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Division:	Pulmonary Sciences and Critical Care Medicine
Presenter's Rank:	Other: PRA
Research Category:	Clinical Research
Title of Abstract:	EFFECTS OF ACETATE ON CEREBRAL BLOOD FLOW AND SYSTEMIC INFLAMMATION IN RECENTLY DETOXIFIED ALCOHOL USERS
Please copy and paste your abstract here: (no more than 300 words):	Purpose: Excess consumption of alcohol is associated with brain injury. Acetate, an energy substrate for the brain, may be neuroprotective by enhancing metabolic reserve and decreasing inflammatory processes. Prior work suggests that acetate increases cerebral blood flow (CBF) in the thalamus in healthy people. We hypothesized that acetate would increase thalamic and cortical CBF and decrease systemic inflammatory mediators in patients with severe AUD. Design: Prospective, double-blinded study comparing intravenous acetate to placebo, within-person, on CBF and inflammatory markers in recently detoxified participants with severe AUD. Methods: Sixteen (15M/1F; age 45.1(9.4)) severe alcohol users (AUDIT Score 28.3(9.1)) received acetate and placebo infusions, counterbalanced for order, separated by 3 hours. CBF was measured using an arterial spin labeling sequence on a 3T MR scanner. Thalamic CBF (ml/min/100g tissue) was extracted using a template-based mask. Whole brain perfusion maps were compared between placebo and acetate using a paired t-test, set at $p<0.05$ cluster-corrected for

multiple comparisons, voxel-level p<0.0001. Plasma was collected before and after placebo and acetate infusions. Cytokines including interleukins (ILs)-1β, -6, -8, -10, TNFα, IL-1 receptor agonist, and monocyte chemoattractant protein-1(MCP) were measured using a multiplex array.

Results: Compared to placebo, acetate infusion resulted in significantly increased CBF values in the thalamus (Left: 512.4 vs. 688.1, p<0.001; Right: 536.6 vs. 696.0; p<0.001). There were widespread increases in CBF in the cortex, cerebellum, and brainstem. Values of cytokines were not appreciably influenced by acetate versus placebo infusions.

Conclusion: Acetate-induced increases in thalamic CBF in AUD participants were consistent with previous findings in controls. Widespread increases in cortical and cerebellar CBF in AUD participants may reflect the brain's compensatory ability to utilize acetate in the setting of chronic alcohol exposure. The lack of change in cytokine values suggests that acetate does not alter inflammatory mediators in the short term.