21st Annual Puerto Rico Neuroscience Conference

Puerto Rico Chapter
Society for Neuroscience

9 Years / 12

Saturday, December 1, 2012

1st December 2012
Center for Puerto Rico, Sila Calderon Foundation
8:00AM to 5:00PM

President's message:

The Puerto Rico Chapter of the Society of Neuroscience unites neuroscientists across the whole island. The Chapter is represented by Río Piedras and Medical Sciences Campuses of the University of Puerto Rico, Institute of Neurobiology, Ponce School of Medicine and Universidad Central del Caribe. The diverse research topics conducted by neuroscientists, from neuronal development to neuronal death and aging, exemplifies the development and progress of neuroscience research in Puerto Rico.

Neuroscience has become the science of the 21st Century. Neuroscience research has transcended the confinements of the bench to be applied in all aspects of our society. From the used of the information gathered by brain scanning or imaging to determine the activity related to a stimulus in a court of law to the understanding of decision making for the outcome of the stock market. The understanding of our brain function goes beyond the generation of new knowledge to the application of that acquired knowledge. Following this trend, this year our chapter has integrated neuroscientists from different institutions, including Carlos Albizu University and Veterans Affairs Caribbean Healthcare System.

This year is the 21st Annual PR Neuroscience Conference. Thus, I hope you join us to celebrate and enjoy 21st century neuroscience research at our 21st annual conference…

Irving E. Vega, PhD
President
PR Neuroscience Chapter
Associate Professor
Department of Biology
UPR-Río Piedras Campus

Conference's Venue
&
Logistics

Center for Puerto Rico, Sila Calderón Foundation

Calle González #1012
Urb. Santa Rita
San Juan, PR. 00925

Parking

Park your vehicle at the “Architecture/Art” lot in the UPR-Río Piedras Campus
(first Gandara Ave. entrance, coming from Barbosa Ave.)

A bus will be available to transport conference’s participants to the meeting venue, starting at 7:30AM

visit prneuroscience.uprrp.edu for more information

Conference's Agenda

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

PROGRAM

8:00AM – 8:45AM	Registration/Breakfast	Main Lobby
8:45AM – 9:00AM	Welcome	Amphitheater
9:00AM – 9:50AM	Dr. Kay M. Tye	Amphitheater

Optogenetic dissection of neural circuits in health and disease

10:00AM – 10:50AM	Dr. Yasmin Hurd	Amphitheater

Neurobiology of Addiction disorders and Psychiatric illnesses

11:00AM – 11:20AM	Jose Rodriguez-Romaguera	Amphitheater

Deep brain stimulation of the ventral striatum enhances extinction of conditioned fear

11:30AM – 11:50AM	William Castro	Amphitheater

Neuroprotection by 4R-Cembranoid evaluated 24H and 7days after ischemic stroke

12:00PM – 1:00PM	Lunch	Main Lobby
1:00PM – 1:50PM	Dr. Alexei Verkratsky	Amphitheater

Physiology and Pathophysiology of Neuroglia

2:00PM – 2:50PM	Dr. José R. Lemos	Amphitheater

Endogenous feedbacks regulate secretion of neuropeptides from Nerve terminals

3:00PM – 5:00PM	Poster Presentation / Refreshements	Main Lobby

Poster's Abstracts

Clinical Neuroscience

CN1 – Alonso, H.
CN2 – Camacho-Mercado CL
CN3 – Faris-De Jesús, V.M.
CN4 – Vergara-Mojica, A.

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A B S T R A C T

Title:

The utility of SPECT-CT and PET-CT in the diagnosis of Traumatic Brain Injury at the VA Caribbean Healthcare System (VACHS): Retrospective Descriptive Study

Author(s):

Irma Molina, MD; **Hector Alonso**; Carlos Quijano; Keryl Motta, MD; Amilcar Matos; Sharyl Valdes; Frances Marrero

Institutional Affiliation:

VA Caribbean Healthcare System

Research Topic:

Clinical Neuroscience

Abstract:
OBJECTIVES. This is a cross sectional, retrospective, descriptive study to obtain preliminary data to characterize the brain injuries of OIF/OEF veterans, group them based on these characteristics and describe the correlation of the neuron-imaging findings and symptomatology (physical, cognitive, and psychological) of veterans who are living with Traumatic Brain Injury (TBI). This data will support evaluation of specific targeted rehabilitation strategies in a future study. METHODOLOGY. Socio-demographic and medical history data were gathered for up to 500 patients that underwent a SPECT CT and/or PET CT due to Brain Trauma. Standard neuro-imaging procedures were used to obtain neurological data related to brain impairments. Quantitative data, including the mechanism of injury and various standardized motor and cognitive tests, was collected to describe the sample and contribute important information related to physical functional ability. FINDINGS. Preliminary results showed the most common physical symptom present in these TBI patients were headaches, most common cognitive symptom was forgetfulness, and most common psychological symptom was irritability. Most common location for TBI lesions using SPECT/CT was the frontal lobe. Socio-demographic trends include a high percentage of veterans receiving some level of college education, as well as over three fourths of the population being married.

Notes:
I would like to be considered for oral short presentation.

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A B S T R A C T

Title:

Profile of Puerto Ricans from metropolitan area diagnosed with Alzheimer Disease (AD)

Author(s):

C. L. Camacho Mercado[1], C. Padovani Lebrón[2], R. Figueroa, H.A. Acosta-Vélez[2] , Arnold, S.E.[3], I.E. Vega Vega[1]

Institutional Affiliation:

[1]Department of Biology, University of Puerto Rico, Río Piedras Campus, San Juan, P.R. [2]Caribbean Center for the Study of Memory and Cognition, Judith Irizarry de Díaz Memory Clinic, San Juan, P.R. [3]Penn Memory Center, Department of Neurology, University of Pennsylvania, Philadelphia

Research Topic:

Clinical Neuroscience

Abstract:
Alzheimer's disease (AD) in Puerto Rico has a higher mortality rate compared with that observed in the United States. From 1999 to 2004, the occurrence of AD mortality in Puerto Rico is higher than in the United States. In Puerto Rico the AD mortality rate has increased 52.8%, while in the United States there was a 31.4% increase. The aim of this study is to elaborate a profile of the Puerto Rico AD patients that may help in the diagnostic of the disease and to have a better understanding of its progression. Different parameters will be considered, including demography, socioeconomics and co-morbidities. The parameters considered were metabolic profile, co-morbidities, demographic information and psychiatric profile. The preliminary data shows that in 432 of records revised from AD patients, the age of onset of AD is 78 years old. The disease is more prevalent in women (63%). The majority of the patients have an education level of high school or higher. Although many diseases are correlated with the AD, we detected that, approximately the 69% of the patients with AD, suffer of high blood pressure and 58% suffer of lipidemia. The results obtained will contribute to improve the diagnosis of AD and to offer a better care, leading toward prevention.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Model of Traumatic Brain Injury Using Imaging, Physiological & Psychosocial Parameters: Descriptive Study

Author(s):

Irma L. Molina, MD, **Verónica M. Faris-DeJesus**, Kathia Y. Jusino, Ivan Velez-Miro, MD; Keryl Motta, MD; Isabel Borras, MD; Carlos Montalvan, MD; Jannette Figueroa, MD; Gerty Jones, MD; Magaly Freytes, Ph. D and Jose Mendez Villarubia, Ph. D

Institutional Affiliation:

VACHS

Research Topic:

Clinical Neuroscience

Abstract:
The goal of this study was to characterize the brain injuries of veterans, categorize them based on these characteristics and describe the psychosocial experiences of veterans with TBI. Standard imaging procedures were used to obtain neurological data related to brain impairments. Qualitative, quantitative and triangulation methods were used to obtain data to develop psychosocial profiles for different types of brain impairments using the brain imaging data. The study population included males and females, older than 21 years. Subjects underwent a SPECT/CT and PET/CT scan within 4 to 6 weeks of TBI diagnosis confirmation. A complete neurological exam and Evoked Potential tests were performed. Qualitative data on the daily life's experiences was obtained using a semi-structured interview methodology. There was a tendency to find perfusion and metabolic findings in the presence of negative CT. Most common injury was located in the frontal lobe. A mismatch between findings of perfusion defects and metabolic defects was observed which suggests the possibility of up regulation of receptors to compensate for the diminished perfusion. The increased severity and number of perfusion defects in comparison to metabolic defects suggests that the etiology of the TBI may be in part related to a vasomotor response or endothelial dysfunction and not solely to the trauma itself. There was a significant correlation showing that the less independent patients according to FIM have more severe TBI and more severe depression. SPECT CT and PET CT could have an add-value in the diagnosis of patients with mild and moderate TBI.

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A B S T R A C T

Title:

Fluoxetine: An open-channel blocker candidate to treat Slow-Channel Congenital Myasthenic Syndome

Author(s):

Vergara-Mojica, Arayoán; Alicea-Vázquez, Vivianette; Lasalde-Dominicci, José*

Institutional Affiliation:

University of Puerto Rico, Río Piedras Campus

Research Topic:

Clinical Neuroscience

Abstract:

The congenital myasthenic syndromes (CMS) are a group of disorders that affect the neuromuscular junction (NMJ). The slow-channel congenital myasthenic syndrome (SCCMS) is one of those disorders, but it differentiates from the others in that it prolongs the open-channel state of the acetylcholine receptor (AChR). Patients suffering from SCCMS are usually treated with quinidine sulfate to treat the general fatigability and muscle weakness. For those patients that do not tolerate quinidine sulfate, fluoxetine is another option. The dose-effect of fluoxetine is not yet fully understood and established. To shed some light in the field we will analyze the effect of different fluoxetine concentrations in the endplate potential current and the AChR open-state time of transgenic mice containing the SCCMS caused by a Î±C418W mutation. For this purpose, the voltage clamp technique will be used on the diaphragm muscle fibers perfused with different concentrations of the drug. In accordance to previous research, we expect fluoxetine to be effective in shortening the open-state length of the AChR several time-folds.

Notes:
Not interested in short presentation.

Neurochemistry Neurobiology

NN1 – Alemán, J.
NN2 – Cátala, A.
NN3 – Cintrón-López, D.
NN4 – Cintrón, G.
NN5 – Cintrón-Colón, R.G.
NN6 – Colón, J.M.
NN7 – Crooke-Rosado, J.L.
NN8 – Dionisio, D.
NN9 – Feliciano, P.
NN10 – Ferrer-Acosta, Y
NN11 – González-Ruiz, A.
NN12 – Jimenez-Nuñez, E.
NN13 – Marrero-Rivera, G.E.
NN14 – Martínez, N.A.
NN15 – Martínez-Rivera, A.
NN16 – Matos-Ocasio, F.
NN17 – Morales-Rivera, A.
NN18 – Orozco-Vega, R.A.
NN19 – Ortíz-Carpena, J.F.
NN20 – Ortíz-Lugo, J.L.
NN21 – Pasaoglu, T.
NN22 – Perez-Carambot, M.
NN23 – Huang, S.Q.
NN24 – Ramos-Ortolaza, D.L.

NN25 – Rivera, Y.
NN26 – Rivera-Aponte, D.E.
NN27 – Rivera-Díaz, M.
NN28 – Rivera-Pagán, A.
NN29 – Rodríguez-Cruz, E.N.
NN30 – Rodríguez-Laureano, L.
NN31 – Rodríguez, N.Y.
NN32 – Rolón, K.
NN33 – Sáenz, J.F.
NN34 – Santiago-Gascot, M.E.
NN35 – Seale, G.E.
NN36 – Sepulveda-Orengo, M.T.
NN37 – Soto-Soto, E.E.
NN38 – Vaquer-Alicea, J.
NN39 – Vázquez-Figueroa, L.D.
NN40 – Vázquez-Rosa, E.F.
NN41 – Vega-Meléndez, G.S.
NN42 – Zayas-Santiago, A.
NN43 – Nogueras-Ortiz, C.J.
NN44 – Flores-Otero, J.
NN45 – Vallejo, D.I.
NN46 – Cruz-Lopez, D
NN47 – Roman-Ortiz, C.

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A B S T R A C T

Title:

A Strategy to Genetically Manipulate Subsets of Circadian Cells in Drosophila melanogaster

Author(s):

J.AlemÃ¡n; N.RodrÃguez; R. Bohm; E. Rivera; M. Reyes; F. Rivera; H. RodrÃguez; J.L.Agosto

Institutional Affiliation:

University of Puerto Rico Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Drosophila has been a cornerstone in our understanding of the genetic basis of circadian rhythms, but the lack of tools to manipulate the circuit has led us to propose an adaptation of the novel mosaic approach for neural circuit mapping known as ET-FLP-induced intersectional GAL80/GAL4 repression (FINGR). This method has two components added to the GAL4/UAS binary system: 1) a library of enhancer-trap Flippase (ET-FLP), transgenic lines that define the overlap expression pattern and; 2) a GAL80 repressor construct that expresses ubiquitously from a tubulin promoter unless Flippase removes this FRT flanked cassette. As a first step for adapting the FINGR system, we have recombined the FRT flanked tubulin-GAL80 construct with the circadian driver timeless-GAL4 and demonstrated that it can suppress the mortality and arrhythmicity linked with expression of the neurotoxic protein causing Machado-Joseph disease (MJD). Moreover, this construct also suppressed the short period phenotype induced by overexpression of the Drosophila homolog GSK-3 (shaggy) as well as the expression of the Green Fluorescent Protein (GFP) in the circadian system. Finally, by incorporating the ET-FLP gene into a transgenic line expressing GAL80 and GAL4 a subgroup of cells within the circadian network was identified. This demonstrates that the adaptation of the FINGR system to the circadian circuit is a viable approach to characterize anatomically and functionally any subgroup of the circadian network. This knowledge will grant insights into the basic mechanisms underlying the circadian control of physiology and may shine light into how circadian dysregulation increases the risk of diseases.

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A B S T R A C T

Title:

The Role of the Fatty Acid Synthase (FASN) in Running-Induced Neurogenesis

Author(s):

Alma Cátala, Janneliz de la Nuez, Joan Liz Morales, Abdier Benitez, Nataliya Chorna, Sandra Peña

Institutional Affiliation:

UPR Río Piedras

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Previously we identify that running exercise increases the activity of fatty acid synthase (FASN) in the hippocampus of middle age male C56Bl/6J mice. Inhibition of FASN with C75 affected spatial learning and memory in the Barnes maze. We next hypothesized that inhibition of FASN will also affect the neurogenesis in the subgranular zone (SGZ) of the dentate gyrus (DG) in running mice. To determine if the inhibition of FASN was linked to the decrease in cell proliferation in the SGZ of the DG of running mice, brain sections were stained for proliferation marker Ki-67. We identify Ki-67+ cells scattered in a clustered pattern in the SGZ within both supra- and infrapyramidal blades of the rostral DG of vehicle injected and sham running groups. In contrast, C75 injected running group had progressive reduction in the number of Ki-67+ cells diffusively scattered without any clustering and similar to those observed in all sedentary groups. Taken together, our data suggest that activation FASN in the hippocampus is relevant for both the increase of proliferation in the DG and enhancement of spatial learning and memory in middle aged male mice.

Notes:
Not interested in short presentation.

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A B S T R A C T

Title:

IMPAIRED EMOTIONAL MEMORY BY ACUTE INJECTIONS OF ANABOLIC STEROIDS IN ADOLESCENT BUT NOT ADULT MALE RATS

Author(s):

Dahima Cintron Lopez, Keyla M. Ramos-Pratts, Nivia L. Perez-Acevedo, Jennifer L. Barreto-Estrada

Institutional Affiliation:

University of Puerto Rico- Rio Piedras, University of Puerto Rico-Medical Sciences Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Anabolic androgenic steroids (AAS) are exogenous androgens able to modulate cognitive-related regions in the brain such as the amygdala and hippocampus. We aimed to assess AAS effects on emotional memory in adolescent male and female rats (PN-42), as well as in adult male rats (PN- 82-92).Using a single trial Inhibitory Avoidance Task (IAT),and after acute exposure to the AAS, 17alpha-methyltestosterone, we measured acquisition to restrain entrance to a dark side chamber where a mild electric foot shock was received. AAS exposure produced significant impairment of inhibitory avoidance learning in males but not female adolescent rats. Generalized anxiety, locomotion, and risk assessment behaviors (RABs) were not affected. In contrast, adult groups showed high heterogeneity in their latency responses. When comparing adult and adolescent control males, we observed higher latency among adults than in their adolescent counterparts. Further studies will attempt to better standardize the IAT for the adult rats. Our results show that among adolescent rats, acute exposure to a pharmacological dose of AAS,exerts sex- specific detrimental cognitive effects without affecting anxiety. Future experiments will address the role of the NPYergic circuit in the amygdala, given that emotional memory is driven by neuro-peptidergic responses in this brain region. Supported by NIH-NCRR (2P20RR016470-12) and NIGMS (8P20 GM103475-12).

Notes:
Not interested in short presentation.

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A B S T R A C T

Author(s):
Gabriel Cintrón, Dennis R. Valentín, Anixa Hernández & Kenira Thompson
Institutional Affiliation:
Ponce School of Medicine and Health Sciences
Research Topic:
Neurochemistry/Neurobiology
Abstract:

Chronic BDNF-induced delay in hippocampal spatial learning

Gabriel Cintrón[1], Dennis R. Valentín[2], Anixa Hernández[1] and Kenira J. Thompson[1].
Department of Physiology[1]; Department of Psychology[2], Ponce School of Medicine and Health Sciences

BDNF has recently been implicated in various learning tasks, including radial arm maze, eye-blink conditioning, and contextual fear conditioning (Linnarson et al., 1997; Bao et al., 1999; Liu et al., 2001). Acute BDNF administration results in CA3 hyperexcitability and long term potentiation (LTP) (Kang et al., 1995; Schuman et al., 1995; Jimenez et al., 2008). Under chronic conditions, BDNF has been shown to decrease hippocampal excitability (Larmet et al., 1995).The effects of chronic BDNF infusions on hippocampal learning are unknown. In this study we wanted to determine if chronic infusions of BDNF protein into the CA3 region of the hippocampus could enhance spatial learning using the Morris Water Maze task. Male Sprague Dawley rats (250-300g) were bilaterally implanted with cannula at the CA3 region of the hippocampus using stereotaxic coordinates. Following 5 days of recovery post-surgery, human recombinant BDNF (Alomone Labs, Jerusalem) was infused bilaterally into the CA3 region of the hippocampus (1 µg/µl, administered at a rate of 0.080 µl/min for 13min) five days prior to exposure to behavioral testing. Our results indicate that chronic infusions of BDNF induce a delay in water maze learning ($p<0.05$), thus suggesting that exogenous application of BDNF may have an adverse effect on hippocampal functioning. Subsequent studies are currently underway to assess the extent of hippocampal damage after BDNF infusions. These data further support the notion that BDNF plays an important regulatory role in plasticity at the mossy fiber synapse.

This work was supported by: *MBRS-SCORE* (S06 GM008239-20S1), RCMI (2G12 RR003050-24), RCMI Behavioral Neuroscience Core.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Molecular characterization of pathological tau transmission

Author(s):

R. G. Cintron-Colon, C. Nogueras-Ortiz, I. E. Vega

Institutional Affiliation:

University of Puerto Rico, Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Taupathies are a family of neurodegenerative diseases, which pathological hallmark is the accumulation of filaments of the microtubule associated protein tau. The best known tauopathy is Alzheimerâ€™s disease (AD). Filamentous tau is characterized by conformational changes due to posttranslational modifications, such as hyperphosphorylation and truncation. The spreading of tau pathology at specific brain regions is known to directly correlate with the progression of cognitive and/or movement decline in tauopathies. Recent studies demonstrated that tau pathology progression is done by transmission of pathological tau proteins from one neuron to a neighboring neuron. The identity of the tau pathological specie and the mechanisms of tau transmission are still poorly understood. Therefore, mouse primary neurons will be treated with recombinant full length tau. The recombinant tau protein will also be subjected to post-translational modifications prior to add it to the cultured cells to determine the molecular requirements for tau transmission. This study will provide useful information in the quest to understand the process of tau transmission.

Notes:
Not interested in short presentation.

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A B S T R A C T

Title:

17β-Estradiol and Tamoxifen Administration Offers Neuroprotection and functional locomotor recovery after Spinal Cord Injury

Author(s):

Laurivette Mosquera[1], Jose M. Santiago[3], Aranza Torrado[1], **Jennifer M. Colon[1]**, Margarita Melendez, Annabell C. Segarra[1], Jose Rodriguez Orengo[2] and Jorge D. Miranda[1]

Institutional Affiliation:

[1]Physiology & [2]Biochemistry Department, University of Puerto Rico, Medical Sciences Campus, San Juan P.R. 00936; [3]Department of Natural Sciences UPR-Carolina Campus, Carolina, P.R. 00984

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Estradiol (E2) is a multi-active steroid that imparts neuroprotection via diverse mechanisms of action. Controversy has been observed when trying to elucidate the mechanisms of action that confer neuroprotection. In this study, the acute and chronic effects of constant E2 treatment after Spinal Cord Injury (SCI) were evaluated. Ovariectomized rats received E2, MPP-dihydrochloride, control implants or Tamoxifen pellets before a contusion to the spinal cord. E2 treatment improved locomotor function significantly at 7, 14, 21, and 28 days post injury (DPI). An enhancement in capacity crossing the round beam and fewer errors during the grid-walk test was also observed. The observed effects were ER-α mediated (Estrogen receptor-α), since functional recovery was blocked by the ER-α antagonist, MPP-dihydrochloride. An up-regulation in ER-α after SCI was observed and this cellular response was augmented after E2 treatment at 14 and 28 DPI. Long-term treatment (28 days) of the injured rats with E2 reduced the extent of the lesion cavity. The antioxidant effects of E2 were seen acutely at 2 DPI (not at 28 DPI) and this acute effect (2 DPI) was not receptor mediated. These results suggest that E2 improved functional outcomes by having acute and chronic protective effects mediated by the ER-α dependent and independent mechanisms. Rats treated with Tamoxifen (a selective estrogen receptor modulator) recovered some locomotor activity at 28 DPI which could be related to the antioxidant protection seen at this time point. Therefore, Tamoxifen could be considered a long-term safer-alternative treatment drug for spinal cord injury conditions.

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A B S T R A C T

Title:

Assessment of effects of heavy metal contaminants in Puerto Rico´s urban rivers on interactive behaviors of the freshwater prawn *Macrobrachium rosenbergii*

Author(s):

Jonathan L. Crooke Rosado, Ana I. Ortiz Colon, Erick X. Perez Guzman, Laura C. Vicente Rodriguez, Eduardo A. Ruiz Rodriguez, Nilsa M. Rivera Cheverez, Liz M. Diaz Vazquez, Alonso Ramirez, and Maria A. Sosa Llorens

Institutional Affiliation:

Department of Anatomy & Neurobiology (School of Medicine) and Institute of Neurobiology, UPR Medical Sciences Campus; Departments of Environmental Sciences, Chemistry, and Psychology, UPR Río Piedras Campus; Departments of Biology and Social Sciences, UPR Cayey Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Fauna found in tropical streams are dealing with anthropogenic environmental changes that may affect their chances for survival in their natural habitat. The aim of this project is to monitor the effects of contaminants in urban rivers of Puerto Rico on interactive behavior and the underlying neural circuits of freshwater prawns. Water and sediment samples were obtained before the start of storm season from two urban rivers, the Rio Piedras and Rio La Plata, and were analyzed with Gas Chromatography-Mass Spectrometry and Inductively Coupled Plasma-MS for detection and quantitation of organic and heavy metal contaminants, respectively. Initial results indicate that water from the Rio Piedras contains at least 35 organic contaminants, including various esters and/or phthalates. Levels of copper in the Rio Piedras were measured as 0.008, 0.014 and 0.029 mg/L in the upper reach, midpoint and lower reach of the river, respectively. Copper in the La Plata river remained constant at 0.004 mg/L throughout its length. Behavior observation experiments were carried out before, during and after exposing pairs of prawns to dibutyl phthalate, pthalic acid, or copper. The prawn´s interactions were recorded and videos were analyzed using two methods, one to calculate an index of dominance and another to determine distinct behavior frequencies using an instantaneous/continuous behavioral ethogram. Preliminary experiments have shown that exposure to copper or dibutyl phthalate does not significantly change the animal´s

dominance index, but exposure to phthalic acid appears to increase the difference in dominance index between dominant and submissive animals of each pair.

Notes:
Not interested in short presentation.

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A B S T R A C T

Title:

The role of Flap Structure-Specific Endonuclease 1 (FEN1), a DNA recombination/repair factor, in Contextual Fear Conditioning Memory

Author(s):

Dawling Dionisio, Marizabeth Pérez, Edgardo Castro, Adrinel Vázquez and Sandra Peña de Ortiz

Institutional Affiliation:

Department of Biology, University of Puerto Rico-Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:

Learning and memory processes are key to healthy, as well as, disordered mental health. Previous studies published from our laboratory were the first to identify a DNA endonuclease, Fen1, as a factor important in long-term memory (LTM) of conditioned taste aversion in the rat (Saavedra-Rodríguez et al., 2009). In the present studies, we propose to address the role of Fen1 in learning and consolidation of LTM of context fear conditioning in C57Bl/6n mice. Our hypothesis is that Fen1 plays a role in LTM by participating in a pathway of DNA recombination and repair activated by context fear conditioning in the amygdala, a brain region required for this behavioral task. Preliminary data shows that fen1 mRNA is rapidly induced in amygdala, but not the hippocampus, of mice after context fear conditioning training. Our ongoing experiments are directed towards determining that such induction is specific to associability by including context-only and shock-only controls. In addition, we will be examining Fen1 protein induction using Western blotting and confocal immunofluorecense analyses. Finally, siRNA technology will be used to assess whether the observed conditioning-related induction of fen1 and Fen1 function are necessary for LTM of context fear conditioning. Such findings would support a more universal role in Fen1 as a LTM factor, as well as of DNA recombination/repair processes in LTM.

This work was supported by SPO grant: 5SC1MH086072.

Notes:

Not interested in short presentation.

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Functional Interaction of Synapsin II and Rab3a in the Regulation of Epileptic Activity and Synaptic Plasticity at Hippocampal Synapses

Author(s):

Pedro Feliciano, Rodrigo Andrade, and Maria Bykhovskaia

Institutional Affiliation:

Universidad Central del Caribe, Neuroscience Department

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Some forms of epilepsy are associated with a deficiency of the synaptic vesicle protein synapsin. Our recent study revealed that seizure phenotype in synapsin II deleted mice (SynII(-)) can be rescued by the gene deletion of Rab3a, a binding partner of synapsin. To elucidate the nature of the synapsin-dependent epileptic phenotype and to understand how the Rab3a deletion can balance epileptic seizures that are typical for SynII(-) animals, we recorded synaptic and epileptiform activity from CA1 pyramidal cells at hippocampus. We found that SynII(-) deficient neurons have increased spontaneous and epileptiform activity, and this effect depend on GABAergic transmission. Next, we investigated if the observed increase in network excitability in SynII(-) is mediated by opposite changes in glutamatergic and GABAergic transmission. We found that the epileptogenic agent 4-aminopyridine(4-AP) promotes action potential evoked glutamatergic transmission in SynII(-) synapses stronger than in WT. In contrast, this treatment had no effect on GABAergic transmission in SynII(-) synapses. The elevation of extracellular Ca2+ mimicked the effect of 4-AP application, suggesting that the observed phenotype in SynII(-) neurons may be produced by modifications in Ca2+ sensitivity of the exocytic pathway. This differential effect of the epileptogenic agent on excitatory and inhibitory transmission could account for overexcitability in SynII(-) networks. To understand how Rab3a deletion can balance the epileptic phenotype observed in SynII(-) neurons, we have repeated the above experiments at Rab3a(-) and Rab3a(-)/SynII(-) hippocampal slices. We have found that Rab3a deletion balances synapsin II deficiency at the level of excitatory but not inhibitory transmission.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Characterization of EFhd2, an amyloid-like protein associated with tau in tauopathies

Author(s):

Yancy Ferrer-Acosta, Eva N. Rodriguez-Cruz, François Orange, Maxime J.F.Guinel and Irving E. Vega

Institutional Affiliation:

University of Puerto Rico, Rio Piedras Campus, San Juan, PR

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Tauopathies are a group of neurodegenerative diseases characterized by intracellular accumulations mainly composed of the Tau protein. EFhd2, a calcium-binding protein, was found to be associated with pathology-related Tau in a Tauopathy mouse model and in humans with AD. EFhd2 contains two calcium-binding domains, a polyalanine stretch and a coiled-coil domain, yet, its function within the CNS is unknown. This protein co-purified with hyperphosphorylated Tau in a detergent-insoluble fraction harboring aggregated brain proteins (P3). Protein aggregates are the consequence of a fibrillation process that forms highly stable Î²-pleated sheet structures. These results led us to hypothesize that EFhd2 could be an amyloid-like protein that aggregates during neurodegeneration. To test this hypothesis, we performed in vitro binding assays with Thioflavin S (ThS), a dye that has affinity for amyloid structures. Results show that EFhd2 forms ThS-affinity structures in a concentration-dependent manner, and these were shown to be fibrils by TEM. In vitro incubation of EFhd2 and Tau show increased ThS binding, demonstrating the formation of amyloid structures between them. Amyloid structures for both EFhd2 and Tau were observed in vivo by immunogold in the P3 fraction from AD brain and by immunofluorescence in AD human brain slices. These results indicate that EFhd2 has amyloid-like properties in vitro and in vivo, introducing it to the family of amyloids within the CNS. Furthermore, aggregation and colocalization of EFhd2 with pathological tau supports the idea that this protein plays an important role in the pathobiology of tau-mediated neurodegeneration.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Characterization of the fusion clamping function at the *Drosophila* neuromuscular synapse

Author(s):

Agustin Gonzalez Ruiz[1], Maria Bykhovskaia[1]

Institutional Affiliation:

[1]Department of Neuroscience, Universidad Central del Caribe, Bayamon, Puerto Rico

Research Topic:

Neurochemistry/Neurobiology

Abstract:

Vesicle fusion is regulated by the SNARE protein complex. The SNARE complex is formed by a vesicle protein synaptobrevin (nSyb), and membrane proteins syntaxin (Syx) and SNAP-25. Complexin (Cpx) is a cytosolic protein that interacts with the SNARE complex and has been implicated to act as a "clamp" preventing exocytosis. However, complexin function and fusion clamp machinery is still under investigation. Basing on molecular modeling, we predicted several mutations in the SNARE proteins and Cpx, which would destabilize the SNARE complex and promote the "unclamped" conformational state thus increasing spontaneous exocytosis. To test these predictions experimentally, we employed focal recording from visualized synaptic boutons at the *Drosophila* neuromuscular junction. We have also investigated a temperature-sensitive paralytic *Drosophila* mutant *syx*$^{3\text{-}69}$, which has a point mutation in Syx. According to our molecular modeling study, the *syx*$^{3\text{-}69}$ point mutation partially mimics the Cpx null (*cpx*$^{-/-}$) phenotype. We found that *syx*$^{3\text{-}69}$ mutant has an increased spontaneous activity similar to the *cpx* null. However, action potential evoked release is unaffected in the *syx*$^{3\text{-}69}$, even

though it is strongly desynchronized in the *cpx*$^{-/-}$. Our results reveal several elements of the Cpx and SNARE structure required for the fusion clamping.

Notes:

Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Enhancements in mossy fibers long-term potentiation (LTP) are neurotrophin-specific

Author(s):

Esther Jimenez-Nuñez*, James T. Porter and Kenira J. Thompson

Institutional Affiliation:

Ponce School of Medicine and Health Science

Research Topic:

Neurochemistry/Neurobiology

Abstract:
In addition to a clearly defined role of BDNF (Brain-derived neurotrophic factor) in cell survival and differentiation, this neurotrophin has recently been implicated in hippocampal plasticity, including LTP. Interestingly, the mossy fiber (MF) projection to pyramidal cells in area CA3 of the hippocampus contains the highest concentration of BDNF in the CNS (Danzer and McNamara, 2004). Also, BDNF dramatically enhances mossy fiber field potentials and LTP in vitro (Jimenez et al 08). In vivo BDNF infusions enhance MF LTP and result in neurogenesis at the MF synapse (Gomez-Palacio and Escobar et al., 2008). In this study we addressed the question of whether other neurothrophins such as NT3 and NGF are involved in LTP at the MF synapse. Using ahippocampal slice preparation, we recorded field potentials at CA3 pyramidal cells following high frequency stimulation (HFS; 2 trains 100 Hz, 1 sec duration) of the MF pathway, and observed the effects of bath application of BDNF. In addition; we recorded field potentials at CA3 pyramidal cells following HFS of the MF pathway and observed the effects of micropipette application of NT3 a tyrosine kinase C and NGF a tyrosine kinase A activators on CNS. We found that BDNF resulted in MF LTP, and this effect was further enhanced if BDNF was bath applied prior to HFS whereas enhancement of LTP was not observed following application of NGF or NT3. Our results indicate that BDNF plays a specific effect in synaptic plasticity and LTP at the MF synapse.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Characterization of the Subcellular Localization of EFHD2 in Tau-Mediated Neurodegeneration

Author(s):

Gabriel E. Marrero-Rivera, Yancy Ferrer Acosta, Irving E. Vega

Institutional Affiliation:

University of Puerto Rico, Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
The novel calcium binding protein EFhd2 was found associated with pathological forms of the microtubule-associated protein tau in the tauopathy mouse model JNPL3 and Alzhemier's disease cases. The association between EFhd2 and tau was only detected in JNPL3 mice at terminally ill stage, suggesting the association between these two proteins is triggered by the neurodegeneration process. Furthermore, EFhd2 copurified in the sarkosyl insoluble fraction with tau proteins. However, the physiological or pathological roles of EFhd2 are not understood. This novel protein is most abundant in the central nervous system. EFhd2 contains a polyalanine motif on its N-terminus, two EF-hand (calcium-binding) motifs, and a coiled-coil domain in the C-terminus. Functional structural analyses demonstrated that the two EF-hand motifs bind calcium cooperatively and that it is predominantly composed of alpha helix and random coil structures. Thermal stability assays indicated that EFhd2 is a thermostable protein, which depends on its N-terminal domain. To obtain insights that contribute to the understanding of the biological function of EFhd2, experiments were designed to determine its subcellular localization in non-transgenic (NTg) and JNPL3 mice. The longitudinal analysis of NTg and JNPL3 mice will also allow us to determine changes in subcellular localization associated to the progression of tau-mediated neurodegeneration. Subcellular fractionation assays and brain region specific immuno assays were performed to characterize the association of EFhd2 with subcellular compartments and its expression profile. The results obtain will provide value information leading to the characterization of the novel protein EFhd2 and its putative role in tau-mediated neurodegeneration.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Caveolin-1 Supports the P2Y2 Receptor Signaling

Author(s):

Namyr A. Martínez, Alondra M. Ayala, Magdiel Martínez, Mónica Quiñones, Jorge D. Miranda & Walter I. Silva

Institutional Affiliation:

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Research Topic:

Neurochemistry/Neurobiology

Abstract:
Damage to the cells of the central nervous system (CNS) can cause a differential spatiotemporal release of multiple factors and chemicals into the extracellular space. Among such factors, nucleotides, and their interaction with $P2Y_2$ nucleotide receptors ($P2Y_2Rs$) have gained prominence as putative modulators of gliotic responses after CNS injury. ATP and UTP can promote reactive astrocytosis, a process that involves astrocyte proliferation and stellation. Reactive astrocytes have been suggested to have a dual role during injury responses as they may promote neuronal growth and guidance to damaged regenerating axons, or inhibit axonal regeneration and remyelination. Nevertheless, the molecular mechanisms underlying these responses remain to be explored. Detergent-free discontinuous sucrose density gradient separation of 1321N1 cells (expressing recombinant $P2Y_2R$-1321N1) homogenates revealed co-fractionation of $P2Y_2Rs$ with cav-1 in light density membrane-raft fractions. Furthermore, laser scanning confocal microscopy of $P2Y_2R$-1321N1 cells revealed that a significant percent of $P2Y_2R$ co-localized with cav-1 in its subcellular distribution. Additionally, blocking cav-1 expression in $P2Y_2$-coding 1321N1 cells elicited abnormal intracellular calcium mobilization responses when stimulated with nucleotide agonists, as determined by microfluorometric calcium imaging analyses. Our findings suggest that $P2Y_2Rs$ reside in membrane caveolae of naive, non-stimulated 1321N1 cells and that this subcellular compartment may couple its downstream signaling machinery. Therefore, we have hypothesized that signaling cascades of the $P2Y_2Rs$ are intimately linked to their expression in cav-1 positive rafts micro-domains. Understanding this receptor's activation and signal transduction is imperative as it might potentially unveil new venues for disease treatment and/or modification.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Role of metabotropic glutamate receptor 5 (mGluR5) within the nucleus accumbens shell and expression patterns of mGluR5 and Homer 1b/c during environmental-elicited cocaine conditioning.

Author(s):

Martinez-Rivera, A., Rodriguez-Borrero,E. and Maldonado-Vlaar, C.S.

Institutional Affiliation:

University of Puerto Rico, Rio Piedras

Research Topic:

Neurochemistry/Neurobiology

Abstract:
The metabotropic glutamate receptors 5 (mGluR5) within the Nucleus Accumbens (NAc) have been implicated in modulating psychostimulant reward. mGluR5 subtypes are associated with Homer proteins, a family of synaptic proteins that anchor mGluR1/5 at the excitatory synapse and that may mediate the effects of cocaine conditioning. Previous evidence proposed that Homer protein family have an important role in memory and learning processes during cocaine exposure. However, how this protein affects the environmental elicited cocaine conditioning still remains unknown. Our experiments examined the effects of blockade of mGluR5 subtype within the NAc shell, especially on the expression of this drug conditioning, and the protein expression patterns of mGluR5 and Homer1b/c during environmental-elicited cocaine conditioning. We hypothesized that blocking mGluR5 subtypes within NAc shell will impair associative learning responsible for the cocaine conditioning state, therefore producing an enhancement of mGluR5 and Homer1b/c at synaptosomal membrane fraction. Rats were implanted cannula within NAc shell, and then separate groups were exposed to a multimodal environment within activity chambers that signaled cocaine (paired) or saline (controls, unpaired). Prior to placing the animals in the chambers, rats received systemic injections of saline or cocaine for 10 consecutive sessions. On the test session (Day 12) separate groups of animals were infused within NAc shell with 2.5, 12 or 25nmol/.5Âµl/side of MPEP, an mGluR5 antagonist. Blockade of mGluR5 subtype with 2.5nmol showed no significance difference in the total move time and in the vertical plane move time. In contrast, mGluR5 blockade with 12nmol or 25nmol decreased conditioned locomotion in the paired groups. These results suggested that mGluR5 within NAc shell play a role in the expression of a conditioned response elicited by cocaine use. Biochemical studies revealed that there are no changes in protein expression of mGluR5 and Homer1b/c during the development of cocaine treatment. Taken together, these results suggest a role of mGluR5 during cocaine conditioning. Moreover, that cocaine conditioning may be modulating the activity of mGlur5 intracellular cascade proteins other than the receptor itself.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

GLT-1 up-regulation impairs learning of a novel object recognition task

Author(s):

Félix Matos-Ocasio, Anixa Hernández, and Kenira J. Thompson

Institutional Affiliation:

Ponce School of Medicine and Health Sciences

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Glutamate transporters (GluTs) are important for maintaining optimal glutamate concentrations at the synapse. This allows proper synaptic response, plasticity and prevents neurotoxicity. It has been shown that the β-lactam antibiotic ceftriaxone (Rocephin) induces an up-regulation of the Glut GLT-1 (Omrani, 2009). This GLT-1 up-regulation blocks the metabotropic glutamate receptor (mGluR) dependent long-term depression (LTD) at the mossy fiber (MF)-CA3 hypocampal synapse. It also has negative effects on long-term potentiation (LTP). Behavioral effects of GLT-1 up-regulation, related to learning and memory on normal rats, are not known. However, GLT-1 up-regulation has been proven to have beneficial effects on Huntington's disease (Miller, 2008) and cocaine addiction (Sari, 2009). In addition, deletion of GLT-1 appears to exacerbate deposition of β-amyloid plaques in animal models of Alzheimer's disease (Mooherjee, 2011). We used male Sprague Dawley rats (2-3 months old) to assess the cognition effects of GLT-1 up-regulation (via IP injections of ceftriaxone) on normal rats. After 8 days of drug administration, we performed a standard novel object recognition task. Drug administered rats showed memory impairments when compared to control rats ($p<0.05$). These findings show that an up-regulation of GLT-1, which in turn causes a decrease of glutamate at the synapse, has detrimental effects on learning and memory. Our results further support the notion that glutamate transporters provide an essential regulatory role in hippocampal learning and memory.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Behavioral effects of brain oxytocin in cocaine seeking behavior and cocaine conditioning.

Author(s):

A. MORALES-RIVERA(1), M. M. HERNANDEZ2, R. RIVERA, C. S. MALDONADO-VLAAR(2)

Institutional Affiliation:

UPR Medical Sciences Campus(1), UPR Rio Piedras(2)

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Addiction is defined as the loss of control over drug use, compulsive seeking and taking drugs despite the negative consequences (Nestler E.J. 2001). Several clinical studies have revealed that the maintenance of cocaine addiction requires three key contributors: (a) the reinforcing effects evoked by cocaine intake, (b) the environmental stimuli associated with the drug, and (c) the stress (Sinha, 2003). Moreover, a stress stimulus triggers the reinstatement of cocaine seeking behavior, following long periods of abstinence in dependent subjects.Oxytocin (OT) is a neuropeptide that has been related to reward, learning, memory and stress, events that previously have been associated with cocaine addiction. Previously published data from our laboratory demonstrated an increase in mRNA OT levels within the NAc by acute and chronic cocaine exposure (Borrero 2009). The working hypothesis for these studies is that centrally administered oxytocin, or an oxytocin receptor agonist, will be modulating the stress/anxiety response elicited by environmentally elicited cocaine seeking behavior (CSB). Specifically, we investigated the effects of OT in the extinction and reinstatement of self administratin paradigm and in the cocaine conditioning paradigm, while micro-injecting OT agonist (T-got) and OT into the Ventricular system (ICV). First, for the CSB, male Sprague Dawley rats were surgically implanted with an intravenous catheter. Following recovery from surgery, rats underwent in a cocaine self administration paradigm. A set of rats for cocaine conditioning were implanted cannula within ICV and separate groups were exposed to a multimodal environment within activity chambers that signaled cocaine (paired) or saline (controls, unpaired). Rats were randomly divided into experimental group and control group, rats received ICVcsf/OT/tgot microinjections (0Î¼mol/Î¼l,

10Î¼mol/10Î¼l) during extinction, reinstatement of the self administration paradigm and expression day of conditioning paradigm, and later elevated plus maze testing. Results showed that during extinction no significant differences between drug treatments were found. These results contrast from our reinstatement data that showed that OT and Tgot treatment diminished reinstatement of cocaine seeking behavior. For the cocaine conditioning paradigm an increase of time in the open arms for the cocaine-oxytocintreated group compared with cocaine-vehicle attenuating the anxiety triggers by the cocaine-paired environment. Thus, with these results, we can suggest OT is a potentialpharmocotherapeutical candidate in treating cocaine addiction.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Age-dependent effects of the anabolic steroid nandrolone in conditioned place preference and anxiety.

Author(s):
Orozco-Vega R.A., Martínez-Rivera F.J., Muñiz-Seda O.A., Natal E. , Martínez-Alicea N.A., Barreto-Estrada J.L.

Institutional Affiliation:

UPR-RCM

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Age-dependent effects of the anabolic steroid nandrolone in conditioned place preference and anxiety Orozco-Vega R.A.4, Martínez-Rivera F.J.1, Muñiz-Seda O.A.3, Natal E. 3, Martínez-Alicea N.A.2, Barreto-Estrada J.L.1 1Departments of Anatomy and Neurobiology, 2Physiology and Biophysics, Medical Sciences Campus-University of Puerto Rico, San Juan, Puerto Rico 00936, 3Department of Biology, RÃo Piedras Campus, University of Puerto Rico 00936, 4Department of Biology, Cayey Campus, University of Puerto Rico 00736. The misuse of anabolic-androgenic steroids (AAS) is widespread among adults and adolescents, representing a considerable public-health problem. It has been suggested that most AAS users initiate misuse for the anabolic effects, but many develop neuropsychological dependence. Previously, we have shown that nandrolone (ND) induced conditioned place preference (CPP) in a dose response manner in the adult male mice without affecting locomotion and anxiety-like behaviors (ALB). However, the rewarding effects of ND during adolescence remain unidentified. In this study we measured CPP, locomotor activity and ALB after exposure to ND (7.5, 0.75, 0.075mg/kg) in adolescent male mice. Elevated plus maze (EPM) was also used to analyze ALB, locomotion, stereotype, and risk assessment (RABs) behaviors. Results showed that ND: i) shifted place preference in adults, but not in adolescents, ii) failed to cause changes in ALB in the adults, whereas it elicited anxiolytic-like behavior in adolescents, iii) did not affect RABs in adults nor in adolescents, iv) increased grooming (stereotype behavior) in adult mice but not in adolescents, v) did not altered body and gonadal weights. Western blots analysis of the nucleus accumbens, a key structure in the reward mesolimbic circuit, showed a decrease in the type 1-dopamine receptor (D1DR) expression only in ND-treated adults. Results suggest that differences in the dopaminergic mesolimbic system might account for the CPP sensitivity throughout

development. Supported by MBRS-RISE-MSC (R25-GM061838), NCRR (2P20RR016470-12), NIGMS (8P20 GM103475-12).

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Developmental Exposure to Lead (Pb^{2+}) Results in Social Interaction Deficits in *D. melanogaster*

Author(s):

Jorge Felipe Ortiz-Carpena[1], Adrinel Vázquez-Montes[1], Amneris Hernández[1], Daniel Vélez-Costas[2], Eduardo Galindez-Cintrón[2], Sandra Peña de Ortiz[1], Humberto Ortiz-Zuazaga[2,3]

Institutional Affiliation:

1.Department of Biology, 2. Computer Science and 3. High Performance Computing Facility, University of Puerto Rico, Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:

"Developmental Exposure to Lead (Pb^{2+}) Results in Social Interaction Deficits in *D. melanogaster* "

Lead (Pb^{+2}) is an environmental contaminant widely dispersed throughout the world. Exposure to Pb^{+2} causes neurological damage in humans and may be linked to neurodevelopmental pathologies such as attention deficit hyperactivity disorder, antisocial behavior, and autistic spectrum disorders (ASD). *D. melanogaster* have been used to understand the behavioral, synaptic and molecular changes after developmental exposure to Pb^{+2} and to study ASD-associated pathology. Here, we used the fruit fly for understanding the molecular mechanisms underlying developmental Pb^{+2}-mediated sociability impairments. Flies were exposed through the mother and until eclosion with either Pb^{+2} or control corn-based media and isolated until beginning of our behavioral studies. Sociability testing was done using a test tube that was divided by a mesh allowing the flies to interact using the olfactory and visual clues while avoiding direct contact as previously described. Multiple fly recordings were made using a high-throughput video system and software analysis. Our findings are that developmental exposure to Pb^{+2} results in dose-dependent developmental delay and decreased social interaction in female flies. Our next experiments will use the microarray approach to identify Pb+2 mediated alterations of gene regulatory networks and their contribution to sociability dysfunction in exposed flies.

Work supported by NIH INBRE grant P20-RR016470, UPRRP MARC Program Grant #5T34GMO7821-32 and SC1GM084708 to SPO.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Effect of Isolation Rearing on Nurr1 Brain Expression and the Sociability of C57BL/6J

Author(s):

Ortiz-Lugo, J.L., Maldonado, P., Escalera, K., Ramírez, T., Vázquez-Montes, A. & Pena de Ortiz Sandra

Institutional Affiliation:

University of Puerto Rico at Rio Piedras

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Nurr1 (Pena de Ortiz and Jamieson 1996) is a member of the nuclear hormone receptors family of transcription factors that has been shown to play a key role in regulation of gene expression in the hippocampus and active neural development. Previous studies, demonstrate that Nurr1 is relevant to long-term memory (LTM) formation(Colon-Cesario et al.,2006) and plays important roles in the molecular machineries regulating complex behaviors (Ressler et al.,2002; Keely et al.,2006). Here,we have focused in determining if the expression of Nurr1 is sensitive to isolation rearing, whether sociability interaction tests (SIT) induce the expression of Nurr1 and in what brain regions, and whether isolation rearing affects sociability behavior. Male C57BL/6J mice were divided into two groups immediately after weaning: Socially Reared (SR) versus Isolated (IR) mice. Animals from both groups were divided in three categories: Naive, Context-Only and SIT. To assess social behavior a three-chambered Ugo Basile sociability apparatus was used. All test sessions were video-recorded and mice were sacrificed 1 h later to extract the brains. Preliminary results of behavioral data shows that SR-SIT mice spent significantly more time interacting with intruder mice, than the IR-SIT animals. Analysis of Nurr1 immunopositive cells shows a significant and specific induction in CA1 ($p<0.005$) in SR mice, but not in IR mice. SIT and CO associated significant changes were also observed in the dorsal and ventral claustrum regions of SR and IR mice due to the trainings. In summary, our results suggest that Nurr1 may play a significant role in sociability behavior.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Quantitative Ultrastructural Analysis of Commissural/associational CA3 Synapses in Wild Type and FGF22 Knock-out Mice

Author(s):

T. Pasaoglu, D. Cruz, L. Qu, T. Schikorski

Institutional Affiliation:

Universidad Central del Caribe

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Synaptic morphology forms the basis of synaptic function. Statistical distributions of the various morphological characteristics of a particular population of synapses are therefore important predictors of the functional properties of a particular synapse population. Here, we present statistical distributions of the morphological properties of associational/commissural synapses in the stratum radiatum of the hippocampal CA3 region. Since synaptic characteristics are determined by pre- and postsynaptic signaling during development and in the mature brain, this histological uniqueness provides the opportunity to investigate to what extend synaptic differentiation varies when the same axon population is connected to two different excitatory target cell populations with different sets of presynaptic organizers. We found that the morphological properties of associational CA3 synapses are statistically similar to their CA1 counterparts, although several differences were apparent. The percentage of boutons that contacted two or more spines was higher in CA3 than in CA1 synapses. In contrast, in CA3, some spines were innervated by two or more boutons. However, the most obvious difference was the lack of a significant correlation between spine volume and postsynaptic density area, that is, all spines in CA3 are similar in size. We also were interested how the lack of the CA3-specific presynaptic organizer FGF 22 affects synaptic morphology. By using the same approach as above, we determined the morphology and density of CA1 and CA3 synapses in FGF22 knock-out mice. The differences to wild type are discussed

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

The apurinic/apyrimidinic endonuclease 1 (apex1) plays a role in long-term memory on context fear conditioning.

Author(s):

M. PEREZ CARAMBOT, A. VÁZQUEZ, N. Y. OCASIO, E. A. PÉREZ-CASTRO, K. P. BETANCOURT, V. RIVERA, X. FIGUEROA, A. CÃ• TALA, S. PEÑA DE ORTIZ**;**

Institutional Affiliation:

Univ. of Puerto Rico, Rio Piedras Campus, San Juan, P.R., Puerto Rico

Research Topic:

Neurochemistry/Neurobiology

The apurinic/apurimidinic endonuclease 1 (apex1) plays a role in long-term memory on context fear conditioning.

Abstract:
Our research focuses on evaluating the role of DNA recombination/repair processes as possible mechanisms of gene regulation required in the formation of long-term memory (LTM). We are examining the role of the apurinic/apyrimidinic endonuclease 1 (Apex1) gene, which also encodes an endonuclease involved in DNA recombination/repair processes, during LTM formation of contextual fear conditioning (CFC). Male adult C57BL/6J mice were subjected to CFC and sacrificed at 30min, 1h, and 3h after training. Naive animals were used as controls. Hippocampal and amygdalar mRNA was isolated for qReal-Time PCR. Additional experiments used Naive, Context and Shock only controls, sacrificed at 30min or 1h to examine the behavioral specificity of mRNA and protein induction, respectively. Current studies use antisense to block Apex1 amygdalar expression in order to determine its relevance on CFC LTM. qReal-time PCR showed a bi-phasic Apex1 amygdalar-specific mRNA induction at 30 min and 3 hr after CFC. Such Apex1 mRNA induction is specific to associative learning. Furthermore, our preliminary immunohistochemistry data suggests that induction of Apex1 protein 1 hr after CFC is restricted to the central and basolateral nuclei of the amygdala. Also, preliminary antisense data suggests that Apex1 is necessary for CFC LTM formation. These studies demonstrate that Apex1 is rapidly induced at the mRNA and protein levels in the amygdala as a result of CFC. Moreover, within the amygdala, Apex1 induction is specific to the basolateral and central amydala. Finally, our ongoing antisense studies suggest that this expression plays a significant role in the formation of CFC LTM.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Characterization of the intestinal tract neuroendocrine cells in normal and regenerating *Holothuria glaberrima*

Author(s):

Sunny Qi Huang and José E. García-Arrarás

Institutional Affiliation:

Undergraduate Student

Research Topic:

Neurochemistry/Neurobiology

Abstract:
The neuroendocrine system is composed of cells that release neurotransmitters and neuropeptide hormones in response to environmental stimuli. In the present study we characterize the neuroendocrine system of the sea cucumber Holothuria glaberrima using immunohistochemistry in normal (non-eviscerated) and regenerating organisms at different stages of regeneration. The intestinal tract of the sea cucumber was divided into four portions: adjacent to the esophagus, descendant and ascendant small intestine, and convoluted large intestine. Specific antisera against-calbindin, -GABA, -Nurr 1, -Galanin, -PAX 6 and -GFSKLYFamide were employed to visualize neuroendocrine cells. These cells are slender in shape, with basally located nucleus, and their main axis perpendicular to the basement membrane. The number of neuroendocrine cells was higher in the descendant portion of the small intestine and in the large intestine than in the ascendant portion of the small intestine. During regeneration, neuroendocrine cells appeared very early with the formation of the luminal layer and were already observed at 10 days post evisceration. In addition, their numbers increased as regeneration proceeded. These results contribute to a better understanding of the neuroendocrine system in the digestive tract of echinoderms and of the cellular events that characterize the regeneration process. **Financial support: NIH (1SC1GM084770-01,1R03NS065275-01), NSF (IOS-0842870), University of Puerto Rico.**

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

FEMALES ARE PROTECTED FROM THE EFFECTS OF CHRONIC MORPHINE, WHILE MALES SHOW IMPAIRMENT IN FEAR EXTINCTION AND RECALL.

Author(s):

DL RAMOS-ORTOLAZA; EM Pérez-Torres; JK Alvarado; A Torres-Reverón; E Santini

Institutional Affiliation:

Nova Southeastern University; Ponce School of Medicine and Health Sciences; University of Puerto Rico in Ponce

Research Topic:

Neurochemistry/Neurobiology

Abstract:

Morphine is an opiate used to treat chronic pain. Its long-term use can lead to the development of dependence and/or addiction. The persistence of addiction is associated with abnormal learning and memory processes and possibly with the development of mood disorders, effects that may be sex-specific. Our study was aimed at determining whether chronic morphine administration affects associative learning differently in males and females. Male and female rats were injected with morphine (5-50 mg/kg) or saline for 10 days. Twenty and ninety six hours after the last injection, rats were observed for somatic signs of withdrawal. Five days after the last injection, rats underwent auditory fear conditioning followed by extinction training and extinction recall 24 and 48 hours later respectively. Vaginal smears were performed daily in female rats throughout the experiment to determine estrous cycle regularity. Morphine disrupted the estrous cycle of female rats, similar to what's documented for female heroin addicts. No sex differences were observed in the somatic signs of spontaneous withdrawal. Morphine did not affect acquisition or recall of fear conditioning in either male or female rats. However, it caused a significant delay in fear extinction and impairment of extinction recall in males. Taken together, our results suggest that there might be a sex-specific involvement of the opiate system in extinction learning that could affect the persistence of morphine addiction and the development of associated mood disorders.

Notes:

Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Spermine and Spermidine permeate Cx43-hemichannels in freshly isolated astrocytes and Mueller glia

Author(s):

Y. RIVERA[1], Y. V. KUCHERYAVYKH[1], J. BENEDIKT[1], R. W. VEH[2], C. G. NICHOLS[3], A. RIVERA[1], M. J. EATON[1], S. N. SKATCHKOV[1]

Institutional Affiliation:

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Research Topic:

Neurochemistry/Neurobiology

Abstract:

Glio-transmitters regulate neuronal behavior. Here we provide evidence that polyamines, spermidine and spermine (SD/SP), are new candidate glio-transmitters. We found that SD/SP: accumulate in glia, are taken up and released from glia, and act on neuronal receptors and channels. We hypothesize that SD/SP can become unbound from acid compounds in the cytoplasm and released via large pores in the membrane. The purpose of this study was to investigate the permeability of SD/SP through the glial membrane. We monitored biotinylated SP uptake, measured SD/SP electrical currents and used a novel SP-biosensor to measure SP release. The SP-biosensor is based on excised patches from Cosm-6 cells transfected with cDNAencoding mutated Kir6.2 [N160D, C166S]/SUR1 channels. We found that biotinylated SP permeates the membrane and accumulates in astrocytes; SD/SP fluxes are blocked by Cx43 siRNA inastrocytes. Comparing freshly isolated astrocytes and Mueller cells with cultured astrocytes, we found that SP electrical currents were 60% less pronounced in isolated cells than in culture, suggesting that enzymatic treatment (papain-DNase) can affect the external Cx43 pore and/or disrupt fine glial processes where Cx43 is predominantly localized. Using SP-biosensor, we measured SP release from cultured astrocytes, retina and brain slices. Depending on stimulation protocols and the amount of SP previously accumulated, release from 4 to 100 ÂµM was observed. We conclude that SD/SP are

novel glio-transmitters which not only modulate synaptic transmission but also can be neuroprotective when blocking Ca2+ permeable channels in neurons.

Acknowlwdgements:

Supported by the NIH grant numbers: R01-NS065201 (to S.N.S); 8G12-MD007583 (for core facilities at UCC). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of NIH.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

High Glucose Decreases Kir4.1 and TREK-2 Potassium Channels Expression and Function in Astrocytes

Author(s):

D.E. Rivera-Aponte, A. Rivera-Pagan, Y. Kucheryavykh, S.N. Skatchkov and M.J. Eaton

Institutional Affiliation:

Department of Biochemistry Universidad Central del Caribe, Bayamón, Puerto Rico

Research Topic:

Neurochemistry/Neurobiology

Abstract:

Both type I and type II diabetes affect the central nervous system (CNS) and are coupled with increased incidence of seizure and stroke. Astrocytes are critical for normal CNS function and dysfunction or loss of astrocytic potassium channels increases seizure susceptibility and is associated with ischemic brain damage. The purpose of the present experiment was to test the hypothesis that high glucose alters the expression of potassium channels in astrocytes. To test this hypothesis, we prepared primary astrocyte cultures from Sprague-Dawley rats and grew them in low (5.5 mM) and high glucose (25 mM)-containing DMEM. After two weeks in culture, we performed RT-PCR and Western blot to determine gene and protein expression of Kir4.1 and TREK-2 potassium channels and whole-cell electrophysiological recording to measure potassium channel currents. For comparison, we determined the mRNA and protein levels of glial fibrillary acidic protein (GFAP); a marker of astrocytes that has been shown to be downregulated in astrocytes grown in high glucose. Our results revealed that astrocytes grown in high glucose had lower levels of Kir4.1, TREK-2 and GFAP mRNA and protein when compared with astrocytes grown in low glucose. Furthermore, electrophysiological studies demonstrated that Kir and TREK channel function is impaired in astrocytes grown in high glucose. Taken together, our results suggest that downregulation of astrocytic potassium channels by elevated glucose may contribute to the underlying pathophysiology of diabetes-induced CNS disorders.

NIH-SC1-GM088019, NIH-NCRR-G12-RR03035, NIH-NIMHD-8G12-MD007583, NIH-NINDS-U54-NS-039408.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

MICRO-RNA EXPRESSION PROFILES IN PUERTO RICAN BRAIN TUMOR SAMPLES

Author(s):

Monica Rivera-DÃaz, Mario Quintero, Rodolfo Alcedo-Guardia & Pablo Vivas-MejÃa

Institutional Affiliation:

University of Puerto Rico, Medical Sciences Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
PURPOSE: Gliomas, the tumor of glial cells, are the most common of the central nervous system malignancies. Glioblastoma multiforme (GBM) or grade IV astrocytoma, is the most common and aggressive glioma. GBM accounts for more than 60% of brain tumors. The median survival for patients diagnosed with GBM has only marginally changed in the last 25 years and still remains 1 year. Thus, there is an urgent need for better prognostic, diagnostic and therapeutic tools to treat this devastating disease. MicroRNAs (miRNAs) are endogenous, short (19â€“24 nucleotides) non-protein-coding RNAs that regulate gene expression at the post-transcriptional level. Evidence indicates that miRNAs regulate about 60% of the human genes. Multiple studies have demonstrated that miRNAs have a causal role in brain tumor initiation and progression, and in drug resistance. However, the precise miRNAs aberrantly expressed in GBM have not been identified. DESIGN METHODS: Archived Puerto Rican brain tumor formalin-fixed paraffin-embedded (FFPE) samples were used for this study. Total RNA was isolated, labeled, and hybridized to Affymetrix miRNA arrays. RESULTS: PARTEK analysis identified 85 miRNAs differentially regulated in grade II, 72 miRNAs in grade III and 118 miRNAs in GBM compared with control samples. We selected the most expressed miRNAs among the samples analyzed to validate using TAqMan-based assays. CONCLUSION: We are currently performing in vitro studies to clarify the role of some of these microRNAs in tumor growth. Future studies will correlate the expression of miRNAs in tumor tissues and in the blood of brain tumor patients.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

ISCHEMIA UPREGULATES TREK-2 POTASSIUM CHANNELS IN ASTROCYTES

Author(s):

A Rivera-Pagán; DE Rivera-Aponte; LY Kucheryavykh; YV Kucheryavykh; LA Cubano; SN Skatchkov; MJ Eaton

Institutional Affiliation:

Universidad Central del Caribe

Research Topic:

Neurochemistry/Neurobiology

Abstract:
PURPOSE: Excitotoxicity due to glutamate receptor over-activation is one of the key mediators of neuronal death after an ischemic insult. Rapid removal of glutamate from the extracellular space by astrocytes is required for the survival and normal function of neurons. The ability of astrocytes to regulate the extracellular glutamate depends upon their hyperpolarized membrane potential conferred by the presence of K+ channels in their membranes. We have previously shown that TREK-2 potassium channels in astrocytes are upregulated by ischemia and may support glutamate clearance by astrocytes during ischemia. The purpose of the present study was to determine the mechanism leading to this upregulation. DESIGN METHODS: We used cultured cortical astrocytes and an anoxia/hypoglycemia model to simulate ischemia. Experimental protocols included RT-PCR, Western blot and whole cell patch clamp recording. RESULTS: Ischemia increased TREK-2 protein, but not mRNA, levels. This increase was reversed by protein synthesis inhibitors. Using a cell surface biotinylation assay, we determined that up-regulated TREK-2 channels were localized in the astrocytic membrane. Using whole cell patch clamp recording, we determined that TREK-2 channels in astrocytes are open/active during ischemic conditions, perhaps due to the ability of astrocytes to maintain ATP levels using anaerobic glycolysis. CONCLUSION: Taken together, these data demonstrate that TREK-2 channels are upregulated in the astrocytic membrane during ischemia through a mechanism requiring de novo protein synthesis. Furthermore, these channels in astrocytes are not inhibited by ischemia making them ideal candidates to maintain glutamate clearance during ischemia and protect neurons from excitotoxic cell death.

Supported by: SC1GM088019, G12RR03035, OADRGS Predoctoral Scholarship and Title V PPOHA grant number P031M105050 from the US Dept. of Education.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Behavioral and Phenotypic Characterization of EF-hand 2 knockout mice

Author(s):

E.N. Rodríguez Cruz, A.S. Laureano-Ruiz, J.A. Ballester, C.L Camacho, and I.E. Vega

Institutional Affiliation:

Department of Biology, University of Puerto Rico, Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
In the quest to gain insights on the mechanisms involved in tau mediated neurodegeneration, previous work from our lab identified a novel tau associated protein, EF hand domain 2 (EFhd2). EFhd2 has been found associated with pathological forms of tau in the tauopathy mouse model JNPL3 and validated in human Alzheimer's Disease and Frontotemporal dementia cases. Although EFHD2 gene is highly conserved, the physiological role of the protein is unknown. To understand the role of Efhd2 in the central nervous system an EFHD2-knockout (KO) mouse has been developed. Characterization of the EFhd2 KO mice line includes analysis of their general health and behavior. The battery of tests performed cover several domains of neurological function such as: autonomic, neuromuscular and sensorimotor function, excitability and physiological measurements. Preliminary results show that EFhd2 deficiency indoes not prevent fertility in both males and females, nor parturition or milk ejection in females analyzed. No gait abnormalities have been detected in efhd2+/- or efhd2-/- animals when compared to control efhd2+/+ animals at 16 weeks of age. We hypothesize that the EFhd2 knockout mice will develop a degenerative phenotype as it ages, as seen in tau knockout mice lines. Generation and characterization of an efhd2 knockout mouse line (efhd-/-) will provide a valuable tool to determine EFhd2's physiological role in CNS development and function. Furthermore, the development study of these mice will help us gain insights in the potential involvement of EFhd2 on the induction and/or progression of neurodegeneration.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Tau Acetylation Triggers Synaptic Dysfunction in Cultured Rat Hippocampal Neurons Related to Neurodegeneration

Author(s):

Lucelenie Rodriguez- Laureano, Tara Tracy, Li Gan

Institutional Affiliation:

University of Puerto Rico Rio Piedras Campus, Gladstone Institute of Neurological Disease, University of California in San Francisco

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Tau Acetylation Triggers Synaptic Dysfunction in Cultured Rat Hippocampal Neurons Related to Neurodegeneration. Tauopathies are a group of progressive neurodegenerative disorders, such as Alzheimerâ€™s disease and Frontotemporal dementia, characterized by abnormal intracellular inclusions of the protein tau. Aberrant acetylation of tau, induces tau aggregation, inhibits its proteasome degradation and causes deficits in AMPA receptor (AMPAR) mediated synaptic transmission; all of which likely contribute to neurodegeneration. Still, the mechanisms by how hyperacetylated tau triggers deficiencies in synaptic function are unknown. Expression of tau mutated to mimic K281 acetylation causes a reduction tendency in the frequency of miniature excitatory postsynaptic currents (mEPSCS). It was expected that the expression of K281Q tau would have caused a synaptic density decrease related to the mEPSCS reduction. A significant change was not found after assessing the number of synapses in the neurons expressing wild type and K281Q tau. Similarly, the expression of the pathogenic P301L tau in rat hippocampal neurons weakens the AMPAR mediated synaptic transmission. To further validate the hyperacetylation of the pathogenic tau mutation as a trigger of synaptic dysfunction primary rat hippocampal culture was infected with lentivirus expressing WT and P301L tau. The expression of WT and P301L tau was expected to occur in neurons rather than in glias from the rat hippocampal culture. Immunohistochemistry analysis confirmed expression of WT and P301L tau in neurons. With these and future studies we will gain more insight about how hyperacetylated tau contribute to synaptic transmission dysfunction and neurodegeneration.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

The role of *PUMILIO* on the sleep-like state of *Drosophila melanogaster*

Author(s):

N.Y.Rodriguez, J.Ortega, H. RodrÃguez, G. Diaz, F. Rivera, M. Reyes,E. Rivera, and J.L.Agosto

Institutional Affiliation:

University of Puerto Rico RÃo Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
The fruit fly Drosophila melanogaster exhibit a sleep-like state that shares many similarities with mammalian sleep and is a powerful model for understanding the genetic bases of sleep and sleep homeostasis. We have previously shown that the anticonvulsant drug carbamazepine (CBZ) dramatically decreases sleep by increasing sleep latency and decreasing sleep duration. CBZâ€™s effects on sleep latency are mediated by inhibition of a Drosophila GABAA receptor subunit called Resistant-to-dieldrin (Rdl) while its effects on sleep duration occurs via an unknown mechanism. While doing sleep deprivation studies with CBZ, we noticed that flies chronically sleep deprived with this agent over several days exhibit a daily compensatory increase in sleep that consists mainly of a decrease in sleep latency and an increase in the number of sleep episodes. In contrast, the inhibitory effect of CBZ on the duration of sleep episodes is maintained throughout the experimental period. Since CBZ acts as an antagonist of the Drosophila GABAA receptor (RDL) and chronic treatment with GABAA receptor antagonists is known to trigger neuronal homeostasis mechanisms to shut down over-activated neural circuits, we hypothesized that the sleep compensation during CBZ-induced sleep deprivation is due to the activation of neuronal homeostasis mechanisms within the sleep/wake circuit. We predicted that altering the expression of genes involved on neuronal homeostasis within the circadian circuit would disrupt sleep homeostasis. Our preliminary findings indicate that pumilio negatively regulate sleep duration and participates on sleep compensatory processes. These results indicate that pumilio plays an important role on sleep regulation and suggest neuronal homeostasis mechanisms within the circadian network regulate sleep duration and homeostasis.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

MIGROGLIA INCREASE GLIOMA CELL Pyk2 SIGNALING AND INVASION

Author(s):

K Rolon; M Mendez; DE Rivera Aponte; SN Skatchkov; MJ Eaton; LY Kucheryavykh

Institutional Affiliation:

Universidad Central del Caribe, Bayamon PR

Research Topic:

Neurochemistry/Neurobiology

Abstract:
MIGROGLIA INCREASE GLIOMA CELL Pyk2 SIGNALING AND INVASION

PURPOSE: Glioblastoma multiforme is one of the most aggressive and fatal brain cancers mostly because of the ability of glioma cells to disperse through the healthy brain stroma what makes surgical and chemotherapeutic treatments mostly ineffective. Microglia infiltrate most gliomas and release factors, which favor tumor growth and dispersal. We hypothesize that microglia stimulate migration of glioma cells through the phospholipase C (PLC)gama1 and proline rich tyrosine kinase 2 (Pyk2) signaling cascade. DESIGN METHODS: We investigated three different human glioma cell lines with varying levels of invasiveness: A172, U-87MG and HS683. Experimental protocols included Western blot, invasion assays and RNA interference. RESULTS: Using Western blot, we demonstrated that treatment of glioma cells with microglia conditioned medium upregulated phosphorylation of Pyk2 protein at Tyr579/580 in all glioma cell lines. Also, using a standard Boyden chamber invasion assay, we demonstrated that microglia significantly activated invasion of glioma cells, and this effect was reversed after knock-down of PLCgama1 and Pyk2 proteins using siRNA in glioma cells. Pharmacological blockers of PLCgama1 (U73872 1nM) and Pyk2/focal adhesion kinase (FAK) (PF562271 20nM) also completely eliminated the ability of microglia to stimulate glioma cell migration. CONCLUSION: Taken together, these data indicate that microglial cells activate glioma cell migration/dispersal through the pro-migratory Pyk2 and PLCgama1 signaling pathway in glioma cells.

Research Support: NIH-NICHD-G11-HD052352, NIH-NCRR-G12-RR03035, NIH-NIMHD-8G12-MD007583, NIH-NINDS-U54-NS039408, Universidad Central del Caribe Pilot Project Program and Title V PPOHA grant number P031M105050 from the US Dept. of Education.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Characterization of enteric system labeling monoclonal antibody the Holothuria glaberrima

Author(s):

Jean F. Sáenz, Griselle Valentín, Jose García Arrarás

Institutional Affiliation:

University of Puerto Rico Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Echinoderms are a phylum of great interest given their status as deuterostomes and close precursors of the chordates. Our laboratory uses the echinoderm Holothuria glaberrima as a model for studying the organization and regeneration of the echinoderm nervous system. One of the main hurdles in this study is the availability of cell markers that can identify specific cell and fiber populations. Monoclonal antibodies provide useful markers to identify specific cell populations. In order to obtain echinoderm cell markers, we injected mice with a intestinal luminal cell homogenate. One of the antibodies obtained was named EN1 (Enteric Nervous-1). EN1 labels cells and fibers of the holothurian intestine that appear to be neuronal in nature. The labeling is restricted to the enteric nervous system and cannot be found in the radial nerves. The labeled cells are found in both epithelial layers and in the connective tissue. Similar resutls were found in other holothurian species. This antibody provides an exceptional tool to classify distinct populations of enteric neurons. In turn, we will be able to obtain a better picture of the holothurians nervous system and its regeneration.

Notes:
Not interested in short presentation.

Title:

The anabolic steroid 17α–methyltestosterone modulates cancer-related proteins in the hypothalamic GT1-7 cell line.

Author(s):

[1]**Santigo-Gascot ME**, [1]Martínez-Rivera F, [2]Sosa J, [3]Pérez-Laspiur J, [3]Rodríguez-Pérez Y, [1]Barreto-Estrada JL.

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Research Topic:

Neurochemistry/Neurobiology

Abstract:
The anabolic steroid 17α–methyltestosterone modulates cancer-related proteins in the hypothalamic GT1-7 cell line.

Alarming reports estimate that adolescents and adults are using supraphysiological doses of anabolic androgenic steroids (AAS) to increase muscle size and induce strength gains through their anti-catabolic / anabolic effects. However, in the last years a diverse spectrum of neuroendocrine disorders including cancer disease of the reproductive system has been reported. The main goal of this study was to determine the cellular mechanisms of AAS in the neuroendocrine system using the immortalized hypothalamic GT1-7cell line through the protein profile of AAS versus vehicle-treated samples. Cells were grown to confluence in steroid-free serum. Afterward, these cells were exposed to the AAS 17α–methyltestosterone (1μM) for 48 hours. Proteomic approaches including one- and two-dimensional difference in gel electrophoresis (2D-DIGE) and mass spectrometry were used. Twelve proteins were found to be differentially expressed: 7 up-regulated and 5 down-regulated. Ontological analysis showed that a large proportion of these proteins are related to cancer and tumor development. Among the identified proteins are: stress-induced phosphoprotein 1, heat shock protein 90B and phosphatidylethanolamine - binding protein. Interaction between these proteins was confirmed by pathway analysis. Further experiments will be validated by western blots. **This project was supported in part by grants from NIH: NCRR (2G12-RR003051) NIMHD (8G12-MT007600), NCRR (2P20RR016470-12) and NIGMS (8P20 GM103475-12).**

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Ethanol Exposure Reduces Synaptic Enrichment of the BK Channel in Hippocampal Neurons

Author(s):

Garrett E. Seale, Jose Garcia, Stephanie Palacio, Guillermo Yudowski, Steven N. Treistman

Institutional Affiliation:

Institute of Neurobiology - University of Puerto Rico

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Research has shown that alcohol tolerance is a major predictor of increased rates of alcohol consumption, potentially leading to alcoholism. Previous studies from our lab have demonstrated that large conductance calcium- and voltage-activated potassium (BK) channels are potentiated by acute ethanol exposure, while longer durations of ethanol exposure result in molecular adaptations to the channel, altering neuronal sensitivity to ethanol. Thus, the BK channel may play a key role in alcohol tolerance, bridging casual alcohol use to severe alcohol abuse. As prior research has shown, we observed enrichment of the BK channel alpha subunit at synapses in cultured hippocampal neurons. Ethanol exposure (1hr, 6hrs, 24hrs) reduced synaptic enrichment of the BK channel alpha subunit. Interestingly, synapses on distal dendrites exhibited greater reduction of BK channel localization within synaptic compartments as compared to synapses on proximal dendrites. These effects of ethanol exposure on BK channel localization were specific to synaptic compartments as measurements of BK channel immunofluorescence along dendritic and somatic areas showed no significant difference between control- and ethanol-treated cultures. In addition, immunoblot experiments on hippocampal culture lysates showed only a moderate decrease of total BK channel alpha subunit protein after 6hrs of ethanol exposure, while 1hr and 24hrs of ethanol exposure did not lead to significant changes in total BK channel alpha subunit levels. Regulation of the BK channel at synapses by alcohol may have profound effects on synaptic communication between neurons in networks that are critical to learning and memory.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Metabotropic glutamate receptor activation is necessary for synaptic plasticity in the infralimbic cortex during fear extinction

Author(s):

M. T. SEPULVEDA-ORENGO[1], G. J. QUIRK[2], J. T. PORTER[1]

Institutional Affiliation:

[1]Ponce School of Medicine and Health Sciences., Ponce, PR; [2]University of Puerto Rico School of Medicine, San Juan, PR

Research Topic:

Neurochemistry/Neurobiology

Abstract:
It is known that IL activation is necessary for the fear extinction memory. Recent work in our laboratory showed that extinction memory requires type 5 metabotropic glutamate receptor (mGluR5) activation. These results suggest that mGluR5 stimulation may lead to IL plasticity needed for fear extinction. We previously showed that fear extinction increases the AMPA/NMDA ratio (SFN 2009, Abstract # 880.6) consistent with insertion of AMPA receptors into IL synapses. Moreover, neurons from extinction-trained rats showed increased AMPA inward rectification compared with neurons from the conditioning group. This suggests that fear extinction induces a switch from AMPA GluR2-containing (calcium impermeable) to AMPA GluR2-lacking (calcium permeable) receptors in IL synapses. To determine whether mGluR5 activation mediates these changes, we measured AMPA and NMDA currents in IL pyramidal neurons in slices from trained rats using whole-cell patch-clamp. On day 1, all rats received 3 tone-shock pairings. On day 2, rats received an intraperitoneal (IP) injection of the mGluR5 blocker MPEP or vehicle 30 minutes before extinction training (15 tone presentations). On day 3, vehicle and MPEP rats were sacrificed immediately after testing fear expression (2 tone trials). Replicating our earlier finding, blockade of mGluR5 prior to fear extinction training with MPEP impaired extinction memory. Furthermore, mGluR5 inactivation prevented the extinction-induced increases in AMPA/NMDA ratio and rectification index. Taken together, these findings suggest that mGluR5 activation leads to consolidation of fear extinction by modifying the composition of AMPA receptors in IL synapses via the insertion of GluR2-lacking AMPA receptors.

21st PR Neuroscience Conference
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December 1st, 2012

A B S T R A C T

Author(s):

Edgardo A. Castro-Perez, EMILIO E. Soto Soto, Dawling A. Dionisio-Santos, Kristian Saied-Santiago, Marizabeth Perez-Carambot and Sandra PeÃ±a de Ortiz.

Institutional Affiliation:

University of Puerto Rico, Rio Piedras Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:
We assess here the importance of genomic rearrangement mechanisms in consolidation of long-term memory. Our hypothesis is that long-term memory formation involves a regulatory mechanism of DNA rearrangement in the brain. We focus our research on DNA recombination machinery, which includes the activation of effector molecules, such as endonucleases, DNA repair factors and DNA ligases. Using context fear conditioning (CFC) as a model of learning in mice, our main goal is to identify molecular factors and changes associated to DNA recombination and their impact on the hippocampus and the amygdala during memory formation. Our previous studies using pharmacological blockade of DNA ligase function, showed an impairment of consolidation of context fear conditioning without interfering short-term memory or reconsolidation. In this research, DNA microarray experiments lead us to preliminary identify the recombination-activating gene 1 (Rag1) as a DNA recombination factor involved in LTM formation in CFC. Here, we report preliminary data indicating that Rag1 mRNA is induced in the amygdala during CFC, thus validating microarrays data. The induction seems to be specific to the conditioned animals, because contex-only and shock-only controls did not show significant differences from naÃ¯ve animals. Preliminary experiments using immunohistochemistry and Western blots also indicate Rag1 protein induction. Our ongoing experiments are lead to validate Rag1 induction at the protein levels by immunohistochemistry and westernblot. In conclusion, our preliminary results support our hypothesis on the role of DNA recombination and repair pathways during memory formation. Further studies will assess the significance of Rag1 in memory consolidation during CFC.

Notes:
I would like to be considered for oral short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Autoantibody profiling for candidate biomarkers of Alzheimer's disease and related tauopathies

Author(s):

J. VAQUER-ALICEA, C. Nogueras-Ortiz, I.E. Vega

Institutional Affiliation:

Univ. of Puerto Rico-Rio Piedras Campus, San Juan, PR

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Alzheimer's disease (AD) is the most common form of dementia affecting approximately 35.6 million people worldwide. AD and many other neurodegenerative diseases, collectively known astauopathies, share a common pathological hallmark: neuronal accumulation of abnormal fibrillar tau protein deposits. One of the greatest challenges in treating patients with AD and relatedtauopathies is the lack of objective and non-invasive diagnostic tools. Surprisingly, biomarkers for diagnosis prior to cognitive decline, when current therapy is likely to have the greatest impact, are currently not available. Recent studies have highlighted the role of the adaptive and innate immune response in neurodegenerative diseases such as AD. These findings suggest the possibility of the use of auto-antibodies as biomarkers for these maladies. The present study undertakes an autoimmune biomarker profiling approach using the JNPL3 tauopathy mouse model bearing the human Tau gene with a P301L mutation. A quantitative immunoblotting approach has been carried out leading to the detection of differentially expressed antigens from mouse brain homogenates recognized by antibodies in the blood sera of the JNPL3 transgenic mice. Subsequent tandem mass spectrometry and bioinformatic analyses will be executed for identification of targeted antigens. Our findings may provide an essential stride in the discovery of specific biomarkers for the diagnosis of tauopathies.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Ganglia, Nuclei and Fiber Tracts in the Echinoderm Nervous System?

Author(s):

Lionel D Vazquez-Figueroa[1], Carlos A Diaz-Balzac[2], Jose E Garcia-Arraras[1]

Institutional Affiliation:

University of Puerto Rico - Rio Piedras Campus[1]

Albert Einstein College of Medicine[2]

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Echinoderms are nonchordate deuterostomes that lie in a key position in the evolutionary tree of vertebrate animals. However, the echinoderm nervous system has not been studied in detail, probably due to a lack of markers that serve to recognize the nervous system components. Our group has developed several markers that recognize neurons, glia and fibers of the nervous system of the sea cucumber Holothuria glaberrima. We now extend our studies to show that most of these markers also serve to recognize neural system components in other echinoderms. We have used indirect immunofluorescence with several antibodies against neurotransmitter systems, calcium binding proteins, transcription factors, or still undetermined epitopes specific to nerve cells, to label neurons and fibers in four different holothurian species: H. glaberrima, H. mexicana, Sclerodactyla briareus and Leptosynapta clarki. Our results show that each antibody labels distinct and specific distributions of fibers and nerve cells in the holothurian radial nerve cords (RNC). Based on this data, our group has begun to characterize the regeneration of injured RNC in H. glaberrima. Further characterization of fiber and cellular distribution is expected to provide a cohesive and comprehensive map of the echinoderm nervous system and its regenerative processes, thus providing critical information for cellular, molecular and evolutionary studies of the deuterostome nervous system.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

***In vitro* phosphorylation of the novel tau-associated protein EFhd2 by CDK5/p25**

Author(s):

Edwin F. Vázquez-Rosa[1,4], Lucelenie Rodríguez-Laureano[3], Tiffany J. Rios-Fuller[2] and Irving E. Vega [3,4]

Institutional Affiliation:

[1]Department of Chemistry, University of Puerto Rico-Rio Piedras,[2] Department of Biology, Universidad Metropolitana, [3]Department of Biology, University of Puerto Rico-Rio Piedras [4]Protein Mass Spectrometry Core Facility, Department of Biology, University of Puerto Rico-Rio Piedras

Research Topic:

Neurochemistry/Neurobiology

Abstract:
EFhd2 protein was first identified in cells of the immune system, but EFhd2 is more abundant in the central nervous system. Previous studies indicated that EFhd2 is associated to pathological forms of the microtubule-associated protein tau in tauopathy animal models and Alzheimer's disease (AD) patients. However, the pathophysiological role of EFhd2 in neurodegeneration is unknown. One important molecular event associated to tau-mediated neurodegeneration is the unregulated activation of kinases that lead to the hyperphosphorylation of tau proteins. A known tau kinase associated to neurodegeneration is the CDK5/p25 complex. In vitro studies were conducted to determine if the CDK5/p25 kinase complex also phosphorylate EFhd2. Biomass analysis revealed the addition of one phosphate group (~79.9 amu) to the molecular mass of HIS-EFhd2 protein. Additionally, we exposed EFhd2 to brain extracts of JNPL3 and CKP25 mouse model and observed the phosphorylation of EFhd2. This phosphorylation could be decreased to background levels using Roscovitine, a potent inhibitor of CDK5, suggesting specificity of this kinase for EFhd2. Tandem mass spectrometry analysis and point mutants were used to identify the site where CDK5/p25 complex phosphorylates HIS-EFhd2 protein. The results indicated that CDK5/p25 phosphorylates EFhd2 on a serine residue at position 74 (S74). The results demonstrated that EFhd2 is a substrate of the kinase complex CDK5/p25 in vitro. CDK5/p25 complex is known to play an important pathological role in tau-mediated neurodegeneration. Thus, the results suggest that the CDK5/p25-mediated phosphorylation of EFhd2 may be an important pathological event in neurological disorders, such as AD.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Ciliary neurotrophic factor (CNTF) affects the speed and number of regenerating axons after optic nerve injury.

Author(s):

G. S. VEGA MELÉNDEZ[1,2], J. M. BLAGBURN[1], R. E. BLANCO[1,2]

Institutional Affiliation:

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Research Topic:

Neurochemistry/Neurobiology

Abstract:
Ciliary neurotrophic factor (CNTF) promotes survival and axonal regeneration after neuronal damage. In the present study we investigate the effects of exogenous application of CNTF on the speed, number and distribution of regenerating axons after optic nerve crush. We are interested in comparing the relative efficacy of this factor when compared with other growth factors like brain-derived neurotrophic factor (BDNF) and basic fibroblast growth factor (FGF-2); and also in determining whether applications intraocularly or to the optic nerve have different effects. To that end we performed optic nerve crush and applied either saline solution or growth factors (CNTF, BDNF or FGF-2) and examined the optic nerves at one, two and three weeks after axotomy. An antibody against GAP-43 protein was used as a marker for regenerating axons. We performed measurements of the length, number and density of axons projecting beyond the lesion. All three factors, whether applied to the optic nerve or intraocularly, significantly enhanced the speed of the regenerating axons, with CNTF doubling the speed of regeneration. The number of regenerating axons at two weeks was also significantly increased by CNTF and FGF-2, but not BDNF. All the effects described above are inhibited by the appropriate receptor-blocking antibodies. We conclude that FGF-2 and CNTF, in particular the latter, play an important role in optic nerve regeneration in the amphibian visual system and we need to further understand the mechanisms of these effects. This work was supported by NIH-GM093869, RCMI-G12 RR03051, NSF-DBI-0959225, and NSF-DBI-0115825.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Unique morpho-physiological characteristics of K^+-channels in retinal Müller cells from the Spectacled caiman.

Author(s):

***A. ZAYAS-SANTIAGO[1],** S. AGTE[2], L. ZUEVA[1], A. SAVVINOV[3], J. BENEDIKT[1], Y. KUCHERYAVYKH[1], Y. RIVERA[1], L.A. CUBANO[1], A. REICHENBACH[2], M.J. EATON[1], S.N. SKATCHKOV[1]

Institutional Affiliation:

[1]Univ. Central Del Caribe, Bayamón, Puerto Rico; [2]Paul Flechsig Inst. of Brain Res., Leipzig Univ., Leipzig, Germany; [3]Univ. de Puerto Rico-Recinto de Río Piedras, Río Piedras, Puerto Rico

Research Topic:

Neurochemistry/Neurobiology

Abstract:
The Spectacled caiman is a unique animal that develops a complex retina and has perfect night and day vision with a large scale of light adaptation. The Müller glial cells in the caiman's retina have developed unique properties. In this study, we describe a pattern of potassium channels that raises doubt about the theory of K^+-buffering. Potassium channel expression in caiman Müller cells was tested by: immunocytochemistry, electrophysiology, and pharmacology. In contrast to most vertebrates, Kir4.1 and Kir6.1 inwardly rectifying potassium channels were not labeled in caiman Müller cells immunocytochemically. The I/V-curves recorded from vitreal endfeet of Müller cells in retinal whole mounts as well as from enzymatically isolated Müller cells demonstrated mostly outwardly rectifying K^+ currents matching behavior of 2P-domain pore (2P) channels. Indeed, markers against 2P-channels TASK-1, -2, and -3, were localized in Müller cells. In addition, cell currents were insensitive to 10-100 µM barium (a key blocker of Kir channels), but sensitive to bupivacaine, a blocker of TASK channels. This is different from Müller cells of other vertebrates in which robust Kir -type inward currents are found. Moreover, the currents in caiman Müller cells were weak in response to increased external potassium, while showing robust outward currents in response to decreased external potassium concentration. This suggests that in the caiman retina 2P-channels may play a major role in glial K^+ homeostasis; however, it does not fit the classical idea of K^+-spatial buffering where Kir channels play the major role.

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A B S T R A C T

Author(s):

CARLOS J. NOGUERAS-ORTIZ and Irving E. Vega

Institutional Affiliation:

University of Puerto Rico, Río Piedras Campus.

Research Topic:

Neurochemistry/Neurobiology

Abstract:
Tauopathies are a group of neurodegenerative diseases that present cognitive and/or motor deficits associated to modifications and aggregation of the microtubule-associated protein tau. Among several altered cellular events associated with tau burden lies synaptic dysfunction which best correlates with cognitive deficits. However, the mechanistic relationship between pathological tau and synaptic dysfunction is still poorly understood. A proteomics approach directed to identify proteome changes in the course of neurodegeneration indicated that the amount of the synaptic protein amphiphysin-1 (AMPH-1) is significantly reduced in tauopathy. AMPH-1 has been demonstrated to be essential for synaptic vesicle endocytosis thus promoting the maintenance of proper neural function. Using a tauopathy mouse model (i.e. JNPL3 mice) and AD post-mortem brains, we found that AMPH-1 protein level reduction occurs by means of proteolysis in neurons without signs of degeneration. We observed a positive correlation between the AMPH-1 depletion and positive indicators for the hyperactivation of calpain proteases, thus suggesting that AMPH-1 is a targeted protein in the process of tau-mediated neurodegeneration. In order to test the hypothesis of a putative involvement of AMPH1 in the pathobiology of tauopathy, we evaluated the presence of anti-AMPH-1 antibodies in blood as a biomarker. Results indicate that antibodies recognizing AMPH-1 are increased in the blood serum of the JNPL3 mouse model when compared to non-transgenic age-matched mice. The study of AMPH-1 depletion in tauopathies suggests that pathological tau has a direct effect on synaptic deregulation and provides the identification of a potential biomarker that could serve as a diagnostic tool.

Notes:
I would like to be considered for oral short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:
Clathrin coated pit lifetime is regulated by cargo activation

Author(s):
JACQUELINE FLORES-OTERO,Ph.D.; Guillermo A. Yudowski, Ph.D.

Institutional Affiliation:
UPR-RCM, Institute of Neurobiology

Research Topic:
Neurochemistry/Neurobiology

Abstract:
*Agonist-induced internalization is an essential component of G protein-coupled receptor biology. Agonist activation of the cannabinoid receptor (CB1R) induces GRK3 and Î²-arrestin2-mediated desensitization and endocytosis. However, it remains unknown if the kinetics of this process is regulated. To investigate whether the internalization kinetics of CB1R is selectively regulated by specific agonists, we examined the effect of CB1R ligands, WIN, 2AG, THC and Rimonabant in transfected HEK293 cells and in hippocampal neurons. We also identify the molecular mechanism mediating CB1R internalization. CB1R endocytic lifetime was investigated by using live-cell total internal reflection fluorescence microscopy (TIRFM). Our results showed that in cells treated with WIN or 2AG, there were two distinct endocytic populations. Cells that were treated with WIN displayed shorter endocytic lifetime (88.5 seconds) while cells that were treated with 2AG displayed longer lifetime (151.5 seconds). We also found that THC and Rimonabant had little or no effect in endocytic pit formation. To test if these endocytic lifetimes are mediated by different molecular mechanisms, we co-transfected HEK293 cells and hippocampal neurons with super-ecliptic pHluorin (SEP)-CB1 receptor and ds-red-clathrin or with SEP-CB1 receptor and mCherry-Caveolin. After agonist treatment, we identified that CB1R internalization, was mediated by clathrin-coated pits (CCPs) and not by Caveolin. Taken together, these findings suggest that the endocytic lifetime of CCPs is differentially regulated by the activation state of the cargo, beyond the receptor or cell type as reported by other studies.Our work provides a molecular framework to understand the interplay between receptor trafficking and biological tolerance and dependence. *

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A B S T R A C T

Author(s):

N. Delgado, **D. I. Vallejo(presenter),** and M.W. Miller 1,2

Institutional Affiliation:

Institute of Neurobiology, University of Puerto Rico Medical Science Campus

Research Topic:

Neurochemistry/Neurobiology

Abstract:

The digenetic trematode Schistosoma mansoni that causes the form of schistosomiasis found in the Western Hemisphere requires the freshwater snail Biomphalaria glabrata as its primary intermediate host. It has been proposed that the transition from the free-living S. mansoni miracidium to parasitic mother sporocyst depends on uptake of biogenic amines, e.g. serotonin, from the snail host. However, little is known about potential sources of serotonin in B. glabrata tissues. This investigation examined the localization of serotonin-like immunoreactivity (5HTli) in the central nervous system (CNS) and peripheral tissues of B. glabrata. Emphasis was placed on the cephalic and anterior pedal regions that are commonly the sites of S. mansoni miracidium penetration. The anterior foot and body wall were densely innervated by 5HTli fibers but no peripheral immunoreactive neuronal somata were detected. Within the CNS, clusters of 5HTli neurons were observed in the cerebral, pedal, left parietal, and visceral ganglia, suggesting that the peripheral seroto- nergic fibers originate from the CNS. Double-labeling experiments (biocytin backfill □ serotonin immunoreac- tivity) of the tentacular nerve and the three major pedal nerves (Pd n. 10, Pd n. 11, and Pd n. 12) disclosed central neurons that project to the cephalopedal periphery. Overall, the central distribution of 5HTli neurons suggests that, as in other gastropods, serotonin regulates the locomotion, reproductive, and feeding systems of Biomphalaria. The projections to the foot and body wall indicate that serotonin may also participate in defensive, nociceptive, or inflammation responses. These observations identify potential sources of host-derived serotonin in this parasite–host system. J. Comp. Neurol. 000:000–000, 2012.

Note:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Author(s):

Didiana Cruz-Lopez; Dianne Ramos- Santiago & Thomas Schikorski PhD

Institutional Affiliation:

Universidad Central del Caribe

Research Topic:

Neurochemistry/Neurobiology

Abstract:
For years, electron microscopists have been on the search for a reporter gene for electron microscopy that has the potential to match the breakthrough of green fluorescent protein (GFP) for light microscopy. Horseradish peroxidase (HRP) has been applied sporadically but was hampered by the lack of reliability when expressed in mammalian cells, particularly in mammalian neurons. We used HRP mutants with elevated enzyme activity and tested their functionality in neuroblastoma cells. A small number of promising mutants were further evolved to optimize their use for HRP expression in mammalian neurons. One HRP variant (eHRP) has better sensitivity when compared to eGFP in the light microscope and provides unambiguous labeling in the electron microscope while the ultrastructure is preserved at the highest standard. This mutant is reliably detected in all mammalian cells tested including neuroblastoma cells and primary hippocampal cell cultures. Next, we tested whether protein chimeras based on HRP can be built to target HRP to various cell-organelles. We developed a HRP chimera with synaptobrevin(neuroHRP) which targets HRP to the lumen of synaptic vesicles. When expressed in cultured hippocampal neurons and in the CNS in vivo neuroHRP is reliably detected in the light and electron microscope. In summary, eHRP and neuroHRP are outstanding candidates for a reporter gene for electron microscopy in all areas of cell biology. More so, they are exciting new tools for presynaptic research and an advanced label for modern electron microscopy.

Notes:
I would like to be considered for oral short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Author(s):

C. BRAVO-RIVERA, C.ROMAN-ORTIZ, E. BRIGNONI-PEREZ, G. J. QUIRK;

Institutional Affiliation:

Depts. of Psychiatry and Anat. & Neurobio., Univ. Puerto Rico Sch. Med., San Juan, Puerto Rico

Research Topic:

Neurochemistry/Neurobiology

Abstract:
We recently developed a simple avoidance task in which rats, trained to press a bar for food, learned to avoid a tone-signaled footshock by stepping onto a nearby platform (Bravo-Rivera et al, 2011, SFN). Remaining on the platform for the duration of the 30 sec tone protected them from the shock, but also prevented their access to food. Here we used pharmacological inactivation to address the neural circuitry involved. After 10 days of avoidance training, rats were infused with saline or GABAA agonist muscimol in the prelimbic cortex (PL), basolateral amygdala (BLA), ventral hippocampus (vHPC), or nucleus accumbens core (NAc), and tested for avoidance. Inactivation of each of the four structures blocked avoidance responses, which returned the following day when tested drug-free (pâ€™s 50% compared to the previous day. Loss of avoidance with a concurrent increase in freezing suggests that inactivation eliminated the avoidance program. Our results suggest that BLA and vHPC contribute an essential fear signal, which drives avoidance. In contrast, PL and NAc contribute an avoidance program which inhibits freezing, perhaps through the CeL (Lázaro-Muñoz et al, 2010). In the absence of PL and NAc, rats resort to freezing as an auxiliary defense mechanism.

Notes:
I would like to be considered for oral short presentation.

Neurodevelopment

ND1 – Castro, W.
ND2 - Colón, L.R.
ND3 – Ferchmin, P.A.
ND4 – Mashanov, V.S.
ND5 – Quiñones-Frías, M.

21st PR Neuroscience Conference
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December 1st, 2012

A B S T R A C T

Title:

NEUROPROTECTION BY 4R-CEMBRANOID EVALUATED 24H AND 7 DAYS AFTER ISCHEMIC STROKE

Author(s):

W. Castro, A.H. Martins, G. Grudziak, P.A. Ferchmin, V. Eterovic

Institutional Affiliation:

Universidad Central del Caribe

Research Topic:

Neurodevelopment

Abstract:

Stroke is one of the leading causes of disability in adults. Neuroprotective compounds that increase the opportunity of recovery have yet to be described. 4R-cembranoid (4R), a compound isolated from tobacco, displays neuroprotective activity against different types of insults to the brain, including the neurodegeneration induced by organophosphate, by the HIV-1 virus and by ischemic events. This work studied the effect of 4R on the neurological damage induced by ischemic stroke in rats, using electrophysiological and histological methods. Methods: Male Sprague Dawley rats were utilized as a stroke model using transient middle cerebral artery occlusion (MCAO) on the left hemisphere. Two hours after MCAO, the rats received a single injection of 4R (6mg/kg, sc) or vehicle (DMSO). Somatosensory evoked potentials were measured at various times before and after MCAO. To assess the acute effects of 4R, rats were euthanized 24 hours after MCAO and the infarct area was determined by staining the tissue tiht 2, 3, 5-triphenyltetrazolium chloride. In order to evaluate the long term neuroprotection of 4R, rats were euthanized 7 days after the injury by perfusion withparaformaldehyde. Histology assays were performed on 40 µm thick brain slices to assess neuronal damage and repair. Results: Twenty four hours after the insult, infarct volume was 50% smaller in animals injected with 4R than in vehicle controls. The 4R animals have also lost less body weight. The amplitude of somatosensory evoked potentials recorded from the ipsilateral hemisphere was significantly decreased after MCAO by comparison with pre-operation values, both in 4R and control groups. In the contralateral hemisphere, both groups displayed an increase in amplitude but that increase was larger in the 4R group. Seven days after MCAO, hematoxylin and eosin staining

revealed an enlarged infarct area infiltrated with leucocytes. Nestin and GFAP positive cells were detected surrounding the infarct area, apparently unable to cross over the lesion. Inside the lesion, was detected the presence of CD68 positive cells, showing microglia and macrophage migration. No significant difference was detected between 4R and vehicle groups. Thus, 4R showed promising neuroprotective properties when measured one day after reperfusion but no improvements were detected after 7 days following a single dose of 4R. This could be due to 4R fast metabolism and excretion from the body. Conclusion: 4R is a promising neuroprotective compound whose long-term effects should be evaluated using multiple injections.

Support: **SNRP Grant 3235-10-AB; Bromedicon Inc.; U54RR022762; Fundacion Segarra**

Notes: **I would like to be considered for oral short presentation.**

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Swimming behaviors in larva and adult zebrafish in the absence of a functional lateral line

Author(s):

Luis R. Colon

Institutional Affiliation:

RCM

Research Topic:

Neurodevelopment

Abstract:

Zebrafish is increasingly becoming a popular animal model to assess subletal effects of aquatic pollution in vertebrates. We are focusing on the mechanosensory organ, which is called the lateral line (LL). This superficial organ is exposed to surrounding waters and consists of stereotypically distributed sensory patches called neuromasts. Each neuromast is composed of centrally located hair cells (HC), and neighboring supporting cells. The function of the LL is to detect water movements, which is important for catching prey, escaping predators, socializing, and rheotactic behavior (stabilization in currents). HC are well known for their sensitivity to a wide array of compounds, therefore providing a putative sensitive readout for water quality. In an effort to link defects in the LL to detectable altered behaviors, we standardized a series of simple behavioral tests in larvae and adult zebrafish, in which the LL has been completely ablated. General and specific swimming behaviors, like the startle response were monitored and quantified in individual and group of fish. We will present our approach and results obtained so far. This work will establish standards, which will be subsequently used for the assessment of swimming behaviors of animals which have been exposed to water samples collected in local rivers, therefore providing a rapid readout of water quality and of its potential repercussion on behaviors mediated via the LL in endogenous species.

21st PR Neuroscience Conference
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December 1st, 2012

A B S T R A C T

Title:

DFP EXCITOTOXICITY AND ITS REVERSAL BY 4R-CEMBRANOID IN THE ACUTE HIPPOCAMPAL SLICES

Author(s):

P.A. Ferchmin, D. Perez, B.L. Cuadrado, M. Carrasco, A.H. Martins, V.A. Eterovic

Institutional Affiliation:

Universidad Central del Caribe

Research Topic:

Neurodevelopment

Abstract:

There is a need to better understand the neuropathology caused by organophosphates (OPs) and to develop new therapies. This effort is still is hampered by an incomplete understanding of the brain pathways responsible for Ops neurotoxicity. It is generally accepted that Ops toxicity is primarily due to inhibition of acetylcholine esterase (AChE) and the consequential buildup of acetylcholine (ACh). However, numerous non cholinergic effects of Ops have been observed. This work was done to study the mechanism of Ops mediated neurotoxicity in conditions in which the effect of AChE inhibition was minimized. We used diisopropylfluorophosphate (DFP) as a surrogate of the chemical war nerve agent sarin. To isolate aspects of DFP neurotoxicity where the direct effect of AChE inhibitoni and ACh accumulation is minimized, we measured the population spikes (PSs) in the acute hippocampla slices. In this preparation most of the soluble AChE is washed away and after DFP application there is only a limited capacity for ACh build up. In these conditions we studied the neuroprotection byt the novel neuroprotectivecompound the 4R-cembranoid (4R). DFP inhibited AChE activity (IC50, 39.2 Î¼M). The 50-fold discrepancy between the two IC 50s suggested that DFP effect on the PS is unrelated to the inhibition of AChE. DFP effect on PS was prevented by APV, a reversible inhibitor of the N-methyl-D-aspartate (NMDA) receptor. This shows that DFP neurotoxicity was mediated byexcitotoxicity. Since, in our preparation, excitotoxicity triggers neuronal apoptosis, we queried whether DFP effect on PS was mediated by apoptosis. The slices were exposed to LEHD-CHO, a cell permeable caspase 9 inhibitor, which protected the PS from DFP toxicity when applied immediately before or after DFP. Therefore, the PS decrease was related to apoptosis. 4R reversed DFP toxic effect

on the PS when applied 30 min after DFP (EC50, 63nM). We have shown previously that 4R protected the PS from excitotoxicity triggered by NMDA by activationAkt/PKB and a pathway that included inhibition of GSK-3Î². Based on these observations, we propose that DFP over activates the NMDA receptors causing excitotoxicity. The role of AChEinhibition, ACh accumulation, and muscarinic rectpro activation in this preparation is still not clear and under investigation. Considering that 4R was shown by us to be neuroprotectiveagainst excitotoxicity it is not surprising that it protects against DFP. The acute hippocampal slice is a promising preparation to study mechanisms of OP toxicity and testing ofneuroprotective drugs.

Support: NIH Grant 1U01NS063555; NIH Grant RCMI 2G12RR03035.

Notes: **I would like to be considered for oral short presentation.**

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Transcriptome analysis of the post-traumatic neurogenesis in an echinoderm

Author(s):

Vladimir S. Mashanov, Olga R. Zueva, Jose E. Garcia-Arraras

Institutional Affiliation:

Department of Biology, University of Puerto Rico, San Juan, PR

Research Topic:

Neurodevelopment

Abstract:
Echinoderms emerge as a promising model system in regenerative biology. First, they are capable of efficient regeneration of virtually any part of their body, including the central nervous system. Second, they occupy a unique phylogenetic position among basal deuterosomes and are closely related to chordates. The combination of these two features makes studies on echinoderms highly relevant both for understanding the evolutionary conserved fundamental mechanisms of neural regeneration and for informing more applied biomedical research. There is a growing body of data on the basic cellular events in neural regeneration in echinoderms, but molecular processes have not been studied at all. In this work, we characterise the transcriptome of the regenerating central nervous system of the sea cucumber Holothuria glaberrima using massively parallel RNA-seq techniques, de novo transcriptome assembly, and gene expression profiling. Our findings show that regeneration in these animals involves large-scale changes in gene expression (4023 and 3257 up-regulated and down-regulated transcripts, respectively), with the highest number of differentially regulated genes being detected at the early-post injury stage. Functional annotation revealed somewhat paradoxical pattern of gene regulation, which is exemplified in concurrent occurrence of opposite processes, such as up-regulation of pro- and anti-apoptotic genes, breakdown and stabilization of extracellular matrix, promotion and suppression of dedifferentiation, positive and negative regulation of cell motility. These data illuminate the complexity of neural regenerative events even in an organism with a relatively simple organization of the central nervous system. **Financial support: NIH (1SC1GM084770-01,1R03NS065275-01), NSF (IOS-0842870), University of Puerto Rico.**

Psychology Behavioral Sciences

PB1 – Cepeda-Rivera, K.M.
PB2 – Chorna, N.
PB3 – Cruz, E.
PB3 – De Jesús-Burgos, M.I.
PB4 – Do Monte, F.H.M.
PB5 – Feliz-Ortíz, A.
PB6 – Giannoni-Guzmán, M.A.
PB7 – Hernández, L.
PB8 – Jimenez, G.L.
PB9 – Maldonado-Catala, P.J.
PB10 – Martínez-Sosa, F.
PB11 – Pérez-Torres, E.M.
PB12 – Ramos-Pratts, K.M.
PB13 – Rivera-Rodríguez, M.
PB14 – Rodriguez-Romaguera, J.
PB15 – Santiago, S.
PB16 – Bermudez-Cruz, N.D.

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Characterization of the holothurian LIM domain binding protein 1 (Ldb1) and its expression during intestinal regeneration.

Author(s):

Mónica C. Quiñones-Frías, Jaime Flores and José E. García-Arrarás

Institutional Affiliation:

Department of Biology, University of Puerto Rico, Río Piedras

Research Topic:

Organogenesis and Development

Abstract:
Holuthuria glaberrima **is an echinoderm known for its regenerative ability. Many of the molecular events that occur during the regeneration of the enteric nervous system in the sea cucumber are still unknown. To elucidate this, we have characterized and identified the expression of LIM homeodomain genes, Islet and Lhx3, expression during intestinal regeneration of the sea cucumber. LIM homeodomain proteins are known to have a prominent role in the regulation of key events of organogenesis as precursor cells enter into cell lineages and in the differentiation of neuronal subtypes. These proteins are dependent on a cofactor called Nuclear LIM Interactor (NLI) or LIM domain binding protein 1 (Ldb1). Here we analyzed contigs from an intestinal cDNA database of *H. glaberrima* and identified sequences corresponding to the holothurian Ldb1. In addition we profiled its expression during intestinal regeneration. The sequence was studied using bioinformatics tools, such as NCBI BLAST, NCBI Conserved Domains and ExPASy Tools. Semi-quantitative PCR was performed to determine the expression of Ldb1 at different intestinal regenerative stages and in control non-eviscerated animals. A decrease in the levels of Ldb1 was observed during the first and second week of regeneration. This apparent downregulation of Ldb1 in the regenerating tissues is surprising in view that its associated protein, Lhx3, is upregulated. This might indicate an unusual path of gene modulation not previously recorded. Future experiments are aimed at exploring this issue and determining whether cells of the enteric nervous system are expressing the given product.** Financial support: NIH (1SC1GM084770-01,1R03NS065275-01), NSF (IOS-0842870), University of Puerto Rico.

21st PR Neuroscience Conference
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A B S T R A C T

Author(s):

Katherine M. Cepeda-Rivera

Institutional Affiliation:

University of Puerto Rico, Río Piedras Campus

Research Topic:

Psychology Behavioral Science

Abstract:
Different Neuropsychiatric disorders are characterized by memory processing aberrations; schizophrenia, bipolar and post-traumatic stress disorders. To find treatment for these mental disorders, it is necessary to understand the requirements for healthy memory functioning. Two stages of declarative memory will be studied in order to explore the functions of the nuclear transcription factor Nurr1 in these processes. Declarative (explicit) memory is the memory of facts and events. It is formed over the course of experience and together with the genome, determines human individuality. Formation of declarative memory involves the acquisition, consolidation, retrieval, and reconsolidation.
Using immunohistochemistry, context fear conditioning and antisense technology, Peña's laboratory demonstrated that the gene Nurr1 is induced and required for memory consolidation in the CA3 region of the hippocampus. However, the scientific community still debates on the different requirements for the consolidation and reconsolidation processes. More research in the field of reconsolidation is still necessary for the eventual conclusion of this debate. The aforementioned techniques will help to explore weather or not Nurr1 is also induced and require during the process of memory reconsolidation. Contrary to what was found in the consolidation experiment, a variation at the site of Nurr1 induction of the hippocampus is expected.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Exercise-induced hippocampal-specific activation of fatty acid synthase and accumulation of stearate is mediated by insulin receptor signaling: Impact on spatial learning and memory

Author(s):

N. CHORNA[1,2,3], A. VAZQUEZ-MONTES[1,3], J. L. MORALES[1], J. DE LA NUEZ[1], N. M. CARBALLEIRA[4], A. P. CHORNYY[5], S. PENA DE ORTIZ[1,2,3];

Institutional Affiliation:

[1]Biol., Univ. Puerto Rico, Rio Piedras Campus, San Juan, PR; [2]Metabolomics Res. Ctr., [3]Functional Genomics Res. Ctr., Univ. of Puerto Rico, Rio Piedras Campus, San Juan, PR; [4]Chem., Univ. of Puerto Rico, San Juan, PR; [5]High Peformance Computing faciity, Univ. of Puerto Rico, Central Admin., San Juan, PR

Research Topic:

Psychology Behavioral Science

Abstract:
In neurons, endogenously synthesized fatty acids (FA) are esterified predominantly into phospholipids that stimulate membrane production or are used for lipid-based post-translational modification of membrane proteins. Since running is known for its beneficial effects in learning and memory, we hypothesized that different life styles, voluntary exercise versus sedentariness, would differ in specific lipid-related pathways associated with improved cognition. Administration of streptozotocin (STZ), an established diabetogenic agent known to block insulin receptor (IR) intracellular signaling upstream of fatty acid synthase (FASN) gene expression, specifically inhibited exercise-induced accumulation of stearate (SA) in the hippocampus previously identified by neurolipidomics. Accordingly, voluntary exercise induced hippocampus-specific increases in the expression of FASN but not elongases 1/6 -enzymes involved in SA biosynthesis via elongation from palmitate, an effect that was also blocked by STZ. Importantly, intracerebroventricular (i.c.v.) injections of C75, an irreversible FASN inhibitor, or STZ, where used to inhibit exercise-induced forebrain FASN activity or upregulation, respectively. Results indicate that directly blocking running associated FASN enzymatic activity with C75 or blocking its exercise-induced upregulation impairs spatial learning and memory in the Barnes Maze. Both treatments increased the escape latency, caused animals to shift from spatial to non-spatial searching strategies, and impaired spatial discrimination memory. Interestingly, in all our behavioral experiments

results indicated that both C75 and STZ interfered with the exercise mediated improvements in learning and memory. Overall, these studies identified a machinery of exercise-induced hippocampal FA regulation, involving FASN and IR-mediated intracellular signaling, relevant for the beneficial effects of exercise in spatial learning and memory

Notes:
I would like to be considered for oral short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

EphB2 down-regulation in IL facilitates fear extinction

Author(s):

Emmanuel Cruz, James Porter

Institutional Affiliation:

Ponce School of Medicine

Research Topic:

Psychology Behavioral Science

Abstract:
Treatments have to be developed for posttraumatic stress disorder (PTSD) patients that do not respond to current therapies. Gene expression studies such as microarrays could identify genes that could be targeted for development of new PTSD treatments. Pavlovian fear conditioning experimentally mimics PTSD and fear extinction mimics exposure therapy used to treat PTSD by reducing the fear response. Since the infralimbic cortex (IL) is critical for the recall of extinction, we used microarray technology to identify gene expression changes in IL after extinction and identify novel molecules associated with fear extinction. Interestingly, EphB2 was down-regulated in IL five hours after fear extinction. Based on these findings, we hypothesized that reducing EphB2 in IL would facilitate fear extinction. To test this hypothesis a plasmid containing EphB2-shRNA was used to reduce expression of the EphB2 receptor. Rats (P30) were fear conditioned on day 1 and received partial extinction on days 2, 4, 5, and 6. On day 2, rats were divided into two groups and received infusions of EphB2-shRNA plasmid (0.50 ng) or scramble-shRNA (0.50 ng) directly into IL at three time-points: (1) immediately after the first partial fear extinction session, (2) the next day in which the subjects rested in their home cages, and (3) after the second partial extinction. Reducing EphB2 with EphB2-shRNA facilitated extinction across days compared to the scramble-shRNA treatment, resulting in significantly less freezing on day 4 of extinction ($p = 0.02$). These results suggest that EphB2 expression in IL plays a role in extinction.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Activation of Group I Metabotropic Glutamate Receptors within the Basolateral Amygdala Produces Paradoxical Effects According to Sex

Author(s):

De Jesús-Burgos MI[1] , González-García S[2], Cruz-Santana Y[3], González-Bouza B[2], Portela-Díaz L[2], Ortíz-Soto L[2] and Pérez-Acevedo N[1].

Institutional Affiliation:

[1]School of Medicine, University of Puerto Rico, Medical Sciences Campus, [2/3]University of Puerto Rico, Río Piedras and Cayey campus.

Research Topic:

Psychology Behavioral Science

Abstract:

Activation of Group I Metabotropic Glutamate Receptors within the Basolateral Amygdala Produces Paradoxical Effects According to Sex

Females are twice more likely to suffer from anxiety than their male counterparts. Modulation of glutamatergic transmission by group I metabotropic glutamate receptors (mGluRs) has been implicated in anxiety. We studied the role of group I mGluRs in anxiety using the Vogel conflict test (VCT). We used ovariectomized female rats with low (OVX) and high (OVX+EB) estradiol levels, to evaluate the role of estradiol, if any, and male rats to evaluate sex differences. (S)-3,5-Dihydroxyphenylglycine-(DHPG), a group I mGluR agonist, was infused into the basolateral amygdala-(BLA), a region implicated in anxiety-responses. We hypothesized that intra-BLA infusion of DHPG produces behavioral effects according to sex and estradiol levels in the female rats. DHPG at 0.1 but not 1.0μM statistically increased the number of shocks in OVX but not OVX+EB. In males, DHPG at 1.0 but not 0.1μM statistically decreased the number of shocks. Sex differences were detected for the number of shocks, recoveries and punished licks, where female displayed more conflict than males. In females, these differences were statistically enhanced by estradiol treatment. Taken together, DHPG produced paradoxical effects that are sex dependent producing anxiolytic-like effects in female, while anxiogenic-like effects in male rats. These results highlight the importance of using female models to underpin the neural circuitry of anxiety according to sex and in the screening of novel anxiolytic compounds.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Post-training recruitment of the paraventricular thalamus into the circuit for retrieval of conditioned fear.

Author(s):

Fabricio H M Do Monte; Gregory J Quirk

Institutional Affiliation:

University of Puerto Rico

Research Topic:

Psychology Behavioral Science

Abstract:
We recently reported that the dorsal part of the midline thalamus (dMT) is critical for retrieval of a well-consolidated auditory fear memory (Padilla-Coreano et al., 2012). Pharmacological inactivation of dMT 24 h after conditioning, but not 2 h after, impaired fear retrieval. One possible explanation is that one or more structures in dMT are recruited into the fear circuit after conditioning. To test this hypothesis, we inactivated dMT at various timepoints after conditioning, and used the neuronal activity marker cFos to assess dMT activity. Rats were fear conditioned to a tone and then tested for fear retrieval 0.5 h, 6 h, 24 h, 7 days, or 28 days later. Inactivation of dMT with fluorescently labeled muscimol impaired fear retrieval at long intervals (24 h, 7 d, 28 d), but not at short intervals (0.5 h, 6 h). This suggests that dMT is recruited for fear retrieval after the initial consolidation and remains mediating the remote memory. We then compared cFos expression at short (6 h) and long (7 d) timepoints. In support of delayed recruitment of dMT, cFos expression in the paraventricular nucleus (PV) of the thalamus was significantly elevated at 7 d, but not 6 h. In contrast, cFos in the mediodorsal nucleus was not increased at either timepoint. PV could facilitate fear retrieval via projections to the central nucleus of the amygdala, which showed a similar time course of cFos activation. Thus, while fear behavior is constant with time, the circuit mediating that behavior is not.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Optogenetic manipulation of amygdala projections to the medial prefrontal cortex and the ventral hippocampus modulate anxiety-like behavior

Author(s):

Ada Felix-Ortiz, Anna Beyeler, Changwoo Seo, Christopher Leppla, and Kay M. Tye

Institutional Affiliation:

Massachusetts Institute of Technology

Research Topic:

Psychology Behavioral Science

Abstract:
Anxiety disorders are the most common psychiatric diseases, with 28% lifetime prevalence. Although it is well-established that the amygdala is important for the generation of anxiety, little is known about the role of connections between the amygdala and downstream regions in anxiety. In the present study, we use optogenetic tools in mice to explore the role of projections from the basolateral nucleus of the amygdala (BLA) to the medial prefrontal cortex (mPFC) and the ventral hippocampus (vHPC) during anxiety-like behavior in the elevated-plus maze and the open field test. BLA pyramidal neurons were transduced using adeno-associated viral vectors containing either channelrhodopsin-2 (ChR2) or halorhodopsin (NpHR), under the control of the CamKIIÎ± promoter to allow for optical excitation or inhibition of neurons with good temporal resolution. Optical fibers were chronically implanted in the mPFC or vHPC to selectively manipulate BLA terminals within these structures. ChR2-mediated activation of either BLA-to-mPFC or BLA-to-vHPC projections significantly increased anxiety-like behavior in both the elevated-plus maze and the open field test relative to control mice expressing only the fluorophore without opsin. In contrast, NpHR-mediated inhibition of each of these BLA projections significantly decreased anxiety. In conclusion, BLA inputs to the mPFC and vHPC are causally and bidirectionally involved in governing anxiety-related behavior.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Plasticity of Light signaling Pathways to the Circadian network in Honey Bees Apis mellifera

Author(s):

Manuel A. Giannoni-Guzmán, Tugrul Giray and Jose L. Agosto-Rivera

Institutional Affiliation:

University of Puerto Rico Rio Piedras Campus

Research Topic:

Psychology Behavioral Science

Abstract:

Although it is clear that light input to the circadian clock is critical for synchronizing internal physiological processes with the external environment, it is unknown whether these light pathways functionally change (are plastic) once they are established. While studying circadian locomotor behavior under constant light conditions, we found that circadian periodicity is highly variable among honey bee foragers of the same colony. This variability included individuals with short period (SP, ≤24hr), long period (LP, >24), arrhythmic (AR) as well as individuals that are initially rhythmic and later arrhythmic (R-AR). We propose that these differences in period are due to changes in the sensitivity of light signaling pathways to the circadian clock. Moreover, based on our preliminary findings, we hypothesize that these changes occur as a function of age or experience. Taken together these results indicate that light signaling pathways to the circadian clock remain plastic even in honey bee foragers. This plasticity may allow more effective synchronization with the environment as a function of age and/or experience, which could improve foraging performance.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Zirconium Phosphate Nanoparticles as a Potential Drug Carrier in Honey Bees

Author(s):

Liz HernÃ¡ndez, JosÃ© L. Agosto-Rivera, Tugrul Giray, Jorge ColÃ³n

Institutional Affiliation:

Universidad de Puerto Rico

Research Topic:

Psychology Behavioral Science

Abstract:
Carbamazepine (CBZ) is a widely used drug in the treatment of multiple mental and neurological disorders, but it has been associated with serious side effects. One side effect of CBZ is the sleep alteration, which has been seen in humans and in Drosophila melanogaster, showing that the drug is active in insects. This study proposes the use of Zirconium phosphate nanoparticles (ZrP) as carriers of CBZ to minimize the side effects of the drug. We hypothesized that if there are doses of ZrP with no effect on the survival of Apis Mallifera (Honey Bee) then, it can be used as a CBZ carrier and minimizes the side effects. Therefore, the survival and the total sleep in bees was monitored at different concentrations of ZrP and CBZ. At 2.5 mg/mL ZrP a significant difference on the survival of individuals was not seen, nor was a difference sleep. In change at a concentration of 2.5 mg/mL CBZ a significant difference on the survival of individuals was not seen, but the sleep was fractionated. Based on these results, we conclude that nanoparticle and the drug at concentrations of 2.5 mg /mL or lower do not affect survival of Honey Bee and can be used without risk of toxicity. Furthermore, since ZrP does not affect sleep, the sleep can be use as an indicator of drug release in the body. Now that a safe dosage of ZrP is known, the dose of CBZ:ZrP complex can be controlled to minimize the risk of toxicity.

Notes:
I would like to be considered for oral short presentation.

21st PR Neuroscience Conference
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December 1st, 2012

A B S T R A C T

Author(s):

GJ Jimenez (Presenter); V Wojna; SF Acevedo

Institutional Affiliation:

Ponce School of Medicine and Health Sciences (GJJ, SFA); NeuroAIDS Program, University of Puerto Rico, Medical Science Campus (VW)

Research Topic:

Psychology Behavioral Science

Abstract:
PURPOSE: Living with HIV/AIDS can be a struggle that endures overcoming many bio-psychosocially influenced risk factors that require adjustments. A Resilience Scales (RS) can be used to measure peopleâ€™s ability to make inferences according to a stressor appraised as an adversity, being able to withstand the difficult experiences or risk and to overcome the stress caused by adversity, in order to modify the stressor and adapt. DESIGN METHODS: The authors examined psychometric properties of a RS for HIV-seropositive Puerto Rican woman; differences between HIV positive and non HIV subjects; and establish association between intensity of depression symptoms and three dimensions of the RS intensity levels. A sample of Puerto Rican women; 45 HIV-positive patients and 17 HIV-seronegative control group were matched for age, education level and socio-economic factors. Participants were evaluated with the resilience scale, the Beck Depression Inventory in Spanish, and Life Orientation Test-Revised. RESULTS: The reliability and validity were evaluated, and reference scores for study samples were calculated. The scale demonstrated adequate psychometric properties. HIV-seropositive women report lower levels of resiliency compared to HIV-seronegative. Results also revealed that higher depression levels were associated with lower resilience levels. DISCUSSION/CONCLUSION: Implications could help recognize patterns on levels of mental distress and identify protective factors used to mitigate vulnerabilities and risk factors such as stigma (which is a human rights issue). Resiliency focus interventions could maximize awareness of current inequalities and implicates pioneering strategies that enhance social justice.

Notes:
I would like to be considered for oral short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Social-dependent Induction of Nurr-1 in the mouse brain

Author(s):

Pablo J Maldonado-Catala[1], Jose Luis Ortiz[2], Adrinel Vazquez[2], Darelys Correa[2], Sandra Peña de Ortiz[2]

Institutional Affiliation:

Universidad Metropolitana[1],University of Puerto Rico-Rio Piedras Campus[2]

Research Topic:

Psychology Behavioral Science

Abstract:
Nurr1 is a transcription factor critical for the maintenance and survival of dopaminergic neurons in regions of the brain. Our previous studies, demonstrate that Nurr1 is relevant to long-term memory formation and disruption of the gene impaired spatial extinction in a manner similar to obsessive compulsive disorder (OCD; Colón-Cesario et al.). Since dopaminergic systems play important roles in sociability we designed a series of experiments to determine if Nurr1 is involved in sociability. Male C57BL/6J were divided into two after weaning: Socially Reared vs. Isolated Mice. Socially Reared mice were housed in groups of 4 for 3 months, Isolated mice remained singly housed for the same period. Mice from both groups were divided in three: Naive (N, mice treated as above but never entering the chamber), context only (CO, mice introduced into empty chamber) and Social Interaction (SI, mice introduced in the chamber which contained one intruder mouse in one of the chambers). IHC was performed to assess brain regions associated with changes in Nurr1 expression following sociability tests. So far, analysis of the number ofimmunopositive Nurr1 nuclei is significantly higher in the dorsal claustrum 1h after test in SI compared to CO mice (N=5 each group; **p<0.005). Our results suggest that 3 months of post weaning isolation results in behavioral changes in sociability. Also, our IHC studies suggest that expression of Nurr1 in the claustrum might be of importance for the modulation of social behavior in mice. This work was supported by NIH (5SC1MH086072, IR25GM097635) ><0.005). Our results suggest that 3 months of post weaning isolation results in behavioral changes in sociability. Also, our IHC studies suggest that expression of Nurr1 in the claustrum might be of importance for the modulation of social behavior in mice. This work was supported by NIH(5SC1MH086072, IR25GM097635)

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Establishment of behavioral and neural structural parameters associated with interactive and agonistic behaviors of local species of freshwater prawns *Macrobrachium faustinum* and *carcinus*

Author(s):

Francelly Martinez Sosa, Frances Ostolaza Santiago, Valeria Salgado, Erick X. Perez Guzman, Laura C. Vicente Rodriguez, Eduardo A. Ruiz Rodriguez, Nilsa M. Rivera Cheverez, and Maria A. Sosa Llorens

Institutional Affiliation:

Department of Anatomy & Neurobiology (School of Medicine) and Institute of Neurobiology, UPR Medical Sciences Campus; Department of Psychology, UPR Rio Piedras Campus; Departments of Biology and Social Sciences, UPR Cayey Campus

Research Topic:

Psychology Behavioral Science

Abstract:
***Macrobrachium faustinum* is one of the most common species of prawns found in the streams and rivers of Puerto Rico and the Caribbean. We will use this prawn to assess the effects of urban river contaminants on the animal´s behavior and neuroanatomy. To do this, we must first characterize these parameters under control conditions in a laboratory setting. Adult prawns were collected from the Rio Salinas and placed in glass aquaria in the lab. The behavior of pairs of prawns was tracked using a video camera, and ethograms were developed to record and quantitate various aspects of their interactions (dominant/submissive postures/movements, fights, escapes, locomotion, and exploratory activities). We found that for each pair, one animal was always more dominant/aggressive than the other, with higher dominance tending to be related with larger sized claws, although pairs with similar sized claws still differed significantly in dominance. The mean levels of dominance of *M faustinum* were lower than those of *M carcinus*, a related species also found in Puerto Rico´s rivers, and similar to those of *M rosenbergii*, a prawn raised in artificial ponds. We also conducted immunohistochemical experiments to characterize the distribution of neurotransmitters/neuropeptides, such as serotonin, dopamine, FMRFamide and proctolin, in the central nervous system of *M faustinum*. The maps of neurons containing these substances are similar, although not identical, to those of related crustacea. We have thus established a set of behavioral and**

structural parameters that can now be used to assess effects of contaminants in Puerto Rico´s urban rivers.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:
ACUTE MORPHINE AFTER FEAR LEARNING:IMPLICATIONS FOR SEX-SPECIFIC PTSD TREATMENT.

Author(s):
EM PÉREZ-TORRES; DL Ramos-Ortolaza; JK Alvarado; E Santini; A Torres-Reverón
Institutional Affiliation:
Ponce School of Medicine and Health Sciences; Nova Southeastern University; University of Puerto Rico in Ponce

Research Topic:
Psychology Behavioral Science

Abstract:
Post-traumatic stress disorder (PTSD) is an anxiety disorder in which people experiencing physical and/or psychological traumas can associate certain stimuli with the trauma such that future exposure to the stimuli triggers intense fear and helplessness. Clinical studies show that acute exposure to morphine immediately after a trauma reduces PTSD episodes. Similarly, post-training acute injections of morphine blocks auditory fear conditioning, an animal model that simulates PTSD. This study was aimed to determine whether acute administration of morphine immediately after conditioning would alter consolidation of conditioned fear and\or extinction differently in males and females. Moreover, since fluctuations in female sexual hormones can alter behavior, we examined the effects of morphine in fear conditioning at stages of the estrous cycle when hormones are either high (proestrus) or low (metaestrus). Male and female rats were used in this study. Vaginal smears were performed in females to determine estrous cycle stages. On day 1, rats underwent auditory fear conditioning. Immediately after, they received subcutaneous injections of morphine (2.5 mg/kg) or saline. On day 2, rats underwent extinction. Acute morphine administration had no effect in fear conditioning, but caused impairment in extinction learning in females during metaestrus. No effects were observed in conditioning or extinction in males and females in proestrus. Our results suggest that phases of low ovarian hormone levels might lead to overconsolidation of fear memories delaying extinction. Furthermore, these results could help develop more effective therapeutic strategies for PTSD in men and women.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Title:

Central administration of the NPYY2 antagonist BIIE0246 modulates sexual behavior in the male rat

Author(s):

Keyla M. Ramos-Pratts and Jennifer L. Barreto-Estrada

Institutional Affiliation:

Department of Anatomy and Neurobiology, Medical Sciences Campus, University of Puerto Rico, San Juan, PR 00936.

Research Topic:

Psychology Behavioral Science

Abstract:
Neuropeptide Y (NPY) is the most abundant neuropeptide in the brain and its neurons abundantly innervate the hypothalamus. It suppresses the gonadotropic axis and is implicated in the inhibition of the sexual response in males. The NPYY2 receptor (Y2R) is the predominant subtype in the brain and is highly expressed in the hypothalamus, however its role on sexual behavior has not been determined. Due to the inhibitory role of NPY in sexual responses, we aimed to determine the pharmacological effect of NPYY2R blockage using BIIE0246, a selective Y2R antagonist. This Y2R antagonist has been shown to participate in the control of the gonadotropic axis depending on gonadal status. In this study, BIIE0246 (1.5 mM, 5 mM, 10 mM) was infused in the third ventricle at the level of the ventromedial nucleus of the hypothalamus (VMN). Male rats infused with BIIE0246 showed a dose response effect in sexual behavior. The frequency of mounts and ejaculations were increased, as well as a decrease in the latency to the first mount and first ejaculation was observed. These behavioral changes occur without body weight or food intake modulation, which are mostly regulated by Y1 and Y5 receptors. These results suggest a key role of the NPYY2R in the hypothalamic NPYergic circuitry and in the regulatory mechanisms of the male sexual response. **Supported by NIH-NCRR (2P20RR016470-012), NIH-NIGMS (8P20GM103475-12).**

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
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December 1st, 2012

A B S T R A C T

Title:

CONFIGURATION AND TESTING OF A MENSTRUAL RELATED EMOTIONAL STROOP TASK PURPOSE

Author(s):

M Rivera Rodríguez; I Flores; SF Acevedo

Institutional Affiliation:

Ponce School of Medicine and Health Sciences

Research Topic:

Psychology Behavioral Science

Abstract:

Configure and test the Emotional Stroop Task (EST) to assess two phases (pre-menstrual, follicular) of the menstrual cycle to determine the differences in the EST to assess the effects of chronic menstrual pain, anxiety and depression in healthy cycling women. We hypothesize that women with a level 6 or higher of pain will present higher levels of anxiety and depression and will be more emotionally receptive and attentive to menstrual related stimuli. METHODS: Using a group of 40 Puerto Rican women between 21 and 51 years of age with healthy menstrual period (28 days to 35 days) will complete a 5-minute questionnaire to assess the socio demographic information regarding age, risky behaviors (nicotine and alcohol consumption) and menstrual pain perception. Additionally, the Beck Anxiety Inventory (BAI) and Beck Depression Inventory (BDI-II) will assess information about anxiety and depression levels. EXPECTED RESULTS: Based on preliminary results, higher levels of pain are associated with anxiety levels in healthy cycling Puerto Rican women and lower reaction time in EST are expected in premenstrual phase compared to follicular phase.
DISCUSSION: **The study offers the opportunity to provide information about the emotional impact of pain and anxiety to the menstrual cycle. In the future we hope to provide management skills for women with high levels of pain and psycho educated women with anxiety about the menstrual cycle, its phases, symptoms and effects in the everyday life to normalize a process that occurs in healthy cycling women every month.**

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December 1st, 2012

A B S T R A C T

Author(s):

Jose Rodriguez-Romaguera, Fabricio H. Do Monte, Yoko Tanimura, Gregory J. Quirk and Suzanne N. Haber

Institutional Affiliation:

UPR School of Medicine

Research Topic:

Psychology Behavioral Science

Abstract:
Deep brain stimulation (DBS) of the VC/VS (ventral capsule/ventral striatum) in humans reduces symptoms of intractable obsessive-compulsive disorder (OCD), but the mechanisms of action are unknown. Using a rat model, we recently reported that DBS of a specific zone of dorsomedial ventral striatum (VS, just above the anterior commissure) facilitated fear extinction, whereas DBS of more ventrolateral sites in VS impaired fear extinction (Rodriguez-Romaguera et al., 2012). This suggests that clinical DBS may act, in part, by facilitating extinction of fear. The marked differences between DBS of dorsomedial and ventrolateral VS, suggest that these regions contain distinct sets of myelinated fibers coming from cortex that are modulated by DBS. To test this hypothesis, we injected retrograde tracer wheat germ agglutinin into the dorsomedial or ventrolateral VS and examined labeling in cortical areas. Using stereological techniques to analyze the number of labeled cell bodies, we found that convergence of multiple structures could be responsible for the DBS-enhancement of extinction. The dorsomedial VS site showed equivalent projections from orbital, insular, and prelimbic cortices. In contrast, the ventromedial VS site received projections predominantly from the ventromedial prefrontal cortex (infralimbic and ventral prelimbic areas), with smaller contributions from orbital and insular areas. Thus, the ability of striatal DBS to reduce fear and strengthen extinction may require coordinated changes within a larger prefrontal-orbital network, as suggested by physiological studies (McCracken and Grace, 2007; 2009).

Notes:
I would like to be considered for oral short presentation.

21st PR Neuroscience Conference
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A B S T R A C T

Title:

Genetic variation of the Serotonin Transporter (5-HTTLPR) in Human Fear Conditioning and Extinction

Author(s):

STEPHANIE SANTIAGO, Francisco Amador, MD, Karen Martinez, MD, MSc

Institutional Affiliation:

University of Puerto Rico, Rio Piedras

Research Topic:

Psychology Behavioral Science

Abstract:
Increased fear conditioning and decreased extinction represent associative models of learning reliably investigated in the etiology of anxiety disorders. Functional neuroimaging during fear learning paradigms reveal that subjects with anxiety disorders show altered activation of brain areas known as the fear circuit. We hypothesize that genetic variations altering protein expression and function related to these fear-interconnected regions might influence fear responses. Altered activity of the Serotonin Transporter (5-HTT) protein has been implicated in the pathophysiology of mood and anxiety disorders. Our objective is to examine if fear responses in humans are influenced by the functional genetic polymorphism of 5-HTT, the Serotonin Transporter Linked Polymorphic Region (5-HTTLPR). Healthy Volunteers aged 21 to 60 were screened using a structured clinical interview (SCID), to rule out any DSM-IV disorder. Included subjects underwent the fear conditioning and extinction paradigm by being exposed to visual cues of a context displayed in a computer monitor. An aversive electrical stimulus after a colored light conditioned participants, and the same colored light in a different context without the electric stimulus introduced an extinction phase. Physiological autonomic responses to fear (i.e. sweating) were measured through skin conductance (SCR). DNA from saliva samples of each subject was analyzed to identify alleles of the 5-HTTLPR. 5-HTTLPR-short allele carriers compared to homozygous for the long allele, showed increased SCR during conditioning and decreased SCR on extinction. Genetic variation in the 5-HTT may risk healthy individuals to develop anxiety disorders.

Notes:
Not interested in short presentation.

21st PR Neuroscience Conference
UPR-Rio Piedras Campus
December 1st, 2012

A B S T R A C T

Author(s):

Nelson D. Cruz Bermudez

Institutional Affiliation:

University of Puerto Rico, Rio Piedras Campus

Research Topic:

Psychology Behavioral Science

Title:

Perception and awareness of neuroscience among students at the University of Puerto Rico, Rio Piedras Campus: Implications for outreach strategies.

Abstract:

Neuroscience-related research is a major area of academic research in Puerto Rico. However, it is unclear how college students perceive neuroscience research. This study, which took place at the UPR Rio Piedras Campus was designed to gather information regarding studentsâ€™ basic knowledge, attitudes and beliefs about neuroscience. We hypothesized that students would have different ideas about neuroscience depending on variables such as high school experience and college major. The questionnaire used includes questions on demographics, general interests in neuroscience and perceptions about the implications of neuroscience for their careers. Results from a pilot study, show that more than 70% of the sample do not know a neuroscientist and do not know about a neuroscience research project or laboratory. Other questions explore whether students believe that knowledge about neuroscience makes them more competitive and whether neuroscience is important for their respective disciplines. Most of the students would like to have additional information about neuroscience and would take a neuroscience course, if available. Quantitative and qualitative analyses of data are included and discussed. The results of this study will be useful to identify misconceptions students have about neuroscience, to evaluate the implementation of interdepartmental neuroscience programs and to develop effective strategies for increasing awareness of neuroscience among college students. This research was approved by IRB and funded by the Department of Psychology and the College of Social Sciences, UPR-Rio Piedras.

Notes:
Not interested in short presentation.