News, Literature, and Events in Braingenethics

View this email in your browser

Braingenethics Update

Vol. 3, No. 1
February 2016
braingenethics.cumc.columbia.edu/

In the Literature

[When Neurogenetics Hurts: Examining the Use of Neuroscience and Genetic Evidence in Sentencing Decisions Through Implicit Bias]

John Pyun

Courts increasingly use neuroscience and genetic evidence to shed light on various aspects of a defendant's mental state and behavior. The evidence is particularly prevalent in cases involving defendants with mental illnesses and is used to determine issues of mental capacity, personal responsibility, and treatability. However, using neurogenetic evidence risks framing mental illness through a narrow explanatory model — one relying solely on biological causes. Such evidence elicits both stigma-reducing and stigma-enhancing implicit biases against

[Top 10 Replicated Findings From Behavioral Genetics]

Robert Plomin, John C. DeFries, Valerie S. Knopik, and Jenae M. Neiderhiser

In the context of current concerns about replication in psychological science, the authors describe 10 findings from behavioral genetic research that have replicated robustly.

mental illness, which can manifest themselves in beliefs that a person with mental illness is less blameworthy for his condition, but also more dangerous and less receptive to treatment. These implicit biases affect jurors (and potentially judges) and may influence sentencing decisions in cases involving defendants with mental illnesses, including ultimate sentencing decisions in capital cases. This commentary argues that the use of neurogenetic evidence in the courtroom may harm defendants with mental illnesses because the nature of the evidence primes negative implicit biases against mental illness. It then explores how this dynamic plays out during the sentencing phase in capital cases involving defendants with mental illnesses.

Stability of Genetic and Environmental Contributions to Anxiety Symptoms in Older Adulthood

Andrew J. Petkus et al.

Anxiety symptoms are common in later life and are associated with diverse adverse health outcomes. Little is known about how genetic and environmental influences on anxiety symptoms might vary across older adulthood. The purpose of this study was to explore change and stability of contributions to anxiety symptoms across older adulthood. The authors examined data from the Swedish Adoption/Twin Study of Aging (SATSA). Between the years 1984 and 2010, 2021 participants (including 753 complete twin pairs) completed up to seven assessments containing two measures of anxiety symptoms.The findings suggest that the heritability of anxiety symptoms may increase later in life, at ages 75 to 80. Physiological factors associated with aging are discussed as potential factors explaining this increase.

Genes, Culture and Conservatism: A Psychometric-Genetic Approach

Inga Schwabe , Wilfried Jonker, and Stéphanie M. van den Berg

These are “big” findings, both in terms of effect size and potential impact on psychological science. They also consider reasons specific to behavioral genetics that might explain why these findings replicate.

Also see:

Weak Genetic Explanation 20 Years Later: Reply to Plomin et al.

Erik Turkheimer

Why Behavioral Genetics Matters: Comment on Plomin et al.

James J. Lee and Matt McGue

Call for Abstracts:

Reductionism and Integration, Bioethics and Behavior: A Conference Celebrating the Career of Kenneth F. Schaffner

Abstract submissions are sought for papers by junior scholars (graduates and postgraduates who have completed their PhDs within the past six years) to be delivered at a conference on September 23-24, 2016, which will celebrate the career of Kenneth F. Schaffner, and

The Wilson–Patterson conservatism scale was evaluated using homogeneity analysis and item response theory models. Results showed that this scale actually measures two different aspects in people: on the one hand people vary in their agreement with either conservative or liberal catchphrases and on the other hand people vary in their use of the "?" response category of the scale. Biometric results showed significant genetic and shared environmental influences, and significant genotype–environment interaction effects, suggesting that individuals with a genetic predisposition for conservatism show more non shared variance but less shared variance than individuals with a genetic predisposition for liberalism.

GWAS of 89,283 Individuals Identifies Genetic Variants Associated with Self-Reporting of Being a Morning Person

Youna Hu et al.

Circadian rhythms are a nearly universal feature of living organisms and affect almost every biological process. Our innate preference for mornings or evenings is determined by the phase of our circadian rhythms. The authors conducted a genome-wide association analysis of self-reported morningness, followed by analyses of biological pathways and related phenotypes, within the 23andMe participant cohort. They find that morningness is associated with insomnia and other sleep phenotypes; and is associated with body mass index and depression, but did not find evidence for a causal relationship in their Mendelian randomization analysis. The findings reinforce current understanding of circadian biology and will guide future studies.

Click here for further reading.

Comorbidity of Intellectual Disability Confounds Ascertainment of Autism: Implications for Genetic

engage intellectually with his forthcoming book, Behaving: What's Genetic, What's Not, and Why Should We Care?

Responses will be delivered by senior scholars. The deadline for abstracts is March 1, 2016.

In the News

Scientists Move Closer to Understanding Schizophrenia's Cause

The New York Times

A landmark study in the journal *Nature* received a great deal of media attention this month. The study outlines the different steps by which genetics influence schizophrenia, which is tied to a process called synaptic pruning, in which weak or redundant neuron connections are shed by the brain as it matures. The researchers are hopeful that this increased understanding of the genetic components of the synaptic pruning process will eventually lead to the discovery of

[Diagnosis]

Andrew Polyak, Richard M. Kubina, and Santhosh Girirajan

Current estimates of autism prevalence fail to take into account comorbidity of genetic factors toward risk of intellectual disability (ID) and epilepsy in autism diagnosis. The authors aimed to assess the effect of comorbidity on the diagnosis and prevalence of autism by analyzing 11 years (2000–2010) of special education enrollment data on approximately 6.2 million children per year. They found a 331 percent increase in the prevalence of autism from 2000 to 2010 within special education, potentially due to a diagnostic recategorization from frequently comorbid features such as ID. Some U.S. states showed significant negative correlations between the prevalence of autism compared to that of ID while others did not, suggesting state-specific health policy to be a major factor in categorizing autism. The results suggest that current ascertainment practices are based on a single facet of autism-specific clinical features and do not consider associated comorbidities that may confound diagnosis.

Click here for further reading.

biomarkers for schizophrenia, which in turn would aid in developing a more defined prognosis for patients.

[The Nature and Nurture of Melody: A Twin Study of Musical Pitch and Rhythm Perception]

Erik Seesjärvi et al.

Both genetic and environmental factors are known to play a role in our ability to perceive music, but the degree to which they influence different aspects of music cognition is still unclear. The author investigated the relative contribution of genetic and environmental effects on melody perception in 384 young adult twins. The participants performed three online music tests requiring the detection of pitch changes in a two-melody comparison task (scale) and key and rhythm incongruities in single-melody perception tasks (out-of-key, off beat). The results

showed predominantly additive genetic effects in the Scale task, shared environmental effects in the Out-of-key task, and non-shared environmental effects in the Off-beat task. This highly different pattern of effects suggests that the contribution of genetic and environmental factors on music perception depends on the degree to which it calls for acquired knowledge of musical tonal and metric structures.

Trim28 Haploinsufficiency Triggers Bi-stable Epigenetic Obesity

Kevin Dalgaard et al.

More than half a billion people are obese, and despite progress in genetic research, much of the heritability of obesity remains enigmatic. In this study, researchers identify an epigenetic 'on/off' switch for obesity. They find that the "obese-on" state is characterized by reduced expression of an imprinted gene network. Additionally, Adipose tissue transcriptome analyses in children indicate that humans cluster into distinct sub-populations, stratified according to Trim28 expression, transcriptome organization, and obesity-associated imprinted gene dysregulation. These data provide evidence of discrete polyphenism in mice and humans and thus carry important implications for complex trait genetics, evolution, and medicine.

Click here for further reading.

The Stress Regulator FKBP51 Drives Chronic Pain by Modulating Spinal Glucocorticoid Signaling

Maria Maiaru et al.

Polymorphisms in FKBP51 are associated with stress-related psychiatric disorders and influence the severity of pain symptoms experienced after trauma. We report that this gene is crucial for the full development and maintenance of long-term pain states, and blocking this

stress-related gene relieves chronic pain. Indeed, FKBP51 knockout mice, as well as mice in which silencing of FKBP51 is restricted to the spinal cord, showed reduced hypersensitivity in several persistent pain models in rodents. The findings suggest that FKBP51 regulates chronic pain by modulation of glucocorticoid signaling. Thus, FKBP51 is a central mediator of chronic pain, likely in humans as well as rodents, and is a new pharmacologically tractable target for the treatment of long-term pain states.

Click here for further reading.

The Phenotypic Legacy of Admixture Between Modern Humans and Neandertals

Corinne N. Simonti et al.

Many modern human genomes retain DNA inherited from interbreeding with archaic hominins, such as Neandertals, yet the influence of this admixture on human traits is largely unknown. The authors analyzed the contribution of common Neandertal variants to over 1000 electronic health record–derived phenotypes in ~28,000 adults of European ancestry. They discovered and replicated associations of Neandertal alleles with neurological, psychiatric, immunological, and dermatological phenotypes. Neandertal alleles together explained a significant fraction of the variation in risk for depression and skin lesions resulting from sun exposure, and individual Neandertal alleles were significantly associated with specific human phenotypes, including hypercoagulation and tobacco use. The results establish that archaic admixture influences disease risk in modern humans, provide hypotheses about the effects of hundreds of Neandertal haplotypes, and demonstrate the utility of EHR data in evolutionary analyses.