

(66.7% of patients), neuropathy (48.8%), muscle pain (44.0%), insomnia (39.3%), and general pain (38.1%). Patients on LOT ≥ 4 had most of these symptoms more often than LOT < 4 (fatigue: 70.6% of patients vs. 60.0%, neuropathy: 71.8% vs. 40.0%, muscle pain: 47.1% vs. 42.2%, insomnia: 35.3% vs. 40.0%, general pain: 47.1% vs. 33.3%). For those on LOT ≥ 4 , 42.9% of survey responses endorsed "somewhat", "quite a bit", or "very much" symptom bother compared to 32.7% for LOT < 4 . QOL was similar between groups. Over many months, patients on LOT ≥ 4 had several persistent symptoms (neuropathy, sadness, insomnia), but even those on LOT < 4 had unmet symptom needs (fatigue, general pain, constipation). DISCUSSION/SIGNIFICANCE: Evidence shows that treatment selection at higher LOT in MM often underrates the impact of cumulative symptom burden. Our study reveals significant longitudinal unmet needs regarding symptom and distress management in MM; understanding this can help guide treatment decisions and palliative care for MM patients with escalating treatment demands.

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Deciphering the role of IL-4 in post-colitis repair*

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OBJECTIVES/GOALS: Incomplete mucosal healing and dysbiosis prevent long-term remission after colitis. IL4 may restore colon homeostasis through its action on immune cells and the microbiome. We will demonstrate this mechanism using genetically modified mice and molecular tools. This may result in target therapies that prolong remission in patients with IBD. METHODS/STUDY POPULATION: Mice were treated with 3% dextran sulfate sodium (DSS) in drinking water for 5 days to induce colitis. Mice were monitored daily for changes in body weight, and to monitor colitis severity. At each endpoint, mice were sacrificed and colon length was measured. For disease severity assessment, mouse colons were prepared in paraffin sections by the 'swiss-rolling' method. For flow cytometry, lamina propria mononuclear cell isolation was performed and cellular populations were stained with fluorophore-conjugated antibodies. IL4-eGFP-expressing (4get) mice were used to analyze the cellular expression of IL4 after colitis. Cell-specific IL4 deletion mice were generated using the cre-lox system. RESULTS/ANTICIPATED RESULTS: IL4-deficient mice had worse colitis compared with wild-type controls. Flow cytometry of lamina propria cells from 4get mice showed that most IL4-producing cells after colitis are eosinophils (CD11b+SiglecF+). Flow cytometry of C57bl6 mice showed an influx of IL4Ra+ monocytes (CD11b+Ly6C+) and macrophages (CD11b+F480+). IL4-stimulated bone marrow-derived macrophages demonstrated an increase in HB-EGF mRNA transcription. Myeloid-specific IL4R deleted mice had no difference in colitis severity compared with controls. Neutrophil-specific IL4R-deleted mice had increased colitis severity and mortality. Co-housing of littermate mice rescued recovery after DSS in IL4 deficient mice. DISCUSSION/SIGNIFICANCE: IL4 appears to play a role in restoring homeostasis after colitis. The mechanism depends on eosinophil-derived IL4, and action through neutrophils. However, the reparative function of IL4 can be shared with deficient mice through the microbiome. I will study the cellular

and microbial mechanism by which IL4 restores homeostasis after colitis.

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Identifying neural and behavioral correlates of social learning and empathetic responding associated with early life adversity

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OBJECTIVES/GOALS: This study seeks to elucidate the relationships between early life adversity (ELA), social learning, and empathic responding. Specifically, it aims to understand the impact of ELA on the expression of empathy and ability to adjust behavior after social observation. METHODS/STUDY POPULATION: 60 healthy participants ages 18-65 will be recruited from the greater Baltimore area. They will undergo a placebo manipulation paradigm with simultaneous EEG recording to capture neural oscillations in frontal and insular cortices and event-related potentials. Participants will observe a demonstrator who indicates pain relief in response to the application of an inert cream. Then, while undergoing heat pain stimulations, the participant will receive the same inert cream and rate their physiological and psychological pain experience using a visual analog scale. The heat stimulations will be lowered without their knowledge to measure placebo response. Participants will also answer a battery of questionnaires which assess personality, psychological factors, life history, empathy, and current social life. RESULTS/ANTICIPATED RESULTS: It is expected that ELA will result in decreased placebo response, interpreted as deficits in social learning. Further, we expect that this effect is moderated by state empathy, empathy in a specific context or moment. We predict that individuals with lower state empathy and exposure to adversity will have greater deficits in social learning. We also expect to see more robust event-related potentials preceding painful stimulations at electrodes corresponding to the medial and ventral prefrontal cortex and insula in ELA-exposed participants. Because these brain regions are connected to anticipatory and predictive circuits, this would indicate that the individual has not adjusted their expectations according to the social information gained via observation. DISCUSSION/SIGNIFICANCE: Results of this study will expand our understanding of how ELA impacts behavior throughout life. Individuals with a history of ELA often face social difficulties and a higher risk of psychiatric disorders. This study will illuminate possible neural correlates of these differences in social behavior and, more generally, the expression of empathy.

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Deformable Medial Modeling to Generate Novel Shape Features of the Placenta Using Automated versus Manual Segmentations*

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OBJECTIVES/GOALS: In this study, we implemented deformable medial modeling as a morphometric approach in first trimester placentas to characterize morphometric differences between fully