tion modeling to summary statistics from genetically correlated traits. With the method we perform exploratory and confirmatory factor analyses, allowing for sample overlap. Our initial analyses include GWAS summary statistics for addictive behaviors (n ~ 157,000), ADHD (n ~ 53,000), alcohol consumption (n ~ 414,000), antisocial behavior (n ~ 16,000), childhood aggression (n ~ 19,000), lifetime cannabis use (n ~ 32,000), impulsivity (N ~ 20,000) and several risk behaviors (n ~ 370,000 – 405,000). We present results from these initial analyses and our planned future directions.
Discussion: We will conclude with details about the Externalizing Consortium, in order to invite groups to participate in the next wave of analyses.

Disclosure: Nothing to disclose.

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SU89
TRAFFIC-RELATED AIR POLLUTION, APOE ε4 STATUS, AND NEURODEVELOPMENTAL OUTCOMES AMONG SCHOOL CHILDREN ENROLLED IN THE BREATHE PROJECT (CATALONIA, SPAIN)

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Background: Traffic-related air pollution is emerging as a risk factor for Alzheimer's disease (AD) and impaired brain development. Individual differences in vulnerability to air pollution may involve the ε4 allele of Apolipoprotein E (APOE) gene, the primary genetic risk factor for AD. We aim to analyze whether the association between traffic air pollution and neurodevelopmental outcomes is modified by APOE ε4 status in children.
Methods: Data on parent-reported behavior problems (total difficulties scores, Strengths and Difficulties Questionnaire), teacher-reported attention-deficit hyperactivity disorder (ADHD) symptom scores, cognitive performance trajectories (computerized tests of inattentiveness and working memory repeated 2–4 times during January 2012–March 2013), and APOE genotypes were obtained for 1,667 children aged 7–11 years attending 39 schools in or near Barcelona. Basal ganglia volume (putamen, caudate, and globus pallidum) was measured in 163 of the children by MRI (October 2012–April 2014.) Average annual outdoor polycyclic aromatic hydrocarbons (PAHs), elemental carbon (EC), and nitrogen dioxide (NO2) concentrations were estimated based on measurements at each school (two 1-week campaigns conducted 6 months apart in 2012).
Results: APOE ε4 allele carriers had significantly higher behavior problem scores than non-carriers, and adverse associations with PAHs and NO2 were stronger or limited to ε4 carriers for behavior problem scores (P-interaction 0.03 and 0.04), caudate volume (P-interaction 0.04 and 0.03), and inattentiveness trajectories (P-interaction 0.15 and 0.08, respectively). Patterns of associations with the same outcomes were similar for EC.
Discussion: PAHs, EC, and NO2 were associated with higher behavior problem scores, smaller reductions in inattentiveness over time, and smaller caudate volume in APOE ε4 allele carriers in our study population, while corresponding associations were weak or absent among ε4 non-carriers. These findings support a potential role of APOE in biological mechanisms that may contribute to associations between air pollution and neurobehavioral outcomes in children. Replication in other population settings is needed to establish the potential role of APOE gene in neurobiological susceptibility to air pollution.

Disclosure: Nothing to disclose.

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SU90
USING MEDICATION DATA TO DEFINE PSYCHIATRIC PHENOTYPES AND RECRUIT LARGE SAMPLES FOR GWAS

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Background: It has become abundantly clear that the key to progress in psychiatric genetics is large samples for GWAS. How to collect these quickly and cheaply?
Methods: We are using data from the Australian Pharmaceutical Benefit Scheme (which centrally registers 99% of all prescriptions) to ascertain cases through their prescription information.
Results: So far, we have collected DNA from >15,000 people with >4 prescriptions for SSRIs of whom 93% report depression as a first diagnosis; 60% also report anxiety. We have also found >2000 ADHD cases through prescriptions for Ritalin etc.
Discussion: This year we are launching recruitment of bipolar cases via lithium prescriptions and schizophrenia via clozapine. We have applied for funding to extend this model to alcoholism [acamprosate, naltrexone] and Alzheimer's disease [memantine].

Disclosure: Nothing to disclose.

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