AIRIG 2024 MEETING REGISTRATION

Registration cost is:

For Faculty & Staff @ $65 each
For Students, Post-Docs & Residents @ $40

To reserve a seat, please register online (www.luc.edu/airig) or send a check (made out to Loyola University Chicago, AIRIG meeting) to: Renita Alis, BSTRI, Bldg 115/CTRE, Room 315, Loyola University Chicago Health Sciences Campus, 2160 South First Avenue, Maywood, IL 60153. (Phone 708-327-2448).

Hotel and local travel information will be posted at a later date.

TRAVEL AWARD AND ABSTRACT SUBMISSION INSTRUCTIONS

Abstracts must be received by Tuesday September 3, 2024 via email to ralis@luc.edu (Renita Alis, 708-327-2448).

Abstract format - Microsoft Word, Times New Roman 12. ≤ 2600 characters (including spaces, author names and address). Abstracts will be published in the journal *Alcohol*. Figures/tables are not allowed.

In addition, include the following:
1) **Address, phone, and email address of the first author** (see demos below).
2) Indicate if you would like your abstract to be considered for a short **oral presentation**.

Travel Award applicants: If you are a student, post-doctoral fellow, resident, or minority scientist, indicate if you would like to be considered for a travel award, include a short note explaining your need for travel funds and your NIH style biosketch (or CV). Travel award applicants should combine documents into a single pdf (letter, abstract, and biosketch (or CV). The pdf file name should be the last name of the applicant (i.e. "Herrnreiter.pdf") and should be sent to ralis@luc.edu.

Example 1:
First author: Caroline Herrnreiter, BS, Bldg 115/CTRE, Room 315, Loyola University Chicago Health Science Division, 2160 South First Avenue, Maywood, IL, USA, Phone: xxx-xxx-2410, email: xxx@LUC.edu

No, I would not like my abstract to be considered for oral presentation.
No, I am not interested in applying for a travel award.

Downregulation of anti-inflammatory miRNAs exacerbate intestinal inflammation after alcohol and burn injury
CJ Herrnreiter, X Li, MA Choudhry
Alcohol Research Program, Burn & Shock Trauma Research Institute, Loyola University Chicago Health Sciences Division, Maywood, IL 60153, USA

Previous findings from our laboratory have shown that alcohol intoxication at the time of burn injury promotes intestinal inflammation and gut barrier disruption. MicroRNAs (miRNAs) are small noncoding RNA molecules that negatively regulate gene expression and play a central role in intestinal epithelial cell homeostasis. In this study, we examined the impact of dysregulated miRNA expression on intestinal epithelial cell inflammatory responses to further understand their contributions to gut barrier disruption after alcohol and burn injury. Utilizing a mouse model of acute alcohol intoxication and burn injury, we found that small intestinal epithelial cell expression of several anti-inflammatory miRNAs (including miR-150, miR-194, and miR-146a) were significantly reduced in alcohol and burn mice compared to vehicle sham ($p < 0.05$). Using different models, others have shown that upregulation of these miRNAs is an important mechanism to control excessive inflammatory responses. In line with this, *in vitro* treatment of MODE-K cells (murine small intestinal epithelia cell line) with 1 ug/mL LPS for 24 hours promoted significant upregulation of miR-150 (~10 fold) and miR-146a (~2.5 fold). To understand the impact that altered miRNA expression could have on intestinal epithelial cell inflammatory signaling, MODE-K cells were transfected with miRNA mimics and the expression and secretion of inflammatory cytokines IL-6 and KC were measured by RT-qPCR and ELISA. Overexpression of miR-146a mimic significantly reduced both IL-6 and KC expression and secretion in response to LPS, while overexpression of miR-150 mimic significantly reduced IL-6, but not KC ($p < 0.05$). Overall, these findings indicate that downregulation of anti-inflammatory miRNAs after alcohol and burn injury could exacerbate intestinal inflammation and contribute to gut barrier disruption. (Supported by T32AA013527, R01GM128242)

Example 2:

First author: Shanawaj Khair, Department of Surgery, GITES Division, University of Colorado Anschutz Medical Campus, 12700 East 19th Ave, RC2 6460D, Mail Stop 8620, Aurora, CO, 80045. Phone: xxx-xxx-8208. Email: XXX@UCDenver.edu

Yes, I would like my abstract to be considered for oral presentation
Yes, I am interested in applying for a travel award
Yes, I hold an identity that the NIH defines as underrepresented in science

Does a single ethanol exposure prior to burn injury in mice worsen pulmonary inflammation as much as episodic exposure of ethanol?

Shanawaj Khair, Brenda J. Curtis, and Elizabeth J. Kovacs
University of Colorado Anschutz Medical Campus, Aurora, CO

Burn injury is a major cause of mortality and morbidity in the US and lungs are often the first organ to fail. Interestingly, patients with alcohol intoxication at the time of burn have worse clinical outcomes, including pulmonary complications. Mechanisms by which alcohol prior to burn injury leads to lung inflammation are still unclear. Previously, we reported that episodic ethanol exposure before burn in a murine model exacerbated pulmonary inflammation, with heightened lung IL-6 levels in mice. Herein, we compare episodic binge ethanol exposure before scald burn injury with a single ethanol exposure to determine if single ethanol exposure was sufficient to create a similar pulmonary response. C57BL/6 male mice were given ethanol (1.2 g/kg) or vehicle 30 min before burn; sham controls were given ethanol or vehicle. IL-6, CXCL1, and neutrophil infiltrate were measured in the lungs at 24 hours after burn and compared using Two-way ANOVA with Tukey's or Sidak's posthoc tests. Mice given burn alone had 4-fold higher lung IL-6 ($p< 0.05$) and those with a single ethanol and burn had an 11-fold increase relative to sham vehicle and ethanol mice ($p<0.05$). When we compared lung IL-6 levels the single ethanol & burn and episodic ethanol & burn groups, there were no significant differences. Next, we assessed pulmonary CXCL1 levels in the single ethanol group and found that mice with burn and ethanol had 14-fold more CXCL1 than in

sham vehicle mice ($p<0.05$). Again, we did not find a difference between the single ethanol exposure and burn to those given multiday ethanol exposure and injury. Given that both CXCL1 and IL-6 are pro-inflammatory mediators, we compared the infiltration of neutrophils into lung tissue after burn and saw 4 times the number of neutrophils in burn injured mice than in sham mice ($p<0.05$) and when ethanol was present at the time of the burn, this was 6 times higher ($p<0.05$). Consistent with prior observations, when the number of neutrophils in burn and ethanol mice were compared between single and episodic ethanol exposure regimens, there was no difference. Taken together, these data show that a single exposure to ethanol prior to burn injury contributes to excessive lung inflammation as seen in episodic multiday ethanol exposure prior to burn. R35 GM131831 (EJK) and VA 1 I01 BX004335 (EJK).

Code of Conduct

The annual AIRIG meeting provides an opportunity for alcohol researchers to share their latest findings in areas of alcohol and immunology along with a platform for networking opportunities. As an AIRIG participant, you are expected to conduct yourself in a professional manner including tolerance and respect for everyone regardless of their background, gender, or status.