will be associated with worse clinical outcomes. (2) To examine the relationship between antipyretics and mortality in mechanically ventilated patients at risk for an acute lung injury. We hypothesize that antipyretics will have no effect on clinical outcomes in mechanically ventilated patients with and without sepsis. METHODS/STUDY POPULATION: This is a retrospective study of a "before and after" observational cohort of 1705 patients with acute initiation of mechanical ventilation in the Emergency Department from September 2009 to March 2016. Data were collected retrospectively on the first 72 hours of temperature and antipyretic medication from the EHR. Temperatures measurements were adjusted based on route of measurement. Patients intubated for cardiac arrest or brain injury were excluded from our primary analysis due to the known damage of hyperthermia in these subsets. Cox proportional hazard models and multivariable linear regression analyzed time-to-event and continuous outcomes, respectively. Predetermined patient demographics were entered into each multivariable model using backward and forward stepwise regression. Models were assessed for collinearity and residual plots were used to assure each model met assumptions. RESULTS/ANTICIPATED RESULTS: Antipyretic administration is currently undergoing analysis. Initial temperature results are reported here. In the overall group, presence of hypothermia or fever within 72 hours of intubation compared with normothermia conferred a hazard ratio (HR) of 1.95 (95% CI: 1.48–2.56) and 1.31 (95% CI: 0.97–1.78), respectively. Presence of hypothermia and fever reduced hospital free days by 3.29 (95% CI: 2.15–4.42) and 2.34 (95% CI: 1.21–3.46), respectively. In our subgroup analysis of patients with sepsis, HR for 28-day mortality 2.57 (95% CI: 1.68–3.93) for hypothermia. Fever had no effect on mortality (HR 1.11, 95% CI: 0.694–1.76). Both hypothermia and fever reduced hospital free days by 5.39 (95% CI: 4.33–7.54) and 3.98 (95% CI: 2.46–5.32) days, respectively. DISCUSSION/SIGNIFICANCE OF IMPACT: As expected, both hypothermia and fever increased 28-day mortality and decreased hospital free days. In our sepsis subgroup, hypothermia again resulted in higher mortality and fewer hospital free days, while fever did not have a survival benefit or cost, but reduced hospital free days. Antipyretic administration complicates these findings, as medication may mask fever or exert an effect on survival. Fever may also affect mechanically ventilated septic patients differently than septic patients not on mechanical ventilation. Continued analysis of this data including antipyretic administration, ventilator free days and progression to ARDS will address these questions.

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The effects of autoimmune inflammation on proliferation, differentiation, and androgen receptor signaling in adult prostate stem cells

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OBJECTIVES/SPECIFIC AIMS: The primary goal of this project is to verify findings from a murine prostatitis model in the human setting. METHODS/STUDY POPULATION: Methods include primary cell isolation and culture, FACS, adoptive transfer, 3D cell culture, histology, immunofluorescence, xenograft, and tissue recombination. The study population includes patients undergoing HoLEP or radical prostatectomy due to hyperplasia or adjacent bladder or prostate cancer. RESULTS/ANTICIPATED RESULTS: Having verified similar sensitivities to androgen receptor (AR) inhibitors between naive murine and human basal prostate stem cells, we anticipate that autoimmune inflammation in humans affects the response of basal prostate stem cells in a manner similar to the murine setting as well. This includes increased proliferation, increased differentiation, and decreased response to AR inhibitors. DISCUSSION/SIGNIFICANCE OF IMPACT: The identification of survival mechanisms used by basal prostate stem cells in an androgen deprived environment may give insight to the process by which prostate cancer becomes androgen independent. The effect of inflammation on proliferation, survival, and AR signaling in these cells may also provide information relevant to cancer initiation and progression.

2483

The Empower Lab: An innovative model for research experience and training for undergraduate, graduate, and medical students

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OBJECTIVES/SPECIFIC AIMS: The Empower Lab was established in 2015 with the goal of providing students with hands-on research experience in sexual and gender-based violence and health. METHODS/STUDY POPULATION: The Empower Lab consists of 10–12 undergraduate, graduate, and medical students at a time. Students undergo a rigorous application process, and agree to volunteer 8 hours per week for at least 1 year. Students are assigned to teams, and learn research skills such as literature searches, systematic reviews, research question generation, study design, IRB procedures, database creation and management, data collection and analysis, oral and poster presentation, manuscript preparation, team collaboration and communication, advocacy, and leadership. Students start as research assistants, and can be promoted to team leader, and associate director of research. Students mentor and teach each other, and are supervised by the principal investigator (PI). A survey skill self-assessment is administered to lab members on entry to the lab, every 4–6 months, and upon exit. RESULTS/ANTICIPATED RESULTS: In total, 20 students have participated in the lab to date, and 12 are currently enrolled. Eighty percent of the lab members are women. The students are 45% undergraduates, 15% graduate (nursing, social work, public health), 20% medical students, and 10% not currently enrolled in school (gap year). Twenty students completed entry surveys, 11 students have completed interim surveys, and 5 students have completed exit surveys. Examination of current surveys indicates that students are gaining skills throughout the lab experience. Free-text feedback provided further insight. Currently, the lab has 5 IRB-approved studies actively recruiting participants, 4 manuscripts being written, and 3 studies in the development phase. Students have presented at three local and 2 national meetings to date. Changes have been made to the lab structure over time in order to provide clear expectations and feedback, and strengthen student performance. DISCUSSION/SIGNIFICANCE OF IMPACT: The Empower Lab is an innovative public health lab that provides opportunities for real-world research experience for students. The teamwork, collaboration, and structure of the lab permit mentoring, support, and teaching from peers, as well as from the PI. The Lab increases the PI's productivity. Students are encouraged to develop and implement their own research ideas, further encouraging independence and initiative. Although the number of surveys is limited to date, they indicate improvement in skills and confidence among lab members. The predominance of women in the lab suggests that this is a strong model for recruitment and retention of women in STEM.

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The microbial-derived short-chain fatty acid butyrate directly and differentially inhibits gut T helper cell subset activation and proliferation

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OBJECTIVES/SPECIFIC AIMS: A hallmark of progressive HIV-1 infection is the massive activation and depletion of the gut barrier protective CD4 T helper subsets (Th17 and Th22) in the intestinal mucosa. The loss of these cells is thought to contribute to microbial translocation and systemic immune activation that occurs during chronic infection. In addition to the loss of protective Th subsets, we previously showed that chronically HIV-1 infected individuals have an altered colonic mucosal microbiome, which is in part characterized by a lower relative abundance of bacteria that produce the short-chain fatty acid butyrate in conjunction with increased relative abundance of gram-negative pathobionts. This dysbiosis was linked to markers of mucosal and systemic immune activation in these individuals. Following up on these clinical observations, we sought to understand how a loss of butyrate might contribute to HIV-associated inflammation. Initial studies showed that the addition of butyrate to cultured lamina propria mononuclear cells (LPMC) resulted in decreased pathobiont-driven gut T cell activation, HIV-1 infection levels and production of IL-17 and IFNy. Since the gut barrier protective Th17 and Th22 subsets are preferentially infected and depleted, which is critical to HIV-1 pathogenesis, we wanted to determine the mechanism by which butyrate modulates activation of these important Th subsets in the gut. METHODS/STUDY POPULATION: Total LPMCs or purified LP CD4 T cells were isolated from human jejunal tissue (n = 3–6), labeled with CFSE and cultured with TCR/CD28 beads to mimic APC driven T cell activation, with the addition of butyrate at physiologic doses(0–2 mM). Four days after culture, secreted cytokine(IL-17 and IFNy) levels were measured by ELISA. Cells were then short-term (4 hr) mitogenically stimulated (PMA/Ionomycin) in the presence of a golgi transport inhibitor. Total CD4 T cell activation (CD38 + /HLA-DR + , CD25 +), proliferation (CFSElow), and frequencies of intracellular cytokines were measured by multi-color flow cytometry. Paired *t*-tests were performed to determine statistical significance. RESULTS/ANTICIPATED RESULTS: Butyrate inhibited LP CD4 T cell activation ($p = 0.013$) and proliferation ($p = 0.015$) within total LPMCs stimulated with TCR/CD28 beads in a dose-dependent manner, with significant activity starting at 0.125 mM. Quantification of total secreted cytokines revealed that butyrate significantly decreased both IL-17 and

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