Abstracts must be received by Thursday, November 2, 2017 via email addressed to ARP@UCDenver.edu. Abstracts will be published in the journal Alcohol.

In addition, include the following:
1) Address, phone, and email address of the corresponding author (see demo abstracts below, and please follow the format).
2) Indicate if you would like your abstract to be considered for a short oral presentation.
3) If you are a student, post-doctoral fellow, resident, minority scientist or early stage investigators, indicate if you would like to be considered for a travel award (pending NIAAA funding). If so, include a short note explaining your need for travel funds and your CV.

Example #1:
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**Alcohol intoxication accelerates aging: Assessment of biomarkers of healthy aging in alcohol consumers**
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Individuals over the age of 65 currently represent a substantial percentage of the population, and their numbers are predicted to increase in the future. Older individuals have an elevated systemic basal inflammatory state referred to as inflamm-aging, which has been implicated in the greater prevalence of chronic diseases associated with aging. Inflamm-aging may also contribute to increased mortality after acute illness, namely infection, among older people. The immune system of older people is impaired; yet, the exact mechanism(s) by which this occurs remain poorly understood, making specific clinical interventions difficult. Another at-risk population in the context of acute illness is alcohol abusers. There is growing interest in detecting behaviors and exposures that lead to premature biological aging, and alcohol abuse is one potential progeroid behavior. Excessive alcohol use and inflamm-aging share many common features, including diminished immune function and increased mortality in the setting of infection. Decreased gut barrier integrity has been linked to reduced immunity due to inflammation in these 2 at-risk populations. Thus, we measured circulating levels of pro-inflammatory cytokines and intestinal fatty acid binding protein (iFABP; a marker of gut permeability)
in younger controls, younger drinkers, and older controls. Alcohol consumption was assessed using the validated Alcohol Use Disorders Identification Test (AUDIT) questionnaire. Interleukin (IL)-8 and IL-6 are increased 2-5-fold, respectively, and levels of iFABP were 50% higher, in the plasma of older non-drinkers and younger alcohol abusers compared to younger non-drinkers. Therefore, markers of systemic inflammation in younger drinkers and older non-drinkers are similar, and elevated in comparison to younger controls, suggesting hazardous alcohol consumption may be a progeroid behavior. Determining levels of these contributors to disease in alcohol abusers and older individuals is an important step in assessing alcohol as an agent that can expedite the onset of complications associated with advanced age. (Supported by NIH R21AA023193 (EJK), R01GM117257 (EJK), R01AG018859 (EJK), R24AA019661 (ELB))

Demo abstract #1, above, is 2515 characters (including spaces, title, authors, affiliations, and funding).

Example #2:
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Yes, I would like my abstract to be considered for oral presentation.

No, I am not interested in applying for a travel award.

**PX-478 Prevents Gut Inflammation and Normalizes the Expression of Tight Junction Proteins Following Ethanol and Burn Injury**

Niya L. Morris, Adam M. Hammer, A. R. Cannon, R. Gagnon, X. Li, M. A. Choudhry Alcohol Research Program, Burn & Shock Trauma Research Institute, Department of Surgery, Integrated Cell Biology Program, Loyola University Chicago Health Sciences Division, Maywood, IL 60153

Ethanol continues to be a major confounding factor in post burn injury pathology. Many of the adverse effects following ethanol and burn injury are associated with impaired intestinal barrier. A compromised barrier could allow bacteria or bacterial products to translocate from the gut into extra-intestinal sites resulting in systemic inflammatory response, sepsis and multiple organ failure. Previous studies from our laboratory have shown that ethanol combined with burn injury results in gut inflammation and barrier disruption. We further observed a significant reduction in intestinal oxygen delivery following ethanol and burn injury. Additionally, hypoxia inducible factor (HIF)-1α, a marker of hypoxia was significantly elevated in small intestinal epithelial cells (IECs) following the combined insult. Since hypoxia can result in increased inflammation and tissue damage, we treated a group of mice with HIF-1α inhibitor PX-478 to determine whether hypoxia modulates tight junction protein expression and inflammation in the intestine following ethanol and burn injury. Briefly, male mice were gavaged with ethanol (~3 mg/kg) 4 hours before receiving a ~12.5% total body surface area full thickness burn injury. Immediately after injury mice were given 5 mg/kg of PX-478 via intraperitoneal injection. One day following injury mice were euthanized, small intestine
was harvested and processed for isolation of epithelial cells. IECs were used to measure tight junction protein expression (occludin and claudin-4) by qRT-PCR. Small intestinal tissue was used to evaluate levels of pro-inflammatory mediators KC and IL-18. We observed a ~2 fold increase in KC and IL-18 levels following ethanol and burn injury. This was accompanied with a ~40% reduction in expression of tight junction proteins occludin and claudin-4. Treatment of mice with PX-478 at the time of burn injury prevented the gut inflammation and normalized occludin and claudin-4 expression to that of sham animals. Together, these data suggest that PX-478 can be used to maintain gut barrier following ethanol and burn injury. (Supported by NIH R01AA015731, R01AA015731S1, T32AA013527 and F31AA024367.)

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