TPS12137 **Poster Session**

Social genomic mechanisms of health disparities among adolescent/young adult survivors of Hodgkin and non-Hodgkin lymphoma: ECOG-ACRIN EAQ211.

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Background: Research in human genomics maps molecular pathways through which social and psychological factors regulate gene expression in immune cells and tumor tissue, thus affecting chronic disease progression, symptom development, antiviral resistance, morbidity, and mortality. In many cases, psychosocial factors trigger neural and endocrine responses that regulate expression of genes involved in cancer progression (inflammation, metastasis, treatment resistance) and immune function (stimulating inflammatory genes and suppressing antiviral gene transcription, as observed in the "Conserved Transcriptional Response to Adversity" / CTRA transcriptome signature). However, nothing is known about how such effects impact AYA cancer survivors. This study aims to identify functional genomic pathways through which psychosocial factors influence gene regulation and alter health outcomes in AYA cancer patients; and define the role of such effects in structuring health disparities in post-treatment survivorship. **Methods:** This longitudinal single cohort study is administered through the ECOG-ACRIN Cancer Research Group. Subjects are accrued through the NCI Community Oncology Research Program (NCORP) or self-refer through a broad network of cancer support organizations and clinical programs that serve the AYA population. Accrual goal is 2,000 survivors of Hodgkin or Non-Hodgkin Lymphoma who have achieved complete response to therapy at time of study registration, aged 15-39 years at time of diagnosis, and recruited within three years following completion of treatment. Current accrual is n=117. Upon enrollment, participants complete an online survey of patient-reported outcome measures of social and psychological risk and resilience factors, including quality of life (QOL), social isolation, socioeconomic status, and exposures to childhood trauma. Clinical records are reviewed for medically reported comorbidities and vital status. Data are collected at baseline and repeated every 6 months for two years. Blood specimens also are collected at each time point. The CTRA transcriptome profile will be assayed using an established 53-gene index comprised of a block of 19 pro-inflammatory genes (e.g., *IL1B, IL6, IL8/CXCL8, TNF*) and 34 genes involved in innate antiviral response (e.g., *IFNA/B, IFI-, OAS-,* and *MX*-family genes), with CTRA representing the difference in average expression of those 2 blocks (inflammatory – interferon). CTRA is a biological intermediate state, which is hypothesized to mediate relationships between proposed psychosocial risk and resilience factors and outcomes (morbidity, mortality, QOL). Defining effects of psychosocial conditions on gene expression and their role in structuring disparities for AYA survivors will fill a critical gap in knowledge that informs risk-based models for cancer survivorship care. Research Sponsor: US Department of Health and Human Services, National Institutes of Health, National Cancer Institute; R01CA261752; ECOG-ACRIN Cancer Research Group (Peter J. O'Dwyer, MD and Mitchell D. Schnall, MD, PhD, Group Co-Chairs); UG1CA189828.