Cognitive Emotion Regulation Traits Predict Inflammation During a Natural Stressor using Intensive Longitudinal Blood Sampling

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Background: Inflammation is often described as a mediator between chronic stress and stress mediated diseases such as depression, cancer, and stroke. As such, psychological traits that modulate how much stressful experiences “get under the skin” can be useful foci for psychoneuroimmunology research focused on buffering the deleterious consequences of stress. One such transdiagnostic factor that is targetable by psychotherapy is cognitive emotion regulation.

Methods: Incoming college students (n=86) completed a 22-day protocol, starting a week before moving onto campus, using the transition to college as an intensive, developmentally critical stressor. Participants completed the Cognitive Emotion Regulation Questionnaire (CERQ) and a blood sample on Day 1 and completed another sample every 3 days (total = 688). Multi-level models with random intercepts tested associations between negative CERQ subscales and CRP, TNF-α, IFN-γ, IL-1β, IL-2, IL-4, IL-6, IL-10, IL-12p70, and IL-17A controlling for day-in-study, sex, BMI, and whether they had caffeine or ate. Participants taking immune medications or with immune illnesses were excluded.

Results: Several cognitive styles that amplified negative emotions were associated with higher levels of inflammatory proteins. Specifically, higher blaming of others predicted higher IL-6 (b=.834, SE=.383, p = .032), IL-1β (b=.337, SE=.139, p = .011), TNF-α (b=.050, SE=.023, p = .037), and IFN-γ (b=.037, SE=.017, p = .037); rumination predicted higher IFN-γ (b=.041, SE=.015, p = .006) and IL-6 (b=.771, SE=.340, p = .026); and self-blame predicted higher IFN-γ (b=.027, SE=.014, p = .0487).

Conclusion: Rumination, self-blame, and blaming other people are plausible psychotherapeutic targets to buffer elevated inflammation during periods of stress. This can guide biologically informed targets in psychotherapy and the development of resiliency programs (e.g., as part of new student orientation) for inflammation-mediated outcomes.