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Phenotypic and genomic relationships between chronic pain and substance use disorders in the All of Us biobank

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Chronic pain is one of the most pressing public health burdens in the United States, affecting up to 20% of the population. Substance use disorders (SUDs) often co-occur with chronic pain. The relationship between chronic pain and opioid use disorder is often attributed to overuse in the context of managing acute pain, but the underlying mechanisms for chronic pain's comorbidity with other SUDs (alcohol, tobacco, cannabis) are unknown. Socioenvironmental factors, including experiencing discrimination, may play a role. Given the role of the brain's reward system in pain and SUDs, it is also plausible that some of the same genetic risk variants contribute to both chronic pain and SUDs. Indeed, genetic correlations between substance use disorders and chronic pain range from r_g = 0.19–0.41. We proposed to examine the phenotypic and genomic relationships amongst chronic pain and SUDs in the All of Us biobank, leveraging data from electronic health records, self-report surveys, and whole genome sequencing. Preliminary data suggest that the prevalence of SUDs ranged from 2.8%–8.9% and chronic pain was indicated in 24.4% of individuals with available electronic health records data in All of Us. Chronic pain was positively correlated with all four SUDs studied (alcohol, tobacco, cannabis, and opioid use disorders), with correlations ranging from 0.25–0.38. An area deprivation index was significantly associated with greater likelihood of reporting opioid use disorder (beta=0.56, SE=0.21, p=0.007), but not chronic pain. Forthcoming analyses will explore the mediating roles of opioid prescriptions, social determinants of health, and shared genetic risk.