**F25. Increased Adverse Childhood Experiences Predict Worse Acute Pain and Psychological Symptoms After Sexual Assault**

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**Background:** Increasing evidence suggests that adverse childhood experiences (ACEs) can result in enduring neurobiological changes that increase pain and psychological vulnerability. To date, the influence of ACEs on acute pain and psychological responses following sexual assault (SA) is unknown.

**Methods:** Women SA survivors ≥18 years of age who presented for emergency care within 72 hours of SA are enrolled into this ongoing study. Acute pain (0-10 scale) is assessed at the time of presentation for emergency care; ACEs (ACE questionnaire) are assessed six weeks after SA, pain, and posttraumatic stress (PTS) symptoms (DSM-IV PCL) are assessed one week after SA.

**Results:** Women presenting for emergency care after SA (n=538) reported substantially and significantly higher ACEs than the general population (mean (SD) 3.87 (2.87)). Number of reported ACEs predicted acute pain severity at the time of presentation for emergency care (r = .155 (p < .001)) and at one week (r = .175 (p < .001)), and predicted PTS symptoms at 1 week (r = .216 (p < .001)). This influence of past ACEs on acute pain and psychological responses persisted after adjustment for age, income, and education (pain at presentation b= .105, (p < .015), (pain at one week b=.110 (p < .014)), (PTS symptoms at one week b=1.24 (p < .001)).

**Conclusions:** Among adult women SA survivors, reported adverse childhood experiences are associated with increased pain and psychological symptoms after SA.

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**Keywords:** Adverse Childhood Experiences, Sexual Assault, PTSD - Posttraumatic Stress Disorder, Pain

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**F26. Exploring Post-Zygotic Variants in Obsessive-Compulsive Disorder**

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**Background:** Obsessive-Compulsive Disorder (OCD) is a debilitating neuropsychiatric disorder that is known to be moderately heritable but has poorly understood pathogenesis, limiting development of novel pharmacologic treatments. Previous studies suggest a significant contribution to OCD risk from de novo germline variants, which arise spontaneously in the parental germ cells or zygote. Recent studies of autism spectrum disorder and intellectual disability suggest a risk contribution from post-zygotic variants (PZVs) arising de novo in multicellular stages of embryogenesis, suggesting these mosaic variants can be used to examine the genetic underpinnings of other neuropsychiatric disorders such as OCD.

**Methods:** We examined whole-exome sequencing (WES) data from peripheral blood of 184 OCD parent-proband trios and 777 control parent-child trios that passed quality control measures. We used the bioinformatics tool MosaicHunter to identify low–allele frequency, potentially mosaic single nucleotide variants (SNVs) in probands and control children, only considering variants with the alternate allele not present in parents and with frequency <0.05 in the dbSNP database to collect SNVs most likely to be true PZVs.

**Results:** The rate of all single-nucleotide PZVs per base pair is not significantly different between OCD probands (5.88 x 10^-9) and controls (5.80 x 10^-9), rate ratio = 1.01 (95% confidence interval = 0.654-1.53), one-sided p = 0.5.

**Conclusions:** We did not detect a higher burden of PZVs in blood in individuals with OCD. However, further studies may benefit from examining a larger sample of families or from looking for PZVs in other tissues.

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**Keywords:** Obsessive Compulsive Disorder (OCD), Genetic Variants, Psychiatric Genetics

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**F27. Heritability of Gamma Butyric Acid: A Mega-Press With Macromolecule Suppression Study**

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**Background:** Gamma-butyric acid (GABA) is the primary inhibitory neurotransmitter in the mammalian brain. It plays a role in plasticity, learning, and memory as well as sensory processing, and is strongly implicated in cognition, normal brain functions, and in multiple neuropsychiatric illnesses. GABA in the brain is emerging as a biomarker for brain health, cognition, and diseases.

**Methods:** To evaluate factors influencing this trait, we measured GABA metabolite in the mediofrontal cortex in a large homogeneous population and estimated the heritability (h2). Our sample included 352 individuals (237 controls, 20 with bipolar disorder,