transgenerational effects of environmental stress on fish health and the gut microbiome. In parallel, we will utilize the natural partial cross-fostering design of a human parent–child adoption study to characterize the interplay of stress, inflammation, and the gut microbiome in adolescents and examine how these physiological processes affect overall physical and behavioral health. Together, these two approaches allow us to not only describe associations between stress, inflammation, and gut health, but also make stronger conclusions regarding causality of disruptions in behavioral and physical health.

Modeling Genomic heterogeneity in cross-disorder risk for psychopathology using partitioned genomic structural equation modeling

Andrew Grotzinger, University of Texas at Austin; Jordan Smoller, Harvard Medical School; Phil Lee, Harvard Medical School; Michel Nivard, Vrije Universiteit Amsterdam; Elliot Tucker-Drob, University of Texas at Austin

Genetic risk for psychiatric disease is shared across a wide variety of disorders (Lee et al. 2019). However, genetic overlap across traits is likely to be heterogeneous across the genome, with certain categories of genes (e.g., genes expressed in the prefrontal cortex) plausibly conferring disproportionate cross-disorder risk. Using GWAS summary statistics, partitioned LD score regression (Finnucane et al. 2015) can currently be used to determine whether heritability is enriched within certain categories of genes expressed in particular cell-types (e.g., hippocampal cells), tissue-types (e.g., central nervous system), or developmental epochs (e.g., genes highly expressed during prenatal development). When this method is expanded to allow for estimation of genetic *covariance* within genomic partitions, Genomic SEM (Grotzinger et al. *in press*) can be used to model the multivariate genetic architecture of traits within multiple, biologically meaningful categories. Here, we introduce partitioned Genomic SEM and report results of a novel application to over a dozen psychiatric disease traits. These results elucidate gene sets that disproportionally confer cross-disorder risk, thereby providing key insights into the biological underpinnings of high levels of psychiatric co-morbidity.

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Differences in pubertal status in genetic and environmental influences on social support and depression among Japanese adolescents

Mami Tanaka, Chiba University; Yusuke Takahashi, Kyoto University; Masumi Sugawara, Ochanomizu University

Social supports, such as parents, teachers, and family, are key factors in helping children and adolescents avoid depression (Gariepy et al. 2016). Correlation between social support and incidence of depression may be accounted for by overlapping genetic and environmental influences in late adolescence (Wang et al. 2017). However, differences in the effects of genetic and environmental influences before and after the onset of puberty are poorly understood. This study aims to examine etiological differences in relationships between sources of social support and depression at onset of puberty, controlling for age and sex of the participants. A total of 401 set of twins ranging in age from 9 to 18 were enrolled. The results of a correlated factor model revealed that genetic effects appeared to be more important after the onset of puberty than before the onset of puberty. Before the onset of puberty, the phenotypic correlations ($r = -0.41$ to -0.35, $p < 0.001$) between sources of social support and depression were explained by shared common and unique environmental factors and were not explained by genetic factors. In contrast, the phenotypic correlations ($r = -0.43$ to -0.26, $p < 0.001$) between sources of social support and depression after the onset of puberty were explained by shared genetic factors ($r_g = -0.86$ to -0.45, which explains 64%–81% of covariance) and unique environmental factors. These results suggest that individual differences in adolescence need to be examined, taking into account an individual's status with respect to the onset of puberty rather than just chronological age.

Genetic associations of borderline personality features with psychiatric and behavioral traits

Lucía Colodro-Conde, QIMR Berghofer Medical Research Institute; Najaf Amin, Erasmus University Rotterdam; Quinta Helmer, Vrije Universiteit Amsterdam; Jouke-Jan Hottenga, Vrije Universiteit Amsterdam; Ian Gizer, University of Missouri; Fabian Streit, University of Mannheim; Timothy Trull, University of Missouri; Cornelia van Duijn, Erasmus University Rotterdam; Gitta Lubke, University of Notre Dame; Marcella Rietschel, University of Mannheim; Nicholas Martin, QIMR Berghofer Medical Research Institute; Dorret Boomsma, Vrije Universiteit Amsterdam; Sarah Medland, QIMR Berghofer Medical Research Institute

Borderline personality features (affective instability, identity disturbance, negative relationships and self-harm) are present at different degrees in non-clinical populations. Borderline features have shown a heritability of $\sim$ 42%. Borderline personality disorder have shown shared genetic variance with schizophrenia, bipolar disorder, and major depressive disorder. Using the results of a GWAS meta-analysis for a total of 17,969 individuals, we investigated the genetic correlations of borderline personality features with schizophrenia, bipolar disorder, major depressive disorder, and other selected psychological and psychiatric traits.

The GWAS meta-analysis was conducted on the continuous scores of the Borderline Features Scale of the Personality Assessment Inventory (PAI-BOR, Morey 1991), which did not detect genome-wide significant hits. The genetic correlations we estimated using LD score regression.

Borderline personality features showed a genetic correlation of 0.81 (SE 0.36, p value 0.03) with borderline personality disorder. I also showed positive genetic correlations with neuroticism, mood instability, autism spectrum disorder, insomnia, and major depressive disorder; and negative correlations with subjective well-being. No significant genetic correlations were observed with schizophrenia or bipolar disorder.

The use of the PAI-BOR to increase our understanding of the etiology of borderline personality seems appropriate and present some advantages (e.g., use in community samples, continuous measure).