Quantitative Evaluation of Cervical Inflammation Among HIV(+) New Orleans Women Co-Infected with Trichomonas vaginalis

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Introduction

The purpose of this study was to quantify the frequency of cervical inflammation in HIV(+) New Orleans women co-infected with Trichomonas vaginalis. We hypothesized that vaginal inflammation is associated with T. vaginalis infection and may contribute to disease progression.

Study Design & Cohort

Study subjects were recruited from the HIV/STI clinic at LSUHSC-Barkley University Medical Center. Women were included if they were HIV(+) and had a positive T. vaginalis culture. Exclusion criteria included current antiretroviral therapy and active sexually transmitted infections other than T. vaginalis.

Methods

Vaginal cultures were performed using modified Thayer-Martin media and examined microscopically for T. vaginalis. Cervical inflammation was quantified using a modified version of the Leishman-Donovan score. Statistical analysis was performed using the chi-square test.

Results

A total of 50 HIV(+) women were enrolled in the study. Of these, 25% were co-infected with T. vaginalis. Cervical inflammation was significantly higher in T. vaginalis-positive women compared to T. vaginalis-negative women (p<0.05). The prevalence of T. vaginalis was higher in women with higher Leishman-Donovan scores (p<0.01).

Conclusions & Discussion

Our findings suggest that T. vaginalis infection is associated with increased cervical inflammation in HIV(+) women. Further studies are needed to determine the role of T. vaginalis in HIV disease progression.

Future Directions

Future studies should focus on determining the mechanisms by which T. vaginalis infection leads to cervical inflammation.

Figure 1: Comparison of cervical inflammation scores between T. vaginalis-positive and T. vaginalis-negative women.

Figure 2: Distribution of Leishman-Donovan scores in T. vaginalis-positive women.
QUANTIFICATION OF NEUTRALIZING ANTIBODIES IN SIV-INFECTED RHESUS MACAQUES EXPOSED TO CHRONIC BINGE ALCOHOL

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Department of Microbiology, Immunology & Parasitology
LSUHSC New Orleans, LA

INTRODUCTION

- HIV/AIDS has been a major global health concern since the first known cases in 1981.
- New HIV cases emerge at a constant rate, and despite effective anti-retroviral treatment, there is still no cure for the disease so the population living with HIV continues to increase.
- Alcohol use disorder (AUD) is also a global health concern and is ranked as the leader in US addictions as well as the top 3 cause of preventable death.
- Epidemiological studies demonstrate that HIV-1 infection is associated with alcohol abuse.
- Alcohol has also been linked to HIV progression however, the specific mechanisms that underlie the biological effects of alcohol on HIV disease are largely unknown.

HYPOTHESIS

We hypothesize that alcohol treatment would adversely affect the humoral immune response to HIV, resulting in lower levels of neutralizing antibodies in those with AUD.

METHODS

- Experimental Design

For our analyses, we utilized the reporter cell line TZMbl, which produce luciferase when infected by HIV or SIV.
- The levels of luciferase are enzymatically determined and used to quantify the magnitude of HIV/SIV infection.
- TZMbl cells with SIV in the presence of
- We infected each of the study animals and plasma obtained from each of the study animals to determine the ability of the neutralizing antibodies to neutralize the virus.
- Plasma samples were determined by

RESULTS

Plasma Viral Load

Neutralizing Antibody Titer

CONCLUSIONS

- Neutralizing antibody levels measured at viral peak post-exposure to SIV animals demonstrate a decrease in antibody response.
- At 8 weeks post-exposure, the antibody response was significantly reduced compared to the control group.
- The findings suggest that alcohol exposure can negatively impact the immune response to SIV infection.
- Further studies are needed to investigate the mechanisms underlying this effect.
Modulation of Inflammatory Genes in Immune Cells and Adipocytes by miR-150

Asha Das1, Emily Ragland2, Sumana Majumder3, Jone Garai2, Li Li2, Nicole Pelligra2, John Estrada2, Melinda Sot hern3, Jovanny Zabaleta2
1Department of Genetics; 2Stanley S. Scott Cancer Center; 3School of Public Health, LSUHSC-NO

Introduction

Reprogramming of gene expression in adipose tissue, and ultimately transfer to different cell types, is a proposed mechanism by which miRNA regulates a number of critical processes in the body. This study aimed to investigate the role of miR-150 in regulating inflammatory gene expression in adipocytes and immune cells. The team of researchers performed a series of experiments to assess the effects of miR-150 on inflammatory gene expression.

Methods

- MicroRNA isolation from human adipose tissue
- qRT-PCR analysis of gene expression
- Western blot analysis
- Transwell assays

Results

I. Diet intervention modifies IL-6 and lipids

<table>
<thead>
<tr>
<th>Variable</th>
<th>Diet</th>
<th>HFD/60g fat/kg</th>
<th>p-value</th>
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<tbody>
<tr>
<td>Weight change</td>
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<td>Body fat%</td>
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<tr>
<td>Triglycerides</td>
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<tr>
<td>HDL cholesterol</td>
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<td>LDL cholesterol</td>
<td></td>
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</tr>
</tbody>
</table>

II. Correlation of changes in TNFα with changes in miR-150

- TNFα correlated with miR-150
- miR-150 levels increased in response to TNFα stimulation

III. Comparison of serum miR-150 in obese and non-obese individuals

- Increased levels of miR-150 in the serum of healthy weight controls when compared to obese individuals

IV. Genes affected by miR-150 OE

- Jurkat cells
- MSCs
- C2C12 muscle cells

Conclusion and Future Directions

The results suggest that miR-150 plays a role in the development of inflammatory responses in obesity. Further investigation is needed to understand the mechanisms of inflammatory gene regulation in response to miR-150.
Role of CRF signaling in the prefrontal cortex in traumatic stress-induced escalation of alcohol drinking

Ashley Augustado, Alyson Schreiber, Nicholas W. Gilpin, Ph.D.
Louisiana State University Health Science Center, Department of Physiology, New Orleans, LA

INTRODUCTION
- Post-Traumatic Stress Disorder (PTSD) affects 7.7 million Americans and is estimated to impact 20.8% of deployed military personnel.
- High-sensitivity CRF (hs-CRF) receptor levels in the prefrontal cortex and hippocampus are increased in PTSD.
- CRF is a stress hormone that plays a role in the hippocampal circuitry.

Hypothesis 1: Traumatic stress in mice increases CRF expression in the prefrontal cortex (PFC) and hippocampus.

Hypothesis 2: High-sensitivity CRF (hs-CRF) receptor levels are increased in PTSD.

ANIMAL MODEL OF PTSD
- Predatory odor stress alters drinking behavior and CRF expression in the PFC.
- CRF expression is increased in the PFC and hippocampus in mice exposed to predator odor.

EXPERIMENTAL DESIGN
- **Experiment 1:**
  - CRF receptor antagonist treatment
  - Predator odor stress
  - Post-treatment alcohol consumption

- **Experiment 2:**
  - CRF receptor antagonist treatment
  - Predator odor stress
  - Post-treatment alcohol consumption

RESULTS
- Predator odor stress increases CRF expression in the PFC and hippocampus.
- CRF receptor antagonist treatment attenuates stress-induced increases in CRF expression.

CONCLUSIONS
- Predator odor stress increases CRF expression in the PFC and hippocampus.
- CRF receptor antagonist treatment attenuates stress-induced increases in CRF expression.

REFERENCES

This research was supported by grants 5R01 AA022134 from the National Institute of Alcohol Abuse and Alcoholism at the National Institutes of Health.
Pegylated Arginase I Blunts T Cell Function Through Inhibition of Dendritic Cell Development

Audrey Lemoine, Paul Kepper, Paul Thevenot, Ph.D, Paulo Rodriguez, Ph.D
Stanley S. Scott Cancer Center, Louisiana State University Health Sciences Center

Abstract:
The development of an immune suppressive environment plays a primary role in the growth of tumors and represents a major obstacle in the success of tumor immunotherapy. The metabolism of the nonessential amino acid L-Arginine (L-Arg) through the enzyme arginase I in myeloid derived suppressor cells (MDSCs) is a fundamental mechanism and prime example of the suppressive immune responses in tumor-bearing hosts. Accordingly, the depletion of L-Arg through a pegylated form of human recombinant arginase I (peg-Arg I) impaired T cell function and delayed the appearance of graft vs. host disease in mice undergoing mismatched bone marrow transplantation (1). Additional results indicated that peg-Arg I therapies induced the accumulation of MDSCs. This study examined the specific effect of peg-Arg I on the maturation and function of myeloid cells in vivo. We hypothesized that l-Arg deprivation by peg-Arg I would alter the maturation and function of dendritic cells. Using cultured human monocytes and monocyte-derived dendritic cells, we determined the effect of peg-Arg I on DC maturation and function. Our results show that peg-Arg I impaired T cell proliferation in vivo through the inhibition of MDSCs. These results indicate a novel potential therapeutic target for cancer immunotherapy and highlight the role of peg-Arg I in immune suppression. MDSCs are a heterogeneous population of cells that can have both inhibitory and stimulatory effects on the immune system. The finding of the effect of peg-Arg I on DC maturation and function suggests a potential therapeutic target for cancer immunotherapy.

Hypothesis:
L-Arg depletion by peg-Arg I blocks the maturation of dendritic cells, leading to the accumulation of these population MDSCs.

Normal Conditions

Bone Marrow

Dendritic Cell

Peg-Arg Induced Conditions

Bone Marrow

Dendritic Cell

Figure 1:
A. Peg-Arg I inhibits T cell proliferation in vivo through the inhibition of MDSCs.
B. Peg-Arg I induces the accumulation of MDSCs in vivo.
C. Peg-Arg I inhibits the accumulation of MDSCs in vivo.

Conclusions & Future Experiments:
1. Peg-Arg I inhibits T cell proliferation in vivo through the degradation of MDSCs.
2. Peg-Arg I induces the accumulation of MDSCs in vivo.
3. Peg-Arg I blocks the ability of dendritic cells to efficiently activate T cells.

Figure 2:
A. Peg-Arg I inhibits T cell proliferation in vivo through the inhibition of MDSCs.
B. Peg-Arg I induces the accumulation of MDSCs in vivo.
C. Peg-Arg I inhibits the accumulation of MDSCs in vivo.

Acknowledgements:
This work was supported by National Institutes of Health grants (CA203801) and (CA117611) to A.S.S. and (CA178196) to P.R.

References:
Refining a Computational Model of CA1 Pyramidal Neurons

Introduction

- If possible, introduce the purpose of the research or the problem that the study addresses.
- Highlight the significance or importance of the research.

Corrected AP Back-Propagation

- Discuss the methodology or techniques used in the experiment.
- Present results or data visualizations related to corrected AP back-propagation.

Achieved Type 1 Excitability

- Explain the outcome of the experiment related to achieved type 1 excitability.
- Compare experimental and model data.

Achieved Type 1 Phase Response Behavior

- Describe the phase response behavior that was achieved.
- Show results or graphs visualizing the phase response behavior.

G-NaP is Responsible for Negative-Slope Conductance

- Discuss how G-NaP contributes to negative-slope conductance.
- Present experimental and model data supporting this finding.

Conclusions

- Summarize the main findings or results.
- Discuss the implications of the study.
- Mention any future directions or potential applications.

Acknowledgments

- Thank contributors or funders who supported the research.
- Acknowledge any important support or resources.

References

- List all sources or studies referenced in the research.
- Include bibliographic information for each reference.
Alcohol Preference in Obesity-Prone and Obesity-Resistant Rats

Brooke S. Lawrence, Jonquil M. Poret, Tony H. Tzeng, & Stefany D. Primeaux, PhD

1Department of Physiology, LSUHSC Health Sciences Center, New Orleans, LA; 2Joint Diabetes, Endocrinology & Metabolism Center, Pennington Biomedical Research Center, Baton Rouge, LA

Introduction

Obesity is a complex disease that can have damaging effects on the body. An increased susceptibility to developing obesity can predispose an individual to a variety of obesity-associated comorbidities, including Type 2 diabetes, cardiovascular disease, depression, and addictive behavior. Addictive behavior, especially with alcohol, is associated with obesity because of one common factor: neuroinflammation. Inflammation of the central nervous system (CNS) is characterized by increased glial activation, pro-inflammatory cytokine concentration, blood-brain barrier permeability, and leptin insulin action. One key player that is believed to drive this neuroinflammatory process is interleukin (IL)-1 beta, a pro-inflammatory cytokine that is up-regulated in Alzheimer's disease (AD), Parkinson's disease, multiple sclerosis, and other neurodegenerative disorders. Alcohol-induced neuroinflammation is mediated by the innate immune system. Within the central nervous system, obesity can lead to an increase in neuroinflammation due to an excess of circulating fatty acids and circulatory inflammatory factors. Local inflammation can cause synaptic remodeling and neurodegeneration within the hypothalamus and have a negative outcome on cognition. Drinking alcohol can be beneficial to health in small amounts, and heavy drinking increases mortality and neurological disorders in the central nervous system. The goal of the current series of experiments was to determine if the susceptibility to developing obesity alters alcohol preference and whether there are innate differences in neuroinflammatory markers in rodents which alter their susceptibility to developing obesity. We predict that an increase in neuroinflammatory markers would alter alcohol preference.

Methods

Animally 8- to 10-week-old male Osborne-Mendel and S/DHR (LSUHSC NO & PBR breeding colonies) rats were used in these studies. Rats were individually housed and maintained on a 12:12 light/dark cycle. All procedures were conducted in an AAALAC-accredited facility and in accordance with the PBRHC & LSUHSC-NO IACUC committees.

Experiment 1: Alcoholic Self-Administration: Rats were given access to two water bottles, one containing 5% saccharin solution and the other containing an alcohol/2% saccharin solution. Alcohol content was increased in steps from 2% alcohol (v/v) to 4% alcohol (v/v), 16% alcohol (v/v), and 16% alcohol (v/v), all of which were decreased to 0% alcohol (v/v). Bottles were counterbalanced to prevent the development of place preferences. Body weight, food intake, saccharin intake and alcohol intake were measured daily. Alcohol intake (g/kg/day) and alcohol preference were determined.

Experiment 2: Neuroinflammatory marker expression: RNA was isolated from the paraventricular nucleus of the hypothalamus, piriform cortex, dorsal striatum, and ventral tegmentum, and the central nucleus of the amygdala using TRIzol. mRNA and microarray (KCl/Qiagen, following refore transmission) results were normalized to the expression level in 6-week-old rats and were normalized to the expression level in 6-week-old rats. Real-time PCR was performed to assess the effects of alcohol (v/v) on gene expression. Relative expression was calculated using the fold-change from the 0% alcohol condition.

Experiment 3: Alcohol Escalation Model: Rats were given 4% alcohol access to two water bottles, one containing water and the other containing 2% alcohol, all days. On Monday, Wednesday, and Friday, for 4 weeks, body weight, food intake, saccharin intake, and alcohol intake and alcohol preference were determined.

Conclusion

This research was supported by grant R35AA0231364 from the National Institute of Alcohol Abuse and Alcoholism at the National Institutes of Health and LSUHSC start-up funds.
Analysis of Drug Resistant Mutations in SIV from Macaques Exposed to Chronic Binge Alcohol
Corey St. Romain, Spencer Robichaux, Whitney Nichols, Alexis Vega, Angela Amedee, PhD.
Department of Microbiology, Immunology, and Parasitology, LSUHSC, New Orleans, LA

Rationale and Background
- The incidence of drug-resistant SIV is rising in the United States and worldwide.
- Drug-resistant SIV is more prevalent in high-risk groups, including those with HIV and other sexually transmitted infections.
- The emergence of drug-resistant SIV is a significant challenge in the treatment of HIV infection.

Methods
- A total of 50 macaques were infected with SIV and treated with two ART regimens.
- Viral loads were measured before and after treatment.
- Drug-resistant mutants were identified using next-generation sequencing.

2. Viral Loads of ART+ Macaques
- Figure 2 illustrates the viral loads before and after treatment for each ART regimen.
- The viral load significantly decreased in all treated groups.
- No significant differences were observed between the two ART regimens.

3. Sequence Comparison
- Figure 3 shows the sequence comparison between the wild-type and drug-resistant SIV.
- The drug-resistant SIV had mutations in multiple regions of the viral genome.
- The sequence of the drug-resistant SIV was compared to the wild-type sequence.

4. Mutation Frequency
- Figure 4 depicts the frequency of all detected mutations in 158 samples from 14 macaques.
- Mutations in red are the functionally established major DRM, those in green are common polymorphisms.

5. Viral Load and DRM Comparison
- Figure 5a shows the correlation between the viral load and the number of DRM.
- Figure 5b evaluates the relationship between the viral load and the average mutation load.
- The viral load and DRM were negatively correlated.

Conclusions
- Drug-resistant mutations in SIV are common and vary depending on the ART regimen.
- The viral load is significantly reduced with ART treatment.
- Further studies are needed to understand the mechanisms of drug resistance and to develop effective strategies to prevent and treat drug-resistant SIV.

National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism
**Mechanisms of Alcohol Induced Cardiac Fibrosis**

**ABSTRACT**

Western Blots

**RESULTS**

Dihydroethidium Staining

**INTRODUCTION**

**METHODS**

- Chronic Alcohol Absence
- Intraperitoneal injection
- Hepatic iron content
- Western Blots
- Dihydroethidium Staining

**CONCLUSIONS**

- Alcohol causes cardiac fibrosis to release pro-inflammatory cytokines (IL-6, IL-1β, TNF-alpha)
- Alcohol also increases oxidative stress and NOX-4 expression, a major source of reactive oxygen species
- Lower doses of alcohol may stimulate pro-fibrotic activities, whereas higher doses may elicit pro-inflammatory responses
- In conclusion, cardiac fibroblasts may also contribute to alcohol-induced cardiac inflammation

**FUTURE DIRECTIONS**

- Determine if alcohol-induced cardiac fibroblast activation and collagen secretion is dependent on inflammatory pathways (NF-kB) and/or oxidative stress pathways (NOX-4)
- Determine the mechanisms of alcohol-induced cytokine secretion by cardiac fibroblasts (oxidative stress, NOX-4)

This research was supported by grant 08251/01364 from the National Institute of Alcohol Abuse and Alcoholism at the National Institutes of Health.
The Effect of PDE5 and nor-NOHA Inhibitors in Mycobacterium Tuberculosis Growth

Dairyn O. Navarro, Mariana Dupont, Matthew Nguyen, Arnold H. Zea

1New Orleans Charter Science and Mathematics High School, 2Baylor University, 3Tulane University, 4Stanley S. Scott Cancer Center/Department of Microbiology, LSUHSC-NO

Introduction

Tuberculosis (TB) is a disease that is caused by a bacterium called Mycobacterium Tuberculosis (Mtbo) that attacks the lungs. Infection with Mtbo begins with latent infection and eventually becomes active. Mtbo has developed a different strategy to survive inside the macrophages. These are the natural host for Mtbo to replicate. Although currently, there are therapies to control tuberculosis, these are expensive, lengthy, and produce different side effects. It has been reported that Mtbo has the capability to induce 3'-5' adenosine phosphates (cAMP) inside macrophages that could inhibit different functions in macrophages such as phagolysosome and cytokine function as a mechanism of Mtbo to survive. Furthermore, cAMP is capable of inducing arginase (ARG) and polyamine synthesis believed to be an important molecule for Mtbo growth and survival (Dr. Zea's preliminary findings). The L-arginine pathway consists of two pathways. One pathway is when L-arginine produces polyamines through the enzyme ARG to help the bacteria grow and replicate. The second pathway is when L-arginine metabolizes inducible nitric oxide synthase (iNOS) to nitric oxide (NO) which helps to stop the production of polyamines resulting in the inhibition of bacterial growth. In this summer we were interested in testing the effect of cAMP and ARG inhibitors in Mtbo growth and to determine what inhibitor is better suitable inhibitor to control Mtbo growth. We did not use live Mtbo as a source for ARG inhibition. We use analogs of cAMP to determine cAMP and ARG activity and polyamine production. These experiments are relevant in the field of mycobacterial infection, since new inhibitors could be the base for the development of new therapeutic venues to control TB, especially in these times when resistant TB can be a dangerous threat in public health.

Objective

To determine the effect of cAMP (PDE5) and arginase inhibitor (nor-NOHA) in murine macrophages stimulated with cAMP analogs or Mtbo antigens.

Acknowledgements

Dr. Fern Tzan, Dr. Paula Gregory, Dr. Arnold H. Zea, LSU HSC, Charter Science and Mathematics High School, and Baptist Community Ministries.
How miR-9 Affects Sound Production in Zebra Finch

D'Andrea Opara1,2, Zhimin Shi1, Dr. XiaoChing Li1
1LSUHSC Neuroscience Center of Excellence, New Orleans, LA,
2Xavier University New Orleans, LA

Abstract

The current study is a novel approach to investigate the role of miR-9 in the development of the zebra finch song system. We found that overexpression of miR-9 in the adult male zebra finch brain leads to a decrease in song duration and pitch. This effect was observed in both control and experimental groups. The results suggest that miR-9 may play a crucial role in the regulation of song production in the zebra finch.

Introduction

The zebra finch is a model system for studying the neural mechanisms underlying vocalization and song production. Previous studies have shown that miR-9 is involved in the development of vocalizations in other species. However, the role of miR-9 in the zebra finch has not been extensively studied.

Methods

The adult male zebra finches were divided into two groups: control and experimental. The experimental group was injected with miR-9, while the control group received a placebo. The birds were observed for changes in song duration and pitch over a period of six weeks. The data was analyzed using statistical methods to determine the significance of the results.

Results

The results showed a significant decrease in song duration and pitch in the experimental group compared to the control group. The mean song duration in the control group was 212.13 seconds, while in the miR-9 group, it was 117.502 seconds. The pitch also showed a significant change, with the control group having a mean pitch of 25.404 Hz, while the miR-9 group had a mean pitch of 34.578 Hz.

Conclusions

Our hypothesis was confirmed because the birds injected with the miR-9 virus had a shorter song duration than the birds injected with the control virus. The birds injected with the miR-9 virus sang for an average of 137.402 seconds, while the birds injected with the control virus sang for an average of 223.157 seconds.

miR-9 is a SUCCESS!!!

References


Figure 1 (Control Virus)

Figure 2 (miR-9 virus)

Legend:

GROUPS
Control (3)
miR-9 (5)

MEAN (seconds)
212.130
117.502
The Use of the Penumbra Aspiration Catheter for Proximal Embolic Protection Device During Intracranial Vertebral Artery Angioplasty and Stenting

Darian J. Harris, Erin S. Famin, Jason D. Wilson, MD
Louisiana State University Health Sciences Center, Department of Neurosurgery

Abstract

Introduction

Methods

Results

Conclusions

Figure 1: Patient is a 69-year-old male with history of MCA stroke and contralateral ischemic changes. He presented with sudden onset of left-sided weakness, left-sided hemiparesis, and right-sided sensory symptoms. The patient was treated with Penumbra aspiration catheter and proximal embolic protection device. The post-procedural MRI showed no evidence of diffusion weight changes.

Table 1: Patient Demographics

<table>
<thead>
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<th>Variable</th>
<th>Frequency (%)</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
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<td>Race</td>
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<td>LOS (days)</td>
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<tr>
<td>Gender</td>
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<td>64.5 (5.13)</td>
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<tr>
<td>Race</td>
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<td>LOS (days)</td>
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Table 2: Modified Rankin Scale

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<th>Standard Deviation</th>
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<td>Procedure</td>
<td>Penumbra (90%)</td>
<td>64.5 (5.13)</td>
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<td></td>
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<tr>
<td>LOS (days)</td>
<td>6.5</td>
<td>5.10</td>
<td>1-16</td>
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</tr>
<tr>
<td>MPA (mmHg)</td>
<td>140</td>
<td>5.10</td>
<td>1-16</td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Modified Rankin Scale

- No symptoms (0)
- Mild symptoms, unable to carry out all usual duties (1)
- Slight disability, unable to carry out all previous activities, but able to look after own affairs (2)
- Moderate disability, requiring help in some daily activities (3)
- Severe disability, unable to walk without assistance (4)
- Total dependence (5)

Table 4: Modified Rankin Scale

- No symptoms (0)
- Minimal symptoms, able to walk with or without assistance (1)
- Moderate disability, requiring help in some daily activities (2)
- Severe disability, unable to walk without assistance (3)
- Total dependence (4)

Conclusions

- Intracranial MCA stenosis was successfully managed with Penumbra aspiration catheter and proximal embolic protection device.
- Intracranial MCA stenosis was successfully managed with Penumbra aspiration catheter and proximal embolic protection device.
- Intracranial MCA stenosis was successfully managed with Penumbra aspiration catheter and proximal embolic protection device.
- Intracranial MCA stenosis was successfully managed with Penumbra aspiration catheter and proximal embolic protection device.

The patient's clinical outcome was excellent, with no residual symptoms or deficits.
The Effect of Fear-Conditioning on the Expression of Endocannabinoid-degrading Enzymes in Cerebellar Neurons

David Le, Christophe J. Dubois, June Liu.
LSU Health Sciences Center, Department of Cell Biology and Anatomy.
Screening Novel Hydrazine Compounds for Anti-parasitic Activity

Evans Wall, Donna M. Neumann, Branko S. Jursic, and Ben L. Kelly

INTRODUCTION

Leishmaniasis is a chronic parasitic disease caused by species of the protozoan parasite Leishmania. It is transmitted via the bite of its phlebotomine sandfly vector (Figure 1). Clinical manifestations range from cutaneous to visceral disease, causing skin sores and fever, anemia, and spleen enlargement. In the absence of drug treatments, such as Amphotericin B, the mortality rate can approach 100%.

Current drug treatments, such as Amphotericin B, are unsatisfactory because they have toxic side-effects. Current, no vaccine against leishmaniasis is available. New, effective, low toxicity drugs are needed to combat leishmaniasis.

Aims of the Study

To identify new compounds as drug leads against leishmaniasis. Since hydrazine compounds have previously shown inhibitory activity against the enzymatic pathway of parasites, they may also have inhibitory properties against Leishmania. The objective of this study is to screen a library of hydrazine compounds for inhibition of promastigote growth in vitro. The selected compounds for inhibition of Leishmania growth in vitro will then be tested for their potential as potential agents against Leishmania and other related parasites.

Experimental Methods

- Seed parasites or 214 t/c plate numbers with compounds at 1x IC50 (growth inhibition).
- Determine parasite density using a hemocytometer after 14 days of culture.

CONCLUSIONS

- New compounds identified as drug leads against leishmaniasis.

Figure 1. L. major life cycle

Figure 2. Reaction scheme analysis of hydrazine compounds with shown chemical structure.
A Descriptive Study of Trinity Medical Brigade:
A Sustainable Physician Run Short Term Medical Mission to Jinotega, Nicaragua

Ferralita Madere1, Elaine Hicks, MS1, Ebony Juakali, BS1, Andrew Delrio, MD2, Lisa Moreno, MD, MS2

1 University of Louisiana, New Orleans, LA 2 Department of Medicine, Section of Emergency Medicine, Louisiana State University Health Sciences Center, New Orleans, LA

Methods:
Trinity Medical Brigade Medical Mission Trip 2015 Jinotega, Nicaragua

Introduction

Background

The goal of the medical mission trip is to provide
free medical care to the underserved in rural communities.

Results:

Gender Frequencies

Conclusions

The percentage of females in the patient population is consistent with what is found in
the majority of countries. The age distribution is consistent with what is found in
the majority of countries. The majority of patients are in the age groups of 15-64 years.

Trinity Medical Brigade Medical Mission Trip
2015 Jinotega, Nicaragua

Notes

The picture captures the essence of the medical mission trip to
Jinotega, Nicaragua, which provides healthcare to underserved communities.

References

1. Madere F, Hicks E, Juakali E, Delrio A, Moreno L. A Descriptive Study of Trinity Medical Brigade:
A Sustainable Physician Run Short Term Medical Mission to Jinotega, Nicaragua. Poster presented at the

2. Madere F, Hicks E, Juakali E, Delrio A, Moreno L. A Descriptive Study of Trinity Medical Brigade:
A Sustainable Physician Run Short Term Medical Mission to Jinotega, Nicaragua. Poster presented at
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Inhibiting Endocannabinoid Degradation Following Traumatic Brain Injury Reduces Astroglialisis

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Introduction

- Traumatic brain injury (TBI) is a leading cause of death and accounts for two-thirds of all military fatalities.
- Annual medical costs of TBIs, according to the CDC, is upward of $75 billion dollars.
- Short-term effects of TBI include neuroinflammation and blood-brain barrier (BBB) dysfunction and long-term effects include cognitive impairment, neuronal damage, and neurodegeneration.
- In TBI, 3-arachidonoylgllycerol (2-AG) and N-acylcanolamine-hydrolysing esterases (AHE) have been implicated in endocannabinoid degradation.
- FAAH family of serine hydrolyases hydrolyzes AEA while MAGL (monoacylglycerol lipase) hydrolyses AGL, which occurs in TBI. This hydrolysis is followed by impaired astrocyte function (EC) or increased ECM degradation, at the site of injury, could improve neuroprotection and reduce the short-term and long-term effects of TBI.
- Inflammation often results from sustained astroglial stimulation.
- Increased gliofibrillary acidic protein (GFAP) expression is a marker of astroglialis.

Hypothesis

We hypothesize that blocking EC degradation enzymes (i.e., FAAH or MAGL) following TBI would result in reduced tissue damage as measured by GFAP, a marker of astroglialisis.

Methods

Methods involved measuring GFAP expression using Western Blot, and using a microarray to analyze gene expression.

Summary

- At 72 hrs in the prefrontal cortex, GFAP expression was increased ~2.5 fold when compared to Sham controls. TBI animals treated with EC degradation inhibitors, JZL 184 and URB 937, significantly reduced in GFAP expression compared to TBI vehicle animals.
- At 72 hrs post-TBI, TBI vehicle animals have ~4 fold increased expression of GFAP in the ipsilateral cortex when compared to Sham and TBI animals treated with EC degradation inhibitors. TBI-L2 and TBI-URB are no longer increased compared to Sham controls.

Future Studies will include extending our treatment of endocannabinoid degradation inhibition to animals with post-TBI alcohol exposure to see if the treatment is effective in reducing the enhanced astroglialis and neuroinflammation following post-TBI alcohol exposure.

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