteínas de choque térmico, entre otras. Se ha reportado que estos DAMPs provocan una inflamación estéril en los pacientes con desordenes psiquiátricos, debido a la activación del factor de transcripción NF-KB. Las proteínas S100 son una familia de DAMPs dependientes de calcio Ca2+ y Zn2+, existen alrededor de 25 proteínas S100, son secretadas por células del sistema inmune y células endoteliales, codificadas en la región 1q21 del cromosoma 1, pueden realizar funciones intra y extracelulares. La proteína S100A4 ha sido descrita en varios tipos de cáncer, relacionada con procesos de metástasis, así como en enfermedades inflamatorias crónicas como artritis reumatoide, se ha reportado a S100A4 como mediador de la respuesta inmune innata, S100A4 induce la producción de citocinas proinflamatorias como TNF-α, IL-1β e IL-6 en células mononucleares. Esta proteína actúa a través de receptores tipo Toll (TLR4) y RAGE para activar a MyD88, al factor de transcripción NF-κB y a las tirosin quinasas ERK1 / 2 y p38, esta transducción de señales conlleva a una respuesta inflamatoria. Recientemente el laboratorio de psicoinmunología del Instituto Nacional de Psiquiatría Ramón de la Fuente Muñiz reportó altos niveles de proteínas de fase aguda S100A4 y S100A9 en pacientes con trastorno depresivo mayor (TDM). El objetivo de este estudio es cuantificar la expresión génica (mRNA) de S100A4 en células mononucleares de sangre periférica (PBMC) de pacientes con TDM, y a su vez determinar si existe correlación entre la severidad de la depresión y la expresión génica de S100A4. Este estudio ampliará los conocimientos sobre la fisiopatología de la depresión y nos ayudará entender más sobre la participación de los sistemas neuroinmunoendocrinológicos que se integran en esordenes psiquiátricos como el trastorno depresivo mayor.

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Caracterización de la expresión de las proteínas s100a8 y s100a9 en células mononucleares.

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Se le conoce como Trastorno Depresivo Mayor (TDM) a una serie de síntomas caracterizados por una prolongada tristeza y melancolía, que duran más de dos semanas y no desaparecen aun cuando sus causas ya no estén (Belmakeret al., 2008). Hasta el momento se ha descrito la participación de tres sistemas que influyen de manera muy característica en el inicio y severidad de este trastorno: el sistema nervioso central, cuya irregularidad consiste en la alteración de la neurotransmisión, lo que ha sentado la base para desarrollar fármacos antidepresivos. El sistema endócrino, a través del eje hipotálamo-pituitaria-adrenal (HPA), se sobre activa provocando el aumento de la hormona cortisol a nivel sistémico, donde células de sangre y tejidos periféricos reprimen la activación de su receptor a manera de regulación, provocando la activación de vías de señalización relacionadas con la inflamación, que fisiológicamente se encuentran reguladas por esta hormona. El último sistema, el sistema inmune, ha cobrado importancia en los últimos años, ya que se ha reportado el incremento de citocinas pro inflamatorias y DAMPs en áreas límbicas del cerebro y sangre periférica, lo que sensibiliza al sistema inmune ante un evento estresor o una infección, los cuales exacerban la respuesta inflamatoria y el comportamiento. Con base en esto, el aumento de los niveles fisiológicos de DAMPs y citocinas pro inflamatorias se relaciona con la severidad de trastornos psiquiátricos como la depresión. Las proteínas S100 pertenecen a una familia de DAMPs altamente conservada en vertebrados, codificada principalmente en la región 1q21 del cromosoma 1 y que presentan ortología con la rata. Existen cuatro grupos principales de proteínas S100 en mamíferos, de los cuales S100A8, S100A9 y S100A12 son los únicos DAMPs asociados con respuesta inflamatoria. S100a8 y s100a9 son proteínas que se encuentran principalmente en neutrófilos y macrófagos y funcionan como sensores de Ca^{++}. Estas proteínas se oligomerizan en el espacio intracelular, donde participan en el rearreglo del citoesqueleto para su secreción al espacio extracelular, donde actúan como DAMPs activando la vía de NFκB mediante su unión a TLR4 y promoviendo la expresión de citocinas proinflamatorias favoreciendo el ambiente pro inflamatorio a nivel periférico y de Sistema Nervioso. En enfermedades inflamatorias, metabólicas, así como Alzheimer, se da una expresión significativa de S100A8 y S100A9. Recientemente el laboratorio de psicoinmunología del Instituto Nacional de Psiquiatría reportó altos niveles de ambas proteínas en pacientes con trastorno depresivo mayor; Gong y colaboradores describieron su participación en la neuroinflamación a través de la activación de la microglía en un modelo murino de depresión inducida por restricción crónica. El objetivo de este trabajo es caracterizar la expresión de S100A8 y S100A9 en células mononucleares de sangre periférica y en cerebro de un modelo murino, lo cual ampliará el conocimiento acerca de la participación del complejo calprotectina (S100A8/A9) en el trastorno depresivo mayor.

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Neuroimmunoendocrinology: a historical perspective.

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"The greatest mistake that physicians make is that they attempt to cure the body without attempting to cure the mind, yet the mind and the body are one and should not be treated separately!"(Plato 370 BC).

Although there is no precise moment or unique event referring to the birth of neuroimmunoendocrinology as a defined area, we can argue that it shares historicity with the development of other medical-biological disciplines. It first started with empirical observations and the postulation of suppositions that did not prevail upon the existing axioms. Despite the rejection, these seeming defeats inspired visionary masters of research who managed to systematize the emerging knowledge and were able to make contributions with real scientific foundations. In consequence, new concepts and ideas arose in areas as physiology, anatomy, endocrinology, and early immunology and gave rise to a budding approach on the integration between the nervous, immune, and endocrine systems. Then, neuroimmunoendocrinology appeared as a discipline integrating a super system of multidirectional interactions that allowed for responding against internal and external threats through soluble mediators. The latter include cytokines, hormones, and neurotransmitters, which take part in different physiological mechanisms of the homeostasis in the organism. Neuroimmunoendocrinology is no longer an area of scientific skepticism and has cemented its place as a biomedical discipline worldwide in the past 70 years.

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Expresión de nAChR α7 en en peces guppy (*Poecilia reticulata*) expuestos a los plaguicidas temefos y spinosad.

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Los receptores nicotínicos de acetilcolina (nAChR) forman parte del sistema colinérgico neuronal, los cuales se expresan en la membrana plasmática, a las que se une específicamente el neurotransmisor acetilcolina (ACh). Estas moléculas llevan a cabo la señalización celular mediante el paso de iones. Los nAChR del subtipo α7, se encuentran ampliamente distribuidos en el sistema nervioso central y periférico, su nivel de expresión en la célula puede verse afectada por sustancias a las que se encuentran expuestos los organismos, como los plaguicidas, provocando alteraciones a nivel neurológico.

El objetivo de este estudio es evaluar la expresión de nAChR α7 de peces guppy expuestos a temefos y spinosad (dos de los plaguicidas utilizados para el control de vectores), a las concentraciones recomendadas por la SSN (10 mg/L y 0.7 mg/L, respectivamente), durante 7, 14 y 21 días. En cada pez se evaluará a través de qPCR la expresión de nACh α7. Los resultados nos permitirán comparar el grado de neurotoxicidad de los plaguicidas en organismos “no blanco”.

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In silico prediction of the CHH / ITP neuropeptide in R. sanguineus, as a study model.

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R. sanguineus is one of the most important vectors of worldwide distribution, causing important diseases that affect the nervous system in humans by zoonoses. (1). The singanglion of ticks has not been fully explored (2), in this study we are interested in knowing if there is the presence of the CHH peptide which are documented and have a central role in the homeostatic regulation of energy metabolism, molting, reproduction and stress responses (3), so in future experimental studies it could be a therapeutic target. The objective is identify the CHH / ITP neuropeptide with *in silico* analysis for future experimental applications in the *R. sanguineus* tick. The Hypothesis is the neuropeptide CHH / ITP is conserved in the *R. sanguineus* tick.

The study carried out by Andrew , 2008, showing transcripts encoding putative ipodoid neuropeptide precursors was taken as a reference, a phylogenetic tree was performed, a BLAST of the reference sequence was performed, online programs were used to identify the signal peptide, possible glycosylation sites as well as determine the possible secondary structure of the peptide.. Both insects such as crustaceans and ticks belong to the same edge so that this possibility is increased if this peptide is conserved, no glycosylated sites were analyzed and the prediction of its secondary structure showed an alpha helix with a turn and 1 beta sheet.

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Systemic immune modulation after inhalation exposure to three ubiquitous phthalates.

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Esters of phthalic acid, also known as phthalates are used in the plastics and resins manufacture, as well as in the cosmetic and pharmaceutical industries as solvents and fixatives (Meeker et al.). Phthalates are classified as endocrine disrupting compounds (Diamanti-Kandarakis et al.). While oral consumption of contaminated food and dermal absorption from personal care products are considered the main exposure routes, phthalates are ubiquitously found in environmental matrices such as in the soil, water bodies and atmosphere (Quintana-Belmares et al.), where Bis-(2-ethylhexyl) phthalate (DEHP), Di-isobutyl phthalate (DiBP), and Di-n-Butyl phthalate (DBP) are the most abundant.

Adult male Wistar rats were exposed to a mix of DEHP, DiBP and DBP at doses ranging from 15 to 45 mg/kg each, through a nose-only inhalation device (SCIREQ Aeroneb). 7 days after exposure, splenocytes were cultured for assessment of lymphocytic proliferation and cytokine production. Exposure to 30 and 45 mg/kg reduced both proliferative index and percentage of divided cells. All doses resulted in a decreased IFN-γ production.

Our results show that inhalation exposure to ubiquitous phthalates affects lymphocytic function in a systemic level.

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