Behav Genet (2008) 38:612–653
DOI 10.1007/s10519-008-9228-x

ABSTRACT

Behavior Genetics Association 38th Annual Meeting Abstracts

Published online: 11 October 2008

Further Evidence for Association Between GABRA2 and Nicotine Dependence in a Sample of U.S. and Australian Adults

Arpana Agrawal[1], Michele Pergadia[1], Sumitra Balasubramanian[1], Anthony Hinrichs[1], Scott F. Saccone[1], Naomi Breslau[2], Dorothy Hatsukami[3], Eric Johnson[4], Nicholas Martin[5], Grant Montgomery[5], Alison Goate[1], John Rice[1], Laura Bierut[1], Pamela A. F. Madden[1]

[1]Washington University in St. Louis; [2]Michigan State University; [3]University of Minnesota; [4]RTI International; [5]Queensland Institute of Medical Research

We recently demonstrated evidence for association between several single nucleotide polymorphisms (SNPs) in the gamma-aminobutyric acid, receptor A, subunit 4 (GABRA4) gene. Evidence was also found for a synonymous polymorphism (rs279858) in the gamma-aminobutyric acid, receptor A, subunit 2 (GABRA2) gene. We used a case/control sample where cases were defined as a score of 4+ on the Fagerstrom Test for Nicotine Dependence (FTND, $N = 1{,}050$ cases), and controls had an FTND score of 0 ($N = 879$ controls) in exposed cigarette smokers (smoked 100+ cigarettes in their lifetime) (Agrawal et al., in press. Addiction). Through acompetitive application to the Center for Inherited Diseases Research (CIDR), we have genotyped 24 and 39 additional SNPS in GABRA4 and GABRA2 in this sample, which contains both U.S. and Australian adults. We tested for association using the computer program PLINK (Purcell et al. Am J Hum Genet 81:559–575, 2007). We found widespread evidence for association with nicotine dependence, with 24 signals in GABRA2 ranging from $P = 0.02$ to 0.002. The GABRA4 results suggest the association was limited to a region we had previously identified—no other SNPs were significant. SNPs in GABRA2 also demonstrated significant interactions with sex, and the odds-ratios more significant in men versus women. No evidence for epistatic interactions were found between GABRA4 and GABRA2. Results from these joint analyses present GABRA2 as an emerging candidate gene for nicotine dependence. This is a particularly interesting finding given several independent replication studies of the association between SNPs in GABRA2 with alcohol and illicit drug dependence.

Early Cannabis Use and Later Stages of Cigarette Smoking: A Twin Study

Arpana Agrawal[1], Michael Lynskey[1], Michele Pergadia[1], Kathleen Bucholz[1], Andrew Heath[1], Nicholas Martin[2], Pamela A. F. Madden[1]

[1]Washington University in St. Louis; [2]Queensland Institute of Medical Research

Evidence suggests that cannabis users are at an increased risk for elevated levels of cigarette smoking. We investigated whether, in those exposed to cigarettes, cannabis use prior to age 17 is associated with an increased likelihood of nicotine dependence and persistent smoking, even after controlling for genetic and environmental factors that contribute to the liability to both early cannabis use and problematic cigarette smoking. A population-based cohort of 24–36 year old Australian male and female twins ($N = 6{,}257$) was used. The cotwin-control method, with twin pairs discordant for early cannabis use, was used to examine whether after controlling for genetic and familial environmental background, there was evidence for an additional influence of early cannabis use on problematic cigarette smoking (nicotine dependence and persistent smoking). The early cannabis-using twin was 1.9 and 2.6 times more likely to report nicotine dependence and persistent smoking respectively, when compared to their co-twin who had experimented with cigarettes but never used cannabis. Even when analyses were restricted to cannabis users, earlier age of onset conferred greater risk (1.7–2.3) for cigarette smoking than did later onset of cannabis use. This association was largely governed by common genetic liability to early cannabis use and problematic cigarette smoking as demonstrated by genetic correlations of 0.41–0.70. Therefore, early-onset cannabis users are at increased risk for progressing to more problematic stages of cigarette smoking, but this risk is largely attributable to common familial vulnerability. There is no evidence for the causal underpinnings of the 'reverse gateway'.

Selection on Rutgers Alcohol Problem Index (RAPI) Scores in Adolescence Predicts High Rates of Alcohol Dependence in Young Adulthood

Fazil Aliev[1], Danielle M. Dick[1], Richard J. Viken[2], Torsten Winter[3], Eero Vuoksimaa[3], Anja Häppölä[3], Mari Siltala[3], Jaakko Kaprio[4], Richard J. Rose[5]

[1]Virginia Institute for Psychiatric and Behavioral Genetics, VCU, Richmond, VA; [2]Indiana University, Bloomington, IN; [3]University of Helsinki; [4]University of Helsinki & National Public Health Institute, Finland; [5]Indiana University, Bloomington, IN

FinnTwin16-25 is a population-based twin study of five consecutive birth cohorts of twins (born 1975–1979), identified through Finland's Central Population Registry. The twins were assessed via self-report questionnaire at ages 16, 17, and 18 and 25. The Rutgers Alcohol Problem Index (RAPI) was used to assess negative consequences of drinking at age 18. The Finnish adaptation of the RAPI contained 22 items, with 4 response alternatives for reporting frequency of each consequence, including items assessing injury to self and others, neglected responsibilities, emotional problems, and personal and interpersonal loss associated with drinking. A subset of the twins ($N = 602$), representing pairs selected for extreme discordance/concordance for elevated RAPI scores at age 18, were enrolled into a laboratory follow-up at mean age 25.6 (st.dev = 1.3), as part of an effort to identify the consequences of adolescent alcohol use (grant AA12502 to RJR). Within a day-long laboratory protocol including ERP and neuropsychological tests, these twins were interviewed with the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA), which permits DSM diagnoses of most major psychiatric disorders. Rates of DSMIII-R alcohol dependence were very high in this sample ($N = 270$; 45%). The correlation between RAPI scores at age 18 and alcohol dependence symptoms assessed at interview 7 years later was 0.50. To our knowledge, this study represents one of the first prospective interview follow-up of individuals selected for problem drinking with RAPI scores in late adolescence, and demonstrates the utility of this measure for identifying individuals at high risk for developing dependence.

Genetic Differences in Childhood Disorders of Self-Regulation

Robert Althoff[1,2], Lynsay Ayer[1,2], Timea Nelson[1,2], Erik Ehli[1,2], Gareth Davies[1,2], James Hudziak[1,2]

[1]University of Vermont College of Medicine, Burlington, VT; [2]Institute of Human Behavioral Genetics, Avera Behavioral Health & South Dakota State University College of Pharmacy, Brookings, SD

Objective As part of our study of children with profound disturbance in self-regulation, we examined two putative candidate genes to see if the genetics of the Child Behavior Checklist-Dysregulation Profile (clinical levels of Attention Problems, Aggression, and Anxious/Depression) was different from DSM disorders. *Method* 449 children from a large family study in Vermont were genotyped on the two allele system of the catechol O-methyltransferase gene (COMT) and the three allele system of the 5-HTTLPR gene. DNA was obtained by cheek swabs. The percentage of children homozygous for the Val allele of the COMT gene and/or homozygous for the short allele was compared between those with the CBCL-DP versus DSM mania or ADHD + ODD. Path analyses were performed in MPlus to control for familial clustering. *Results* The CBCL-DP group was significantly different from DSM mania on the COMT gene with more val homozygotes in the mania group. The CBCL-DP group was significantly different from the ADHD + ODD group on the 5-HTTLPR with fewer short allele homozygotes in the ADHD + ODD group. *Conclusions* There is preliminary evidence that the CBCL-DP is different on the molecular level from DSM mania and from ADHD + ODD. This is consistent with the hypothesis that this profile is a measure of poor self regulation rather than mania and is not simply "comorbidity". However, more work with more subjects is needed to have the power to confirm these findings.

How do High School Students Learn? Genetics of Academic Performance, Learning Attitude, and School Environment

Juko Ando[1], Kou Murayama[2], Shinji Yamagata[2], Chizuru Shikishima[1], Yusuke Takahashi[3], Koken Ozaki[4], Koichi Nonaka[5]

[1]Faculty of Letters, Keio University; [2]Japan Society for the Promotion of Science; [3]Graduate school of arts and sciences, University of Tokyo; [4]Japan Science and Technology Agency; [5]Wako University

Academic performance (AP) is affected by many factors such as students' motivation, learning styles, efforts, personality traits, and teachers' styles of teaching. The current study investigated how these factors are related genetically and environmentally by 490 pairs of Japanese junior high twins (289 MZ, 101 same sex DZ, 101 opposite sex DZ) and 395 pairs of senior high twins (228 MZ, 79 same sex DZ, 88 opposite sex DZ). Intrinsic motivation (IM) and competitive attitude (CA) were the best predictors for AP, and additive genetic factor mediated for IM and shared environment mediated for CA. Students' effortful attitude and perception of teachers' intimacy were genetic correlates for AP. Conscientiousness environmentally correlated with AP. Study time was affected by additive genetic when students were in junior high school, and affected by shared environment when they were in senior high school. Educational implications will be discussed.

Intelligence and Semen Quality are Positively Correlated

Rosalind Arden[1], Linda S. Gottfredson[2], Geoffrey Miller[3], Arand Pierce[3]

[1]King's College London; [2]University of Delaware; [3]University of New Mexico

Why is general intelligence correlated with so many important health outcomes? If the positive correlations among cognitive abilities are part of a larger matrix of positive correlations among fitness-related traits, then intelligence ought to correlate with important but seemingly unrelated traits—such as semen quality. As hypothesized, we found significant positive correlations between intelligence and 3 indices of semen quality (sperm concentration, sperm count and sperm motility) in a sample of US Veterans. These correlations were not mediated by age, body mass index, days of sexual abstinence, service in Vietnam, or use of alcohol, tobacco, marijuana, or hard drugs. These results suggest that the link between intelligence and physical health may reflect general phenotypic condition, and not just lifestyle factors.

Etiology of the Stability of Reading Deficits: An Update

Raven L. Astrom, Sally J. Wadsworth, John C. DeFries

Institute for Behavioral Genetics, University of Colorado, Boulder

Previous longitudinal studies of reading difficulties have indicated that reading deficits are generally stable. Nonetheless, relatively little is know about the etiology of this stability. In an earlier analysis of data from participants in the Longitudinal Twin Study of Reading Disabilities (Astrom et al. Twin Res Hum Genet 10:434–439, 2007), we observed substantial and significant longitudinal stability and bivariate heritability for reading deficits. However, the sample of proband pairs is now approximately 25% larger. Thus, the primary objectives of this present study are to assess genetic and environmental influences on the stability of reading deficits using data from this larger sample, and to estimate bivariate heritability using a bivariate extension of the Purcell and Sham (Behav Genet 33, 271–278, 2003) model-fitting implementation of the DeFries-Fulker multiple regression model for analysis of selected twin data (DeFries et al. Behav Genet 15:467–473, 1985). Data were analyzed from 71 twin pairs, (21 MZ and 50 DZ), in which at least one member of each pair was classified as reading-disabled and on whom follow-up data were available. The twins were tested at two time points (average age of 9.9 years at initial assessment and 15.4 years at follow-up). A composite measure of reading performance (Reading Recognition, Reading Comprehension and Spelling) was highly stable, with a stability correlation of 0.85. When data from the initial time point were analyzed, the resulting estimate of heritability of the group deficit (h2 g) was 0.83 ($\pm$0.29, $P < 0.006$). When the initial and follow-up data were fitted to a bivariate extension, bivariate heritability was estimated at 0.73, suggesting that more than two-thirds of the proband reading deficit at initial assessment is due to genetic factors that also influence reading difficulties at follow-up.

COMT is Not Associated with Externalizing Behavior in Children with ADHD

Lynsay Ayer[1,2], Robert Althoff[1,2], Timea Nelson[1,2], Erik Ehli[1,2], Gareth Davies[1,2], James Hudziak[1,2]

[1]University of Vermont College of Medicine, Burlington, VT; [2]Avera Institute for Human Behavioral Genetics, Avera Behavioral Health Center, Sioux Falls, SD & South Dakota State University College of Pharmacy, Brookings, SD

Objective To test whether recent findings (Caspi et al. Arch Gen Psychiatry 65:203–210, 2008) that carriers of the valine/valine (Val/Val) polymorphism of the catechol O-methyltransferase gene (COMT) exhibit more symptoms of conduct disorder and aggressive behavior would replicate in a sample of 211 children with ADHD. *Method* A total of 196 children with ADHD participated in this study. ADHD and conduct disorder symptoms were measured using the Vermont Structured Diagnostic Interview (VSDI). The Child Behavior Checklist (CBCL) Aggressive Behavior, Delinquent Behavior, Attention Problems, and Externalizing Problems raw scale scores were used to assess non-DSM externalizing behaviors. DNA was obtained by cheek swabs from 100% of the children included in this study. Following DNA isolation, samples were genotyped for the COMT val158met polymorphism using established methods. Path analyses were used to test whether children homozygous for the Val allele are more likely to exhibit conduct problems. *Results* Neither conduct disorder symptoms nor the CBCL measures of externalizing behavior were related to COMT. *Conclusions* Caspi's findings were not replicated using CBCL measures of externalizing behavior. Potential explanations for these findings include: (1) differences in sample size, (2) differences in DSM-IV interviews, (3) inclusion of clinic-referred children and their non-referred siblings in our sample, (4) over-sampling of children with aggression and inattention in our sample.

Examination of the Developmental Change in Genetic Risk for Substance Use from Early Adolescence to Young Adulthood

Jessica Baker[1], Hermine Maes[1], Paul Lichtenstein[2], Kenneth Kendler[1]

[1]Virginia Institute for Psychiatric and Behavioral Genetics; [2]Karolinska Institutet

Objective Previous twin studies have demonstrated that the genetic and environmental influences on the development of substance use and disorders varies across development (e.g. Hicks et al. 2007; Rose et al. 2001) and that the genetic risk for substance use may not be substance specific, but general across substances (e.g. Kendler et al. 2003). Despite this much less is known about whether a developmental trajectory is seen across this general substance use factor. *Methods* Self-reported substance use (nicotine, alcohol, and illicit drug) was obtained at ages 13–14, 16–17, and 19–20 from 1,480 twin pairs in the Swedish Twin Study of child and Adolescent Development. Multivariate longitudinal twin analysis was conducted with Mx (Neale 1997). A model allowing for quantitative and qualitative sex-effects will be later examined. *Results* Results indicate a common factor is largely responsible for substance use at all three time points, regardless of substance. A stable cross-time factor structure was shown for illicit substance use however, decreased cross-time for nicotine and alcohol use. Generally, as age increased, common genetic effects declined and became more substance specific. However, genetic effects did increase in importance from mid-adolescence to young adulthood. In contrast, the impact of common unique environmental influences increased with age while common shared environmental influences remained relatively stable until young adulthood during which almost no common or specific shared environmental effects were indicated. *Discussion* Genetic and environmental influences for substance use are partly mediated through a common factor and are also partly substance-specific. Over development, genetic effects seem to be responsible for changes in type of substance use. Common shared environmental effects appear less important with increasing age while common unique environmental effects increase in importance.

A Genome-Wide Linkage Scan for Wellbeing

Meike Bartels[1], Marleen H. M. de Moor[1], Gonneke Willemsen[1], Jouke-Jan Hottenga[1], Danielle Posthuma[1], James J. Hudziak[2], Dorret I. Boomsma[1], Eco J. C. de Geus[1]

[1]Department of Biological Psychology, VU University, Amsterdam, the Netherlands; [2]Departments of Psychiatry and Medicine (Division of Human Genetics), Center for Children, Youth and Families, University of Vermont, College of Medicine Burlington, Burlington, Vermont USA

While it is commonly accepted that genetic factors influence variation in psychopathology, the suggestion that genetic factors also determine psychological wellbeing often elicits surprised reactions. So far, a few studies investigated sources of variance in well-being from the perspective of behavior genetics. Overall these studies show substantial heritability with estimates in the range of 40–50%, while the

remaining variance is accounted for by environmental influences unique to an individual (See among others: Stubbe et al. Psychol Med 35:1581–1588, 2005). In an attempt to identify the genes underlying the heritability of wellbeing we used twin-family data from families participating in the 2002 wave from an on-going biennial survey study on health and lifestyle in the Netherlands Twin Register (NTR; Boomsma et al. Twin Res Hum Genet 9:849–857, 2006). Wellbeing was measured with the Satisfaction with Life Scale (Diener et al. J Pers Assess 49:71–75, 1985) and the Subjective Happiness Scale (Lyobomirsky and Lepper, Soc Indic Res 46:137–155, 1999). Genetic factor analyses will be used to obtain a genetic factor score for wellbeing based on these two measures. A univariate variance components-based linkage scan, using the genetic factor score and marker data from a genome-wide 10.6-cM microsatellite scan, is carried out to localize regions significant for wellbeing.

Dyslexia and SLI: Evidence for Substantial Genetic Independence

Timothy Bates[1], Michelle Luciano[1], Penelope Lind[2], Margie Wright[2], Nick Martin[2]

[1]University of Edinburgh; [2]QIMR

Dyslexia and SLI are common, heritable communication disorders. It has been suggested that they define severe and mild deficits on a common language-function dimension, or alternatively that they define two quadrants of a 2-dimensional space of comprehension and phonological processing. Here we propose that SLI and dyslexia are largely unrelated disorders of speech-sound processing and grapheme processing respectively, and that their comorbidity arises because both disorders are exacerbated (and are diagnosed more often) in the presence of poor comprehension: specifically impoverished semantic representation and low working memory. Multivariate genetic modelling is used to test this hypothesis.

Testing the Social Intelligence Hypothesis of Human Brain Evolution

Timothy C. Bates

University of Edinburgh

Brain volume has roughly tripled in humans compared to their nearest primate relatives, the great apes. This poses a problem for adaptationists: what selective benefit favored the increase in size of this expensive tissue? Several hypotheses have been advanced, from language and tool use, to simple allometric scaling. Among primates, large differences in brain size/body weight ratios have been observed, and this increasing encephalisation correlates with social group-size, measured for instance by the size of grooming cliques. Byrne, Dunbar and others have suggested that the selective pressure on brain volume reflects benefits of large group sizes: the "social intelligence hypothesis". Here, I take advantage of the fact that human brain size shows significant heritable variance, to test the social correlates of human head size. Data are presented replicating the link between head-size and cognitive ability, and demonstrating a link between cognition and entrepreneurism, but finding no relationship between group size and either head size or intelligence. Follow-up experiments confirmed this lack of association between IQ and group size, and between IQ and ability to form groups over a 6-month novel social-environment situation. Instead variance in group size was related to personality and empathy measures, which mostly have been argued to be selectively neutral. Other potential dimensions of human selection were examined—among them altruism, temperance, fortitude, time-discounting, self-control, and humility. These are largely unrelated to variance in head size, but cross- species comparisons indicate that these may have been selected in the hominid line, perhaps forming a context which supports positive selection favouring for neocortical volumes and general cognitive ability.

Genetic Epidemiology of Nicotine Dependence

Andrew W Bergen[1], David V. Conti[2], David Van Den Berg[2], Wonho Lee[2], Jinghua Liu[2], Dalin Li[2], Nan Guo[1], Huaiyu Mi[1], Paul D. Thomas[1], Faith Allen[3], Christina Lessov-Schlagger[1], Ruth Krasnow[1], Ruhong Jiang[1], Denise Nishita[1], Yungang He[1], Judy Andrews[4], Elizabeth Tildesley[4], Hyman Hops[4], Rachel Tyndale[5], Neal L. Benowitz[3], Caryn Lerman[6], Gary E. Swan[1]

[1]SRI International; [2]USC; [3]UCSF; [4]Oregon Research Institute; [5]University of Toronto; [6]University of Pennsylvania

The Fagerström Test for Nicotine Dependence (FTND) assesses quantity of cigarettes smoked and time- and place-dependent characteristics of the respondent's smoking behavior. The present candidate gene association analysis of FTND utilized $N = 833$ treatment seeking cigarette smokers (50% female) enrolled in either of two smoking cessation trials and who completed at least one clinic visit. In this sample, the mean (SD) FTND assessed at baseline (before quitting) was 5.3 (2.2), and, in univariate analysis, FTND was significantly ($P < 0.01$) associated with age, depression, education, sex and study site. Germline and whole genome amplified (WGA) DNA from these individuals was genotyped at $N = 1303$ single nucleotide polymorphisms (SNPs) at 57 monoamine, neuropeptide, nicotinic and intracellular signaling candidate genes using the BeadArray platform from Illumina. After genotype and sample QC, the final analytic sample was comprised of $N = 793$ individuals and $N = 1214$ SNPs. Multivariate logistic regression analysis of association to FTND was performed and SNPs from dopamine transporter gene (SLC6A3) were found to be significantly ($P < 0.01$) associated with FTND. Analyses stratified by sex revealed that SNPs at the dopamine transporter and at the erythrocyte binding protein 4.1 like 2 loci were significantly associated to FTND in the female strata, and SNPs at the D3 dopamine receptor locus were significantly associated to FTND in the male strata. A second, community based, pedigree cohort ($N = 149$ pedigrees), selected on the basis of a high density of smoking (at least 3 ever smokers per pedigree), was analyzed using family based association analysis to evaluate the association of SNPs found to be significantly associated with FTND in the treatment seeking smoker sample. Three of four dopamine transporter SNPs found to be significantly associated with FTND in the treatment seeking smoker sample were found to be significantly associated with FTND in the community based smoker sample.

Smoking Initiation and Persistence: First Results for Genome-Wide Association Analyses in Dutch Adults

D. I. Boomsma[1], J. M. Vink[1], P. Sullivan[2], J. H. Smit[3], W. Hoogendijk[3], J. J. Hottenga[1], G. Willemsen[1], E. J. C. de Geus[1], B. W. Penninx[4]

[1]Dept Biol Psychol, VU University, Amsterdam, Netherlands; [2]Dept Genetics, Psychiatry, Epidemiology, Univ North Carolina, Chapel Hill, NC, USA; [3]Dept Psychiatry, EMGO Inst, Inst Neuroscience, VU

Medical Center, Amsterdam, The Netherlands; [4]Dept Psychiatry, EMGO Inst, Inst Neuroscience, VU Medical Center, Amsterdam; Dept Psychiatry, Univ Medical Center Groningen, Univ Groningen, Groningen; Dept Psychiatry, Leiden University Medical Center, Leiden, The Netherlands

The influence of genetic factors on several aspects of smoking behavior has been well documented. In order to identify the genetic variants that may explain the heritability for smoking behavior we carried out a genome-wide association analysis (GWA) for "ever-smoking" and "current-smoking". Phenotype and genotype data were available for 3469 unrelated subjects. Subjects were recruited through the Netherlands Twin register (NTR; Boomsma et al. Twin Res Hum Genet 9(6):849–857, 2006) and the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al. Int J Meth Psychiatry Res, in press). Genotyping was funded through the GAIN initiative (US Foundation for the National Institutes of Health Genetic Association Information Network) and was carried out at Perlegen Sciences. After quality control, 75% of SNPs were retained for analyses (427,037 autosomal SNPs). Logistic regression analyses with covariates sex and age were carried out in Plink. First, we selected all SNPs with a P-value <0.005 in either the GWA for current-smoking or the GWA for ever-smoking and explored which SNPs were located in a gene. We have compared those results to results of previous GWA or candidate gene studies. We replicated 53 genes (with $P < 0.005$ for 1 or more SNPs) that were associated with nicotine dependence or addiction vulnerability in other studies. Next we focused on the most significant findings (SNPs with a P-value of <10–14 for either ever-smoking or current-smoking). Some of these top SNPs are located within candidate genes for smoking. All SNPs with $P < 10–14$ will be explored in independent replication samples.

Impulsive Buying and Personality: A Croatian Twin Study

Denis Bratko[1], Ana Butkovic[1], Michael Bošnjak[2]

[1]Department of Psychology, Faculty of Humanities and Social Sciences, Croatia; [2]School of Economics and Management, Free University of Bozen-Bolzano, Italy

Contrary to approaches favoring rational processes in consumer decision making, human buying behavior is often guided by spontaneously activated wants, needs, emotions, and moods. A variety of these apparently 'irrational' antecedents of consumption have been summarized under the rubric impulsive buying, which is basically characterized by intense emotions and a lack of planning and deliberation. While prior research has indentified two core domains associated with the propensity to buy impulsively, namely extraversion and conscientiousness, the etiology of impulsive buying tendencies remains unclear. Specifically, the amount of genetic versus environmental factors contributing to the development of impulsive buying tendencies is largely unexplored. To fill this gap, we employed a classical twin design. The sample consisted of 330 twin pairs aged between 16 and 22 years. Several instruments were employed: (1) The impulsive buying scale measuring impulsive buying tendencies and encompassing cognitive as well as emotional components, (2) Measure of five broad personality domains (Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness), and (3) Measure of the lower-level impulsivity trait. To estimate the relative genetic/environmental contribution to individual differences in impulsive buying and to estimate genetic/environmental overlap between impulsive buying and personality, both univariate as well as bivariate behavioral genetic analyses were performed.

Early Risk Behaviors and Later Psychological Outcomes: A Discordant Twin Design

Jacob P. Buchanan, Matt McGue, William G. Iacono

Department of Psychology, University of Minnesota

Participation in problematic and risky behaviors during adolescence indicates a high, generalized risk of developing adult psychopathology (McGue and Iacono, Am J Psychiatry 162:1118–1124, 2005). Risky behaviors during adolescence may be early indicators of underlying genetic liability or possible causal (environmental) mechanisms for the development of later psychopathology. To help disambiguate these mechanisms, we identified 49 monozygotic twin pairs within the Minnesota Twin and Family Study (supported in part by USPHS grants AA09367 and DA05147) who were discordant for engaging in six early risk behaviors during adolescence, including alcohol use, nicotine use, cannabis use, other drug use, police contact, and sexual intercourse. As compared to their control co-twins, defined as having participated in none of the previously listed early risk behaviors, the twins who had engaged in at least two of those early risky behaviors showed significantly more symptoms of Adult Antisocial Behavior ($t(48) = -2.932$, $P = 0.005$) and Major Depression ($t(48) = -3.595$, $P = 0.001$) and marginally more symptoms for Alcohol Abuse and Dependence ($t(48) = -2.001$, $P = 0.051$) by age 20. Mean differences for symptoms of Nicotine Dependence, Cannabis Abuse and Dependence, and Other Drug Abuse and Dependence were not significant, but do vary in the expect direction. Our findings are supported with correlations between the difference in early risk behaviors engaged in and the difference in psychopathological symptom outcomes within twin pairs with an average r of 0.16. These correlations remained robust when variance due to the difference in Attention Deficit/Hyperactivity Disorder symptoms occurring before age 7 in the twin pairs was partialled out. Our discordant twin analysis is consistent with the hypothesis that early problem behavior is a marker of both genetic vulnerability and environmental risk for later mental health and substance abuse problems.

A Fresh Look at the Shared Environment: Results from a Large Meta-Analysis of Twin and Adoption Studies Examining Child and Adolescent Psychopathology

S. Alexandra Burt

Department of Psychology, Michigan State University

Behavioral genetic research has generally concluded that the more important environmental influences on psychological and behavioral disorders result in differences between siblings (i.e., non-shared or child-specific; E). By contrast, environmental influences that create similarities between siblings (i.e., shared or family-wide; C) have historically been found to be statistically indistinguishable from zero and have thus been largely dismissed. Importantly, however, there is mounting evidence that, during childhood and adolescence in particular, shared environmental influences may make important contributions to most forms of psychopathology. The aim of the current study was to explicitly evaluate this hypothesis. I thus conducted a meta-analysis of all twin and adoption studies ($n = 498$) of internalizing and externalizing psychopathology during childhood and adolescence. Results revealed the presence of moderate and significant shared environmental influences on conduct disorder, oppositional defiant disorder, anxiety, depression, and broad internalizing and externalizing disorders, with estimates ranging from 10% to 19% of the total variance. Importantly, these estimates did not vary

across twin and adoption studies, suggesting that they are not a function of passive rGE, but instead reflect "true" environmental influences. Moreover, shared environmental influences persisted across age, informant, and to a lesser extent, assessment method, suggesting that such findings are robust to study design and methodology. Indeed, the only disorder examined that did not demonstrate significant C was attention-deficit/hyperactivity disorder, which instead appeared to be largely genetic (and particularly non-additive genetic) in origin. Implications of these findings will be discussed.

Cooperation Development in Young Twins

Katie Butera, Lisabeth F. DiLalla

This study examined the difference between the cooperation levels of monozygotic (MZ) twins and dizygotic (DZ) twins, with previous research showing conflicting findings. It was hypothesized that MZ twins would exhibit higher levels of cooperation than DZ twins and that cooperation development would contain a genetic component. The sample was from the Southern Illinois Twins and Siblings Study (SITSS; DiLalla, Twin Res 5(15):468–471, 2002) and consisted of 86 twin pairs (27 MZ; 40 same-sex DZ; 19 opposite-sex DZ). Participants engaged in a video-recorded triadic interaction with their co-twin and parent, later coded for cooperative and non-cooperative behaviors. Correlations showed that similarity in cooperation was highly significant for all twin pair types (MZ, same-sex DZ, and opposite-sex DZ). However, there were no significant differences across zygosity types. Non-cooperation correlations showed that MZ twins were more similar to each other than were same-sex DZ twins, suggesting a heritable component. In addition, non-cooperation similarity for opposite-sex DZ twins was higher than for same-sex DZ twins, suggesting that sex of co-twin has an effect on non-cooperation levels. In addition, it was found that a lower Theory of Mind perspective-taking ability resulted in an increased level of general non-cooperation. Thus, although cooperation was similar for all types of twins, non-cooperation appears to reflect a more genetically influenced behavior that also is influenced by cognitive ability.

The Effects of Siblings on Twins and Singletons in Theory of Mind Tasks

Katie Butera, Ashley L. Melby, Kristin Bell, Sara W. Biebl, Sarah Long, Angela Phillips, Lisabeth F. DiLalla

Individual differences related to familial characteristics may be influential factors in theory of mind (ToM) development. Recently, a few researchers have attempted to investigate whether or not twins, in spending a significant amount of time with one another, are more likely to develop theory of mind sooner than other sibling pairs. Some findings have suggested that there are no significant differences between twins and other sibling pairs in the development of ToM (Cassidy et al. Child Dev 76(1):97–106, 2005), whereas other studies have noted that close-in-age siblings may increase ToM performance (Peterson, Cognitive Dev 15:435–455, 2000). We examined this more thoroughly. The sample consisted of 65 twin pairs (20 MZ, 45 DZ) and 74 singleton children between the ages of 3 and 5 years. Each child was tested on ToM false belief tasks. There was no significant difference between MZ twins, DZ twins, and singletons on ToM, or between children with and without siblings. However, having a greater number of younger siblings was related to an increased ToM. In addition, a larger age difference between the child and younger sibling was related to increases in ToM. Increased parental sensitivity also was related to enhanced ToM performance for the twins in this sample. Therefore, having a twin does not appear to enhance ToM. However, having younger siblings does appear to have an effect, possibly because children need to use ToM skills in order to communicate effectively with younger siblings.

Gene-Environment Interaction Problems

Gregory Carey

Department of Psychology and Institute for Behavioral Genetics, University of Colorado

There are several different models for gene-environment interaction when genotype is a latent variable and the "environment" is a continuous moderating phenotype. These are the regression model of Labuda, DeFries, and Fulker; the Mx definition-variable approach used by Dick, Turkheimer, and others; and an analytical formulation by Purcell. Unfortunately, these do not give the same answers when applied to the same simulated data set. A new approach is developed within the context of traditional, linear, path-models based on solving for expected values and variance-covariance matrices using a conditional likelihood. This approach can be generalized to the multivariate case of more than two relatives in a pedigree, more than one moderating phenotype, and more than one phenotype of interest.

Genes for g: A Novel Method for Analyzing Data from Genome-Wide Association Studies

Gregory Carey

Department of Psychology and Institute for Behavioral Genetics, University of Colorado

With the decreasing cost of DNA arrays, it will not be long before it is economically feasible to genotype individuals on hundreds of thousands of single-nucleotide polymorphisms and g will be one of the first behavioral traits studied. There is also an increasing likelihood that many normal behavioral traits are "hyperpolygenic," i.e., the maximum variance accounted for by a locus is less than 1%. Under these circumstances, analytical methods for data analysis must be as powerful as possible. Here I describe a new approach to the analysis of genome-wide association data on a continuous phenotype. This approach is based on distributional mixture-models of summary statistics and it may identify up to 5-fold more trait-loci than traditional methods that use corrected P values or false discovery rates.

Heritability of Economic Behavior: Field and Laboratory Evidence from Sweden

David Cesarini[1], Paul Lichtenstein[2], Magnus Johannesson[3], Bjorn Wallace[3], Orjan Sandewall[3]

[1]Department of Economics, MIT; [2]Department of Medical, Epidemiology and Biostatistics, Karolinska Institutet; [3]Department of Economics, Stockholm School of Economics

Merging data from the Swedish Twin Registry with data from Statistics Sweden, the National Service Administration and The Swedish Social Insurance Agency, we report estimates of heritability for a variety of socioeconomic indicators, financial risk-taking as well as their bivariate heritabilities with IQ. Our results are consistent with

prior literature suggesting a moderate to strong genetic component for a variety of socioeconomic outcomes. In previous papers, we have shown that economic preferences elicited experimentally are under moderate genetic influence. Here, we extend this work in two directions. First, we provide new measurement-error corrected estimates of heritability, based on new laboratory data from subjects retested in the spring of 2008. Second, we examine the extent to which laboratory measures of preferences predict economic decisions made outside the laboratory (e.g. pension-fund investments).

Get Rid of Tedious Coding for Activity Level?

Sonia Chawla, Manjie Wang, Kimberly J. Saudino

Boston University

Measurement of temperament has been the subject of some controversy, with differing opinion over the detail necessary for behavior ratings (Goldsmith et al. Child Dev 58:505–529, 1987). The analyses presented address whether global behavioral tendencies are interchangeable at the level of etiology with specific behavioral counts as measures of temperament. If fine-grained behavior counts, a laborious and time-consuming method, show no substantial etiological difference from global ratings, it is reasonable to primarily employ global ratings. We compared two measures of activity level (AL) on the same twins in the same situation: the Bayley (1969, Psych Corp, NY) Infant Behavior Record (IBR), a global tester-rated measure; and a composite score of discrete behavior counts over four episodes of the Laboratory Temperament Assessment Battery (LabTAB; Goldsmith & Rothbart, 1988, Manual for the Lab-TAB). The IBR was completed by testers following the four AL LabTAB episodes. Videotapes of LabTAB AL were employed by trained and reliable observers to code discrete behaviors. Both measures of AL were significantly correlated ($r = 0.64$). Intra-class correlations suggested genetic factors influenced individual differences in IBR AL, whereas shared environmental influences largely influenced coded LabTAB AL. Cross-twin cross-measure correlations suggested that these two etiological factors contributed very little to the overlap between measures. These results were confirmed in bivariate model-fitting analyses. The only significant source of overlap in the two measures of AL was non-shared environment. The findings of no significant genetic variance for the coded LabTAB AL and no significant shared environment for IBR AL explains the lack of overlap in either of these two factors. Therefore, global and fine-grained analyses of AL behaviors, although substantially correlated, cannot be assumed to tap the same genetic effects.

Do Temperament and Behavior Problems Share a Common Etiology at Age 2?

Sonia Chawla, Kimberly J. Saudino

Boston University

Temperament is a reliable predictor of behavior problems in children as young as 42 months (Lemery et al. Child Dev 73:867–882, 2002). Substantial etiological overlap has also been shown between temperament and behavior problems (Deater-Deckard et al. J Child Psychol Psychiatry 48:80–87, 2007; Schmitz et al. Int J Behav Dev 23:333–355, 1999). The present research extends these findings to younger children by exploring genetic and environmental overlap in temperament and behavior problems in a sample of 314 twin pairs (143 MZ, 164 DZ) assessed at age 2. Tester ratings of affect/extraversion, task orientation, and activity level were obtained using the Infant Behavior Record (IBR). Parents rated twins' externalizing and internalizing behavior problems on the CBCL. Multiple regression analyses indicated that affect/extraversion significantly predicted internalizing problems and activity level significantly predicted externalizing problems. Task orientation did not uniquely predict either internalizing or externalizing problems. Intraclass correlations were larger for MZ twins than DZ twins for all temperament dimensions and behavior problems suggesting genetic influences. However, cross-trait, cross-twin correlations did not provide evidence of genetic or shared environmental overlap between temperament and behavior. This was confirmed via multivariate model-fitting analyses. Although both observed temperament and parent-rated behavior problems were genetically influenced at 2 years of age, the two domains do not appear to share common genetic etiologies. These results with observed temperament contrast markedly with Schmitz et al. (1999) who found substantial genetic overlap between parent-rated temperament and behavior problems at 24 months of age. The finding of higher phenotypic and genetic covariance between temperament and behavior problems when both are assessed via parent ratings may reflect overlap in items used to assess the two domains.

Genome-Wide Association Study of Hirschsprung's Disease

S. S. Cherny[1], M. M. Garcia-Barceló[2], C. S. Tang[3], P. C. Sham[1], P. K. Tam[2]

[1]Department of Psychiatry, Genome Research Centre, and The State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong; [2]Department of Surgery, The University of Hong Kong; [3]Department of Psychiatry, The University of Hong Kong

Hirschsprung's disease (HSCR), or aganglionic megacolon, is a congenital disorder characterized by the absence of enteric ganglia in variable portions of the distal intestine. RET is a well-established susceptibility locus, although existing evidence strongly suggests additional loci contributing to sporadic HSCR. To identify these additional genetic loci, we carried out a genome-wide association study using the Affymetrix 500 K marker set. We genotyped 493,840 single-nucleotide polymorphisms (SNPs) in 200 Chinese subjects with sporadic HSCR and 306 ethnically matched control subjects. The SNPs most associated with HSCR were genotyped in an independent set of 190 HSCR and 510 control subjects. Aside from SNPs in RET, the strongest overall associations were found for two SNPs on chromosome 8p, yielding odds ratios of 1.68 [CI95%:(1.40,2.00), $P = 1.88 \times 10–8$] and 1.98 [CI95%:(1.59,2.47), $P = 6.12 \times 10–10$], respectively, for the heterozygous risk genotypes under an additive model. There was also a significant interaction between RET and the 8p locus ($P = 0.0095$), increasing the odds ratio 2.3-fold to 19.53 for the RET rs2435357 risk genotype (TT) in the presence of the 8p risk heterozygote. Our highly significant association findings are backed-up by the important role of the identified gene as a regulator of the development of the enteric ganglia precursors.

Genetically Capitalist? Survival of the Richest in the Pre-Industrial Era and Modern Human Nature

Gregory Clark

Department of Economics, University of California Davis

Before 1800 all societies, including England, were Malthusian. The average man had 2 surviving children. Such societies were also

Darwinian. There was a competition over who reproduced most successfully. But it can be shown in at least some settled agrarian societies, including England after 1250, this selection amongst men was based on economic success, not success in violence as in hunter-gatherer society, and that this success was hereditable. The richest men left more than twice as many children as the poorest. The modern English population thus largely descended from the medieval economic upper classes. At the same time, from 1150 to 1800 economic preferences in England changed towards more "capitalist" attitudes. The highly capitalistic nature of English society by 1800—individualism, low time preference rates, long work hours, high levels of human capital—may thus stem from the nature of the Darwinian struggle in a very stable agrarian society in the long run. The triumph of capitalism may lie as much in genes as in ideology or rationality.

Measuring Persistent Antisocial Drug Dependence: Effects of Incarceration

Robin P. Corley, Sally-Ann Rhea, Tanya M. Button, Michael C. Stallings

Institute for Behavioral Genetics, University of Colorado, Boulder, CO, 80309

Antisocial drug dependence during adolescence has been operationalized as a composite of lifetime measures of conduct disorder symptoms and substance dependence symptoms, standardized against the behavior of community adolescents (Stallings et al. Arch Gen Psychiatry 62:1042–1051, 2005). To measure persistent antisocial drug dependence, the Colorado Center for Antisocial Drug Dependence has retested clinical probands from residential and outpatient treatment facilities for substance abuse and delinquent behavior. Through March 31, 2008, a total of 247 probands have been reinterviewed, with 65 interviewed while incarcerated in jail or prison. Twelve full siblings of the probands have also been interviewed while incarcerated. Based on self-report data on age of arrests, we estimate that more than 60% of the clinical probands have been arrested as adults (since their 18th birthdays). Based on living arrangement data, the mean length of continuous incarceration for the probands was 25.4 months. The average interval between original and follow-up interviews for all probands was 95.0 months, with a mean interval for incarcerated probands of 116.4 months. Incarcerated probands report dependence symptoms in significantly fewer drug classes over the past year than probands interviewed in other settings, although lifetime measures of dependence are comparable, suggesting that incarceration has interrupted probands' antisocial drug dependence trajectories. We examine differences between probands interviewed in prison and probands interviewed in other settings on measures of antisocial drug dependence across three reporting intervals: lifetime, past year, and last year before incarceration (using recency data for symptoms and symptom classes). Estimates of sibling resemblance for these measures will also be presented.

Do the Genetic Effects for Literacy in Early Childhood Differ Across Sex or Across the Disabled and Normal Range?

William L. Coventry[1], Brian Byrne[1], Richard K. Olson[2], Stefan Samuelsson[3], Robin Corley[2], Sally Wadsworth[2], John C. DeFries[2]

[1]School of Behavioural, Cognitive and Social Sciences, University of New England, Armidale NSW; [2]Department of Psychology and Institute for Behavioral Genetics, University of Colorado, Boulder CO 80309, USA; [3]Department of Behavioral Sciences, Linköping University, 581 83 Linköping, Sweden, and Stavanger University

To date, research shows that the genetic etiology of reading disability is not dissimilar to that observed for the normal range, supporting the generalist genes hypothesis (Plomin and Kovas 2005). However, findings on the genetic etiology of reading disability in boys versus girls are mixed. Some observe greater heritability in boys (Harlaar et al. 2005; Stevenson 1992), while others do not (Wadsworth and DeFries 2005). We explored these issues for reading measured with the TOWRE at Grade 1 with a dataset compiled across Australia and the US. The full distribution of the sample comprised 413 MZs and 420 DZs. The top and bottom probands were those with scores greater than 1 SD either above or below the mean. For the bottom proband, the estimates of A, D, C and E were 53, 0, 26 and 21%; for the full distribution they were 77, 0, 7 and 16%; and for the top proband they were 72, 17, 0 and 11%. Through not significant, this shows a trend whereby, when explaining differences between high end reading ability and the normal range, genetic effects were more important, but when explaining differences between reading disability and the normal range, environmental effects played more of a role. While inconsistent with previous research, our trend may be from detrimental environmental effects that impact low but not high end reading ability, rather than differential genetic effects, so our results are not inconsistent with the generalist genes hypothesis. An analysis of the bottom proband separately for males and females showed slightly stronger genetic effects in males (effects of A, C and E were 45, 21 and 24%) than females (effects of A, C and E were 63, 21 and 16%). These differences were not significant thought were in the same direction as Harlaar et al. (2005) and Stevenson (1992) but not Wadsworth and DeFries (2005).

Puberty and the Genetic Diathesis of Disordered Eating: A Replication Study

Kristen M. Culbert, S. Alexandra Burt, Kelly L. Klump

Department of Psychology, Michigan State University, East Lansing, MI

Previous research has shown differential heritability of eating pathology across pubertal development. While genetic effects are negligible before puberty, genes account for approximately 50% of the variance in disordered eating after puberty onset (Klump et al. Psychol Med 37:627–634, 2007; Klump et al. Arch Gen Psychiatry 64:1409–1415, 2007). This study aimed to replicate these results using an independent sample of female twins from the Michigan State University Twin Registry (Klump and Burt, Twin Res Hum Genet 9:971–977, 2006). Participants included 530 same-sex female twins ages 7–28. Pubertal development was assessed using the Pubertal Development Scale (Petersen et al. J Youth Adol 17:117–133, 1988) for twins ages 10–15. Age was used as a proxy for pubertal development status for twins <9 years (i.e., pre-pubertal) or >16 years (i.e., pubertal/post-pubertal) old. Disordered eating was assessed with the total score from the Minnesota Eating Behaviors Survey (von Ranson et al. Eat Behav 6:373–392, 2005). Findings replicate previous research suggesting that puberty moderates genetic effects on disordered eating, where significant increases in genetic effects are observed after the onset of puberty. This is the first replication of differential genetic effects on disordered eating across puberty. Findings continue to implicate this key developmental stage (i.e., puberty) and the corresponding biological changes (e.g., ovarian hormones) in the genetic diathesis of eating disorders.

Maternal Smoking During Pregnancy and Offspring Criminal Convictions: Disconfirming a Causal Inference

Brian M. D'Onofrio[1], Amber L. Singh[1], Anastasia Iliadou[2], Mats Lambe[2], Christina M. Hultman[2], Martin Grann[3], Jenae M. Neiderhiser[4], Niklas Långström[2], Paul Lichtenstein[2]

[1]Indiana University; [2]Karolinska Institutet; [3]Swedish Prison Service; [4]Pennsylvania State University

Epidemiological and basic-science research has suggested that maternal smoking during pregnancy (SDP) causes increased antisocial behavior in offspring. However, most research to date has been unable to account for unmeasured environmental and genetic selection factors that could confound the association. The current study explored the possible underlying processes responsible for the association between SDP and offspring criminal convictions using national registries in Sweden that included all children ($n = 778{,}000$) born between 1983 and 1991. Maternal SDP was based on assessments during prenatal care visits (from the Swedish Medical Birth Register) and offspring antisocial behavior was based on criminal convictions from the Swedish National Crime Register. The analyses used statistical controls and a quasi-experimental approach, the comparison of siblings differentially exposed to SDP, to account for selection factors. Although SDP was associated with a 3-fold increase in risk for criminal convictions for violent crimes (Hazard Ratio = 3.05) when comparing unrelated individuals, the comparison of differentially exposed siblings found no association (HR = 0.96). Similar results were obtained when studying non-violent and drug-related convictions. As such, the results strongly suggest that SDP does not cause the increased risk of criminal convictions; rather, risk factors that co-occur with maternal SDP are responsible.

Louisville Twin Study: Past, Present and Future

Deborah Winders Davis[1], Barbara M. Burns[2], Toni P. Miles[3]

Departments of [1]Pediatrics, [2]Psychological & Brain Sciences, and [3]Family & Geriatric Medicine, University of Louisville

The Louisville Twin Study (LTS) was a longitudinal study of child health and development (1957–2003). The LTS cohort is positioned to evaluate combined genetic and environmental influences on behavior, health, & development. The purpose of this poster is to highlight previous work, describe the current status of these data, and describe plans to re-engage the cohort as well as engage potential research collaborators. In the U.S., longitudinal studies of twins are limited by the availability of well-defined cohorts and sophisticated statistical methods. The generally low rate of mobility out of the recruitment area for this cohort means that new studies of disease incidences and mortality are possible in a large, U.S.-based cohort. In addition to serial measurements of participating twin pairs, the LTS data also contain in-person interviews and genetic data from related family members. This provides the opportunity for the application of twin *and* family statistical methods. The LTS study team is designing a follow-up study to locate participants and to examine current health status and health behaviors of twins and family members. Other studies of predictors of health and developmental outcomes are under development. Researchers within 3 departments are beginning to identify specific areas for future research and to identify potential collaborators. We seek to open discussions with potential collaborators about research questions answerable through these data.

An Interaction Between the R7 Allele of the DRD4 Gene and the Number of Friendships Young Adults Report is Associated with Political Orientations

Christopher Dawes

University of California, San Diego

Previous studies have found that political orientations are heritable (Alford et al. 2005). Using the National Longitudinal Study of Adolescent Health, we find that an interaction between the R7 allele of the DRD4 gene and the number of friends a young adult has is associated with political orientation. Specifically, younger adults possessing R7 alleles and reporting more friendships in the second wave of the study go on to report being more liberal in the third wave. Our results are based on a within-family test of association (QTDT) and therefore are unlikely due to population stratification.

Genetic Variation in the Development of Voting Behavior

Christopher Dawes

University of California, San Diego

This paper studies whether variation in the development of voting behavior as individuals age can be attributed to genetic factors. I construct a Bayesian biometric latent growth curve model, using validated turnout data from 5 elections, to estimate the degree to which initial propensity to vote, and the subsequent growth in the propensity to vote, is heritable. I find that nearly a quarter of the variation in initial turnout can be explained by genetic factors, more than 4 times the proportion that can be explained by parental socialization. The results also show that about a third of the variation in the growth of the propensity to vote can be attributed to genetic factors. These findings suggest that socialization gets too much credit in developmental theories of voting behavior due to confounding with genetic factors.

Partisanship, Voting, and the Dopamine D2 Receptor Gene

Christopher T. Dawes, James H. Fowler

UCSD Political Science, La Jolla, CA 92093, USA

Previous studies have found that both political orientations (Alford et al. Am Polit Sci Rev 99:153–167, 2005) and voting behavior (Fowler and Dawes, J Polit 70, 2008, in press) are heritable. In this article we study genetic variation in another important political behavior: partisan attachment. Using the National Longitudinal Study of Adolescent Health, we show that individuals with the A1 allele of the D2 dopamine receptor gene are significantly less likely to identify as a partisan than those with the A2 allele. Further, we find that this gene's association with partisanship also mediates an indirect association between the A1 allele and voter abstention. These results are the first to identify a specific gene that may contribute to the tendency to join political groups, and they may help to explain correlation in parent and child partisanship and the persistence of partisan behavior over time.

Genome-Wide Association Study of Leisure-Time Exercise Behavior

Marleen H. M. de Moor[1], Danielle Posthuma[1], Patrick Sullivan[2], Dorret I. Boomsma[1], Eco J. C. de Geus[1]

[1]Department of Biological Psychology, VU University, Amsterdam, The Netherlands; [2]Department of Genetics, Psychiatry, & Epidemiology, University of North Carolina, Chapel Hill, NC, USA

Twin (family) studies have shown a significant contribution of genetic effects to variation in adult leisure-time exercise participation and physical activity, with heritability estimates ranging from 25% to 75% (Stubbe et al. 2006, PLoS ONE 1, e22). Not much is known yet about the actual genetic variants involved in exercise behavior. Three genome-wide linkage studies and six association studies on exercise participation and physical activity phenotypes have been conducted so far (Cai et al. Obes Res 14:1596–1604, 2006; De Moor et al. Eur J Hum Genet 15:1252–1259, 2007; Loos et al. Int J Obes Relat Metab Disord 29:420–428, 2005; Wolfarth et al. Med Sci Sports Exerc 37:881–903, 2005). Overlap in findings is generally lacking, with the exception of the MC4R gene. Here, we report the results of a genome-wide association study in a sample of 1,772 unrelated Dutch adults from the Netherlands Twin Registry. Leisure-time exercise behavior was based on survey data from 2002 and 2004 on type, frequency and duration of exercise. We classified individuals into six categories of weekly METhours (metabolic equivalent*hours/week). All individuals were genotyped using the Perlegen 600 k SNP chip. After quality control, 427,037 autosomal SNPs were left for further analysis. To enable future comparison with replication samples using different genotyping platforms, we imputed over 2 million SNPs that were not on the Perlegen chip using HAPMAP data and the software program IMPUTE. Results from genome-wide association analyses in SNPTEST, accounting for the uncertainty of imputed SNPs and with sex and age as covariates, will be presented.

Causality in Genetic Epidemiology: An Application to Exercise and Depression

Marleen H. M. de Moor, Dorret I. Boomsma, Janine H. Stubbe, Gonneke Willemsen, Eco J. C. de Geus

Department of Biological Psychology, VU University, Amsterdam, The Netherlands

There is a long history of scientific and philosophical debates on causality. Within the social and life sciences nowadays, a randomized controlled trial is commonly seen as the preferable method to study causality. Correlational studies can help gaining insight into causal mechanisms, at the condition that possibly confounding factors are corrected for. Correlational studies are further thought to become more powerful when using a longitudinal design, as opposed to a cross-sectional design. Within genetic epidemiology, different methods have been proposed to study causality in a cross-sectional design, such as the MZ discordant twin design (Cederlöf et al. Acta Medica Scandinavica Supplementum 1–128, 1977; Kendler et al. Arch Gen Psychiatry 50:36–43, 1993) and direction-of-causation models (Duffy and Martin, Genet Epidemiol 11:483–502, 1994; Heath et al. Behav Genet 23:29–50, 1993). These methods have their own strengths and limitations. We propose that a combination of different methods is to be preferred when studying causality in genetic epidemiology. A combination of methods that focus on falsifying the causal hypothesis instead of determining the direction of causation is presented. This approach will be illustrated using longitudinal data (1991–2002) on leisure-time exercise behavior and anxious depressive symptoms from 5,952 adult twins and 2,606 of their relatives from the Netherlands Twin Registry. It is shown that the population association between exercise and symptoms of anxiety and depression is not explained by a causal effect of exercise on symptoms. Implications of this finding and discrepancies with findings from previous experimental studies are discussed.

Sex-Specific Moderation of Substance Use by Deviant Peer Association in Late Adolescence

Jaime Derringer, Robert F. Krueger, William G. Iacono, Matt McGue

Department of Psychology, University of Minnesota

Previous research shows that both substance use (SU) and deviant peer affiliation (DPA) differ in variance attributable to genetic, common environmental, and unique environmental factors at different points throughout adolescence (Derringer et al. Behav Genet 37:747, 2007; Kendler et al. Arch Gen Psychiatry 64:958–965, 2007). Additionally, DPA shares common environmental variance with adolescent SU (Walden et al. J Abnorm Soc Psych 113:440–450, 2004) and moderates variance components of adolescent conduct disorder symptoms (TMM Button et al. J Abnorm Soc Psych 116:554–564, 2007). However, the relationship between DPA and SU differs as a function of sex (Dick et al. Alcohol Clin Exp Res 31:2012–2019, 2007). The current study sought to integrate these previous findings by examining the potential moderating effects of both DPA and positive peer affiliation (PPA) on SU, as well as investigating whether these moderating effects differed by age and sex. The sample consisted of 687 same-sex twin pairs participating in the longitudinal Minnesota Twin and Family Study. During day-long in-person assessments at ages 14 and 17, participants were administered both a computerized SU questionnaire and a survey regarding DPA and PPA. A moderated Cholesky model, in which genetic and environmental factors were free to be correlated, was fit to substance use diversity counts for eight groups, defined by age, sex, and moderator (DPA, PPA). Model fits indicated that PPA did not moderate SU in any of the four age-by-sex groups and DPA did not moderate SU in either sex at age 14. DPA did moderate SU at age 17 for both sexes. However, only the genetic component was moderated in 17-year-old males (with greater DPA indicating less genetic influence) while both the common and unique environmental components to SU were moderated in 17-year-old females (with greater DPA resulting in reduced variance due to common and unique environment).

Genetic Influences on Developmental Trajectories of Externalizing Behavior: Data from the Child Development Project

Danielle M. Dick[1], Shawn J. Latendresse[1], John Budde[2], Alison Goate[2], Jennifer E. Lansford[3], Ken A. Dodge[3], Greg S. Pettit[4], John E. Bates[5]

[1]Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University; [2]Washington University, St. Louis; [3]Center for Child and Family Policy, Duke University; [4]Department of Human Development and Family Studies, Auburn University; [5]Department of Psychological and Brain Sciences, Indiana University, Bloomington

The Child Development Project (CDP) is a representative, community-based sample of individuals who were enrolled into the study when they entered kindergarten. Just under 500 individuals have been followed annually for 20 years (to age 25), with comprehensive developmental

assessments, including family, school, peer, neighborhood, and child characteristics. Recognizing the potential of this sample to examine the interplay of genetic and environmental influences on trajectories of development, we added DNA collection via saliva sample to the 2006 follow-up, with a 93% response rate of the target sample ($N = 452$). We have genotyped a small number of candidate genes in the sample, to conduct more detailed studies of their influence across development. Initial analyses focused on GABRA2, which has been associated with adult alcohol dependence across multiple samples (initial report from COGA data: Edenberg et al. Am J Hum Genet 74:705–714, 2004). There was also evidence in COGA that GABRA2 was associated with conduct disorder, using cross-sectional data from COGA children (Dick et al. Behav Genet 36:577–590, 2006). The CDP collected data on externalizing behavior using Achenbach's Youth/Young Adult Self Report at ages 12, 14, 15, 16, 17, 19, 20, 21, and 22. We used latent class growth analysis to identify patterns of externalizing behavior within the CDP sample, from which two developmental profiles of antisocial behavior emerged: 83% of the sample showed moderate, decreasing levels of externalizing from ages 12–22, whereas 17% showed persistent high levels. GABRA2 genotypes discriminated between individuals showing different trajectories of antisocial behavior. These results replicate the previous association between GABRA2 and conduct disorder symptoms reported in cross-sectional COGA data, and importantly, extend these analyses to examine trajectories of behavior across development.

What is g?

William T. Dickens, Thomas C. Schelling

School of Public Policy, University of Maryland

Large secular gains in cognitive ability (the Flynn Effect) show that large, environmentally induced changes in measured cognitive ability are possible, but several studies have suggested that secular gains are not gains in general cognitive ability and this has led some to conclude that they are therefore not substantive. This paper extends the model of a single cognitive ability presented by Dickens and Flynn (2001) to multiple abilities. It shows that such a model can account for all the important facts about general cognitive ability without postulating any common underlying physiological cause for different mental abilities. A general intelligence factor arises in the model because people who are better at any cognitive skill are more likely to end up in environments that cause them to develop all skills. Scores on the resulting general ability factor can be highly heritable even while they are potentially subject to considerable environmental influence. Loadings of subtest scores on the general ability factor can be positively correlated with subtest heritabilities. In the model, discrimination against a social group in access to cognitively demanding environments can produce subtest score differences from other groups that are strongly correlated with both the g loadings and heritabilities of those subtests. Despite this, there is no reason to expect that meaningful secular gains should be correlated with g loadings across subtests.

Parenting and the DRD4 Genotype: Interaction and Effects on Aggressive and Prosocial Behaviors in Preschoolers

Lisabeth F. DiLalla[1], Kit K. Elam[2]

[1]Southern Illinois University School of Medicine; [2]Southern Illinois University

The present study examined effects from a specific dopamine receptor gene (DRD4) and environmental influences from parents and peers, as well as the interaction between them, on both aggressive and prosocial behaviors of preschoolers. The total sample for whom DRD4 data were available was 118 children. Children were classified as DRD4-L if they had at least one DRD4 allele with 7 or 8 repeats and as DRD4-S if they did not. Parent-child interaction data were collected when children were 3–4 years old. Peer interaction data and parent questionnaires on temperament and behavior problems were collected at age 5. Results showed that DRD4-L children were less cooperative ($t = 2.70$, $P < 0.01$) and their parents were less sensitive ($t = 2.61$, $P < 0.05$) during parent-child interactions. In addition, genotype interacted with peer aggression to affect children's aggression during a peer play interaction at age 5 (beta $= 0.59$, $P < 0.05$), and genotype interacted with prior parental sensitivity behaviors to affect later externalizing problem behaviors (beta $= 0.42$, $P < 0.01$). Thus, there appear to be several factors that interact with genotype (specifically DRD4) to influence child social behaviors. Specifically, this study found that warm, sensitive parenting appears to be a protective factor for children with this at-risk genotype, and interacting with aggressive peers may be an added risk factor.

Genetic Overlap Between Borderline Personality Disorder and Personality Traits

Marijn A. Distel[1], Timothy J. Trull[2], Gonneke Willemsen[1], Catherine A. Derom[3], and Dorret I. Boomsma[1]

[1]Department of Biological Psychology, VU University, Amsterdam, The Netherlands; [2]Department of Psychological Sciences, University of Missouri-Columbia, Columbia, MO, USA; [3]Department of Human Genetics, Katholieke Universiteit Leuven, Belgium

Recently, the nature of personality disorders and their relationship to normal personality has received extensive attention. The FFM of personality, consisting of the dimensions neuroticism (N), extraversion (E), openness to experience (O), agreeableness (A) and conscientiousness (C), is one of the proposed models to conceptualize personality disorders as maladaptive variants of continuously distributed personality traits. The heritability of the FFM scales has intensively been studied while genetic studies of Borderline Personality Disorder (BPD) remain scarce. Recently, a twin study suggested a heritability for BPD features of 42% (Distel et al. 2008). Evidence for linkage was found on chromosome 9 (Distel et al. Psychiat Genet, in press). Epidemiological studies into the relationship between the FFM and BPD show that BPD patients tend to score high on N and low on A and C (Costa and Widiger. Personality disorders and the five factor model of personality. Washington, DC, APA). The aim of the present study was to explain the genetic etiology of the relationship between BPD and the FFM. The Personality Assessment Inventory—Borderline scale (Morey 1991) and the NEO-FFI (Costa and McCrae 1992) were mailed to twins and siblings registered with the Netherlands Twin Register and the East Flanders Prospective Twin Survey. Data were available for 2,669 MZ twins (969 pairs), 2,371 DZ twins (668 pairs) and 1,291 siblings from 4,029 families. Phenotypic correlations between BPD and N, E, O, A and C were 0.64, −0.21, 0.08, −0.29 and −0.31, respectively. Four bivariate factor models specifying genetic and environmental influences on BPD, N, E, A and C was fitted to the data using MX. The additive genetic correlations were 1, −1, −0.74 and −0.63, respectively. In addition there were non-additive genetic effects on each trait and moderate to strong environmental correlations. These results support the hypothesis that BPD can be described in terms of dimensional trait profiles.

The Consequences of the Mutualistic Theory of "g" for QTL Searches. Turkheimer Symposium

Conor Dolan, Kees Jan Kan, Han van der Maas

Dept of Psychology, University of Amsterdam

As a latent phenotype derived from factor analyses, g is often interpreted as a quantitative inter-individual-differences dimension. This interpretation sits well with the simple view of QTLs are contributors of small amounts of variance with respect to g. However, the exact nature of g as quantitative dimension remains elusive, and attempts to identify QTLs contributing specifically to g have met with little success. A possible reason for this may lie in the conceptualization of g. Recently van der Maas et al (2005) presented a developmental theory of g, in which g is attributable to mutualistic interaction among basic processes. Mutualism gives rise to the positive manifold, and thus to a dominant first eigenvalue. However, this theory renders the realist interpretation of g questionable. The aim of the present talk is to discuss the implication of the mutualistic interpretation of g for research aimed at detecting QTLs underlying g.

Genetic and Environmental Contributions to BMI in Adolescent and Young Adult Female Twins

Alexis E. Duncan, Arpana Agrawal, Julia D. Grant, Pamela A. F. Madden, Andrew C. Heath

Midwest Alcoholism Research Center, Department of Psychiatry, Washington University School of Medicine, St. Louis, MO

While several studies have examined genetic and environmental contributions to the variance in BMI over time in men, there has been little research on this topic in women. The objective of this current study was to determine the genetic and environmental contributions to BMI over time in adolescent and young adult female twins using data from a longitudinal epidemiologic study of female twins. Twins (1244 complete pairs: 703 MZ and 541 DZ) were aged 13–23 years at the adolescent baseline interview and 18–28 years at young adult follow-up. BMI (kg/m^2) was based on self-reported weights and heights at two time points and categorized into underweight (<18.5 kg/m^2), ideal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2) and obese (=30 kg/m^2). Bivariate Cholesky decomposition models were fit to the data, with thresholds adjusted for age and pregnancy and postpartum status at interviews. The heritability of BMI category was high at both time points (80% for both), with the remaining variance attributable to non-shared environment. Genetic and non-shared environment correlations between adolescent and young adult BMI category were 0.84 and 0.31, respectively. Thus, although there was evidence for a substantial genetic overlap for BMI measured across a 5-year interval, individual-specific environmental factors were largely specific to each time-point. While the error in BMI assessment (influencing unique environmental specificity) and potential gene-environment interactions exist, our results imply that intervention efforts targeted at modification of the individual-specific environment may impact the high rates of overweight and obesity in young adult women.

Looking for Politically Relevant Genes

Lindon Eaves[1], Peter Hatemi[1], Nathan Gillespie[1], Nicholas Martin[2]

[1]Virginia Institute for Psychiatric and Behavioral Genetics; [2]Queensland Institute of Medical Research

Previous studies consistently indicate a strong heritable component for political and social attitudes, offering a reason to begin to identify which genes may be partially responsible for the sizable heritability estimates. However, only two studies have attempted to identify which genes may influence political behavior, focusing on 5HTTPLR and MAOA (Fowler and Dawes 2007; Hatemi et al. 2007), and none so far have employed genome wide methods. In this paper, we report the findings from a large population based genome-wide linkage scan of twins and relatives for a limited conception of political ideology, civic participation, social attitudes and other personality phenotypes. Complex genetic traits such as political orientations entail so many genes, so many biological systems, a vast array of environmental influences, and so many potential gene-environment interactions that one gene, or even a small group of individual genes is unlikely to produce significant results. Initial investigations support this assertion and reveal no clear genetic focal point for civic participation. However political temperaments and ideological positions may be another matter; results to be discussed.

The Etiology of Autism-Like Behaviors in Very Young Twins

Lisa R. Edelson, Kimberly J. Saudino

Department of Psychology, Boston University

Autistic symptoms are present in individuals without a diagnosable autism spectrum disorder and are normally distributed in the general population (Hoekstra et al. Arch Pediat Adol Med 161:372–377, 2007), thus it is reasonable to explore the etiology of these behaviors in non-clinical samples. The present study examined genetic and environmental contributions to individual differences in autistic-like behaviors in a population sample of 312 2-year-old twin pairs. This age is particularly salient because it is frequently acknowledged that this is the age when autism can first be reliably diagnosed (DiCicco-Bloom et al. J Neurosci 26:6897–6906, 2006). Autistic-like behaviors were assessed via parent ratings on the pervasive developmental problems subscale of the CBCL (Achenbach and Rescorla 2000). Tester ratings on the orientation/engagement subscale of the Behavior Rating Scale (BRS; Bayley 1993) provided an observational measure of social approach. Both autistic-like behaviors and social approach were moderately heritable (0.39 and 0.30, respectively). There were also modest, but significant shared environmental effects (0.21 and 0.12, respectively). Further, these genetic and environmental influences overlapped greatly between the two phenotypes. These findings suggest that autistic-like behaviors exist on a continuum in the general population with individuals with diagnosable autism at one extreme. These behaviors are largely genetic in etiology, but are also influenced by a shared environmental component in early childhood. Moreover, autistic-like behaviors have many influences in common with social approach behaviors. Molecular genetic research that finds genes that affect one phenotype would inform which genes might affect the other.

The Cys23Ser Polymorphism Contributes to Personality Traits of Harm Avoidance and Self-Directedness in Young Males From the Russian Population

M. S. Egorova[1], G. I. Korovaitseva[2], O. V. Parshikova[1], T. V. Lezheiko[2], M. A. Alfimova[2], E. A. Polezhaeva[1], V. E. Golimbet[2]

[1]Moscow State University, Department of Psychology; [2]Mental Health Research Center, Russian Academy of Medical Science

Serotonin receptor type 2C gene located at the Xq chromosome has been reported to be associated with different behavioral traits. Several lines of evidence support its relation to personality. The Cys23Ser polymorphism was tested for association with personality traits in some studies but the results were inconsistent. We compared personality traits assessed by the Temperament and Character Inventory (TCI-125) and traits measured by the Self-Monitoring Scale in people with different Cys23Ser genotypes from the Russian population. The measures, used in the study, allow to compare three levels in personality structure—temperament, character and the most complex behavior traits (sub-scales of self-monitoring). The latter reflect the ability of a person to manipulate some others' behaviour, are connected with individual differences in goals and at the same time have moderate correlations with four Big 5 factors. We studied 314 males, students of the Military University, aged 16–26 years. Our specific hypothesis was that the Ser23 allele which was constitutively more active than the Cys23 allele might be associated with anxiety-related traits. The distribution of genotypes in the sample was as follows: the Cys—85% and the Ser—15% that was consistent with the results reported earlier for Caucasians. There was no association between the Cys23Ser polymorphism and Self-monitoring. At the same time, personality factors assessed by TCI-125 were related to genotype (MANCOVA $F = 2.2$; df $= 7$; $P = 0.036$). Post hoc analysis showed the association between the Cys23Ser polymorphism and such factors as Harm avoidance ($P = 0.004$) and Self-directedness ($P = 0.042$). The carriers of the Ser23 allele had higher scores on Harm Avoidance that was in accordance with our hypothesis and lower scores on Self-directedness. The results confirm biosocial model: the higher the level of the trait in personality structure (and, accordingly, the cultural influences) the less is genetic influence.

A Method for Obtaining High Quality Human Genomic DNA from 11-Year Old Cotton Spit Wads for Genetic Studies of Behavioral Disorders

Erik A. Ehli[1], Timea Lengyel-Nelson[1], James J. Hudziak[2], Gareth E. Davies[3]

[1]Avera Institute for Human Behavioral Genetics, Avera Behavioral Health Center; [2]Vermont Center for Children, Youth, and Families at the University of Vermont College of Medicine, Burlington, VT; [3]South Dakota State University, College of Pharmacy, & Department of Psychiatry, Sanford School of Medicine, University of South Dakota

Objective To obtain high quality Human genomic DNA from saliva saturated cotton spit wads stored at −20°C for approximately 11 years for use in genetic studies of Behavioral Disorders. *Methods* 783 spit wad samples were collected from an ADHD sample population (Vermont Family Study) during 1996–2000. Human genomic DNA was extracted from the spit wads using a commercially available kit; QIAamp DNA Blood Midi Kit (Qiagen, Inc., Valencia, CA.) with a few modifications. DNA concentration and purity were determined using UV spectrophotometry. The quality of genomic DNA was also measured using PCR success of the 5HTTLPR polymorphism. *Results* The resulting DNA yield was more than adequate for genetic analysis and ranged from approximately 1 μg to a total of 80 μg (mean 17.3 μg ± 11.9 μg). A260/A280 ratios for the human genomic DNA extracted from the spit wads was consistently within the generally acceptable values of 1.7–2.0. The mean A260/A280 ratio for the 783 samples was determined to be 1.937 ± 0.226. The DNA also was suitable for PCR reactions as evidenced by the amplification of the Serotonin Transporter promoter region, 5HTTLPR. Using PCR analysis of the 5HTTLPR polymorphism, we identified that 770 DNA samples (98.3%), were successful for amplification of the 5HTTLPR repeat polymorphism using fragment analysis on the ABI 3130 Genetic Analyzer (Applied Biosystems). In addition, the genomic DNA can be extracted from the saliva saturated cotton spit wad in less than 3 h and many samples can be processed at the same time. *Conclusions* This report describes the effectiveness of a commercially available kit in extracting and purifying human genomic DNA from saliva saturated cotton spit wads, stored at −20 for approximately 11 years. We found that DNA obtained from the spit wads was of adequate quantity and quality for use in downstream genetic studies.

Neuronal Nicotinic Receptor Genes and Associations with Smoking and Alcohol Behaviors

Marissa A. Ehringer[1], Isabel R. Schlaepfer[1], Nicole R. Hoft[2]

[1]Institute for Behavioral Genetics and Department of Integrative Physiology, University of Colorado, Boulder, CO; [2]Institute for Behavioral Genetics, University of Colorado, Boulder, CO

Alcoholics are more likely to be dependent on nicotine and likewise, nicotine dependence is correlated with increased severity of alcohol dependence (Falk et al. Alcohol Res Health 29:162–71, 2006). Furthermore, there is strong evidence that shared genetic factors contribute to this co-morbidity (Kendler et al. Arch Gen Psychiatry 64:1313–1320, 2007). The nAChRs are ligand-gated ion channels which are the primary targets for nicotine and the endogenous agonist acytelcholine. Recent work from our group and others has provided convincing evidence for an association of several of the nAChR genes with alcohol and nicotine behaviors in humans. A single nucleotide polymorphism (SNP) in the 5′UTR of the human CHRNB2 gene has been associated with subjective responses to alcohol and nicotine (Ehringer et al. Am J Med Genet B Neuropsychiatr Genet, 2007). Likewise, the CHRNB3 5′UTR and promoter region have been associated with subjective responses to tobacco (Zeiger et al. Hum Mol Genet 17:724–734, 2008), and with tobacco dependence (Bierut et al. Hum Mol Genet 16:24–35, 2007; Saccone et al. Hum Mol Genet 16:36–49, 2007). In addition, the gene cluster containing CHRNA5-A3-B4 has been associated with nicotine dependence (Bierut et al. Hum Mol Genet 16:24–35, 2007; Saccone et al. Hum Mol Genet 16:36–49, 2007), age of initiation of alcohol and tobacco (Schlaepfer et al. Biol Psychiatry, 2007), and heavy smoking (Berrettini et al. Mol Psychiatry, 2008). The biological significances of these genetic associations are being pursued in our lab using cultured neuronal cell lines which are known to express nAChR subunits. We hope these approaches will provide a better understanding of how these variations lead to differences in the underlying molecular mechanisms of these genes.

Microorganismal Pheromones

Lee Ehrman

State University of New York at Purchase

The data tables below indicate that when the Drosophia pualistorum superspecies' omnipresent endosymbiont is virtually eliminated by ingested antibiotics, behavior is altered. Additional data, not presented here, identified the cause as pheromones borne on the cuticular skeleton of these insects. Thus a cytoplasmic microorganism, genus Wolbachia, is directing its perpetuation, in bravura co-evolution. Current and original isolation indices, ranging from −1.00 (preferences for unlikes, heterogamy) through 0 (random mating) to +1.00 (preferences for likes homogamy between Amazonian and Orinocan Drosophila paulistorum semispecies. Treated = ten generations on 0.01 Rifampicin, an antibiotic versus Wolbachia endosymbionts. Five replicas and 120 matings were scores (12A female +12B female +12A male +12B male differentiated by rotated wing clips for each row, totaling 720 matings. Controls, untreated 1965 (Ehrman, Evolution 19:459–464, 1965) Sympatric strains 0.75 ± 0.07, Allopatric strains 0.61 ± 0.07, 2008 Allopatric strains 0.70 ± 0.004, Experimental, treated both strains allopatric, 2008 Amazonian treated 0.00 ± 0.007 (Orinocan males mated significantly more than opposing Amazonian ones; here Orinocan was normal and untreated. All other male and female copulae numbers were equivalent.), 2008 Orinocan treated 0.54 ± 0.006, 2008 Am. + Or. treated 0.86 ± 0.002.

Genetic Influences on Attentional Response to Emotional Faces in 5-Year-Old Twins

Kit K. Elam[1], Josh Carlson[1], Dr. Karen R. Pressley[2], Dr. Lisabeth F. DiLalla[1]

[1]Southern Illinois University Carbondale; [2]University of Illinois Springfield

Behavioral studies reveal an attentional bias to the location of threatening stimuli in children as young as 9 years old. As early as age 4, children demonstrate an ability to discriminate between happy and sad emotional faces. However, it is unclear whether preschoolers' developing abilities to discriminate emotional faces may contribute to decreased spatial attention to emotional faces, and we know little about the role of genetics in this developing ability. The present project addressed this by studying 12 5-year-old twin pairs who performed a modified dot probe task. Two faces (from Gur et al. J Neurosci Methods 115:137–143, 2002) were simultaneously presented (500 ms) to the left and right of fixation and were followed by a target in one of the two locations that children subsequently responded to. Spatial attention trials consisted of one emotional face (fearful or positive) and one neutral face. Children produced faster reaction times (RT) (m = 905.91 ms) for emotional congruent (target cartoon appearing on the same side as the emotional face) compared to incongruent (m = 993.12 ms) trials in the right visual field (RVF). MZ correlations were larger than DZ correlations, indicating a possible genetic influence for a number of trial types. In addition, a significant MZ versus DZ mean RT difference was found for all trial types except those in the right visual field (RVF). This is the first such study of 5-year-olds or of twins. Most adult spatial attention studies have found congruency effects either bilaterally or in the LVF and none have investigated genetic effects. The presence of a RVF effect in preschoolers may represent an early left-hemisphere mechanism that matures as processing becomes more automatic while the LVF effects may indicate a stronger genetic influence on the right hemisphere.

Genetic and Environmental Overlap Among Schizophrenia Spectrum Endophenotypes and Schizophrenia Liability During Childhood and Adolescence

Marissa Ericson[1], Catherine Tuvblad[1], Laura Baker[1], Adrian Raine[2]

[1]Department of Psychology, University of Southern California; [2]Departments of Criminology, Psychiatry and Psychology, University of Pennsylvania

Objective In recent years, the p50, p300 amplitude and latency, and Mismatch Negativity (MMN) event related potential components have been proposed as potential endophenoytpes for schizophrenia spectrum disorders on the basis of twin and family studies. To date, there have been no previous studies investigating the genetic overlap between event-related potential indices and schizophenia liability in childhood and adolescence. *Method* P50 sensory gating, p300 amplitude and latency, MMN, and schizotypal traits were measured in a community sample of 605 9–11 year old twin pairs. Structural equation modeling was used to quantify the genetic and environmental contributions to the covariance between schizotypal personality and each of the event-related potential endophenotypes. *Preliminary Results* Moderate phenotypic correlations were found among the measures, ranging between 0.19 and 0.24, considered for psychophysiological data to be quite strong. Subsequent analyses are currently in progress.

Retrospective Perceptions of Parenting and Marital Satisfaction: Genetic and Environmental Influences

Hilah K. Evrony[1], Jody M. Ganiban[1], J. Ulbricht[1], Suzanne K. Haddad[1], Paul Lichtenstein[2], Erica L. Spotts[3], David Reiss[4], Jenae M. Neiderhiser[5]

[1]Department of Psychology, The George Washington University, Washington, DC, [2]Karolinska Institutet, Stockholm, Sweden; [3]National Institute of Aging, Bethesda, Maryland; [4]Yale University, New Haven, CT; [5]Department of Psychology, Pennsylvania State University, College Station, PA

Attachment theory puts forth that individuals develop internal working models of themselves and others based on their early experiences with their caregivers. These models subsequently guide expectations and perceptions of romantic relationships (Truant et al. Can J Psychiatry 32:87–92, 1987). The present study examined the relationship between recalled experiences of parenting and marital satisfaction. Genetic and environmental sources of variation within these associations were examined for male twins and female twins. It was hypothesized that recollections of maternal and paternal care and overprotection would be significantly associated with one's self-reported marital satisfaction. Based upon attachment theory, we hypothesized that these associations would be explained by nonshared or shared environmental factors. Data for this study were from The Twin/Offspring Study in Sweden (TOSS), a sample consisting of 909 male and female same-sex twin pairs and their spouses. Utilizing a correlated factors model, the relationships between husbands' and wives' scores on the care and overprotection dimensions of the Parental Bonding Instrument (PBI), a self-report measure of one's prior parenting experiences, and a composite measure of marital quality, were examined. Contrary to expectations, it was found that the association between retrospective perceptions of parenting and marital satisfaction is almost entirely explained by genetic factors. This hints that a relationship between recalled parenting and marital

quality may be the result of genetically influenced characteristics, such as personality, which may elicit both parental and spousal warmth. Alternatively, parents' characteristics may impact how interactions with parents and spouses are experienced and interpreted. These findings have implications for understanding marital quality and how internal representations of parents influence subsequent relationships.

Stability of Mental and Motor Abilities in Very Young Children

David L. Fask, K. Paige Harden, Jennifer E. Hill, Eric Turkheimer

Department of Psychology, University of Virginia, Charlottesville, VA

A number of genetically informed studies have examined the factors contributing to the stability of scores on the Bayley Scales of Infant Development (BSID). Early studies (e.g., Wilson and Harpring, Dev Psychol 7:277–287, 1972) suggested that the stability is largely mediated by genetics, rather than by shared or nonshared environment. Cherny et al. (Psychol Sci 5:354–360, 1994) suggested that a substantial portion of the stability in mental ability scores on the BSID may be accounted for by genetics and shared environment at ages 14, 20, and 24 months, with new genetic variation introduced at 24 months. In the present study, we analyzed Bayley Short Form Research Edition scores in 485 sibling pairs from the Early Childhood Longitudinal Study: Birth Cohort (National Center for Education Statistics, 2007, *Early Childhood Longitudinal Study: Birth Cohort* [machine-readable data file and documentation], U. S. Department of Education, Washington, DC), a representative sample of American children, in two waves (9 months and 2 years). The primary purpose of this study was to attempt to replicate the finding that the stability of mental and motor abilities in early childhood is largely genetic. The results suggested that stability from 9 months to 2 years is indeed largely genetic for both phenotypes. It also suggested that genetic and shared environmental effects are highly correlated between both mental and motor scores at both 9 months and 2 years. Furthermore, substantial new genetic and shared environmental (as well as nonshared environmental) variation was introduced at 2 years, particularly in the case of the mental scores.

Self-Regulatory Problem Behaviors in 5-Year Old Twins with Stuttering and High Normal Nonfluency

S. Felsenfeld[1], C. E. M. van Beijsterveldt[2], D. I. Boomsma[2]

[1]Department of Communication Disorders, Southern Connecticut State University and the Child Study Center at Yale University, New Haven, CT, USA; [2]Department of Biological Psychology, Vrije universiteit, Amsterdam, The Netherlands

Using a population sample of 10,683 twin pairs participating in the Netherlands Twin Registry, this study examined whether fluency control in young children might be related to other self-regulatory problem behaviors; specifically, attention, emotional reactivity, and obsessive-compulsive tendencies. Maternal, paternal, and teacher ratings of these problem behaviors were obtained from twins at ages five and seven. On the basis of responses to a health questionnaire at age five, subjects were placed into one of three fluency subgroups: highly normally nonfluent children ($N = 547$, 2.6% of the sample); children who were probable for stuttering ($N = 826$, 4.0% of the sample); and typically fluent children ($N = 19{,}072$, 93% of the sample). The primary aim of this investigation was to examine whether children in the two fluency-affected groups received significantly different scores from each other and from the large group of typically fluent children on maternal, paternal, and teacher reports of these problem behaviors. As a secondary objective, a discordant cotwin-control design was used to examine the possibility that common genes might be influencing these self-regulatory phenotypes. Results revealed that, when compared with the control children, subjects in both of the fluency groups were rated by mothers and fathers as displaying significantly more attention and emotional problems at age five and more attention problems and obsessive-compulsive tendencies at age seven. Teacher reports for attention at age seven were generally consistent with the parental findings. Results from the discordant cotwin control analyses supported the existence of a relationship between nonfluency, attention, and emotional reactivity whose origin was likely due to both genetic and shared environmental factors. This study is the first to provide evidence for the presence of a common etiological association between disrupted fluency and other non-speech self-regulatory problem behaviors. Although the mechanisms that underlie this complex association are not yet understood, one hypothesis is that these behaviors may arise from a shared underlying deficit in sensorimotor gating or executive functioning that affects multiple performance domains.

Spontaneous Vocal and Gestural Imitation as Separate Predictors of Cognitive Ability and Elicited Imitation Performance

Susan K. Fenstermacher, Kimberley J. Saudino

Boston University, Boston, MA

Prior research (Fenstermacher and Saudino, Behav Genet 37:751–752, 2007) has shown genetic overlap between elicited imitation and imitation that occurs spontaneously in the absence of explicit modeling. However, it is not known to what degree this relation may be separately driven by vocal or gestural behaviors, or how it might be mediated through influences on general cognitive ability. The present study extends previous findings to examine genetic and environmental factors underlying relations between spontaneous vocal imitation, spontaneous gestural imitation, elicited imitation, and cognitive performance. Imitation and mental development (MDI; Bayley 1994, Bayley Scales of Infant Development-II) data were obtained from 314 pairs of 24-month old twins (MZ = 145, DZ = 169). Multivariate model-fitting analyses revealed substantial genetic overlap between the MDI and all imitation variables. Interestingly, no genetic covariance was found between the imitation variables beyond that mediated through the MDI. Significant unique genetic variance was found for both spontaneous vocal and elicited imitation, explaining 77% and 57% of the total genetic variance on those variables, respectively. Shared environmental covariance was found only between MDI and elicited imitation; nonshared environmental covariance was found between MDI and spontaneous vocal imitation, and between spontaneous vocal and gestural imitation. Thus it appears that the relation between spontaneous vocal and gestural imitation is mediated through genetic and nonshared environmental influences, whereas the relation between elicited and spontaneous imitation is entirely due to genetic factors. Covariance between MDI and spontaneous imitation is primarily due to genetic influences, with some minor nonshared (for vocal) and shared (for gestural) environmental overlap. Finally, the relation between MDI and elicited imitation can be attributed to both genetic and shared environmental factors.

Genetic Effects Explain the Stability of Psychopathic Personality from Mid- to Late Adolescence

Mats Forsman[1], Paul Lichtenstein[1], Henrik Andershed[2], Henrik Larsson[1]

[1]Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden; [2]Center for Research on Criminological psychology, Örebro University, Örebro, Sweden

This study examined the importance of genetic and environmental influence for the stability of psychopathic personality between mid- and late adolescence. The target sample consisted of all 1,480 male and female twin pairs born in Sweden between 1985 and 1986. Psychopathic personality was measured with the Youth Psychopathic traits Inventory (YPI) when the participants were 16 and 19 years old. Results showed that the three psychopathic personality dimensions were stable at different levels of analysis and linked to a stable higher-order general factor (i.e., psychopathic personality factor). Genetic factors contributed substantially to the stability of this general higher-order factor, whereas environmental factors were of little importance. However, we also found specific genetic stability in the Callous/unemotional and Impulsive/irresponsible dimension. Thus, our model provides evidence for etiologic generality and etiologic specificity for the stability of psychopathic personality between mid- and late adolescence.

Elucidating Genetic and Environmental Influences on Adult Attachment Style, Anxiety, and Depression in Middle-Aged Men

Carol E. Franz[1], Timothy York[2], Lindon Eaves[2], Seth A. Eisen[3], Kristen Jacobson[4], Michael J. Lyons[5], Matthew Panizzon[1], William S. Kremen[1]

[1]University of California, San Diego; [2]Virginia Commonwealth University; [3]Washington University; [4]University of Chicago; [5]Boston University

Adult intimate attachment relationships play an important role in providing emotional comfort, affect regulation, feelings of security and are associated with successful aging. However, little genetically informed research examines adult attachment style or its relationship with indicators of adjustment such as depression, or neuroticism. About 1,236 male twins, ages 51–60, participating in the Vietnam Era Twin Study of Aging completed self-report measures assessing adult romantic attachment style, depressed mood (CES-D), and two neuroticism subscales from Tellegen's Multidimensional Personality Questionnaire (anxiety and alienation). Heritability estimates were 0.22 (depression), 0.49 (anxiety), 0.32 (anxious attachment), 0.27 (avoidant attachment), and 0.28 (alienation). Common environment did not contribute significantly to the variance of any measure. In multivariate analyses, overall covariance among the measures was highly significant. The best-fitting model allowed for a single overall genetic factor (with variable specific genetic effects) plus another genetic factor explaining overlap between the attachment measures. There was no support for A and E comprising one latent variable; much of the unique environmental influences (E) were measure specific, especially for attachment style. The persistence of vulnerability to negative emotion (whether expressed by depression, anxiety, or dysfunctional attachments) is largely due to common underlying genetic influences. Evidence from the analyses of shared and unique environmental factors, however, suggests different etiologies based on life experiences and different manifestations in social relationships or generalized anxious responses to situations.

Multivariate Behavioral Genetic Analyses of the Lifetime History of Aggression Questionnaire

Michelle T. Fung, Emil F. Coccaro, Kristen C. Jacobson

CNPRU, Department of Psychiatry, University of Chicago

Behavior genetics models were fit to responses on the Lifetime History of Aggression Questionnaire (LHA) from 3,035 adult twins (1,626 MZ, 1,409 DZ) from the Pennsylvania Twins Cohort. This study examined the five items of the a priori Aggression subscale of the LHA. Phenotypic factor analyses on these items revealed 2 correlated factors ($r = 0.49$), 'non-person-directed aggression' (temper tantrums, verbal fights, and striking objects), and 'person-directed physical aggression' (physical fights and hitting others).

Univariate analyses of the total 5-item scale and the two factors showed significant genetic (0.32–0.50) and nonshared environmental effects (0.50–0.68), and negligible shared environmental effects. A bivariate Cholesky decomposition of the 3- and 2-item factors indicated significant sex differences. For males, heritabilities of the factors were 0.48 and 0.41 (respectively) and the genetic correlation was significantly less than unity ($rg = 0.56$). For females, heritabilities of the factors were 0.35 and 0.32 (respectively) and the genetic correlation was not significantly different from 1.0 ($rg = 0.82$). Next, a series of multivariate genetic factor models were run on the 5 individual items. A 2-factor common pathway model was the best model for both sexes. The factor structures appeared to be similar in both sexes, with one general aggression factor and a second factor indexing only the physical aggression items. Heritabilities of the two latent factors ranged from 0.33 to 0.67 with no significant effects for shared environment. Additional models suggest that variance parameters cannot be equated across sex. Taken together, these preliminary phenotypic and genetic factor analyses suggest that the LHA Aggression subscale may index two factors but that there may be differences in factor structure for males and females. Future analyses will continue to explore sex differences in the genetic and environmental architecture of the LHA Aggression subscale.

A Behavior Genetic Investigation of Laboratory-Assessed Anger and Inhibitory Control in Infancy and Early Childhood

Jeffrey R. Gagne[1], Michele M. Volbrecht[2], H. Hill Goldsmith[1]

[1]Department of Psychology, University of Wisconsin, Madison, WI; [2]Department of Educational Psychology, University of Wisconsin, Madison, WI

Inhibitory control (IC) is an individual differences variable that emerges around 24 months of age and involves the self-regulation of behavior. Previous research using parent report indicated that anger in infancy was associated with IC in toddlerhood and early childhood (Gagne and Goldsmith 2007). All behaviors were genetically influenced, and genetic influences overlapped across early anger and later IC. The present investigation focuses on similar analyses using a laboratory assessment of temperament. Examining early anger and later IC in the laboratory will clarify if associations are similar across assessments, and genetic analyses will allow for the estimation of genetic and environmental influences, as well as genetic and environmental covariance. Participants included 97 MZ and 201 DZ twin pairs from the Genetic of Emotional Ontogeny (GEO) Project at the University of Wisconsin. Temperament was assessed with the prelocomotor (12 months) and preschool (36 months) versions of the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith et al.

Unpublished manuscript, 1995). Phenotypic correlations between observed anger at 12 and 36 months, and observed IC at 36 months were significant although much smaller than in similar parent-rated behaviors. 12-month anger was positively related to 36-month IC, and 36-month anger was negatively related to 36-month IC. MZ correlations for the three variables exceeded DZ correlations, suggesting the presence of genetic influences. A multivariate analysis was conducted using a Cholesky decomposition model. Results indicated that genetic variation accounts for around one-third of variance (30–35%) in all three behaviors, with significant shared environmental effects present for 36-month anger and IC. Unlike previous parent-rated findings, genetic correlations between all three variables were non-significant.

Family Influences on Personality

Jody M. Ganiban[1], Tova Jacobs[1], Erica L. Spotts[2], Paul Lichtenstein[3], David Reiss[4], Jenae Neiderhiser[5]

[1]Department of Psychology, George Washington University, Washington, DC; [2]Behavioral and Social Science Research Program, National Institute of Aging, Bethesda MD; [3]Department of Medical Epidemiology and Statistics, Karolinska Institutet, Stockholm, Sweden; [4]Yale University, New Haven CT; [5]Department of Psychology, Pennsylvania State University, State College, PA

Current personality theories propose that while personality is heritable, one's environment can regulate how personality is expressed. This study explored the degree to which family environment affects genetic and environmental contributions to personality within a subsample of 544 female twins (258 Monozygotic, 286 Dizygotic pairs) drawn from the Twin and Offspring Study in Sweden. The average age of the twins was 43.6 (+4.49) years, and each twin was in a stable romantic relationship and had at least one adolescent child (mean child age = 15.8 ± 2.4 years). All twins completed the Karolinska Scales of Personality (KSP) and the Temperament and Character Inventory (TCI). The twins also completed the family conflict, cohesion and control subscales of the Family Environment Scale (FES). Factor analysis with the KSP and TCI yielded three personality factors (anxiety, aggression, and sociability) that demonstrated significant additive genetic (h2 range 0.50 to 0.54) and nonshared environmental variance (e2 range 0.46–0.50). Additional analyses indicated that as family conflict increases, shared environmental contributions to anxiety and aggression also increase. Similarly, when there are higher levels of control within the family, shared and nonshared environmental contributions to aggression increase, while genetic contributions decrease. Thus, when extremes in family environments are present, the relative importance of environmental factors increases, even if genetic contributions remain constant. Moreover, much of the increasing influence of environmental factors on personality reflects shared environmental factors. Because shared environment most likely includes experiences the twins shared when they were children and living in the same family, these findings suggest that lessons learned early in development emerge and affect the expression of aggression and anxiety when adults are under stress.

Genetic Overlap Between Alcohol Consumption Indices and Alcohol Dependence: Can Consumption be Used as an Index of Genetic Risk for Dependence?

Julia D. Grant[1], Andrew C. Heath[1], Kathleen K. Bucholz[1], Pamela A.F. Madden[1], Nicholas G. Martin[2]

[1]Department of Psychiatry, Washington University School of Medicine, St. Louis, MO; [2]Queensland Institute of Medical Research, Brisbane, Australia

Identification of quantitative indices of alcohol consumption that are closely related to risk for alcohol dependence (AD) would afford increased power for the detection of genetic linkage and association. We examined the extent to which genetic influences on alcohol consumption overlap with DSM-IV AD symptom count and DSM-IV AD symptom clustering. Data from 6257 individuals who participated in a telephone diagnostic interview for the Australian Twin Study were included ("1989 cohort", $M = 29.9$ years; 2,761 complete pairs: MZF = 698, MZM = 494, DZF = 513, DZM = 395, DZO = 661, 735 singletons). Factor analyses indicated that a single alcohol consumption factor captures the association between maximum 24-h consumption, maximum tolerance, and regarding the period of heaviest use: typical weekly consumption, frequency of heavy drinking, and frequency of drinking to intoxication. Quantitative genetic analyses were conducted to examine the extent of genetic overlap between AD symptoms (0, 1, 2, 3, 4+), a 7-level alcohol consumption score (created from the factor score, with individuals having 3+ symptoms set to missing), and AD symptom clustering (missing for those with <3 symptoms). Genetic and nonshared environmental influences were significant for all three measures ($a2 = 0.34$, 0.49, 0.32 and $e2 = 0.55$, 0.40, 0.55 for AD symptoms, alcohol consumption, and AD symptom clustering respectively). Modest nonsignificant shared environmental influences were also observed ($c2 = 0.11$, 0.12, and 0.13, respectively). All genetic influences on AD clustering were overlapping with those on AD symptoms (there were genetic influences specific to the consumption score). These analyses indicate that: (1) AD symptom count can provide a meaningful index of genetic vulnerability to AD (rG = 1.0 between symptoms and clustering), and (2) even when consumption is treated as an unknown for AD individuals, consumption remains a useful index of genetic vulnerability to AD (rG = 0.95).

Genome-Wide Association for Loci Influencing Entrepreneurial Behavior: The Rotterdam Study

P. J. F. Groenen[1], A. Hofman[2], P. Koellinger[1], M. J. H. M. van der Loos[1], F. Rivadeneira[3], F. van Rooij[2], R. Thurik[1], A. Uitterlinden[2,3]

[1]Economics, Erasmus University, Rotterdam, The Netherlands; [2]Epidemiology and Biostatistics, Erasmus Medical Centre, Rotterdam, the Netherlands; [3]Internal Medicine, Erasmus Medical Centre, Rotterdam, The Netherlands

Entrepreneurship is one of the scarcest and most essential input factors of the modern economy. The economics and management literature has identified numerous socio-demographic factors and personality characteristics that are associated with entrepreneurial behavior (Grilo and Thurik, 2008, Industrial and Corporate Change, forthcoming). However, endogeneity problems and unobserved heterogeneity often impede the establishment of causality. Yet, the systematic occurrence of intergenerational effects points at a new avenue of research. The propensity to become self-employed has recently been shown to be highly heritable (Nicolaou et al. Manage Sci 54(1):167–179, 2008). However, the particular genes and the causal pathways influencing the genetic predisposition of entrepreneurial activity remain largely unknown. We therefore carried out genome-wide association analyses (GWAs) for entrepreneurial activity using data from a large population-based prospective cohort of elderly Caucasians (Hofman et al. Eur J Epidemiol 22:819–829, 2007). Information on entrepreneurial activity was available in 5,375 individuals and is defined in three different ways as individuals who were: (1) at least once self-employed ($n = 532$); (2) at least two times self-employed ($n = 402$); and (3) never anything else than self-employed ($n = 140$). Genotyping was done with the Illumina

550 K Single Nucleotide Polymorphism (SNP) arrays and available for 5,373 phenotyped individuals. All subjects are Dutch Caucasians as determined by Identity-by-State (IBS) clustering analysis of genotype data, and after exclusion of 102 genetic outliers during quality control (mean genotype call rate 99.5%). After filtering and genotype pruning (MAF < 0.05, HWE < 0.0001, SNPcall rate 95%), 503618 SNPs were analyzed in men and women separately and tested for association using permutation (PLINK software). Prevalence of entrepreneurial behavior ranged between 3% and 14% and was significantly different across genders except for those "never anything else than self-employed" ($n = 70/70$). Individual genotype-phenotype associations did not reach genome-wide significance (lowest $P = 1.4 \times 10^{-6}$), while Q–Q plots show that multiple loci are associated. We therefore went on to identify loci with associations across the different definitions of entrepreneurship. In males, 11 loci from 16 SNPs and in women 19 loci from 21 SNPs were associated consistently at $P < 0.001$ across all trait definitions. One such locus was replicated across genders. Odds ratios for markers in these loci ranged between 1.3 and 3.2 and likelihood increased with increasing entrepreneurial activity. Replication of these findings is currently underway.

Infant Temperament, Marital Relationship, and Parenting in Adoptive Families

Nastassia J. Hajal[1], Ginger A. Moore[1], Daniel S. Shaw[2], Leslie D. Leve[3], Jenae M. Neiderhiser[1], David Reiss[4]

[1]The Pennsylvania State University, Department of Psychology, University Park, PA; [2]University of Pittsburgh, Department of Psychology, Pittsburgh, PA; [3]Oregon Social Learning Center, Eugene, OR; [4]Yale Child Study Center, Yale University, New Haven, CT

Various factors within individuals and the family have been found to influence parenting. We hypothesized that infant temperament, reflecting genetic and environmental factors, would evoke over-reactive parenting (OP; reflecting anger, meanness, and irritability) differentially for mothers and fathers. We also expected that marital warmth would impact the evocative effects of temperament on parenting. In a sample of 355 adoptive families with 9-mo-old infants, mothers and fathers reported on marital warmth, infant temperament (IBQ), and parenting, and birth mothers completed personality questionnaires (Adult Temperament Scale). Mother and father reports of Infant Smiling and Laughter (SAL) and Marital Warmth were combined to decrease method variance. To create a measure of infant temperament correlated with genetic factors, SAL was regressed onto birth mothers' sociability (BM-SOC) and predicted scores saved. Residual scores measured infant temperament independent of variability related to BM-SOC. Mothers' and fathers' OP was predicted by composite SAL (βs $= -0.28$, -0.15, both $P < 0.01$) and by that portion of SAL independent of BM-SOC (βs $= -0.25$, -0.15, both $P < 0.05$). Marital warmth explained significant additional variance only for fathers when predicting OP from all measures of infants' SAL (βs $= -0.19$, -0.25, -0.26, all $P < 0.01$) and attenuated the relation between OP and SAL independent of BM-SOC to non-significance. Results suggest adoptive parents respond to aspects of infant temperament that are uncorrelated with a theoretically similar measure of birth mother's personality. However, the family affective environment affects mothers' and fathers' parenting differently. Adoptive fathers' parenting is influenced by marital warmth, while adoptive mothers' parenting seems to be primarily influenced by environmentally-related child characteristics. Additional analyses will examine the role of adoptive parents' temperament as a contributor to parenting.

Religiosity and Adolescent Antisocial Behavior

K. Paige Harden, Elliot M. Tucker-Drob, Jennifer E. Hill, Jane Mendle, Eric Turkheimer

University of Virginia

Antisocial behavior (ASB)—defined as behavior that violates laws, social norms, and/or the rights of others—is so common in adolescence that some authors have suggested that it should be considered developmentally normative (e.g., Moffitt, Psychol Rev 100:674–701, 1993). Normative or not, adolescent ASB negatively impacts victims, who bear the burden of damaged property, medical expenses, lost wages, and diminished quality of life, as well as society as a whole. Moreover, ASB can be entrapping for adolescents, who must deal with the consequences of teenage childbearing, substance dependence, and criminal records for far longer than their teenage years. Sociologists have long observed that adolescents who are affiliated with and involved in religious organizations demonstrate less ASB; however, the processes underlying this relation are unclear. Religious adolescents may be less antisocial because of some causal mechanism, as posited by most theoretical work, or this association may be an artifact of uncontrolled genetic and environmental differences between religious and non-religious families. The current project examines the relation between religiosity and antisocial behavior using a sample of sibling pairs from the National Longitudinal Study of Adolescent Health. Participants were 11–21 years old at the study's beginning, and they were assessed on three occasions over six years. Latent growth curve models indicated that there were significant inter-individual differences in both initial level and intra-individual change in ASB. Moreover, religiosity significantly predicted both initial level and change in ASB, after controlling for genetic and shared environmental confounds, consistent with theories suggesting that religiosity causally protects against adolescent ASB.

Genetic Influences on Language, Reading, and Mathematic Skills in a National Sample: A Selected and Unselected Analysis in the NLSY

Sara A. Hart, Stephen A. Petrill, Claire M. Kamp Dush

The Ohio State University

Purpose The present study is an examination of the genetic effects in both a selected and unselected sample across multiple domains of cognitive achievement. Furthermore, this study uses a largely representative population sample of children to determine if the conclusions from twin and adoption studies can be generalized. *Methods* A kinship algorithm which assigned degree of genetic relatedness to all available pairings (Rodgers et al. Intelligence 19:157–177, 1994) was applied to the 1994 wave of the National Survey of Youth Children's sample. Four cognitive achievement outcomes related to language, reading and mathematics were analyzed across the general sample, as well as for children selected below the lowest 20%tile. *Results* The tests of receptive vocabulary, decoding, reading comprehension and mathematics all suggested estimates of group heritability and full sample heritability of moderate effect sizes, and all estimates were significant. Furthermore, all estimates were within standard errors of previously reported estimates from twin and adoption studies. *Conclusions* The present study gives support for significant genetic effects across low and wide range of specific achievement. Moreover, this study supports that genetic influences on reading, language, and mathematics are generalizabile beyond twin and adoption studies.

Is There a Party in Your Genes?

Peter K. Hatemi[1], Sarah E. Medland[1], Nicholas G. Martin[2], Lindon J. Eaves[1]

[1]Virginia Institute for Psychiatric and Behavioral Genetics, Richmond, Virg., USA; [2]Queensland Institute of Medical Research, Brisbane, Australia

Using data from a population based sample of twin families, we examine the sources of party identification as well as the intensity of that identification. The results indicate genetics exerts little, if any, influence on party identification, directly or indirectly. However, genes do play a pivotal role in shaping the intensity of an individual's party identification, even after modeling the influence of personality and religiosity. Together with recent examinations of political attitudes and vote choice, these findings provide a more complete picture of the source of partisanship and the complex "nature" of the political phenotype.

Genetic Etiologies of Parent- and Teacher-Rated ADHD Symptom Dimensions

Jesse L. Hawke[1], John C. DeFries[1], Bruce F. Pennington[2], Sally J. Wadsworth[1], Erik G. Willcutt[1]

[1]Institute for Behavioral Genetics, University of Colorado, Boulder CO; [2]University of Denver, Denver CO

In order to assess the genetic etiologies of DSM-IV ADHD symptom dimensions, both parent- and teacher-rated data from 59–106 monozygotic and 47–98 same-sex dizygotic selected twin pairs were analyzed using DeFries-Fulker regression analysis (DF analysis; DeFries and Fulker, Behav Genet 15:467–473, 1985). Univariate basic and extended DF models (e.g., Hawke et al. Dyslexia 12:21–29, 2006) were fitted to parent- and teacher-rated inattention (IN), hyperactivity-impulsivity (HI), and combined ADHD (Combined) data. Phenotypic correlations between parent and teacher ratings for IN, HI, and Combined were 0.61, 0.50, and 0.60, respectively. Results obtained from the extended DF analyses provided no evidence for a differential genetic etiology as a function of gender for any of the parent- or teacher-rated phenotypes. Therefore, heritability (h_g^2) estimates were pooled for males and females (Parent/IN: $h_g^2 = 0.80$; Parent/HI: $h_g^2 = 0.71$; Parent/Combined: $h_g^2 = 0.73$; Teacher/IN: $h_g^2 = 0.53$; Teacher/HI: $h_g^2 = 0.35$; Teacher/Combined: $h_g^2 = 0.45$). In addition, when bivariate basic and extended DF models were fitted to the data, no evidence was obtained for a differential genetic etiology as a function of gender. Bivariate h_g^2 estimates pooled across males and females ranged from moderate (0.31) to high (0.72). Genetic correlations were then computed from the univariate and bivariate h_g^2 estimates using the method of Knopik et al. (Behav Genet 27:447–453, 1997) (Parent-IN/Parent-HI: $r_g = 0.53$; Teacher-IN/Teacher-HI: $r_g = 0.79$; Parent-IN/Teacher-IN: $r_g = 0.94$; Parent-HI/Teacher-HI: $r_g = 0.83$; Parent-Combined/Teacher-Combined: $r_g = 1.0$). In conclusion, results obtained from these analyses suggest that parent- and teacher-rated DSM-IV ADHD symptom dimensions are highly heritable and that their observed correlations are due substantially to genetic influences (This article is supported by NICHD Center Grant HD-27802).

The Effects of Peer Group Behavior on Delinquency in Adolescent Twins

Jennifer Hill, K. Paige Harden, Robert Emery, Eric Turkheimer

University of Virginia

Peer delinquency is one of the strongest correlates of delinquency in adolescence (e.g. Dishion and Dodge, J Abnorm Child psychol 33:395–400, 2005), and may influence adolescents directly through peer pressure or indirectly by defining normative behavior. However, behavior genetic research has implicated genetic confounds in the relationship between best friend and adolescent alcohol use and suggest that best friend behavior may only influence behavior for susceptible individuals (Harden et al. Behav Genet, in press). This paper uses longitudinal and behavior genetic designs to address the potential genetic and environmental confounds in the association between peer group and adolescent behavior. This paper uses a sample of 1,820 twin and sibling pairs from three waves of the National Longitudinal Study of Adolescent Health (Udry 2003, Add Health User Guides). Peer delinquency is measured using peer self-report and peer groups were defined by target nomination of friends and friend nominations of the target. Delinquency was modeled from early adolescence into early adulthood using latent growth curve modeling. Peer problem behavior was modeled as risk in the multivariate twin and sibling model; the growth components were regressed onto the ACE variance components for the peers. Genetic and shared environmental components completely accounted for the mean level relationship between peer group and mean level adolescent problem behavior. However, peer group delinquency accounted for additional variance in the trajectory of adolescent problem behavior after considering confounds. Greater peer group problem behavior resulted in a more pronounced spike in delinquency in mid and late adolescence. Involvement in a delinquent peer group may influence problem behavior in adolescence, but may not be related to delinquency characterized by early involvement and continuation into adulthood.

Genetic Influences on Extreme Autistic Traits and Intellectual Impairment: Overlap and Specificity

Rosa A. Hoekstra[1], Angelica Ronald[2], Francesca Happé[3], Simon Baron-Cohen[1], Robert Plomin[3]

[1]Autism Research Centre, Section of Developmental Psychiatry, University of Cambridge, Cambridge, United Kingdom; [2]Birkbeck College, London, United Kingdom; [3]Institute of Psychiatry, King's College, London, United Kingdom

Autism spectrum disorders (ASDs) are associated with various degrees of intellectual impairment. The origin of this association is not well understood. Recently it has been suggested that the prevalence of severe intellectual impairment in ASD may be overestimated due to ascertainment bias (Skuse 2007). The current project aimed to investigate the association between autistic traits and intellectual impairment in children selected from a community based sample, and to examine whether overlap between these traits can be explained by genetic factors. The most extreme scoring 5% on a measure of autistic traits, cognitive ability and academic achievement were selected from 7,965 7-year-old twin pairs and from 3,687 pairs when the twins were 9 years old. Phenotypic associations between extreme autistic traits and intellectual

impairment were compared with associations among the full range scores. Bivariate DeFries-Fulker extreme analyses were employed to assess the genetic associations between these traits. Results showed that endorsement of autistic traits is modestly related to cognitive disability and academic underachievement, both in the extreme scoring groups and in the sample as a whole. This association is mainly reflected in autistic trait items assessing communication difficulties. The association was observed both in teacher and parental ratings of autistic traits and at either time point. Bivariate DeFries-Fulker analyses showed that there is a degree of genetic overlap between intellectual impairment and extreme autistic traits, but also substantial genetic specificity. These results suggest that the relationship between autistic traits and intellectual impairment is modest; driven by communication problems characteristic for autism; and due to a modest degree of shared genetic influences. These observations largely agree with Skuse's proposition of an inflated prevalence statistic of intellectual impairment in ASDs due to ascertainment bias.

Serotonin Transporter Three Allele System Behaves Like Three Different Functional Alleles

James J. Hudziak[1,2], Lynsay A. Ayer[1,2], Masha Y. Ivanova[1,2], David C. Rettew[1,2], Robert R. Althoff[1,2], Erik A. Ehli[1,2], Timea Lengyel-Nelson[1,2], Gareth E. Davies[1,2]

[1]Vermont Center for Children, Youth, and Families at the University of Vermont College of Medicine, Burlington, VT; [2]Avera Institute for Human Behavioral Genetics, Avera Behavioral Health Center, Sioux Falls, SD

Objective Recently, the serotonin transporter gene (SERT) has been characterized as a three allele system in which the long allele is divided into an La (functioning as a 'long allele'), and an Lg (reportedly functioning as a 'short allele'). This research aims to determine if the Lg allele indeed functions as an S allele in relation to internalizing psychopathology. *Method* 420 children (60% male, mean age = 10.94, age range = 5–18) from a large family study in Vermont were genotyped on a three allele system of the 5-HTTLPR gene. DNA was obtained by cheek swabs. The Child Behavior Checklist (CBCL) and Vermont Structured Diagnostic Interview (VSDI) were used to assess internalizing. *T*-tests were conducted to examine differences in psychopathology between the following groups of children: (1) S/S ($N = 82$) vs. S/Lg ($N = 23$), (2) S/Lg ($N = 23$) vs. S/La ($N = 176$), (3) S/La ($N = 176$) vs. Lg/La ($N = 34$), and (4) La/La ($N = 105$) vs. Lg/La ($N = 34$). *Results* There were no differences in internalizing scores between the S/S and S/Lg groups. Children with the S/Lg genotype had higher scores on the CBCL Somatic Complaints scale relative to those with the S/La genotype. Youth with the Lg/La genotype had higher CBCL Anxious/Depressed and Internalizing scores, and more DSM-IV Dysthymia symptoms relative to those with the S/La genotype. Finally, the Lg/La genotype was also associated with higher CBCL Anxious/Depressed and Internalizing scores and more DSM-IV Major Depressive Disorder symptoms when compared to the La/La genotype. *Conclusions* Lg may predict greater risk for internalizing relative to the S and La alleles. These data should be interpreted carefully, however, as unequal Ns and non-independence of the data (i.e., multiple children within families) were not accounted for. These results suggest that the SERT S, La, and Lg function as three discrete alleles. Analyses that collapse Lg and S as a single allele may need to be reconsidered.

Assortative Mating for Psychopathology, Personality Traits, and Candidate Genes as Evidence of an Active Gene-Environment Correlation

Mikhila Humbad[1], M. Brent Donnellan[1], Matthew McGue[2], William G. Iacono[2], S. Alexandra Burt[1]

[1]Michigan State University; [2]University of Minnesota

Assortative mating is often assumed to result from active gene-environment correlations (active rGE) whereby individuals ultimately select genetically similar partners based on phenotypic similarity. However, the broad assumption that genetic factors underlie phenotypic similarity is rarely evaluated empirically. The current study aims to address this gap by exploring assortative mating on both phenotypic and genetic levels using data from 1,806 married couples from three studies housed at the University of Minnesota. We will first determine the degree of spousal similarity on both psychopathology (i.e., Conduct Disorder, Alcohol Dependence, Major Depressive Disorder, and adult symptoms of Antisocial Personality Disorder) and personality traits (i.e., Positive Emotionality, Negative Emotionality, and Constraint). We will also evaluate whether or not spousal convergence (whereby individuals become more similar to their spouses over time) is a viable explanation for spousal similarity. Next, we will seek to identify associations between three candidate genes (i.e., serotonin intron 2 VNTR, monamine oxidase A, and the dopamine d4 receptor) and the above phenotypes for couples with complete genetic data ($n = 176$ couples). Finally, given the expectation of assortative mating at the phenotypic level, we will examine whether these associations are explained by actual genetic covariation between spouses. Specifically, we will estimate the proportion of phenotypic similarity that can be explained by genetic covariation using structural equation modeling. In this way, we hope to determine whether assortative mating is indeed a function of active rGE, and perhaps more importantly, provide a statistical framework for future such examinations.

Genetic and Environmental Covariations Among Obsessive-Compulsive Symptoms, Neuroticism, and Extraversion in South Korean Adolescent and Young Adult Twins

Yoon-Mi Hur[1], Hoe-Uk Jeong[2]

[1]Chonnam National University, Gwangju, South Korea; [2]Mokpo National University, Mokpo, South Korea

Patients with obsessive compulsive (OC) disorder typically show higher level of neuroticism and lower level of extraversion, as compared to normal controls. The present study explored genetic and environmental contributions to the covariations among OC symptoms and two major personality dimensions, neuroticism and extraversion. Five hundred and twenty-four pairs of monozygotic (MZ) and 228 pairs of dizygotic (DZ) twins from the South Korean Twin Registry completed Maudsley Obsessive Compulsive Inventory (MOCI) and Neuroticism and Extraversion scales of the Eysenck Personality Inventory. Consistent with literature, the correlation between the MOCI and Neuroticism was high and positive ($r = 0.44$), while the correlation between the MOCI and Extraversion was low and negative ($r = 0.10$). The cross-twin cross-trait correlations between Neuroticism and MOCI were significantly higher for MZ than for DZ twins, while those between Extraversion and MOCI were not significantly

different between the two types of the twins. Trivariate cholesky model was applied to the twin data. The model-fitting results indicated that the significant phenotypic association between Neuroticism and the MOCI was largely due to shared genetic factors, and that the small phenotypic correlation between the MOCI and Extraversion was largely mediated by correlated individual specific environment. The results of the present study suggest that Neuroticism may be an endophenotype of OC symptoms.

Heritability of Skin Conductance Reactivity in Children

Joshua Isen[1], Catherine Tuvblad[1], Laura Baker[1], Adrian Raine[2], Dora I. Lozano[3]

[1]Department of Psychology, University of Southern California, Los Angeles CA; [2]Departments of Criminology, Psychiatry, Psychology, University of Pennsylvania, Philadelphia PA; [3]Universidad Autónoma de Ciudad Juárez, Mexico

This study assessed the genetic covariance between various measures of phasic skin conductance activity, including response amplitude and frequency of responding. A few studies have investigated the etiology of skin conductance reactivity (e.g., Lykken et al. Psychophysiology 25:4–15, 1988), but none have been conducted with children. Given that deficits in skin conductance orienting are associated with psychosis-proneness and conduct problems, it is important to understand the genetic and environmental contributions to skin conductance reactivity in children. Subjects for this study were 800 male and female twins, aged 9–10, who passively listened to stimuli during an orienting task. The stimuli consisted of tones of moderate intensity (75 dB), as well as different types of socially meaningful sounds (e.g. baby cries and speech-like stimuli). Skin conductance response magnitude, averaged across all stimuli, was substantially heritable. Genetic model-fitting was used to determine if the variation in reactivity across the different types of stimuli can be explained by a single latent factor. Furthermore, there was a moderate phenotypic correlation between a continuous measure of reactivity (i.e. response amplitude) and a more categorical measure of skin conductance (i.e. frequency of responding). This association was not genetically mediated, suggesting a theoretical distinction between hyporeactivity and nonresponding.

Evaluation of the Correlation Between Heritability (h2) and g-Loadings in Psychometric Intelligence Tests

Kees-Jan Kan, Han van der Maas

Department of Psychological Methods, University of Amsterdam

In psychometric intelligence testing, the method of correlated vectors has been used to investigate relationship between the general factor of intelligence (g) and heritability of IQ subtest scores. The crux of this method is the correlation between the subtests' g-factor loadings (obtained in a factor analyses) and the subtests' heritability coefficients (h2; obtained for instance in a twin study). As an empirical finding, it is often considered that this correlation is a significant and well-established fact, which is taken to suggest a high influence of the genetic component of g. Such a finding would be problematic for alternative explanations (sampling theory, the Dickens & Flynn model of intelligence, and the mutualism model of intelligence), which do not include a general factor as an underlying quantitative variable, but which do give rise to the positive manifold. In this presentation, we review the relevant literature. Secondly, we present simulation studies to investigate the relation between h2 and g-loading. We end with a discussion concerning the consequences and implications for intelligence research.

Evaluation of the Cascade Model: A New Extended Twin Family Model

M. C. Keller, S. E. Medland

Extended twin family designs offer scientists a much more precise assessment of the genetic and environmental factors affecting trait variation compared to traditional (e.g., twin or adoption) behavioral genetic designs. This increase in precision comes at a cost of increased complexity, making it difficult to gauge how the models are actually performing at capturing reality. Here, we evaluate the performance of the cascade model by comparing its results to simulation data from the GeneEvolve program, and we compare this performance to the Stealth model, nuclear twin family model, and classical twin design.

A Role of Aggressive Behavior in Sexual Isolation Between *Drosophila pseudoobscura* and *Drosophila persimilis*

Yong-Kyu Kim, Kyle F. Abramowicz, Wyatt W. Anderson

Department of Genetics, University of Georgia, Athens, GA 30602, USA

Drosophila pseudoobscura and *Drosophila persimilis* are ideal models for the study of speciation. They are of particular interest because of their natural distribution: the range of *D. persimilis* is entirely within that of *D. pseudoobscura*. No hybrids between these two species are found in nature (Dobzhansky and Powell 1975). Anderson and Kim (2005) have demonstrated strong sexual isolation between sympatric and allopatric populations. Aggressive behavior has recently been reported in several Drosophila species, and serves for the acquisition or defense of food resources, as well as in access to mates in nature (Jacobs 1960; Spieth 1974; Dow and von Schilcher 1975; Hoffmann 1987; Boake et al. 1997; Lee and Hall 2000; Chen et al. 2002; Nilsen et al. 2004; Dierick and Greenspan 2006; Edwards et al. 2006). In this experiment, aggressive behavior of these two species was observed to investigate its roles in sexual isolation between them. Six aggressive behaviors were observed—wing threat, fencing, lunging, boxing, holding, and chasing. With sympatric and allopatric pairs of *D. pseudoobscura* and *D. persimilis*, aggressive behavior between heterospecific males occurred. Preliminary data show that *D. pseudoobscura* males, in sympatry with *D. persimilis*, are more aggressive, although not significantly so, than *D. pseudoobscura* males in allopatry. These results suggest potential roles of aggressive behavior in reproductive isolation, and are consistent with an increase in female *D. pseudoobscura* discrimination in sympatry, where fewer heterospecific matings occurred (Noor and Ortíz-Barrientos 2006). Both male aggressiveness and female discrimination may contribute to sexual isolation between two species in sympatry.

CHRM2 Polymorphisms and Cognitive Ability

Robert M. Kirkpatrick, Matt McGue, Daniel Irons, William G. Iacono

Department of Psychology, University of Minnesota

Although it is well established that cognitive ability is substantially heritable, few specific genetic factors have been identified. CHRM2, which codes for the cholinergic muscarinic receptor 2, is a candidate gene for cognitive ability. CHRM2 polymorphisms have shown

association with the IQ phenotype in four different studies by independent research teams (Dick et al. Behav Genet 37:265–272, 2007). The present study investigates the association of CHRM2 variants with IQ in a sample of 2,543 individuals combined from two longitudinal family studies, one of twins and the other of adopted siblings. Participants were genotyped on 6 SNPs in and around the CHRM2 gene. Cognitive functioning of participants was assessed at intake in both longitudinal studies using an abbreviated version (2 Performance subtests, 2 Verbal subtests) of the WAIS-R or WISC-R, as age-appropriate. The results of a family-based quantitative association test in PLINK (Purcell 2008, PLINK (Version 1.03) [Computer software and manual]. Retrieved June 11, 2008, from http://pngu.mgh.harvard.edu/purcell/plink/; also see Purcell et al. Am J Hum Genet 81, 2007) do not support an association of these CHRM2 polymorphisms with any assessed IQ phenotype.

Phenotypic and Genetic Associations Between Binge Eating and Ovarian Hormones

Kelly L. Klump[1], Pamela K. Keel[2], Kristen M. Culbert[1], Crystal Edler[2], S. Alexandra Burt[1]

[1]Department of Psychology, Michigan State University; [2]Department of Psychology, University of Iowa

Observed increases in genetic effects on disordered eating during puberty (Klump et al. Psychol Med 37:627–634, 2007) indirectly implicate ovarian hormones in the genetic diathesis of eating pathology. Ovarian hormones (estrogen and progesterone) are activated at puberty, regulate gene transcription, and cause predictable changes in food intake in a variety of species. Nonetheless, few studies have examined ovarian hormone influences on disordered eating, and none have examined genetic mediation of these effects. Changes in ovarian hormones across the menstrual cycle provide a powerful test of these influences—if hormones are genetically associated with disordered eating, then menstrual cycle changes in disordered eating and hormones should show phenotypic and genetic associations. In study 1, we examined both types of associations in an on-going pilot study of female twins who are completing measures of binge eating and provided salivary hormone samples daily for 65 days. In study 2, we replicated results by examining genetic mediation in a sample of 42 twins assessed during the mid-luteal phase of the menstrual cycle only. Twins for both studies were drawn from the Michigan State University Twin Registry (Klump and Burt, Twin Res Hum Genet 9:971–977, 2006). Preliminary findings from study 1 revealed significant phenotypic associations between binge eating and ovarian hormones across the menstrual cycle. Twin correlations in studies 1 and 2 indicated that these phenotypic relationships were due to shared genetic effects; MZ cross-twin, cross-trait correlations approached the within-person correlation and were significantly greater than those of DZ twins. Overall, findings confirm the likely presence of genetic associations between ovarian hormones and disordered eating. Larger scale twin studies are needed to estimate the magnitude of shared genetic effects using more sophisticated analytic techniques (e.g., dynamical systems models).

Testing the Equal Environments Assumption in the Children of Twins design

Laura B. Koenig, Theodore Jacob

Veterans Affairs Palo Alto Health Care System, Menlo Park, CA

The Children of Twins (COT) design, where the environmental and genetic risk transmitted to a child is dependent upon the status of both the father and the father's cotwin, is important in investigating the environmental impact of living with a problem parent. As in other behavior genetic designs, the logic used in the COT method breaks down if the zygosity of the twin pair is confounded with the risk environment provided to the child (a version of the Equal Environment Assumption). In the COT design, for discordant monozygotic (MZ, identical) twin pairs a child of the unaffected twin with an affected uncle has high genetic but low environmental risk; for discordant dizygotic (DZ, fraternal) twins, however, a child of the unaffected twin has moderate genetic and low environmental risk. It has been hypothesized that MZ twins see each other more often than DZ twins, and when the uncle of a child is the affected member of the twin pair this increases the environmental risk of children of MZ twins but not DZ twins. The current study was designed to test this environmental assumption of the COT design, with affected twins having a lifetime diagnosis of alcohol dependence (AD). Results indicated that MZ twins do have more contact than DZ twins, but that this contact did not have a main effect on child's behavior. To examine if contact interacts with group status to predict child outcomes, regression and ANOVA analyses were conducted on several child outcomes (alcohol dependence, conduct disorder, and nicotine dependence symptoms and diagnoses). Again, contact seemed to have little effect on differences seen in discordant MZ vs DZ twins' children. Interesting interactions were found, however, for some child outcomes when comparing the control group's children to discordant twins' children, with the effect being driven by differences in child outcome for high and low contact in the control group. Implications of the findings for the COT design will be discussed.

A Twin Study of APOE Genotype and Regional Cortical Thickness in Middle Age

William S. Kremen[1], Matthew S. Panizzon[2], Christine Fennema-Notestine[1], Lisa T. Eyler[1], Kristen C. Jacobson[3], Michael C. Neale[4], Carol E. Franz[2], Michael J. Lyons[5], Carol E. Franz[2], Hong Xian[6], Seth A. Eisen[6], Bruce Fischl[7], Larry J. Seidman[7], Nikos Makris[7], Ming T. Tsuang[2], Anders M. Dale[2]

[1]UC San Diego & San Diego, VA; [2]UC San Diego; [3]University of Chicago; [4]Virginia Commonwealth University; [5]Boston University; [6]Washington University & St. Louis, VA; [7]Harvard Medical School

The APOE-e4 allele increases risk for Alzheimer's disease and perhaps mild cognitive impairment (MCI). Neuroimaging studies of APOE genotype tend to focus on older adults and on medial temporal (primarily hippocampal) brain regions. Little is known about earlier effects of e4 on brain anatomy and the extent to which brain regions other than hippocampus may be affected. In wave 1 of the Vietnam Era Twin Study of Aging (VETSA), we examined brain structure and APOE genotype in 406 middle-aged male twins (110 MZ pairs, 93 DZ pairs; age range = 51–59). We compared MRI-assessed hippocampal volume as well as cortical thickness in 64 regions of interests (ROIs)—half in each hemisphere—in e4 + vs. e4- subjects. Using classical twin analyses with moderated means for APOE genotype, we determined: (1) genetic and environmental influences on the brain volume and thickness ROIs; (2) whether ROI size differed as a function of APOE genotype; and (3) the amount of variance accounted for by APOE genotype. Heritability of left and right hippocampal volume was 0.65 and 0.67, respectively. Average cortical ROI heritabilities were 0.49 in the left and 0.47 in the right hemisphere. There were no medial temporal (hippocampus, parahippocampal gyrus, entorhinal cortex) differences based on APOE

genotype, but e4 + subjects had highly significantly thinner cortices in large portions of prefrontal cortex. Thickness differences were even stronger on a continuous surface map of cortical thickness which is unconstrained by boundaries imposed by the ROIs. APOE genotype accounted for up to about 1% of the phenotypic variance and up to about 2% of the genetic variance in cortical thickness for ROIs that differed on the basis of e4 status. Results are consistent with APOE-e4 affecting brain structure in midlife, well before onset of dementia or even MCI. Consistent with growing evidence for non-mnestic forms of MCI, many e4 + subjects may be at risk for executive function deficits as they age.

Do Genetic Influences Among Specific Brain Regions Parallel Brain Development and Brain Aging?

William S. Kremen[1], Lars Rimol[2], Elizabeth Prom-Wormley[3], J. Eric Schmitt[3], Matthew S. Panizzon[2], Christine Fennema-Notestine[1], Lisa T. Eyler[1], Michael C. Neale[3], Carol E. Franz[2], Michael J. Lyons[4], Hong Xian[5], Seth A. Eisen[5], Bruce Fischl[6], Larry J. Seidman[6], Nikos Makris[6], Ming T. Tsuang[2], Anders M. Dale[2]

[1]UC San Diego & San Diego, VA; [2]UC San Diego; [3]Virginia Commonwealth University; [4]Boston University; [5]Washington University & St. Louis, VA; [6]Harvard Medical School

To understand brain aging, we must elucidate genetic and environmental influences on regional brain anatomy, but little is known about the patterning of those influences. Previous MRI twin studies had few subjects or used mostly broad brain measures. A recent more comprehensive study was conducted in children. We present the first large-scale study of the heritability of specific brain regions of interest (ROIs) to cover the entire brain in an adult sample. Subjects were men in wave 1 of the Vietnam Era Twin Study of Aging (VETSA): 406 VETSA twins had MRIs (110 MZ & 93 DZ pairs; ages 51–59). We measured of 16 subcortical ROI volumes and 64 cortical ROI thicknesses. We also created continuous genetic correlation (Rg) maps of cortical thickness via 3 seed point analyses in which Rgs between thickness at the seed point and at all other points across the cortex—unconstrained by ROIs—were mapped. Mean heritability of subcortical ROIs was 0.75 in the left and 0.76 in the right hemisphere; mean cortical ROI heritabilities were 0.49 (left) and 0.47 (right). The highest cortical heritability (0.76 in superior frontal gyrus) equaled the average of subcortical ROIs. A primary visual cortex seed point showed strong Rgs with other primary sensory and motor—but not association—areas. A mid-frontal seed point showed strong Rgs with inferior parietal cortex. An anterior temporal seed point showed strong Rgs with inferior and orbital frontal cortices. Cortical-subcortical differences suggest greater genetic control of earlier developing regions, although not in all cases. Seed point analyses provide partial support for genetic influences patterned according to brain development (sensory vs. association areas; myelination patterns) or functional connectivity. They argue against brain anatomy being influenced by a single genetic factor, suggesting that whole brain measures may be suboptimal phenotypes for brain aging. Follow-ups will examine how these patterns change with age.

Etiology of Adult ADHD

H. Larsson[1], J.-O. Larsson[2], P. Lichtenstein[1]

[1]Department of Medical Epidemiology and Biostatistics, Karolinska Institute, Sweden; [2]Department of Woman and Child Health, Child and Adolescent Psychiatric Unit, Karolinska Institute, Sweden

Until recently, there was a general view that Attention-Deficit/Hyperactivity Disorder (ADHD) was limited to childhood, but longitudinal studies have shown a degree of stability across time. Twin studies of ADHD in childhood samples have shown that the heritability is around 55–90%. However, the etiology of adult ADHD is not well understood. We used the population based Swedish Twin study of Adults: Genes and Environments, including data from more than 20,000 20–45-year-old Swedish male and female twins. Inattentive and hyperactive-impulsive symptoms of ADHD were assessed via a DSM-IV based self-report instrument. The aim was to conduct a bivariate genetic analysis to estimate the genetic and environmental contribution to the inattentive and hyperactive-impulsive dimension and to explore the extent to which these two dimensions share the same genetic basis. Our results showed that genetic effects contributed moderately to the inattentive (35%) and hyperactive-impulsive (37%) dimensions. Non-shared environmental effects explained all of the remaining variance. The genetic correlation between the inattentive and hyperactive-impulsive dimensions was estimated at .63. Age stratified analyses showed that the magnitude of the genetic and non-shared environmental effects were similar across different age groups. In contrast to the substantial genetic component found in studies of childhood ADHD, the genetic effects influencing adult ADHD seem to be lower. There are several potential reasons for this. For example, our study used a self report instrument to assess adult ADHD symptoms, whereas most childhood studies rely on parent or teacher reports. However, the high genetic correlation between the inattentive and hyperactive-impulsive dimensions corresponds well with studies of childhood ADHD. This result predicts that more than half of the genes found to be associated with inattention will also be associated with hyperactivity-impulsivity.

Genotypic associations with externalizing trajectories: Examining moderation by adverse socialization environments

S. J. Latendresse[1], John Budde[2], Alison Goate[2], J. E. Lansford[3], K. A. Dodge[3], G. S. Pettit[4], J. E. Bates[5], D. M. Dick[1]

[1]Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University; [2]Washington University, St. Louis; [3]Center for Child and Family Policy, Duke University; [4]Department of Human Development and Family Studies, Auburn University; [5]Department of Psychological and Brain Sciences, Indiana University, Bloomington

Many studies have prospectively linked childhood conduct disorder with later alcohol dependence (e.g., Kuperman et al. Am J Psychiat 158:2022–2026, 2001). Evidence from twin studies suggests that this association is largely due to shared genetic liability (e.g., Kendler et al. Arch Gen Psychiatry 60:929–937, 2003). Moreover, family-based studies have implicated specific genes, including GABRA2 (Edenberg et al. Am J Hum Genet 74:705–714, 2004) and CHRM2 (Wang et al. Hum Mol Genet 13:1903–1911, 2004), in adult alcohol dependence. There is less evidence for the heritability of symptoms of dependence at earlier stages of development; however, genetic influences on conduct disorder are robust, including identified links with both GABRA2 (Dick et al. Behav Genet 36:577–590, 2006) and CHRM2 (Dick et al. Arch Gen Psychiatry 65:310–318, 2008), suggesting that these genes may contribute to alcohol dependence via predisposition to a broad range of externalizing psychopathology. In addition, adverse socialization environments, such as harsh parental discipline (Bender et al. Dev Psychopathol 19:227–242, 2007), low parental monitoring (Pettit et al. Child Dev 72:583–598, 2001), and affiliation with deviant peers (Barnow et al. Aggressive Behav 31:

24–39, 2005), have been associated with increased risk for adolescent externalizing problems. In the present study, we use data from a subset of 452 individuals (i.e., those from whom genetic data were collected) from the Child Development Project, an on-going epidemiological study of individuals followed annually from kindergarten through age 25, to test whether precarious socialization environments moderate the influence of GABRA2 and CHRM2 on trajectories of self- and maternal-reported externalizing behavior across childhood and adolescence. Findings yield potential targets for prevention programming among individuals with a genetic propensity for subsequent alcohol dependence.

Impact of Age at First Drink and Stressful Life Events on Alcohol Use: A Replication Using a Population-Based Sample of Female Twins

Lewina O. Lee, Kenneth S. Kendler, Carol A. Prescott

Past studies of stress and drinking have found inconsistent results and suggest that the association is modified by factors such as stressor types, coping styles, expectancies, gender and age. While early age at first drink is linked to higher incidence of alcohol use disorders, only one study to our knowledge has investigated the impact of drinking onset on stress-reactive alcohol use in a population-based sample (Dawson et al. ACER 31:69–77, 2007). Dawson et al. reported that early onset of alcohol use increased the association between number of past-year stressful life events (SLE) and ethanol consumption in adults. We attempted to replicate this finding using data from 1,382 female twins in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders. We extended the analyses in 3 ways: by looking separately at SLE that were unlikely to be an outcome of heavy drinking, by including SLE severity, and by examining the genetic and environmental basis underlying these associations. We found a strong main effect for onset age: early ($\leq$age 14) and middle (ages 15–17) drinking onset were associated with higher past-year alcohol consumption (a weighted quantity x frequency measure). The finding by Dawson et al. that early drinking onset moderates the association between number of SLE and past-year drinking density was only replicated in dependent SLE of high or severe threat. Results from analyses of the twin-pair data show that genetic influences on alcohol consumption are mediated by age at first drink, SLE and demographic covariates. Our findings confirm that early initiators are more likely to respond to life stressors by drinking than individuals with later drinking onset and some of the genetic influence on adult drinking is mediated by stress-reactive drinking. These findings suggest that it may be useful to direct prevention efforts toward providing adolescents with alternative means of coping with stressors.

Teen Pregnancy and Early Motherhood: Predictors and Outcomes

Lisa N. Legrand, Margaret Keyes, Matt McGue, Stephen M. Malone, William G. Iacono

Department of Psychology, University of Minnesota

Early motherhood has consistently been associated with lower educational achievement, reduced occupational attainment, and higher rates of poverty (Boden et al. J Child Psychol Psychiatry 49:727–742, 2008). The mechanisms that underlie these associations, however, remain unclear. They could owe to the limits early motherhood places on opportunities for educational and economic advancement; alternatively, early motherhood may be a marker for a group of young women at elevated risk for non-optimal outcomes. We tested these competing hypotheses using a longitudinal sample of 1434 female twins (including 455 monozygotic pairs) from two cohorts of the Minnesota Twin Family Study. Ours is one of only a few prospective studies on this topic, is the only prospective U.S. study, and the only study involving twins. Our findings replicated and extended previous research by showing, in our younger cohort, that teen pregnancy is prospectively predicted by lower parental SES, lower IQ, more externalizing behavior, and higher levels of negative affect. Early motherhood in our older-cohort sample was associated with a greater likelihood of being on welfare, self-reported financial problems, lower educational and occupational attainment, lower income, as well as a smaller social-support network. After covarying the predictors of teen pregnancy, all outcomes but income remained significantly associated with early motherhood. This suggests these outcomes are causally affected by early motherhood and not simply a byproduct of young mothers being an at-risk group. Moreover, while these results did not reach conventional standards of significance within the smaller subsample of discordant, older-cohort, monozygotic pairs ($N = 19$ pairs), effect size differences between the sisters experiencing and not experiencing early motherhood were in the expected direction and small to moderate in size. Supported by NIH grants AA09367, and DA05147.

School Readiness, ADHD, and Academic Achievement: Common and Specific Genetic and Environmental Etiology

Jean-Pascal Lemelin[1], Nadine Forget-Dubois[2], Ginette Dionne[2], Amélie Petitclerc[2], Frank Vitaro[3], Mara Brendgen[4], Richard E. Tremblay[3], Daniel Pérusse[3], Michel Boivin[2]

[1]Université de Sherbrooke, Sherbrooke, Québec, Canada; [2]Université Laval, Québec, Québec, Canada; [3]Université de Montréal, Montréal, Québec, Canada ; [4]Université du Québec à Montréal, Montréal, Québec, Canada

The negative association between attention-deficit/hyperactivity disorder (ADHD) and academic achievement is well documented (Frazier et al. J Learn Disabil 40:49–65, 2007). In a recent study, Saudino and Plomin (Child Dev 78:972–986, 2007) investigated the genetic and environmental mechanisms responsible for this association. They showed that genetic and nonshared environmental factors significantly mediated the covariance between the two phenotypes in 7 year-old twins. However, little is still known about the mechanisms responsible for the covariance between school readiness, ADHD, and academic achievement across time. The current study aims to examine the genetic and environmental factors responsible for the covariance between school readiness, ADHD, and academic achievement in a longitudinal design, using a sample of 910 twins. At 60 months, twins' school readiness was assessed with the Lollipop test (Chew and Morris, Educ Psychol Meas 44:987–991, 1984). Twins' ADHD symptoms were assessed by their teachers at 84 months using the Social Behavior Questionnaire (Tremblay et al. J Abnorm Child Psychol 19:285–300, 1991). At 108 months, twins' achievement in reading, writing and math was rated by different teachers. School readiness and ADHD both contributed to the prediction of school achievement. Results indicated significant genetic influence on school readiness (38% of the variance), ADHD (66%), and academic achievement (64%). Shared environment represented 36% of the variance in school readiness but was nonsignificant for the other two phenotypes. Strong genetic correlations between the three phenotypes (from .50 to .63 in absolute values) as well as a significant nonshared environment correlation between school readiness and

achievement (re = 0.37) were observed. These results suggest that the relation between school readiness, ADHD, and academic achievement is largely mediated by genetic factors.

Marijuana Use and BMI in the Add Health Sample

Jeffrey M. Lessem[1], Brett C. Haberstick[1], John K. Hewitt[1], Christian J. Hopfer[2], Matthew B. McQueen[1]

[1]University of Colorado; [2]University of Colorado Health Science Center

The relationship between marijuana use and BMI is examined in The National Longitudinal Study of Adolescent Health. At Wave 1 (mean age 16.2, $N = 18496$) and Wave 2 (mean age of 16.7, $N = 13641$) neither use of marijuana, nor the frequency of marijuana use is related to BMI or obesity (BMI of 30 or higher). At Wave 3 (mean age 22.5, $N = 13554$) both the use of marijuana and the frequency of marijuana use are related to BMI and obesity. At Wave 3 marijuana users are on average 0.90 BMI units below non-marijuana users ($t = -8.27$, $P < 0.0001$), while controlling for sex, age, and nicotine use. Each use of marijuana in the 30 days prior to testing predicts a 0.03 unit decrease in BMI ($t = -5.19$, $P < 0.0001$), controlling for the same variables. Use of marijuana is inversely associated with obesity (odds ratio of 0.711, 95% CI 0.643–0.787). BMI increases between Wave 2 and Wave 3, but each additional use of marijuana in the 30 day period prior to Wave 3 over the 30 day period prior to Wave 2 predicts a 0.02 decrease in the rise of BMI compared to the mean rise from Wave 2 to Wave 3.

Genetic and Environmental Influences on Nicotine Pharmacokinetic and Pharmacodynamic Phenotypes

Christina N. Lessov-Schlaggar[1], Gary E. Swan[2], Andrew W. Bergen[2], Yungang He[2], Kirk Wilhelmsen[3], Rachel F. Tyndale[4], Neal L. Benowitz[5]

[1]Department of Psychiatry, Washington University, School of Medicine, St. Louis, MO; [2]Center for Health Sciences, SRI International, Menlo Park, CA; [3]Department of Genetics, University of North Carolina, Chapel Hill, NC; [4]Department of Pharmacology, University of Toronto, Canada; [5]Division of Clinical Pharmacology, Departments of Medicine and Biopharmaceutical Sciences, University of California San Francisco, San Francisco, CA

Nicotine metabolism (pharmacokinetics; PK) and subsequent impact on the cardiovascular system (pharmacodynamics; PD) may play a role in processes leading to addiction. This talk summarizes results of biometric analyses of PK phenotypes including: hepatic nicotine and cotinine clearance, renal clearance of nicotine, the ratio of trans-3′hydroxycotinine/cotinine (3HC/COT) measured in plasma and urine, and the PD phenotype of heart rate response to infused nicotine. One hundred thirty nine twin pairs (110 MZ and 29 DZ) underwent a 30-min infusion of stable isotope-labeled nicotine and its major metabolite, cotinine, followed by an 8-h in-hospital stay. Blood and urine samples were taken at regular intervals for analysis of nicotine, cotinine, and metabolites. Heart rate was measured pre-infusion and at regular intervals for 60 min thereafter. DNA was genotyped to confirm zygosity and for variation in the gene for the primary nicotine metabolic enzyme, CYP2A6 (variants genotyped: *1 (A and B), *1 × 2, *2, *4, *9, *12). Univariate analyses quantified genetic and environmental influences on each measure in the presence and absence of covariates, including measured CYP2A6 genotype. There was a substantial amount of variation in most plasma PK phenotypes attributable to additive genetic influences (~60%) which was reduced somewhat after inclusion of covariates including sex, age, smoking status, and CYP2A6 genotype status (~50%). Variation in the heart rate response to nicotine attributable to additive genetic sources was smaller (~30%), suggesting that PD phenotypes may be more sensitive to environmental sources of variation at the time of measurement. Implications for the nomination of selected PK measures as "candidate endophenotypes" for inclusion in future genomic studies will be discussed.

Overreactive Parenting during Toddlerhood: An Examination of Continuity, Genetic Influences, and Outcomes using an Adoption Design

Leslie D. Leve[1], Jenae M. Neiderhiser[2], Xiaojia Ge[3], Gordon Harold[4], Daniel Shaw[5], David Reiss[6]

[1]Oregon Social Learning Center, Eugene, OR; [2]The Pennsylvania State University, University Park, PA; [3]Institute of Child Development, University of Minnesota, Minneapolis, MN; [4]School of Psychology, Cardiff University, Cardiff, Wales, UK; [5]University of Pittsburgh, Pittsburgh, PA; [6]Yale University, New Haven, CT

This study examined the influences on and outcomes of overreactive parenting during toddlerhood using a prospective adoption design. The sample included 359 sets of adoptive parents, their adopted child, and the child's birth parent(s) assessed during infancy (child age 9 months) and toddlerhood (child age 18 months). Overreactive parenting was the focus of the study due to the potential for informing the development of genetically-informed psychosocial interventions targeting parenting. Hypothesized predictors of overreactive parenting at child age 18 months included overreactive parenting at 9 months (continuity in parenting), marital conflict at 9 months (spillover effects from the marital relationship to the parenting relationship), and birth mother externalizing behavior (to examine genetic effects). Overreactive parenting was measured using a composite of adoptive mother- and father-reported overreactive parenting; marital conflict was measured using a composite of adoptive mother- and father-reported marital hostility and low marital warmth; birth mother externalizing behavior was measured using a composite of birth mother delinquency, drug use, and novelty seeking. Openness in the adoption relationship (measured using a composite of birth parent and adoptive parent-report) was included as a covariate. Preliminary results from a stepwise regression analysis indicated that the final model (Step 3) was significant, $F(4, 303) = 48.92$, $P < 0.001$, $R^2 = 0.39$, with the following significant predictors: overreactive parenting at 9 months ($\beta = 0.56$, $P < 0.0005$) and marital conflict at 9 months ($\beta = 0.15$, $P < 0.001$). The significant, inverse effect of birth mother externalizing behavior in Step 2 ($\beta = -0.13$, $P < 0.05$) was reduced to a trend in the final model. Overreactive parenting was concurrently related to parenting efficacy ($r = -0.23$), marital hostility ($r = 0.24$), and parent-rated child externalizing behavior ($r = 0.17$).

Detection of Gene–Gene Interaction Among CHRNA4, CHRNB2, BDNF and NTRK2 in Nicotine Dependence

Ming D. Li

Department of Psychiatry and Neurobehavioral Sciences, University of Virginia, Charlottesville, VA, USA

Extensive epidemiological data indicate that vulnerabilities to nicotine dependence (ND) are complex traits influenced by genes,

environments, and their interaction. Recent evidence supports a genetic association of the nicotinic receptor alpha 4 subunit (CHRNA4), brain-derived neurotrophic factor (BDNF), and neurotrophic tyrosine kinase receptor 2 (NTRK2) with ND. Although the interacting effects of BDNF with NTRK2 and CHRNA4 with CHRNB2 have been established experimentally using in vitro and animal models, no human genetic study is reported demonstrating that BDNF interacts with NTRK2 or CHRNA4 with CHRNB2 in affecting smoking behavior. To determine if the four genes are affecting ND, we genotyped 6 SNPs for CHRNA4 and BDNF, 9 SNPs for NTRK2, and 4 SNPs for CHRNB2 in a case-control sample containing 275 unrelated smokers with a FTND score of 4.0 or more and 348 unrelated nonsmokers. By using a newly developed algorithm by this group, called generalized multifactor dimensionality reduction (GMDR) method, we found highly significant gene interaction effects on ND for the gene pairs of CHRNA4 and CHRNB2, CHRNA4 and NTRK2, CHRNB2 and NTRK2, and BDNF and NTRK2. Furthermore, we found a significant gene interaction of CHRNA4 and BDNF on ND. No significant interaction was detected for the gene pair CHRNB2 and BDNF. Together, this study provides evidence on the presence of gene-gene interaction among the four genes in affecting ND. Although CHRNB2 alone was not associated with ND in several previously reported association studies on ND, we found it affects ND through interaction with CHRNA4 and NTRK2.

An Analytical Strategy for Modeling Count (Poisson) Distributed Phenotypes with Genetically Informative Twin and Family Data

Levente Littvay

University of Nebraska-Lincoln and Central European University

The available strategies for modeling heritability with continuous, dichotomous, and ordered categorical phenotypes with genetically informative family data are widely used and assessed in the literature. Less attention is attributed to multinomial and count (Poisson) distributed phenotypes. This study strives to fill this hole in part by presenting one easily accessible approach to modeling the heritability of count distributed phenotypes made available with Mplus version 5.1. The study utilizes a Monte Carlo simulation to explore the biases that stem from treating count distributed data as continuous or ordered categorical (with transformations) and not modeling them as Poisson. Beyond the simulations, the analytical strategy is demonstrated using the twin data of the freely available Midlife Development in the United States dataset. The extension of the demonstrated twin model to extended family data is strait forward. Simulations show that the selection of distributional assumptions drastically influences the results.

Some Effects of Assumptions in a GxE Modeling of Cognitive Skill

John C. Loehlin

University of Texas

Two particular assumptions, random parental mating and gene-income independence, were investigated in a slightly simplified version of a model that had been employed to assess GxE interaction in twin data (Harden et al. Behav Genet 37:373–383, 2007). Neither assumption proved to have a major impact on the GxE estimate, although they affected other aspects of the results—this was especially true for the assumption of gene-income independence.

The Impact of Birth Complications on Aggression in Twins at age 5

Sarah A. Long, Lisabeth F. DiLalla

Southern Illinois University

The purpose of this study was to examine the impact of birth complications on behavioral outcomes in preschoolers. Previous research on singleton children has shown that birth complications are related to impulsivity and aggressive behavior at school age and into adolescence. This study expanded on this by examining preschool twins. Twins were drawn from the Southern Illinois Twins and Siblings Study (SITSS; DiLalla, Twin Res 5:468–471, 2002). Birth complication data were collected on 103 twin pairs (65 MZ children; 102 same-sex DZ children; 39 opposite-sex DZ children) who participated at age 5 in a peer play study, and parent-rated behavior measures were collected at that time. Results showed that severe birth complications were significantly related to parent-rated child aggression. In addition, significant heritability was found for parent-rated aggression but not for observer-rated measures of aggression. DeFries-Fulker regression analyses (DeFries and Fulker, Acta Geneticae Medicae et Gemellologiae 37:205–216, 1988) were used to determine if birth complications predicted aggression above and beyond genetic effects. Results showed that after accounting for genetic effects, birth complications did not account for significant variance. These findings demonstrate that although birth complications appear to be an important predictor of aggression, genetic effects actually appear to be more important in the development of aggressive behavior.

Using Observed Genetic Variables to Predict Latent Class Membership: A Comparison of Two Methods

Gitta Lubke[1], Gregory Carey[2], Jeff Lessem[2], John Hewitt[2]

[1]University of Notre Dame; [2]University of Colorado at Boulder

Factor mixture modeling or classic latent class modeling can be used to investigate qualitatively or quantitatively distinct subtypes of psychiatric disorders. A series of alternative models is fitted to questionnaire data or symptom endorsements, and a best fitting model is selected using measures of goodness of fit. In addition to modeling the differences in means and/or covariance structures between latent classes, these methods permit the assignment of individuals to their most likely latent class (e.g., affected vs. unaffected). In case genetic data such as gene candidates are available, two different approaches may be used to relate the genetic information to latent class membership. In a two-step approach, individuals are first assigned to a class, and class membership is subsequently regressed on the observed genetic variables. Alternatively, genetic variables can be integrated as covariates when fitting a latent class model. The two approaches are applied to data from Add Health concerning antisocial behavior, and compared in a simulation study. Our empirical analysis is an attempt to replicate previous results where DRD2 and DRD4 have been reported to distinguish between violent and nonviolent antisocial behaviors. The simulation study quantifies the effect of different factors that may influence the performance of the two approaches to relate gene candidates to latent class membership. The two-step approach is based on modal class assignment using the highest posterior probability of belonging to a class, and does not take into account the size of the posterior class probabilities (e.g., in a 2-class model, probabilities of .51 and .99 lead to the same class assignment), or the uncertainty of these probabilities (e.g., how precise is the value .51). It is investigated whether neglecting these aspects actually results in substantial under-performance when compared to a single step approach.

Increasing Heritability on Measures of Family Environment During Adolescence: A Twin Study

Steven Ludeke, Matt McGue, Bill Iacono

Department of Psychology, University of Minnesota

The Minnesota Twin Family Study (MTFS) collected data on a self-report measure of the parent-child relationship from a cohort of twins at ages eleven, fourteen and seventeen. 1,021 participants provided information at all three assessment points, and an additional 357 provided information at two of the three assessments. We will summarize findings from MTFS that suggest that: (1) The marked deterioration in parent-offspring relations between ages 11 and 14 observed by McGue et al. (2005) does not continue between 14 and 17; (2) The heritability of parent-offspring relations increases steadily across the three time points measured; (3) This increase in heritability appears to largely result from amplification processes, as suggested by growth curve modeling, which showed minimal genetic contributions to slope but substantial positive correlation between the genetic factors contributing to the intercept and slope. Supported by U. S. Public Health Service Grants AA09367 and DA05147.

Behavior Genetic Contributions to the Genetic Basis of Smoking Behavior

H. H. Maes[1,2], M. C. Neale[1]

[1]Virginia Institute of Psychiatric and Behavioral Genetics, Virginia Commonwealth University; [2]Massey Cancer Center, Virginia Commonwealth University

Genetic studies to date have provided strong evidence that heritable factors generate individual differences smoking behavior. Shared environmental factors appear to play a larger role in tobacco use at earlier ages. We review genetic studies of twins and families which demonstrate the cumulative evidence both from data on adolescents and adults on the role of genetic and environmental factors in the liability to smoking initiation and progression to nicotine dependence. We also review the major regions on the chromosomes and candidate genes that have been implicated thus far with respect to smoking behavior. We illustrate how results from twin analyses can be used to optimize the phenotype which can be used in molecular genetic studies to identify susceptibility genes for smoking behavior.

Flexible Mx Specification of Various Extended Twin (ET) Kinship Designs

H. H. Maes[1,4], M. C. Neale[1], S. E. Medland[1,2], M. C. Keller[3], L. J. Eaves[1]

[1]Virginia Institute of Psychiatric and Behavioral Genetics, Virginia Commonwealth University; [2]Genetic Epidemiology, Queensland Institute of Medical Research; [3]Department of Psychology, University of Colorado Boulder; [4]Massey Cancer Center, Virginia Commonwealth University

The extended twin kinship design allows the simultaneous testing of additive and non-additive genetic, shared and individual-specific environmental factors, as well as sex differences in the expression of genes and environment in the presence of assortative mating and combined genetic and cultural transmission. It also handles their contribution to the (co)variation of multiple phenotypes. As such, it provides a general framework which can easily be reduced to fit subsets of data such as twin-parent data, children-of-twins data, etc. A flexible Mx specification of this model that allows handling of these various designs is presented and applied to data from the Virginia 30,000. Future extensions of the model are discussed.

Familial Transmission of Borderline Personality Disorder Features

Marijn A. Distel[1], Irene Rebollo-Mesa[1], Gonneke Willemsen[1], Catherine A. Derom[2], Timothy J. Trull[3], Dorret I. Boomsma[1]

[1]Department of Biological Psychology, VU University, Amsterdam, The Netherlands; [2]Department of Human Genetics, Katholieke Universiteit Leuven, Leuven, Belgium; [3]Department of Psychological Sciences, University of Missouri-Columbia, Columbia, MO, USA

Borderline personality disorder (BPD) is a severe personality disorder characterized by disturbances in emotional regulation, impulse control, interpersonal relationships, and identity. Several family studies report increased rates of BPD in relatives of individuals with BPD, but genetic studies of BPD remain relatively scarce, when compared to the number of studies of other disorders in psychiatric genetics. Recently, a large scale multinational twin study suggested a heritability for BPD features of 42% (Distel et al. Psychol Med, in press). No non-additive genetic effects were detected, although the twin correlations suggested a contribution of non-additive genetic influence. In the present study we examined the role of genetic and environmental effects on differences in borderline personality disorder features using an extended families design. Adding parents and siblings to the classic twin design allows to distinguish reliably between additive and non-additive (dominant) genetic effects. When analyzing the data, we took into account possible effects of assortative mating, cultural transmission and genotype-environment covariance. The Personality Assessment Inventory – Borderline scale (PAI-BOR) (Morey 1991, The Personality Assessment Inventory: Professional manual, Odessa, FL, Psychological Assessment Resources) was mailed to twins and their family members registered with the Netherlands Twin Register and the East Flanders Prospective Twin Survey. In total, data were available for 2,669 monozygotic twins (969 complete pairs), 2,371 dizygotic twins (668 complete pairs), 1,291 siblings, 3,121 parents and 948 spouses of twins from 4,029 families. The data for (co) twin and spouses indicated phenotypic assortment. Analysis in MX suggest that major influences on borderline personality disorder consist of additive (22%) and dominant (24%) effects and unique environmental experiences (54%). Assortment explains 1% of the variance in BPD features.

A Twin Study of Economic Risk Taking

Nick Martin[1], Wendy Slutske[2], Madeline Meier[2], Paul Miller[3], Daniele Terlizzese[4], Megan Ferguson[1], Gu Zhu[1]

[1]Queensland Institute of Medical Research, Brisbane, Australia; [2]Dept of Psychology, University of Missouri, Columbia MO, USA; [3]Dept of Economics, University of Western Australia, Perth, Australia; [4]Banca d'Italia, Rome, Italy

Entrepreneurship and economic risk taking are essential to economic growth and the advance of civilization. Clark has proposed that for at least 30 generations leading up to the industrial revolution, such traits were under intense directional selection. With relaxed selection following improved health, widespread limitation of fertility and the introduction of socialism it is likely that there is considerable residual

genetic variation for these traits although the direction of selection in contemporary society remains unclear. In the context of a study of addictive gambling, we have attempted to measure aspects of economic risk taking in a large sample ($N = 4700$) of Australian twins born 1964–1972 and approaching the height of their economic powers. Behaviors probed include investment in shares, real estate, futures and options, the extent of risk tolerated in these investments, and the perception of excessive risk and over-exposure. There was widespread variation in these behaviors and genetic and environmental analyses of the data will be presented.

Models for Extended Pedigrees: Model Specification and the Power Associated with Different Family Structures

S. E. Medland[1,2], M. C. Keller[3], M. C. Neale[1], L. J. Eaves[1], H. H. Maes[1]

[1]Virginia Institute of Psychiatric and Behavioral Genetics, Virginia Commonwealth University; [2]Genetic Epidemiology, Queensland Institute of Medical Research; [3]Department of Psychology, University of Colorado Boulder

Modelling the data from extended twin pedigrees allows the estimation of increasing complex covariance relationships in which the effects of cultural transmission, non-random mating and genotype x environment covariation can be incorporated. This presentation will outline the major models used with extended twin pedigree data and their inherent assumptions. Using the Cascade model as an example the effect of family structure, (ie the ratio of MZ to DZ families and the importance of cousins vs. avuncular relatives) on power will also be examined.

Mutual Mate Choice Can Drive the Evolution of Costly Fitness Indicators in Both Sexes, Even Under Perfect Monogamy, As Long As Mutations Keep Arising

Geoffrey Miller, Paul Hooper

University of New Mexico

Sexual selection models have emphasized the evolution of fitness indicators (e.g. peacock tails) in males through female mate choice. Yet such models can't explain human mental traits such as intelligence, language, and kindness, which are sexually attractive to both sexes, and which show low sex differences. We thought that mutual mate choice by both sexes could favor the evolution of such fitness indicators equally in both sexes, under certain conditions. So, we simulated the evolution of condition-dependent investment in a fitness indicator, under conditions of perfect monogamy, equal initial offspring number, and differential offspring mortality based on inherited genetic quality minus indicator cost (Hooper and Miller, Adapt Behav 16:53–70, 2008). The model shows that fitness indicators can evolve quickly through mutual mate choice, even when they reduce survival prospects, whenever two conditions are met. First, mutation-selection balance must maintain a positive equilibrium mutation load, e.g. by new mutations arising as old mutations are purged by purifying selection. Second, the fitness indicator must reflect an individual's genetic quality clearly enough to allow some positive assortative mating for 'good genes'. Fitness indicators such as intelligence or kindness can evolve equally in both sexes, purely through sexual attractiveness, even despite moderate survival costs, under evolutionarily plausible conditions. If fitness indicators reveal general genetic quality, they will show high heritabilities and genetic correlations, as found for human mental traits in twins studies. Yet it will be very hard for molecular genetic studies to find replicable QTLs that account for genetic variance in these fitness indicators, since general genetic quality is undermined by different evolutionarily transient mutations at different loci in different populations.

A Behavioral Genetic Analysis of Child Attention Span/Persistence and Maternal Negativity

Paula Y. Mullineaux[1], Kirby Deater-Deckard[1], Stephen A. Petrill[2], Lee A. Thompson[3]

[1]Virginia Polytechnic Institute and State University; [2]Ohio State University; [3]Case Western Reserve University

Tention regulation is an important component of children's development. Children who are less able to regulate their attention may experience more difficulties than those children who are better able to regulate their attention. For example, lower attention regulation is linked to higher rates of externalizing behavioral problems in childhood (Caspi et al. Child Dev 66:55–68, 1995). Attention regulation in childhood also has been associated with parent negativity (Deater-Deckard et al. Dev Sci 4:F1–F6, 2001). To better understand the link between attention regulation and parenting, we examined the relation between maternal negative feelings and child attention span/persistence. Mothers' self-reported ratings of negative feelings from the Parents' Feelings Questionnaire (Deater-Deckard et al. 1997, Parent-child interaction coding system. London, UK: Institute of Psychiatry) and a multi-informant attention span/persistence composite based on teacher, parent, and observer ratings were available for 252 twin families (101 MZ; 151 same-sex DZ; 58% female; Mean age = 6.08 at the first assessment). The children were assessed across several occasions. Modeling results indicated evidence of both common and independent effects on the relation between maternal negativity and child attention span/persistence. A genetic correlation of 0.39 was found indicating modest genetic overlap across maternal negative feelings and the attention span/persistence composite. There was no evidence of overlapping shared environment or nonshared environment effects. Additionally, there was evidence of independent genetic and nonshared effects for both maternal negativity and attention span/persistence. Thus, the link between maternal negative feelings and child attention span/persistence are being influenced by both shared and independent genetic effects.

Genetic and Environmental Influences on the Structure of Temperament in Childhood

Paula Y. Mullineaux[1], Kirby Deater-Deckard[1], Stephen A. Petrill[2], Lee A. Thompson[3]

[1]Virginia Polytechnic Institute and State University; [2]Ohio State University; [3]Case Western Reserve University

A number of studies examining temperament in infancy and early childhood have consistently indicated genetic and environmental influences on a wide array of temperamental attributes. However, little is known about the genetic and environmental relations among the facets of childhood temperament. Thus, in the current study we examined the measurement model and the underlying sources of genetic and environmental covariation among the facets of Rothbert's posited higher-order factors of childhood temperament: Negative Affectivity, Surgency/Extraversion, and Effortful Control (Rothbart et al. Child Dev 72:1394–1408, 2001). Mothers and fathers completed the Child Behavior Checklist-Short Form (Putnam and Rothbart, J Pers Assess 87:102–112, 2006) in 194 families with same-sex

school-age twins (45% MZ twins; 53% female; Mean age = 7.96). These ratings were averaged across mothers and fathers for each item. The data conformed to the published measurement model. Modest to substantial genetic effects and modest to moderate nonshared effects were indicated in the best fitting models for Negative Affectivity, Surgency/Extraversion, and Effortful Control. Shared environmental effects, which ranged from moderate to substantial, were also implicated for Effortful Control. Among the facets for each of the higher-order factors, modest to substantial genetic correlations and modest to moderate nonshared environment correlations were indicated. Additionally, moderate to substantial shared environment correlations were indicated for the facets of the Effortful Control factor. Therefore, genetic and environmental influences both were crucial to the measurement model, but the specific pattern of effects was distinct for each of the three factors, suggesting that there is not a monolithic underlying genetic and environmental etiology of individual differences in temperament in childhood.

Genetic and Environmental Relationships among Disordered Eating and Internalizing Symptoms in Female Twins

Melissa A. Munn, Soo H. Rhee, Robin P. Corley, Michael C. Stallings

Institute for Behavioral Genetics, University of Colorado, Boulder, CO 80309

The comorbidity between eating pathology and internalizing symptoms, such as major depressive (MD) and generalized anxiety disorder (GAD) symptoms, is well documented in both clinical and community samples. However, research on the extent to which these relationships are due to common genetic and environmental influences is limited. We examined the extent to which etiological factors of internalizing symptoms could explain genetic and environmental influences between two disordered eating characteristics: anorexia-like tendencies (ANT) and bulimia-like tendencies (BNT). Participants included 914 female adolescent and young adult twins (579 monozygotic, 335 dizygotic) between the ages of 16 and 27 years. Seven "yes/no" items assessed ANT and BNT, while internalizing symptoms were assessed using lifetime MD and GAD modules of a diagnostic interview. A Cholesky trivariate decomposition threshold model was used to estimate the contribution of genetic and environmental risk to ANT, BNT, and internalizing symptoms. Polychoric correlations suggested that there were no genetic influences on MD symptoms. Although there were additive genetic and shared environmental influences on ANT, BNT, and GAD symptoms, trivariate analyses revealed that variation in GAD symptoms explained little of the covariation between ANT and BNT. While phenotypic correlations exist among eating pathology and internalizing symptoms, other anxiety symptoms may better explain the relationship between ANT and BNT.

Challenges in the Further Development of Gene-Environment Models for Extended Pedigrees

Michael C. Neale

The contributions of, e.g., Rice, Reich and Cloninger, in the late 1970s to modeling of the joint transmission of genetic and environmental variance components were both elegant and generalizable through recurrence relations. However, complexities such as phenotypic homogamy, phenotype to environment transmission, genotype-environment covariance and multivariate analysis make the model difficult to generalize to arbitrary pedigree structures. Some progress has been made in some of these areas, using the 'Stealth' or 'Cascade' models, but much work remains to be done. There is also a need to implement factor structures and to model developmental and cohort effects on an individual basis. Unfortunately, this presentation will not provide a simple general solution to these problems. Some possibilities for ways to proceed will be considered, including applications of the mixed model, marginal likelihood, reducing model complexity, and exploitation of parallel computing facilities. The contributions of the symposium co-presenters will be discussed.

Maximum Likelihood Estimation of Genetic, Environmental and Phenotypic Factor Scores

Michael C. Neale, Paul W. Andrews, Jack M. Hettema

VIPBG Virginia Commonwealth University

Multivariate data collected from genetically informative constellations of relatives, such as MZ and DZ twins, permit the fitting of a wide variety of factor models. Currently popular are (i) the common pathway model, where latent genetic and environmental variance components cause variation in one or more latent phenotypic factors, which in turn influence the observed variables; and (ii) the independent pathway model in which the latent genetic and environmental factors directly influence the observed variables. It is noted that the single factor common pathway model is a submodel of the single factors independent pathway model, which in turn is a submodel of the three-factor common pathway model. By considering the distribution of the observed variables conditional on the distribution of the latent factors, it is possible to estimate factor scores for either model using maximum likelihood. Furthermore, the factor score estimation may take account of covariates that generate either differences in factor means and variances, or measurement non-invariance of the factor loadings, item means or item variances. Though not perfect, this approach provides one avenue by which factor scores can be derived for the purposes of selecting subjects for case-control GWAS studies, or for refining the phenotype in those datasets that have already been collected. Its marriage with bootstrap estimation also permits some accounting for the statistical imprecision of model parameters.

Internalizing, Externalizing, and ADHD in Middle Childhood: The Role of Mothering and Fathering in Toddlerhood

T. Caitlin O'Brien[1], Jodi Swanson[1], Kathryn Lemery-Chalfant[1], H. Hill Goldsmith[2]

[1]Arizona State University; [2]University of Wisconsin-Madison

Child psychopathology symptoms stem from genetic (Lemery and Doelger, 2005. In Hankin and Abela, eds., Development of psychopathology: A vulnerability-stress perspective, 161–198) and environmental factors (e.g., parenting practices; Aunola and Nurmi, Child Dev 16:1144–1159, 2005), yet, parenting and child behavior associations over time are unclear. The parenting literature underscores the independent contributions of mothering and fathering to child development (Park and Buriel, 2006. In Eisenberg, ed., Handbook of child psychology: Vol. 3. Social, emotional, and personality development, pp. 429–504). Thus, we longitudinally examined

parental warmth and control and children's psychopathology with data from the Wisconsin Twin Project. Parents were interviewed when twins (328 MZ, 533 DZ twin pairs [50% same-sex]) were age two (Time 1; T1) to assess parental warmth and control (Block Child Rearing Practices Report) and again when twins were age eight (Time 2; T2) to assess symptoms of internalizing, externalizing, and ADHD (MacArthur Health & Behavior Questionnaire). Univariate ACE models indicated genetic and unique (but not shared) environmental influences on all symptoms. Bivariate analyses supported a common set of genetic and nonshared environmental influences on symptoms across time. Multilevel models (controlling for zygosity, child sex) showed at T1 both parents' control related to externalizing and ADHD and fathers' warmth related to externalizing. Mother's T2 warmth related to all symptoms. Longitudinally, fathers' warmth predicted decreased externalizing and mothers' control marginally predicted increased internalizing and ADHD. In family level analyses, maternal warmth most consistently predicted children's symptoms. Parenting did not relate to MZ differences in symptoms; thus, parenting may not account for nonshared environmental influences. These results illustrate the dynamic relations between parenting and child psychopathology.

Heritability of Breastfeeding Behavior

Juan R. Ordoñana[1], Irene Rebollo-Mesa[2], Lucia Colodro[1], Francisco Perez-Riquelme[1], Juan F. Sanchez-Romera[1], Jose M. Martinez-Selva[1]

[1]Murcia Twin Registry, Area of Psychobiology, University of Murcia (Spain); [2]Department of Biological Psychology, VU University, Amsterdam (The Netherlands)

Introduction Natural breastfeeding has been related to general infant health, disease protection and child cognitive development. It is also an important element in mother-child relationship. Willingness to breastfeed is affected by several physiological, psychological and environmental factors. However, there is no information about the relative impact of genetic and environmental factors on natural breastfeeding. Our objective was to analyze the heritability of breastfeeding behavior. *Methods* The data comprised adult female twins from the Murcia Twin Register (Spain), when both twins had been mothers (302 MZ and 330 DZ). Mean age = 51.6 (Range = 41–67). Information was based on self-report. Breastfeeding length was recorded in months. Zygosity was ascertained by questionnaire. Quantitative genetic analyses were conducted to quantify genetic and environmental influences on variation in length of breastfeeding behavior. *Results* The mean number of children was 2.56 (SD: 1.14). Seventeen per cent of the mothers had not breastfed any of their children. The mean length of breastfeeding for the first child was 4.33 months (SD = 5.32), with a range of 0–36 months. The average length of breastfeeding across all children was 5.2 months (SD = 4.7). Breastfeeding duration for the first child correlated higher for MZ mothers (0.464) than for DZ mothers (0.212). A smaller difference was found for average breastfeeding length across all children (MZ: 0.474, DZ: 0.329). Concordance rates for breastfeeding more than 6 months to the first child were considerably higher for MZ mothers (Tau-b: .407) than for DZ mothers (Tau-b: .160). *Conclusion* Preliminary results suggest that individual differences in breastfeeding behavior may be moderately influenced by genetic factors, with heritability estimates ranging from 0.5 for first child breastfeeding length and 0.3 for average breastfeeding duration.

Heritability of Adherence to Health Recommendations in Adult Women

Juan R. Ordoñana[1], Irene Rebollo-Mesa[2], Francisco Perez-Riquelme[1], Juan F. Sanchez-Romera[1], Lucia Colodro[1], Jose M. Martinez-Selva[1]

[1]Murcia Twin Registry. Area of Psychobiology. University of Murcia (Spain).; [2]Department of Biological Psychology, VU University, Amsterdam (The Netherlands)

Introduction Adherence to health recommendations is a key issue in health promotion. Compliance with disease detection and prevention programs is influenced by health policies and by social and individual factors. In this context, early breast cancer detection behaviors are relevant actions in terms of public health. There is evidence that individual differences in the liability to these behaviors could be related to genetic factors (Treloar et al. Twin Res 2:33–42, 1999). Our objective was to estimate genetic and environmental effects on individual differences in early breast cancer detection behaviors of adult women. *Methods* The data comprised adult female twins from the Murcia Twin Register (Spain) (213 MZ and 236 DZ pairs). Mean age = 51.4 (Range = 41–67). Mammogram use and breast self-examination was based on self-report. Zygosity was ascertained by questionnaire. Age dependent quantitative genetic analyses were conducted to quantify genetic and environmental influences on variation in these behaviors. *Results* Most of the sample had had a mammogram at least once (73.5%) but only 15.5% checked their own breasts systematically. Tetrachoric correlations for mammogram use were higher for MZ twins [MZ: 0.746 (IC 95%: 0.569, 0.866); DZ: 0.543 (IC 95%: 0.279, 0.742)], and the difference increased when women over 50 were ruled out [MZ: 0.556 (IC 95%: 0.254, 0.774); DZ: 0.268 (IC 95%: −0.115, 0.593)]. *Conclusion* Preliminary results suggest that genetic differences may affect the probability of attendance to mammographic screening, with heritability estimations ranging from 0.41 for the total sample and 0.56 for younger women. This difference reflects the effect of public screening programs for women over 50, which shows up as a shared environmental component. Our results confirm the importance of genetic influences on breast cancer detection behaviors.

Specification of Univariate ACDE Model Using Higher Order Moments

Koken Ozaki[1], Hideki Toyoda[2], Juko Ando[3]

[1]Japan Science and Technology Agency; [2]Waseda University; [3]Keio University

Current behavior genetic models are specified using 1st and 2nd order moments, namely means and covariances. A limitation of ordinal structural equation modeling is that it cannot specify ACDE model in the univariate case, because the model cannot be identified. Therefore in cases where we are to specify an univariate model, ACE, AE, CE, DE, and ADE models are compared from the viewpoint of goodness of fit. However, sometimes, in reality, all of the above models may not be appropriate and ACDE model best fits the data. In this paper this limitation is overcome using 3rd order moments as well as 2nd order moments. Simulation study is conducted to examine estimation accuracy.

Disentangling the Genetic and Environmental Vulnerabilities Underpinning the Risk of Substance Involvement in Young Adulthood: A Classical Twin Design

Rohan H. C. Palmer[1], Robin, P. Corley[1], Christian J. Hopfer[2], Michael C. Stallings[1], Susan E. Young[1], Thomas J. Crowley[2], John K. Hewitt[1]

[1]Institute for Behavioral Genetics, University of Colorado, Boulder, CO, 80309; [2]University of Colorado Health Science Center, Denver, CO 80262

A question of great interest in drug research is the extent to which the underlying risk for substance abuse is generalized or specific across substances, especially given the genetic risk that has been confirmed in adolescent (Hopfer et al. J Am Acad Child Adolesc Psychiatry 42:710–719, 2003; Young et al. Behav Genet 36:603–615, 2006) and adult populations (McGue et al. Am J Med Genet 60:1256–1264, 2000; Goldman and Bergen, Arch Gen Psychiatry 55:964–965, 1998). We report on data collected during the second wave of the Center for Antisocial Drug Dependence at the University of Colorado. Preliminary analyses on data from 1,733 respondents who have completed both waves of assessment revealed a generalized risk for substance problems in young adulthood, such that, over and above the observed specific risk, involvement with any substance in adolescence increased the risk for problems in young adulthood. Similarly, there was an increasing tendency for twins to use and be diagnosed with problems on multiple substances as age increased. Using data collected on 2,020 twins in young adulthood we examined the genetic and environment etiology of alcohol, tobacco, and cannabis lifetime reports of initiation, use, and DSM-IV disorder diagnosis allowing for sex-limitation. Model fitting revealed moderate to strong additive genetic influences on alcohol initiation and use with varied levels in each gender; strong additive genetic influences on all levels of tobacco involvement for each gender; and evidence of both additive genetic and shared environmental influences on each level of cannabis involvement but to different degrees in each gender. We will also report on the extent to which the genetic risk is correlated across substances.

The Aetiology of the Covariance Between Reading Difficulties and DSM-IV ADHD Symptom Dimensions of Inattention and Hyperactivity-Impulsivity

Yannis Paloyelis, Fruhling Rijsdijk, Alexis Wood, Philip Asherson, Jonna Kuntsi

MRC Social Genetic Developmental Psychiatry Centre, Institute of Psychiatry, King's College London

Co-occurrence of specific reading disability and attention deficit hyperactivity disorder (ADHD) is relatively high for both clinical and population samples. In this study we aimed to investigate the aetiological architecture of the co-occurrence between reading difficulties (RD) and the ADHD symptom dimensions of inattention (IN) and hyperactivity-impulsivity (H/I), taking into account general cognitive ability and possible gender differences. We carried out multivariate structural equation modelling on data from a general population twin sample of 672 twin pairs. Phenotypic correlations showed that the association between IN and RD ($r = 0.51$) was more than twice as high as that with H/I ($r = 0.20$). Further, there was no evidence that this association was mediated by IQ. Univariate analyses showed that individual differences in all phenotypes were largely driven by genetic effects (heritabilities estimated as 74% for RD, 55% for IN, 72% for H/I and 61% for IQ), with a significant shared environmental contribution emerging only for IQ. There was no evidence of sex differences. Multivariate analyses revealed that the genetic correlation between IN and RD was high ($r_g = 0.60$), about three times the size of the genetic correlation with H/I ($r_g = 0.24$). For both ADHD symptom dimensions, however, their covariance with RD was largely attributed to shared genetic effects (75% for IN and 92% for HI). Moreover, we found that 42% of the heritability for RD was due to genetic effects not related to the covariation with IN, H/I or IQ, while 45% of the covariance between RD and IN was due to genetic effects not shared with IQ or H/I. These results suggest that the strong phenotypic association between RD and symptoms of inattention is largely genetic. It is driven both by unique genetic effects not shared with IQ or H/I, and, to a smaller extent, by a shared genetic component impacting on all phenotypes.

Comparing Alternative Approaches to Stage Models of Substance Use Phenotypes

Matthew S. Panizzon[1], Michael J. Lyons[2], Michael D. Grant[2], William S. Kremen[1], Ming Tsuang[1]

[1]UC San Diego; [2]Boston University

Stage models have emerged as a means of accurately capturing the covariance between substance initiation and other substance use outcomes. Two approaches have been proposed, the causal-contingent-common pathway (CCC) model (Kendler et al. 1999), and a variation of the bivariate Cholesky model (Heath et al. 2002). In order to examine the compatibility of these approaches we applied both to three common substance use phenotypes (nicotine, alcohol, and cannabis dependence). Participants were 3,356 twin pairs from the Vietnam Era Twin Registry. Mean age was 42 years (range 33–53). Information about lifetime substance use was obtained from an administration of the Diagnostic Interview Schedule (Robins et al. 1987). Substance initiation was defined as having ever used the substance; substance dependence was defined by DSM-III-R criteria. The CCC and Cholesky models were fit to raw data using Mx. Data for subjects who never initiated substance use was missing for dependence. Initiation data for the Cholesky approach were divided into three categories based on age of initiation. Variance components for the initiation stages were roughly equivalent. Under the CCC and Cholesky approaches, nicotine initiation accounted for similar proportions of the phenotypic variance in dependence (.41 and .38 respectively). For alcohol and cannabis dependence the variance accounted for by their initiation stages differed across the two approaches (Alcohol: 35% vs. 12%; Cannabis: 68% vs. 16%). For each of the phenotypes, a combined liability model was suggested by the CCC approach, while the Cholesky approach suggested a single liability for nicotine and cannabis. A comparison of the two approaches to stage modeling demonstrates that they are not directly compatible. The observed inconsistency may in part be do to the poor fit of the three category initiation variables to a bivariate normal distribution. This brings into question the validity of the Cholesky approach.

Is Cortical Volume the Correct Phenotype for Studying the Aging Brain?

Matthew S. Panizzon[1], Christine Fennema-Notestine[1], Lisa T. Eyler[1], Elizabeth Prom-Wormley[2], Michael Neale[2], Kristen Jacobson[3],

Michael J. Lyons[4], Carol E. Franz[1], Bruce Fischl[5], Larry J. Seidman[5], Anders Dale[1], William S. Kremen[1]

[1]UC San Diego; [2]Virginia Commonwealth University; [3]University of Chicago; [4]Boston University; [5]Harvard Medical School

Neuroimaging studies examining the effects of aging on the cerebral cortex have largely been based on measures of volume. While recent studies have examined cortical thickness, the two measures have been used almost interchangeably in the literature, with little discussion as to how they may be distinguished from one another. Given that volume is a product of thickness and surface area, likely representing different cellular processes, it is plausible that measures of volume capture two distinct sets of genetic influences. Therefore, the aim of the present study is to examine the genetic relationships between measures of surface area and thickness. Participants were men in the Vietnam Era Twin Study of Aging (110 MZ pairs, 93 DZ pairs). Mean age was 56 years (range: 51–59). T1-weighted MR images were obtained on 1.5 Tesla scanners. Cortical regions of interest (ROIs) were defined utilizing an automated gyral-based labeling system. Bivariate Cholesky models were fit to raw data in Mx. Analyses included the relationship between: (1) total cortical surface area and average cortical thickness; and (2) surface area and thickness across 20 ROIs. Total cortical surface area and average cortical thickness were both highly heritable (0.89 and 0.81, respectively); however, the phenotypic and genetic correlations between the two measures were very small (rp = 0.02, rg = 0.08). At the ROI level a similar pattern was observed. Despite significant heritability estimates for all ROI-based measures (range: 0.18–0.79), the majority of genetic correlations failed to exceed a magnitude of r = 0.20. These results demonstrate that within a middle-aged cohort the use of cortical volume measures combine at least two distinct sources of genetic influences: those affecting cortical thickness and those affecting cortical surface area. Using volume as the phenotype may obscure the underlying genetic architecture, and may be less likely to elucidate genetic influences on neuroanatomical aging.

Genetic Linkage and Association Findings for DSM-IV Major Depressive Disorder: Is the Metabotropic Glutamate Receptor 7 Gene (*GRM7*) an Important Risk Factor for Depression in Smokers?

Michele L. Pergadia[1], Anne L. Glowinski[1], Arpana Agrawal[1], Grant W. Montgomery[6], Anu Loukola[3,4], Ulla Broms[5,7], Scott F. Saccone[1], Tellervo Korhonen[5], Jen C. Wang[1], Julia D. Grant[1], Christina N. Lessov-Schalggar[1], Alexandre A. Todorov[1], Naomi R. Wray[6], Kauko Heikkilä[5], Dixie J. Statham[6], Anjali Henders[6], Megan Campbell[6], John P. Rice[1,2], Richard D. Todd[1,2], Alison M. Goate[1,2], Leena Peltonen[3], Andrew C. Heath[1,2], Jaakko Kaprio [5,7], Nicholas G. Martin[6], Pamela A. F. Madden[1]

[1]Department of Psychiatry, Washington University School of Medicine, Saint Louis, U.S.A.; [2]Department of Genetics, Washington University School of Medicine, Saint Louis, U.S.A.; [3]Department of Molecular Medicine, National Public Health Institute, Helsinki, Finland; [4]Finnish Genome Center, University of Helsinki, Helsinki, Finland; [5]Department of Public Health, University of Helsinki, Helsinki, Finland; [6]Queensland Institute of Medical Research, Brisbane, Australia; [7]Department of Mental Health and Alcohol Research, National Public Health Institute, Helsinki, Finland

A handful of linkage studies have reported findings on major depression. One of them reported on neuroticism in a nicotine dependent sample (Neale et al. 2005). The first part of this study conducted genetic linkage using a DSM-IV Major Depressive Disorder (MDD) phenotype in two samples that are part of an international consortium. Genome-wide scans (using 381 autosomal microsatellite markers) and telephone diagnostic interviews were conducted on 289 Australian (AUS) and 161 Finnish [FIN, combined $N = 450$ families] families ascertained from twin registries through index-cases with a lifetime history of cigarette smoking. We used an affected sib-pair design, where at least two adult offspring reported a history of DSM-IV MDD per family (212 informative sib-pairs), and tested for linkage analyses using MERLIN. We found one linkage signal with a LOD score greater than 3.0. This peak was located on chromosome 3 in the AUS subsample. The multipoint LOD score of 3.9 at 24.9 cM met genome-wide significance ($P = 0.009$ with 1,000 simulations). The highest single-point in this region was associated with a LOD = 3.4. This marker, D3S1304, lies within an interesting candidate gene: the metabotropic glutamate receptor 7 gene (*GRM7*). The second part of this study explored the association between 175 SNPs localized within *GRM7* and MDD from an independent genome-wide association effort in 471 Australian MZF twin-pairs. Twenty-two (or 13%) of the SNPs tested within *GRM7* were nominally associated with MDD ($P < .05$), and these effects were generally stronger in lifetime smokers [e.g. rs10490861 OR = 6.21 (95%CI: 1.6–24.0)] compared to the entire sample [OR = 1.7 (95%CI: 0.9–3.2)]. Recently published reports suggest that polymorphisms in other glutamate receptor genes (i.e., *GRIK2, GRIA3* and *GRIK4*) are associated with antidepressant treatment response (Laje et al. 2007; Paddock et al. 2007), lending additional credence to our observed association.

A Genome-Wide Association Study for Educational Attainment

D. Posthuma[1], G. Willemsen[2], S. van der Sluis[2], P. Sullivan[3], J. H. Smit[4], T. Zandbelt[4], E. J. C de Geus[2], D. I Boomsma[2], B. W. Penninx[5]

[1]Department Biological Psychology, VU University, Amsterdam, The Netherlands, & Dept of Medical Genomics, VU Medical Centre, Amsterdam, The Netherlands; [2]Department Biological Psychology, VU University, Amsterdam, The Netherlands; [3]Department Genetics, Psychiatry, & Epidemiology, University of North Carolina, Chapel Hill, NC, USA; [4]Department of Psychiatry, EMGO Institute, Institute of Neuroscience, VU Medical Center, Amsterdam, The Netherlands; [5]Department of Psychiatry, EMGO Institute, Institute of Neuroscience, VU Medical Center, Amsterdam, The Netherlands, Dept Psychiatry, University Medical Center Groningen, University of Groningen, Groningen, Netherlands, & Department of Psychiatry, Leiden University Medical Center, Leiden, The Netherlands

Educational attainment is a highly heritable trait and correlates strongly with general intelligence, which in turn is mainly due to shared genes (Wainwright et al. Twin Res Hum Genet 8(6):602–608, 2005). The first whole genome linkage study for educational attainment suggested several genomic areas that are also of importance for general intelligence (Wainwright et al. Behav Genet 35(2):133–145, 2005). To identify the genetic variants that may explain the heritability of educational attainment, a genome-wide association analysis (GWA) for educational attainment measured on a 5 point-scale was carried out. Subjects were recruited through the Netherlands Twin register (NTR; Boomsma et al. Twin Res Hum Genet 9(6):849–857, 2006) and the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al. Int J Meth Psychiatry Res, in press). Genotyping was funded through the GAIN initiative (US Foundation for the National Institutes of Health Genetic Association Information Network) and was carried out at Perlegen Sciences. After quality control,

75% of SNPs were retained for analyses (427,037 autosomal SNPs). Phenotype and genotype data were available for 3,317 unrelated subjects. Linear regression analyses with covariates sex and age were carried out in Plink (Purcell et al. Am J Hum Genet 81(3):559–575, 2007). We initially selected all SNPs with a *P*-value <0.002 and which were located in a gene. We then compared this list of top-SNPs to results of previous candidate gene studies for related phenotypes, including intelligence. We found that some of our top SNPs were located within candidate genomic regions for intelligence. All SNPs with $P < 10–4$ will be explored in independent replication samples.

Genetic Overlap of Attention Deficit Hyperactivity and Oppositional Defiant Symptoms with Tobacco Use and Dependence in Male Twins

Carol A. Prescott[1], Susan E. Luczak[1], E. Lenn Murrelle[2], Kenneth S. Kendler[3]

[1]Department of Psychology, University of Southern California, Los Angeles CA USA; [2]Department of Epidemiology, Virginia Commonwealth University, Richmond, VA, USA; [3]Department of Psychiatry, Virginia Commonwealth University, Richmond, VA, USA

Attention deficit hyperactivity disorder (ADHD) is strongly associated with tobacco use and dependence, but little is known about the genetic basis for this overlap. We obtained reports of lifetime tobacco use and childhood ADHD and oppositional defiant disorder (ODD) symptoms from 1,793 adult male twins participating in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders. Four symptom factors, Inattention, Hyperactivity/Impulsivity, Forgetfulness, and Irritability were identified from the ADHD and ODD symptom items. These had similar genetic contributions (heritability = 0.34–0.38) and moderate genetic overlap, but in survival analyses related differently to tobacco use characteristics. Age at onset of tobacco use was most strongly predicted by Inattention, whereas later milestones and nicotine dependence were most strongly related to Irritability. Results from correlated frailty models of the twin-pair data indicate that up to 15% of variation in tobacco use overlapped with the symptom factors, and about half of this variation was attributable to genetic factors. The findings are consistent with tobacco use as a self-medication for Irritability symptoms, and suggest there may be genetic factors which predispose both to tobacco use and to symptoms of ADHD and ODD.

Diurnal Regulation and Responsivity of Cortisol Levels: Evidence for Different Genetic Factors

Elizabeth Prom-Wormley[1], Carol E. Franz[2], Sally Mendoza[3], Timothy York[1], Lindon Eaves[1], Seth Eisen[4], Kristen Jacobson[5], Seymour Levine[3], Michael J. Lyons[6], William S. Kremen[2]

[1]Virginia Commonwealth University; [2]University of California San Diego; [3]University of California Davis; [4]Washington University; [5]University of Chicago; [6]Boston University

Chronic hypothalamic-pituitary-adrenal (HPA) axis dysregulation as measured by cortisol levels has been associated with medical conditions and behavioral outcomes. However, the genetic and environmental contributions to these associations are unclear. The genetic architecture underlying the variance and covariance of cortisol levels for daily functioning and in the presence of a stressful environment was assessed. Genetic and environmental factors were characterized to account for diurnal regulation and responsivity of cortisol in older adults. A sample of 785 men ages 51–60 from the Vietnam Era Twin Study of Aging was measured for daily concentrations of salivary cortisol. Fifteen samples were systematically collected across 3 non-consecutive days, specifically 2 "typical" days in the home and 1 "test" day in the laboratory. Cortisol was measured at awakening, awake plus 30 minutes, 10 AM, 3 PM, and bedtime on all days. Cortisol concentrations demonstrated expected diurnal patterns, peaking at 30 minutes after awakening then declining to their lowest points at bedtime across all three days. Multivariate analysis of the genetic architecture of daily cortisol levels indicated a typical day genetic factor, an awakening test day genetic factor and an afternoon test day genetic factor. This implies that a set of genes controls diurnal cortisol regulation which is distinct from the genetic factors controlling response to stress. The two test day genetic factors suggest that cortisol functions differently in response to awakening or throughout the day. Additionally, the test day factors may indicate different genetic factors for reactivity in the presence of a stressful environment. Genetic factors were moderately correlated with each other, indicating some common genetic effects across factors although the genetic influences on diurnal cortisol regulation are distinct from those influencing the regulation of cortisol stress responses.

The Moderating Effects of Impulsivity and Dietary Restraint on Associations Between Serotonin Genes and Bulimic Symptoms

Sarah E. Racine[1], Kristen M. Culbert[1], Christine L. Larson[2], Kelly L. Klump[1]

[1]Department of Psychology, Michigan State University, East Lansing, MI; [2]Department of Psychology, University of Wisconsin Milwaukee, Milwaukee, WI

The heritability of bulimia nervosa (BN) is well-established (Bulik et al. Bio Psychiatry 44:1210–1218, 1998); however, the identification of risk genes that replicate across studies has been difficult. These difficulties are likely due to a number of limitations, including the use of small samples, a failure to examine the influence of quantitative traits in gene-BN associations, and the lack of consideration of gene-environment interactions (Klump and Culbert, Curr Dir Psychol Sci 16:37–41). The current study will improve upon past work by investigating whether quantitative traits (impulsivity) and environmental risk factors (dieting) moderate associations between serotonin (5-HT) genes and BN symptoms in a large community sample of women ($N = 344$). BN symptoms, dietary restraint, and impulsivity were assessed using well-validated self-report questionnaires. Saliva samples were genotyped for the 5-HT2a receptor T102C polymorphism and 5-HT transporter promoter polymorphism. Data collection for this project is complete, but analyses are on-going. We are using principal components analysis to create a composite dietary restraint factor using items from several restraint scales. Hierarchical multiple regression analyses will then be used to examine the main effects, two-way interactions, and three-way interaction of impulsivity, dieting, and genotype on BN symptoms. Finally, bivariate regression models will be used to control for possible gene-environment correlations by examining whether genotype predicts levels of dietary restraint. Overall, we expect that associations between 5-HT genes and BN symptoms will be stronger in women who are impulsive, who diet, and/or who have both risk factors, and that results will not be due to gene-environment correlations. Findings from these analyses will increase understanding of candidate genes for BN and the temperamental and environmental circumstances in which they are expressed.

Differential Parenting in Fathers by Offspring COMT Genotype

David C. Rettew[1,2], Lynsay Ayer[1,2], Robert Althoff[1,2], Timea Nelson[1,2], Erik Ehli[1,2], Gareth Davies[1,2], James Hudziak[1,2]

[1]University of Vermont College of Medicine, Burlington, VT; [2]Institute of Human Behavioral Genetics, Avera Behavioral Health & South Dakota State University College of Pharmacy, Brookings, SD

Objectives Variations in the catechol O-methyltransferase (COMT) gene in addition to parenting behaviors such as inconsistent discipline have been linked to child psychopathology. To test for the presence of gene-environment correlations, we analyzed differences in levels of parental inconsistent discipline between siblings who varied in their COMT genotype. *Method* Data are drawn from a family study in Vermont who were genotyped on the two allele system of the COMT gene, using cheek swabs. Out of 205 families, we selected 44 families in which one sibling was homozygous at the valine/methionine polymorphism of codon 158 for the val/val while the other possessed with met/met or val/val genotype. Data from fathers was available for 18 of those families. Parents completed the Alabama Parenting Questionnaire regarding their parental behavior specific to each child. Paired *t*-test were performed for mothers and fathers to analyze differences between siblings in levels of inconsistent discipline. *Results* The val/val group was 57% female with a mean age of 11.2 years. The val/met or met/met group was 49% female and had a mean age of 11.0 years. Correlations between levels of inconsistent parenting were high between siblings for both mothers ($r = 0.80$, $P < 0.001$) and fathers ($r = 0.90$, $P < 0.001$). For mothers, there were no significant differences in the levels of inconsistent parenting between their val/val child and non val/val sibling. Fathers, however, reported higher levels of inconsistent parenting for their non val/val child in comparison to their val/val child ($t(17) = 2.14$; $P = 0.047$). *Conclusions* There is preliminary evidence in this small study of a gene-environment correlation in which fathers have higher levels of inconsistent parenting between different siblings based on the COMT genotype.

Autistic Traits and Birth Problems: A Study Suggesting that Both Genes and Environment Play a Part in Their Association

Angelica Ronald[1], Francesca Happé[2], Katharina Dworzynski[3], Patrick F. Bolton[4], Robert Plomin[4]

[1]Birkbeck College, London; [2]Social Genetic and Developmental Psychiatry (SGDP) Centre, Institute of Psychiatry, King's College London; [3]King's College London; [4]SGDP Centre, Institute of Psychiatry, King's College London

Autistic spectrum disorders (ASD) are thought to be caused by a combination of genetic and environmental risk factors, but identifying specific environmental triggers has proved difficult. Problems during the birth process have been shown to be associated with ASD but whether these problems are an environmental cause of ASD, or a consequence of the genetic liability for ASD, is unknown. The current project explored whether perinatal problems are associated with dimensional measures of autistic behaviours, whether birth problems are themselves influenced by genetic factors, and whether there is evidence that the association between birth problems and autistic traits could be due to environmental influences. Parents of over 10,000 twin pairs from the Twins Early Development Study, a UK-based longitudinal twin cohort, reported on birth-related problems one year after the twins were born. When the twins reached middle childhood, parents and teachers of over 6,000 pairs reported on the twins' autistic behaviours. Weak but significant and positive correlations were observed between birth problems and dimensional measures of autistic behaviours at ages 7 and 8, after controlling for cognitive ability and socio-economic status. Twin model-fitting revealed that child-specific birth problems were modestly heritable (12%), as well as showing significant shared and nonshared environmental influences. Within monozygotic twins, the twin with more autistic traits had more problems at birth. These data suggest both genes and nonshared environment may play a role in the association between birth problems and autistic traits.

The Genetics and Evolution of the General Factor of Personality

J. Philippe Rushton[1], Trudy Ann Bons[1], Yoon-Mi Hur[2]

[1]University of Western Ontario, London, Canada; [2]Chonnam National University, Gwangju, South Korea

Three studies tested the hypothesis that a general factor of personality (GFP) underlies diverse individual differences including altruism, the Big Five factors of Openness, Conscientiousness, Extraversion, Agreeableness, and Emotional Stability, and the EAS temperament traits of Emotional Stability, Activity, and Sociability. In Study 1, 214 university students completed 36 personality scales. In Study 2, 322 pairs of monozygotic (MZ) and dizygotic (DZ) twins completed 29 5-point rating scales plus questionnaires. In Study 3, 575 pairs of 2- to 9-year-old Korean twins were rated by their mothers on 25 temperament scales. Factor analyses revealed a hierarchical organization with GFP at the apex and the Big Five and/or EAS temperament scales intermediate. The twin data show GFP has an early age of onset with 50% of the variance attributable to non-additive (dominance) genetic influence and 50% to unique, non-shared environmental influence. We discuss a life history matrix encompassing brain size, maturational speed, and longevity, plus emotional intelligence and the personality disorders, and suggest natural selection acted directionally to endow people with more cooperative and less contentious personalities than their archaic ancestors, or nearest living relatives, the chimpanzees.

Sources of Continuity and Change in Activity Level in Early Childhood: Different Situations, Different Results (Partly)

Kimberly J. Saudino

Boston University

Activity level (AL) is a pivotal temperament dimension in infancy and early childhood, but little is known about the role that genetic factors play in the development of AL. The few longitudinal studies exploring genetic and environmental influences on continuity and change in AL have relied mainly on parent or observer ratings which yield equivocal conclusions about developmental change. The present study used mechanical motion recorders (actigraphs) to explore genetic influences on developmental change in AL in early childhood. The sample comprised over 300 twin pairs assessed at 2 and 3 years of age. At both ages, twins wore actigraphs for a 48-h period in the home and in laboratory test and play episodes. Stability for AL ranged from 0.32 in the play situation to 0.43 in the home, indicating both continuity and change

in the rank ordering of AL across early childhood. Longitudinal model-fitting analyses revealed that AL was genetically influenced at all ages and in all situations. Moreover, there was little evidence of differential heritability across age. For AL in the home, genetic influences accounted for 31% of the variance at age 2% and 25% of the variance at age 3. Shared environmental influences also contributed significantly to AL in the home (Age 2 c2 = 0.56; Age 3 c2 = 0.55). In the play situation, genetic influences accounted for 49% and 48% of the variance at ages 2 and 3, respectively. The remaining variance was due to nonshared environmental influences. Similar results emerged for AL in the test situation (Age 2 a2 = 0.55; Age 3 a2 = 0.54). The genetic factors that influence AL in the home covaried perfectly across age (rG = 1.0); however, this was not the case for AL in the lab (Play AL rG = 0.57; Test AL rG = 0.72). In fact, for both the lab play and situations, significant new genetic variance emerged at age 3. The new genetic effects in the lab situations may reflect increased self-control/self-regulation or adaptability to novel social situations.

Genetic and Environmental Contributions to Political Attitudes

Julie Aitken Schermer[1], Ed Bell[2], Philip A. Vernon[1]

[1]The University of Western Ontario; [2]Brescia University College

The genetic and environmental contributions to political attitudes were assessed in a sample of 253 pairs of adult twins (192 MZ pairs and 61 DZ pairs). Univariate genetic analyses were conducted on three specific items (general political interest, probability of voting in the next election, and left versus right wing) and seven political attitude scales. Genetic effects were found for general political interest and the degree to which individuals stated that they were left or right wing, with heritabilities of 62% and 42%, respectively. The probability of voting in the next election was found to be best explained by environmental factors. For the seven political attitude scales, only two, Capitalism and Wages, were found to be best fit by a genetic and environmental (AE) model, with heritability values of 51% and 57%, respectively. The remaining five scales, measuring Religion, Environment, Laws, Minorities, and the Government, were best fit by an environmental model.

Phenotypic, Genetic, and Environmental Properties of the Portrait Values Questionnaire

Julie Aitken Schermer[1], N. T. Feather[2], Gu Zhu[3], Nicholas G. Martin[3]

[1]The University of Western Ontario; [2]Flinders University; [3]Queensland Institute of Medical Research

The purpose of the present study was to examine the scale properties of the Portrait Values Questionnaire (PVQ; Schwartz et al. 2001) both at the phenotypic (observed) level as well as the genetic and environmental level. The PVQ measures 10 value types. Australian twins ($N = 695$) completed the PVQ as part of a larger questionnaire battery. Internal consistency estimates were found to be acceptable. Nine of the values types were found to have a genetic component with heritability estimates ranging from 10.8% to 38%. The achievement scale was best explained by environmental factors. The inter-scale correlations were found to range from 0.01 to 0.70 at the phenotypic level. Of these 45 correlations, 15 were found to be explained by overlapping genetic factors and almost all (41) were found to have significant environment correlations.

Phenotypic, Genetic, and Environmental Correlations Between Reaction Times and Working Memory in Young Twin Children

Julie Aitken Schermer[1], Philip A. Vernon[1], Kerry L. Jang[2]

[1]The University of Western Ontario; [2]University of British Columbia

Phenotypic, genetic, and environmental correlations between various reaction time and working memory measures were examined in a sample of 4- to 6-year-old twin children ($N = 680$ individuals). Univariate genetic analyses conducted on the same-sex pairs (101 MZ pairs and 132 same-sex DZ pairs) demonstrated that four of the eight reaction time measures had a genetic component (ranging from 38% to 47%) and that three of the four working memory tests had a genetic component (ranging from 54% to 73%). At the phenotypic level, most of the reaction time measures had significant negative correlations with the working memory measures. Multivariate genetic analyses revealed that some of the observed phenotypic correlations could be explained by common genetic factors, but that most were explained by overlapping common environmental effects.

A Behavior Genetic Analysis of Vocational Interests using a Modified Version of the Jackson Vocational Interest Survey

Julie Aitken Schermer, Philip A. Vernon

The University of Western Ontario

The purpose of the present study was to examine the scale properties of a modified version of the Jackson Vocational Interest Survey (JVIS; Jackson 1977) and to examine the heritability estimates of the 34 vocational interest scales and resulting factor scores. Two modifications to the questionnaire were conducted. The number of items was reduced to 170 (from 289 pairs) and the ipsative response format was changed to a 5-point Likert scale. Results based on 742 individuals suggest that the revised scales have good scale properties with an average Cronbach's alpha of .76 even though each scale consists of only five items. Thirty of the 34 vocational interests were found to have a genetic component with heritability values ranging from 37% to 61%. Four of the interest scales were found to have common environment effects between 28% and 46%. An exploratory factor analysis found that the modified interest scales could be reduced to seven factors. Univariate genetic analyses of the factor scale scores demonstrated that six of the seven had a genetic component and one was found to be best explained by common and unique environmental effects.

Associations Between Family Conflict and Child Internalizing and Externalizing Problems: A Children of Twins Approach

Alice C. Schermerhorn[1], Brian M. D'Onofrio[1], Jody M. Ganiban[2], Erica L. Spotts[3], Paul Lichtenstein[4], David Reiss[5], Jenae Neiderhiser[3]

[1]Department of Psychological and Brain Sciences, Indiana University; [2]Department of Psychology, George Washington University; [3]Center for Family Research, Department of Psychiatry & Behavioral Sciences, George Washington University; [4]Department of Medical Epidemiology and Biostatistics, Karolinska Institutet; [5]Yale Child Study Center, Yale University

Although previous work has documented links between family functioning and child adjustment, many questions remain regarding

the processes accounting for these associations. The current study utilized the children of twins design to explore whether genetic or shared environmental factors confound the intergenerational associations related to family conflict in a sample of twins and their offspring from the Twin and Offspring Study in Sweden (TOSS). The sample includes 872 twin families (386 MZ), with 898 boys and 846 girls; mean child age was 15.74 years (SD = 2.41). The family conflict correlations between co-twin families were larger for MZ twins (rMZ = 0.237, $P < .001$) than for DZ twins (rDZ = 0.050, $P > .10$), suggesting a genetic factor may underlie similarities in levels of conflict. We ran a series of multivariate behavior genetic models, including Cholesky decomposition and direct effect models, to examine the processes that account for the associations between family conflict and offspring psychopathology. The results suggest that the association between family conflict and child functioning is explained almost entirely by direct exposure to family conflict (or other factors that vary within twin families).

Chinese Twin Children Reared Apart and Together: A Prospective Study of Medical, Intellectual and Life History Characteristics

Nancy L. Segal, Joanne H. Stohs, Vanessa A. Harris, Kara Evans

California State University, Fullerton

China's One-Child Policy, implemented in 1979–1980, led to the abandonment and eventual adoption of thousands of infants, mostly female. Included among these infants were twins, adopted by different families and reared in separate homes. Eight sets of reunited twins (MZ: $n = 3$ pairs and DZ: $n = 5$ pairs) have participated in the first prospective study of twins reared apart. The mean age of the reared apart twin sample is 5.20 years, SD = 1.64, range: 3.26–7.83. This twin group is contrasted with samples of Chinese twins adopted together ($n = 17$ pairs, mean age = 6.94 years, SD = 2.17) and virtual twins (VTs; same-age unrelated siblings reared together from early infancy) $n = 123$ pairs, mean age = 5.62 years, SD = 1.72). (VTs were age-matched to the Chinese twin samples so represent a subgroup of the larger VT sample.) The families with Chinese twins completed a comprehensive test battery administered by California State University, Fullerton between February 2006 and February 2008. The battery included a Family Background Information Form, Medical History Questionnaire, Dental Questionnaire, Twin/Sibling Relationship Survey, School History Timeline, Physical Facilities in the Home Checklist, Child and Family Activities Survey, Child Behavior Checklist and Adoption History form, although forms are tailored to the specific kinships. Each twin completed the age-appropriate version of the Wechsler Intelligence Scale. Families with virtual twins have participated in the Fullerton Virtual Twin Study since its inception in 1991. The present research report provides descriptive characteristics and preliminary findings regarding the twins' resemblance in selected physical and behavioral traits (e.g., height, weight, IQ). Given that the study is ongoing, additional pairs will be included. Important issues regarding zygosity determination of the reared apart twins are highlighted, and future goals for this ongoing study are outlined.

Bereavement-Related Responses Following the Loss of an MZ or DZ Twin

Nancy L. Segal, Vanessa A. Harris

California State University, Fullerton

An informative variant of the classic twin design compares social-interactional outcomes and processes of MZ and DZ twins. This allows tests of evolutionary-based hypotheses concerning social relatedness. The present study compared the grief reactions of surviving MZ and DZ twins following the loss of their co-twin. The study also compared these responses with the twins' reactions to losing a non-twin relative, friend or spouse. Hypotheses derived from Hamilton's (1964) theory of inclusive fitness. Hamilton reasoned that cooperation should vary with genetic relatedness, as a way of indirectly transmitting one's genes into future generations. It was expected that (1) MZ twins would experience more severe grief reactions at the time of loss than DZ twins, and (2) loss of a twin would be perceived as more psychologically damaging than the loss of a non-twin relative. The sample was mostly recruited from the membership of the Twinless Twins National Support Group, supplemented by personal referrals. The mean age of the MZ twins ($n = 394$) and DZ twins ($n = 202$) at the time of loss was 39.98 years (SD = 16.11) and 37.77 years (SD = 16.19), and ranged from 15–87 years and 15–79 years, respectively. The mean age for completion of the survey for the MZ and DZ twins was 46.93 years (SD = 15.65) and 44.38 years (SD = 15.43), and ranged from 16–94 years and 18–83 years, respectively. Bereavement responses were assessed with a comprehensive Twin Loss Survey, one component of which was a Grief Intensity Scale (1 = No Grief to 7 = Total Devastation/Suicide Point). MZ twins showed significantly higher grief intensity ratings than DZ twins, as expected [$F(1,587) = 11.14$, $P < .001$]. In addition, the loss of a twin was rated significantly higher than the loss of all other relatives, except for the spouse. The findings are discussed with reference to theoretical and applied perspectives on bereavement.

Tacit Coordination in Monozygotic, Dizygotic and Virtual Twin Pairs: A Mechanism for Social Relatedness?

Nancy L. Segal[1], Shirley A. McGuire[2], Steven A, Miller[1], June Havlena[1]

[1]California State University, Fullerton; [2]University of San Francisco

Game theorists have a long-standing interest in why some partners are more likely to achieve mutual goals than others. However, behavioral genetic and evolutionary approaches have (with a few exceptions) been generally absent from experimental research in decision-making. The present twin and adoption study was undertaken to determine if tacit coordination varies as a function of the genetic relatedness between the social partners. Tacit coordination refers to circumstances in which "two parties have identical interests and face the problem not of reconciling interests but only of coordinating their actions for their mutual benefit when communication is impossible" (Schelling 1960, p. 54). The sample included 7–13-year-old monozygotic (MZ) twin pairs ($n = 53$), dizygotic (DZ) twin pairs ($n = 85$) and virtual twin (VT) pairs ($n = 42$; same-age unrelated siblings). Working independently, each child completed a list of questions under two conditions: individual and coordination. In the individual condition, they were instructed to simply answer the questions; in the coordination condition they were instructed to answer such that they and their co-twin/sib would produce the same solution. Co-twin consensus on questions completed under these two separate conditions was compared across the three sibling groups. MZ twins demonstrated significantly greater overall agreement than both DZ twins and VTs, as anticipated, using both broad match and exact match criteria. The implications of these results are examined with reference to behavioral genetic and evolutionary theories of behavior.

The Heritability of Partisan Attachment

Jaime E. Settle, Chris T. Dawes, Dr. James H. Fowler

University of California, San Diego

One of the strongest regularities in the empirical political science literature is the well-known correlation in parent and child partisan behavior. Until recently this phenomenon was thought to result solely from parental socialization, but new evidence on genetic sources of behavior suggests it might also be due to heritability. In this article we hypothesize that genes contribute to variation in a general tendency toward strength of partisanship. Using data collected at the Twins Days Festival in Twinsburg, Ohio, in 2006 and 2007, we compare the similarity of partisan strength in identical twins who share all of their genes to the similarity of partisan strength in non-identical twins who share only half. The results show that heritability accounts for almost half of the variance in strength of partisan attachment, and they suggest that we should pay closer attention to the role of biology in the expression of important political behaviors.

The Use of Latent Class Analysis in a Family-Based Association Including Haplotype Block Analyses in ADHD and Its Comorbidity with Reading Disability

Abdullah Sheikhi, Jan Piek, Neilson Martin, David Hay

School of Psychology, Curtin University of Technology, Perth- Western Australia

Attention Deficit Hyperactivity Disorder (ADHD) and Reading Disability (RD) are common nuero-behavioural disorders that frequently overlap together. However, this comorbidity is still not well understood. This study aimed to investigate the genetic components for ADHD subtypes, and RD separately, and ADHD RD comorbidty. Three approaches were applied to data from 2610 Australian twin families. (1) Latent Class Analysis (LCA) was applied to generate genetically independent classes that defined ADHD subtypes and RD. (2) Genetic modelling was used to examine whether children identified with comorbid ADHD-RD are a genetically distinct group from those who have only ADHD without RD. (3) family-based genetic association including haplotype analysis was also included, for 190 individuals, testing 21SNPs from five ADHD candidate genes (DRD4, DAT1, SNAP25, COMT, and HTR1B), and four RD candidate genes MRS2L, KIAA0319, TTRAP, and THEM2) from the 6p22.2 region. This approach was applied to compare the efficacy of DSM-IV diagnostic criteria and LCA in the genotyping analysis, and to detect some of the risk alleles of ADHD alone, RD alone, and comorbid ADHD-RD. Univariate and bivariate results indicated the presence of genetic components on each ADHD subtype and RD category, and also showed the existence of genetic factors in the ADHD/RD comorbidity. LCA produced nine ADHD/RD latent classes. Haplotype analysis detected one significant haplotype block containing two htSNPs (rs4680 and rs165599), of the COMT gene, with three risk alleles associated with some phenotypic RD components. This study found that the use of ADHD-RD latent classes is more suitable for performing genetic association studies and haplotype block analysis than is DSM-IV-defined ADHD and RD definitions. Furthermore, there is an overlapping of genetic effect, as ADHD candidate genes contributed to RD phenotypes and vice versa. Lastly, ADHD-RD comorbidity is might be caused by both ADHD and RD candidate genes.

An International Comparison of Etiologies of Remembered Parenting Using Japanese and Swedish Twins

Chizuru Shikishima[1], Kai Hiraishi[2], Shinji Yamagata[3], Yutaka Ono[4], Juko Ando[5], Jenae M. Neiderhiser[6]

[1]Graduate School of Human Relations, Keio University, Tokyo, Japan; [2]Kokoro Research Center, Kyoto University, Kyoto, Japan; [3]Japan Society for the Promotion of Science, Tokyo, Japan; [4]Health Center, Keio University, Tokyo, Japan; [5]Faculty of Letters, Keio University, Tokyo, Japan; [6]Department of Psychology, The Pennsylvania State University, PA, USA

With the use of the Parental Bonding Instrument [Parker et al. Br J Med Psycho1 52:1–10, 1979], etiologies of remembered parenting were compared between Japan and Sweden. Participants were 720 (MZf: 334, MZm: 158, DZf: 96, DZm: 48, DZo: 84) pairs of young adult male and female Japanese twins from the Keio Twin Project (KTP) and 824 (MZf: 245, MZm: 124, DZf: 267, DZm: 188) pairs of adult male and female Swedish twins from the Twin and Offspring Study in Sweden (TOSS) Project. For both maternal and paternal parenting for both samples, very similar phenotypic factor structure was produced from each of 25 items of the PBI, and the three factors were labeled Warmth, Protectiveness, and Authoritarianism. However, the relative genetic and environmental influences were different between the two samples; remembered parenting in the Japanese sample showed more genetic influences, while in the Swedish sample was due more to shared environmental influences. This tendency was particularly conspicuous in Authoritarianism. The multivariate genetic analysis using the three dimensions for both parents (i.e. maternal Warmth, maternal Protectiveness, maternal Authoritarianism, paternal Warmth, paternal Protectiveness, and paternal Authoritarianism) revealed that the six dimensions of parenting were more genetically correlated in Japan, while correlated with more shared environment in Sweden. These results could reflect possible differences in ways of parenting between Japanese and Western societies and/or they could be due to the different ages of the samples. Cross cultural implications with regard to parent-offspring involvement will be discussed.

Using Genetic and Environmental influences to Test the External Validity of Childhood Mood and Anxiety Disorders

Amber L. Singh[1], Irwin D. Waldman[2], Carol A. Van Hulle[3], Brian M. D'Onofrio[1], Benjamin B. Lahey[4]

[1]Indiana University; [2]Emory University; [3]University of Wisconsin – Madison; [4]University of Chicago

A valid taxonomy of mental disorders must demonstrate external validity. This study tested the external validity of the DSM-IV dimensions of anxiety and depression using etiologic influences as the external criterion variables. Participants were drawn from the Tennessee Twin Study ($N = 2{,}022$ twin pairs), a representative sample of 6–17-year-old twins born in Tennessee and still living in one of the states five MSAs (Nashville, Memphis, Knoxville, Chattanooga, and Bristol). Childhood mood and anxiety disorders were assessed with the Child and Adolescent Psychopathology Scale, which assesses DSM-IV symptoms of major depressive disorder, dysthymia, separation anxiety disorder, social phobia, specific phobia, agoraphobia,

and obsessive-compulsive disorder. Multivariate behavior genetic model-fitting analyses were conducted to test competing models of the common and unique genetic and environmental influences underlying the DSM-IV internalizing disorders. With the exception of major depressive disorder, each symptom dimension showed at least some unique genetic and environmental influences. All of the genetic influences underlying depression overlapped with those on generalized anxiety disorder, but each had unique environmental influences. The pattern of shared genetic and environmental influences underlying childhood mood and anxiety disorders parallels the phenotypic factor structure that has emerged in both exploratory and confirmatory factor analyses and largely supports the external validity of the DSM-IV emotional disorder taxonomy.

Genetic Relationships between Disordered Eating, Internalizing Symptoms, and Externalizing Behaviors in Adult Male and Female Twins

Jennifer D. Slane, S. Alexandra Burt, Kelly L. Klump

Michigan State University, Department of Psychology

Despite the significant sex difference in prevalence, relatively few studies have examined gender differences in genetic and environmental risk factors for eating disorders (T Reichborn-Kjennerud et al. Acta Pscychiatr Scand 108:196–202, 2003). Examining these sex differences may increase understanding of factors that create differential risk across gender. This study examined sex differences in genetic and environmental effects on (1) disordered eating (DE); and (2) phenotypic associations between DE and key comorbid symptoms. Participants were 146 female and 83 male same-sex adult twin pairs from the Michigan State University Twin Registry (Klump and Burt, Twin Res Hum Genet 9:971–977, 2006). Self report questionnaires were used to assess DE (e.g., weight preoccupation, binge eating), internalizing symptoms, (e.g., depression, perfectionism) and externalizing behaviors (e.g., aggression, alcohol use). Univariate, sex differences models were used to examine genetic and environmental influences on DE, internalizing symptoms, and externalizing behaviors in men versus women. Bivariate, Cholesky decomposition models were then used to examine genetic and environmental factors underlying associations between DE and these symptom dimensions. The univariate models indicated few sex differences in the magnitude of genetic and environmental influences on DE and related phenotypes. Heritability estimates were generally greater than 50% with the remaining variance due to nonshared environmental factors. Bivariate models further indicated that common genetic factors account for phenotypic relationships between DE and all internalizing and externalizing symptoms. Overall, findings suggest few sex differences in genetic risk factors for DE. Studies of opposite-sex twins are needed to determine whether sex differences in DE prevalence are due to differences in the types of genes contributing to the symptoms or differential exposure to putative risk factors in women versus men.

Nonshared Environment and Disordered Eating: A Longitudinal Investigation of Parent-Child Conflict

Alexia Spanos[1], Kelly L. Klump[1], S. Alexandra Burt[1], Matt McGue[2], William G. Iacono[2]

[1]Department of Psychology, Michigan State University, East Lansing MI; [2]Department of Psychology, University of Minnesota, Minneapolis MN

Twin studies suggest that nonshared environmental factors contribute to the development of eating disorders (Klump et al. Int J Eat Disord 31:118–135, 2002). However, few studies have sought to identify these nonshared environmental risk factors, and none have done so with a longitudinal design. The current project thus employed a longitudinal, MZ twin differences design to examine parent-child conflict as a nonshared environmental risk factor for disordered eating across adolescence and young adulthood (i.e., ages 11, 14, 17 and 20). Participants included 234 female MZ twin pairs from the Minnesota Twin Family Study. Disordered eating was assessed with the Total Score, Weight Preoccupation and Binge Eating subscales of the Minnesota Eating Behaviors Survey (MEBS; von Ranson et al. Eat Behav 6:373–392, 2005). Parent-child conflict was assessed with the Parental Environment Questionnaire (PEQ). Twin discordance was defined using twin difference scores on both the MEBS and PEQ. Pearson correlations were used to examine initial associations between twin differences in disordered eating and parent-child conflict. Cross-lagged models were then used to examine longitudinal associations among these variables, controlling for within-age associations (Burt et al. Dev Psychol 42:1289–1298, 2006). Across time, only the association between twin differences in disordered eating at age 14 and twin differences in parental conflict at age 17 was significant. Interestingly, twin differences in parental conflict were not risk factors for later disordered eating; rather, twin differences in disordered eating predicted later differences in parental conflict. Findings suggest that differences between genetically identical siblings on measures of parental conflict may be a consequence of, rather than a risk factor for, differences in disordered eating. Additional longitudinal research is needed to replicate and extend these results using other measures of nonshared environment.

Parental Divorce and Bulimic Symptoms: Evidence for a Gene-Environment Interaction?

Jessica L. Suisman, S. Alexandra Burt, Kelly L. Klump

Michigan State University, East Lansing, MI

Parental divorce and marital discord predict symptoms of bulimia nervosa (BN; Wade et al. Int J Eat Disord 30:389–400, 2001). However, previous studies have only examined the main effects of parental divorce on BN; interactions with parental divorce and other important risk factors have not been investigated. Given the significant heritability of BN (Bulik et al. Biol Psychiatry 15: 1210–1218, 1998), the effects of parental divorce may operate through gene-environment interactions, such that parental divorce increases risk for BN only in those at elevated genetic risk. The current study will directly examine this possibility by investigating whether and how parental divorce moderates genetic vulnerabilities for bulimic symptoms. Participants include 294 same-sex adult female twins (ages 18–28 years) from the Michigan State University Twin Registry (MSUTR; Klump and Burt, Twin Res Hum Genet 9:971–977, 2006). Bulimic symptoms (i.e., binge eating, weight preoccupation, use of compensatory behaviors) were measured using the Minnesota Eating Behavior Survey (MEBS; von Ranson et al. Eat Behav 6:373–392, 2005). Parental marital status (divorced versus intact) was measured via twin self-report. Gene-environment interaction models (Purcell, Twin Res 5:554–571, 2002) will be used to examine the moderating effects of divorce on the heritability of bulimic symptoms. Data collection for this project has been completed, although analyses are on-going. Findings are expected to increase understanding of the specific pathways by which parental divorce influences risk for bulimic symptoms.

Genetic and Environmental Influence on Reading Abilities in Young Twins: Preliminary Examination of Racial/Ethnic Differences

Jeanette Taylor, Chris Schatschneider

Department of Psychology, Florida State University

Historically, much of the twin research on reading related constructs has been conducted on older children and adolescents. Even recent reports on young twins have lacked information on the etiological architecture of various reading abilities across racial/ethnic groups. To address these two gaps simultaneously, data are presented from twins in Kindergarten and 1st grade (ages 5–7). Overall, univariate models for standardized test data assessed in Kindergarten suggested little additive genetic influence on anything but the most complex phonological task (decoding nonsense words), but differences in types and magnitudes of etiological influences were found across racial/ethnic groups for some measures. Univariate models for standardized test data assessed in 1st grade suggested moderate genetic influence across tasks. Though racial/ethnic groups were more consistent with regard to which model fit best for each measure, variance in parameters could be fully constrained across groups only for nonsense word reading. Phenotypic correlations among reading measures were medium in magnitude in Kindergarten and small to medium in 1st grade. Based on cross-twin cross-trait correlations, shared and non-shared environment appeared important in explaining associations among variables in both Kindergarten and 1st grade with the exception of the association between decoding measures in 1st grade, which evidenced genetic influence. The pattern of correlations was similar across racial/ethnic groups. Shared environment could be a target for programs aimed at improving early reading skills, which predict later reading and impact school success.

A Neural Network Twin Model: Simulating Heritability in Language Development

Michael S. C. Thomas, Angelica Ronald

Developmental Neurocognition Lab, Birkbeck College, University of London, UK

Using the twin study design (among others), behavioral genetics has demonstrated that many aspects of cognitive and language development show marked heritability, that is, the influence of genes on individual variation. Recently, it has been argued that such variation is the accumulated influence of many genes, each with small effect size (Kovas and Plomin 2006). Indeed, in this view, both developmental disability and giftedness are the extremes of normal variation rather than being qualitatively different phenomena. However, to date, few attempts have been made to link a genetic level of description with mechanistic models of cognitive development, and even fewer that do so in order to explain individual variability rather than normative development. In this presentation, we report on a model combining genetic algorithms with artificial neural network models of language acquisition to simulate both genetic and environmental sources of variation in English past tense acquisition. These initial results demonstrate how the framework produces a population distribution of performance, and how this distribution changes both across development and according to the measure used. We present a simulated twin study design that demonstrates the heritability of past tense acquisition in an artificial neural network system, and test the performance of the model against real twin data of past tense acquisition in childhood (Bishop 2005).

Nonlinear Factor Analysis and Genetic and Environmental Influences on Intelligence

Elliot M. Tucker-Drob, K. Paige Harden, Edison Choe, Eric Turkheimer

Department of Psychology, University of Virginia

Conventional factor analytic models presume that the functions governing the correspondences between unobserved and observed variables are linear in form. However recent statistical and computational developments now allow for examinations of the possibility that these relations take on other functional forms. In fact, a recent application of nonlinear factor analysis to multivariate cognitive ability data suggests that the relations among cognitive abilities differ according to ability level (c.f. Detterman and Daniel, Intellect 13:349–359, 1989). The current paper reports the results of an application of nonlinear factor models to genetically informative twin data. This approach gives new insights into the recent findings that socioeconomic status modifies the genetic and environmental influences on intelligence (e.g. Turkheimer et al. Psychol Sci 14:623–628, 2003; Harden et al. Behav Genet 37:273–283, 2007.

Heritability of Stuttering and Non-Fluency in 5-Year-Old Dutch Twins

C. E. M. van Beijsterveldt[1], S. Felsenfeld[2], D. I. Boomsma[1]

[1]Department of Biological Psychology, Vrije universiteit, Amsterdam, The Netherlands; [2]Department of Communication Disorders, Southern Connecticut State University, New Haven, USA

Speech fluency is a skill for which there is considerable individual variation. At one extreme is the clinical disorder known as stuttering and at the other extreme is the speech produced by professional voice users, that is often remarkably free of speech disruptions. The role that genes may play in determining how fluent a speaker will be is largely unknown, particularly for the vast majority of speakers whose fluency falls somewhere along the continuum of normal functioning. The primary aim of the present study was to examine the heritability of stuttering and speech non-fluency. Information on speech fluency was obtained by maternal reports for about 10,500 5-years-old twin pairs born between 1989 and 1999. On the basis of maternal responses to six survey items children were classified as highly non-fluent and as probable for stuttering. The overall prevalence for probable stuttering was 4% (1.5:1 ratio of male: female affected). For highly non-fluent it was 4.5% (1.7:1 ratio of male: female affected). Results of the genetic analyses revealed that both phenotypes were moderately heritable, with heritability estimates of .42 and .49 for probable stuttering and high non-fluency, respectively. Shared environmental factors were also significant (0.44 for probable stuttering and 0.20 for non-fluency). For both phenotypes, the magnitude of the genetic and environmental influences did not differ between boys and girls. The phenotypic correlation between the two phenotypes was 0.72. The bivariate genetic analysis showed that this correlation was primarily due to shared genetic factors (47%) and shared common environmental factors (33%).

Environmental Moderation of the Heritability of IQ in Adults

Sophie van der Sluis, Gonneke Willemsen, Eco J. C. de Geus, Dorret I. Boomsma, Danielle Posthuma

VU University, Amsterdam

The notion that cognitive ability is under genetic pressure is no longer in dispute. Heritability estimates are often assumed constant across different subgroups. However, several studies suggest that in children, heritability of cognitive ability is moderated by e.g. parental income (Harden and Turkheimer, Behav Genet 37:273–283, 2007), parental socioeconomic status (SES, Turkheimer et al. Psychol Sci 14(6):623–628, 2003), and parental educational level (Rowe et al. Child Dev 70(5):1151–1162, 1999). In the present study, we investigated the effect of parental educational level on the heritability of cognitive ability in adult males and females. In addition, we studied the moderation effect of environmental characteristics that are informative of the participants' present environment: partner's educational level, and urbanization level and mean real estate price of the participant's residential area (the latter as rough indication of income). Data on IQ (Dutch WAIS-III) and these moderators were collected in over 700 Dutch monozygotic and dizygotic twins and their siblings. Our analyses showed increased common environmental variation in older males whose parents were more highly educated, and increased unique environmental effects in older males living in more affluent areas. On average, FSIQ scores were roughly 5 points higher in participants with highly educated parents, compared to participants whose parents were less well educated. In older participants, IQ scores were about 2 points higher when their partners were highly educated. Finally, higher urbanization levels were associated with higher FSIQ scores in younger males. Most results were replicated for VIQ and PIQ.

Cognitive, Affective and Behavioral Components of Empathy in Middle Childhood: A Comparison of Two Twin Samples

Carol A. Van Hulle[1], Jane E. Schreiber[1], Kathryn Lemery-Chalfant[2], H. Hill Goldsmith[1], Carolyn Zahn-Waxler[1]

[1]University of Wisconsin, Madison WI; [2]Arizona State University, Tempe AZ

Empathy involves the ability to ascertain (cognitive) and share (affective) others' emotional states, and may lead to prosocial actions to alleviate others' distress (Knafo et al. submitted). Twin studies have shown that familial factors, particularly heritable factors, account for individual differences in global ratings of empathy. Few studies of empathy have targeted middle childhood, and fewer have examined affective and cognitive components of empathy. We explored the genetic and environmental influences on observed reactions to a stranger's distress in 7–8 year old twins drawn from two studies. Sample 1 was drawn from 173 MZ and 136 same-sex DZ twin pairs participating in the Colorado Longitudinal Twin Study. Sample 2 was drawn from 99 MZ and 187 DZ twin pairs participating in the Wisconsin Twin Project. In both samples, children took part in behavioral assessments in their homes, during which an examiner pretended to pinch her finger in a clip board. Children's videotaped reactions were coded for the following: hypothesis testing, verbal and non-verbal expressions of concern, number of prosocial acts, positive affect (sample 2 only) and presence of callous behaviors. Children also received a global empathy rating. Given similarities between the two studies we expected to find some consensus in the variation accounted for by genetic and environmental influences. Instead we found substantial differences across studies, particularly for prosocial behavior and callous responses. In general, shared and non-shared environmental factors were more salient than genetic factors, with non-shared environment (and measurement error) accounting for a substantial portion of the variability. In Sample 1, variation in callousness was entirely due to non-shared environment whereas in Sample 2, 57% of the variation in callousness was due to shared environmental influences. Modest shared environmental influences were found for prosocial acts in Sample 1 while heritable factors contributed to variation in prosocial acts in sample 2. The provide a cautionary example of against generalizing from a single measure or a single study.

Gender Differences in the Genetics of Sexual Desire

Markus Varjonen, Pekka Santtila, N. Kenneth Sandnabba

Åbo Akademi University

Previous research has consistently shown gender differences in the level of sexual desire, with men scoring higher than women on a range of measures aimed at quantifying sexual desire (for a review, see Baumeister et al. Pers Soc Psychol Rev 5:242–273, 2001). The present study explored the genetic and environmental etiology of differences in sexual desire in a population sample of Finnish twins and their siblings ($N = 9{,}160$). Men reported a higher level of desire than women did ($P < 0.001$), while the variance was greater for women when compared to men ($P < 0.001$). Additive genetic effects accounted for 41% of the variance for sexual desire in women, and for 38% of the variance in men, with unique environmental influences underlying the remaining variance. This difference, although small, was still statistically significant. There were also qualitative gender differences in the genetic effects, with the genetic correlation between opposite sex siblings estimated at .231. This indicates that partly different genes cause the variance in sexual desire for men and for women. These results highlight the need to view male and female sexual desire, or problems related to desire, from different perspectives.

Does the Heritability of Child Externalizing or Internalizing Differ Based on Rural vs. Urban Residence?

Matthew K. Vendlinski[1], Jennifer L. Bagley[1], Nicole L. Schmidt[1], Carol A. Van Hulle[1], Kathryn Lemery-Chalfant[2], H. Hill Goldsmith[1]

[1]Waisman Center, University of Wisconsin—Madison; [2]Arizona State University

Genetic variation contributes to child externalizing and, to a lesser extent internalizing. However, heritability may differ as a function of population density (Legrand et al. Psychol Med 1:1–10, 2007). We tested whether heritability of child externalizing and internalizing differs for rural vs. urban children. Behaviors were measured in 599 (217 MZs, 202 SSDZs, 180 OSDZs), 8 y/o twin pairs using a composite of self, parent, and observer reports. A US 2000 census classification system determined rural (44.9%) vs. urban status. Using HLM, rural residence was not linked to externalizing (coefficient $= -0.04$, $P = 0.33$) or internalizing (coefficient $= -0.03$, $P = 0.40$). Biometric parameters were first calculated in Mx using a multi-group model. Estimates for externalizing were A = 0.83, C = 0, and E = .17 for rural children and A = 0.67, C = 0, and E = 0.33 for urban children (model fit: χ^2 (df) = 5.74 (6), $P = 0.45$). Estimates for internalizing were A = 0.61, C = 0, and E = 0.39 for rural children and A = 0.56, C = 0, and E = 0.44 for urban children (model fit: χ^2 (df) = 5.15 (6), $P = 0.53$). The model could not be constrained so that estimates were equal across rural and urban groups without significant loss of fit for either externalizing ($\Delta\chi^2$ (df) = 9.22

(3), $P = 0.03$) or internalizing ($\Delta\chi^2$ (df) = 9.39 (3), $P = 0.03$). Heritability of externalizing in first graders varied across rural vs. urban contexts. Contrary to findings for adolescent males (Legrand et al. 2007) child externalizing was more heritable in rural environments. Although fit statistics suggest that the heritability of internalizing differs based on residence, parameter estimates were similar across groups. It is possible that fit was different between the two internalizing models due to relatively large variance in internalizing among rural DZ children vs. MZs and urban DZ children. This finding should be viewed skeptically until this issue is resolved.

Genetic Heterogeneity in the MMPI Clinical Scales: A Twin Study of the Harris-Lingoes and Subtle-Obvious Subscales

Richard J. Viken, Richard J. Rose

Indiana University

The clinical scales of the Minnesota Multiphasic Personality Inventory were developed by a blind-empirical criterion-keyed approach. Almost from the beginning, there has been concern that this development approach resulted in scales that were symptomatically and etiologically heterogeneous. One strategy for dealing with this heterogeneity has been to develop content-based subscales. Here, we consider as an example of the content based approach the Harris-Lingoes subscales. Another strategy has been to develop subscales based on measurement considerations. For instance, the Subtle-Obvious subscales were developed by dividing the items in each clinical scale into those for which the meaning would be obvious to the layperson, and those for which the meaning would not be obvious (subtle items). We use a sample of 491 pairs of same-sex twins to evaluate each of these attempts subdivide the MMPI clinical scales. Results show that the Subtle and Obvious components of each clinical scale generally had little genetic influence in common (mean rg = −0.04). Indeed, when there were significant genetic associations between the Subtle and Obvious components of a given scale the associations were generally negative. Similarly the Harris-Lingoes content approach appeared to be successful in identifying components of the clinical scales that were genetically distinct. The results suggest that the full MMPI clinical scales each comprise several etiologically distinct symptom clusters.

Extreme Male/Female Brain Defined with Two Neuropsychological Tests

Eero Vuoksimaa[1], Annamari Tuulio-Henriksson[2], Laura Hokkanen[1], Jaakko Kaprio[3], Richard J. Rose[4]

[1]Department of Public Health, University of Helsinki, Finland; [2]Department of Mental Health and Alcohol Research, National Public Health Institute, Helsinki, Finland & Department of Psychology, University of Helsinki, Finland; [3]Department of Public Health, University of Helsinki, Finland & Department of Mental Health and Alcohol Research, National Public Health Institute, Helsinki, Finland; [4]Department of Psychological and Brain Sciences, Indiana University, USA

On average, males outperform females in the Mental Rotation Test (MRT) while the converse holds for Digit Symbol (DS). We adapted the extreme male brain theory of autism (Baron-Cohen et al. Science 310:819–823, 2005) and distinguished extreme male/female brain with performance differences in MRT and DS. We then tested whether opposite-sex (OS) twins differ from same-sex (SS) twins. Our sample of 397 twins, aged 21–24, was from ongoing FinnTwin12-22 studies and included 191 SS females, 103 SS males, 54 OS females and 49 OS males. We calculated the difference between DS and MRT Z-scores. Subjects whose DS score was 2 standard deviations (SD) higher than their MRT score were classified into the extreme female brain (EFB) group. Subjects whose DS score was 1 SD higher than their MRT score formed the female brain (FB) group. Those with less than 1 SD discrepancy between DS and MRT scores formed the neutral group. The male brain (MB) group had a MRT score 1 SD higher than their DS score, and the extreme male brain (EMB) group comprised those with MRT score 2 SD higher than their DS score. Brain type differed significantly between sexes ($P < 0.0001$): 15% of males and 0.8% of females were categorized in EMB group. The MB group included 25% of males and 9% of females. Conversely, 9% of females were categorized in the EFB group, against only 0.7% of males. The FB group included 25% of females and 6% of males. Females from OS and SS pairs did not differ in membership in different brain type groups. But female twins from OS pairs, compared to females from SS pairs, were more likely to have performed well in both MRT and DS ($P < 0.05$). When we analysed the absolute difference between DS and MRT as a continuous variable, the results were essentially unchanged. Further, there was a significant difference between SS and OS males in their continuous difference scores ($P < 0.01$), indicating that OS males exhibit a more masculine pattern than males from SS pairs.

Reproductive Onset and Substance Use/Disorder: Genetically-Informed Associations

Mary Waldron[1], Andrew C. Heath[1], Michael T. Lynskey[1], Kathleen K. Bucholz[1], Pamela A. F. Madden[1], Nicholas G. Martin[2]

[1]Department of Psychiatry, Washington University, St. Louis, MO; [2]Queensland Institute of Medical Research, Brisbane, Australia

We examine genetic influences contributing to previously documented associations between reproductive timing and lifetime history of alcohol dependence, regular smoking, nicotine dependence, cannabis use, and problem use of cannabis. Data from 2658 twin pairs (n female MZ = 676, n male MZ = 475, n female DZ = 493, n male DZ = 377, n opposite-sex DZ = 637) were drawn from a young cohort of Australian twins born between 1964 and 1971. Survival analyses were conducted using Cox proportional hazards regression models predicting age at first childbirth from substance use/disorder and dummy variables for co-twin substance use/disorder, zygosity, and their interaction, the latter a test of genetic effects. For alcohol dependence, nicotine dependence, and cannabis ab/use, interactions were small and non-significant. In women, zygosity*co-twin regular smoking was a strong predictor of reproductive onset [Hazard Ratio (HR) = 1.87, 95% CI: 1.14–3.06]. While age at first childbirth and alcohol, smoking, and illicit drug use phenotypes show moderate to substantial univariate heritability, results suggest little to no effect of a common inherited liability to reproductive timing and substance use/disorder.

The Etiology of Hostile Perceptual Biases and their Relation with Children's Aggression

Irwin D. Waldman

Emory University

Twin and adoption studies have found both genetic and environmental influences to underlie aggression and antisocial behavior, as well as their overlap with Attention Deficit Hyperactivity Disorder (ADHD),

although the specific etiological mechanisms accounting for these influences are largely unknown. Developmental psychopathology researchers have posited that misperceptions of others' emotions and intentions represent an important specific etiological mechanism underlying children's aggression, one which is due to various aspects of the environments in which children are raised. Despite this hypothesis, no study has yet tested whether such hostile perceptual biases might have a genetic component, and be influenced by the same genes that underlie children's ADHD, aggression, and antisocial behavior. In this study, we use data from 180 twins in 90 twin pairs on whom we collected DNA and tested on a lab measure of intention-cue detection. Participating twins and their parents were genotyped for several candidate genes that underlie various aspects of catecholaminergic function. Specifically, we examine the relation of hostile perceptual biases with children's ADHD, aggression, and antisocial behavior; estimate genetic and environmental influences on hostile perceptual biases; and test whether candidate genes that underlie catecholaminergic function are associated with hostile perceptual biases and children's aggression and antisocial behavior. These findings should help illuminate the causes of children's hostile perceptual biases, their etiological role in aggression and antisocial behavior, and the substantial overlap of these comorbid conditions with ADHD.

Shared Genetic Influences and Causal Effects Between Hyperactivity-Impulsivity and Novelty Seeking

Alexis C. Wood, Fruhling Rijsdijk, Jenny Van Heerden, Philip Asherson, Jonna Kuntsi

MRC Social, Genetic and developmental Psychiatry Centre, Institute of Psychiatry, London UK

Previous research suggests an association between hyperactivity-impulsivity—one of the two behavioural dimensions that form attention deficit hyperactivity disorder—and the temperament characteristic of novelty seeking. We aimed to examine etiological links underlying the co-occurrence between these behaviours using a general population sample of 668 twin pairs for whom we obtained parent ratings in middle childhood. Our analyses confirmed the phenotypic link between the two behaviours ($r = 0.36$) and showed that much of the covariation can be accounted for by shared genetic effects, with over 80% of the genetic influences shared. We further showed that a portion of the shared genetic influences may arise out of phenotypic causal influences between novelty seeking and hyperactivity-impulsivity, suggesting a complex interplay between these behaviours.

Drinking Alcohol to Improve Mood Partially Mediates the Relation Between Major Depression and Alcohol Dependence

Kelly C. Young-Wolff[1], Kenneth S. Kendler[2], Carol A. Prescott[1]

[1]Department of Psychology, University of Southern California, Los Angeles, CA; [2]Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond, Virginia

Previous studies have indicated that major depression (MD) can increase the risk for alcohol dependence (AD), and that individuals who drink to regulate and improve their affective state drink more frequently and may be at a higher risk for developing AD than individuals without this motivation. Both MD and drinking to manage mood have been shown to share genetic risk with AD, but the role of mood-related drinking motives as a mediator of the association between MD and AD has not been studied. We examined to what degree mood-related drinking motives explain the phenotypic and genetic overlap of MD and AD. Lifetime diagnoses of DSM-IV MD and AD were assessed using structured clinical interviews with 6818 participants in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD; Kendler and Prescott, 2006, Genes, Environment, and Psychopathology, Guiford, New York) a population-based sample of adult twin pairs aged 18–56. Mood-related drinking motives were measured using the Managing Mood (MOOD) subscale of the Alcohol Use Inventory (Horn and Wanberg, Am Psychol 38:1055–1069, 1983). The correlations between risk for lifetime MD and AD were moderate and MOOD scores accounted for about one-third of the individual differences in risk for AD in both females and males. Results from individual-level analyses indicated that a substantial proportion of the MD-AD association was mediated by MOOD scores. The results of twin models indicate that the familial basis of MD-AD comorbidity is substantially mediated by the motive of drinking to improve mood. This study expands the current literature on the association between MD and AD and suggests that treatments that focus on changing mood-related drinking motives may be effective in reducing risk among individuals whose alcoholism is comorbid with depression.

Multivariate Genetic and Environmental Modeling among Childhood Externalizing Disorders across Different Modality

Mo Zheng, Laura A. Baker, Catherine Tuvblad

University of Southern California

Previous research has shown the importance of genetic factors in understanding the etiology of co-morbidity among childhood externalizing disorders, but results have not been consistent across studies in terms of relative weight assigned to genetic, shared and non-shared environmental factors. Specifically, heritability estimates may vary across different measurements, different time points and different informants. This study aimed to use multivariate genetic and environmental modeling to find best fitting models for co-morbid externalizing disorders, including attention deficit-hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) and conduct disorder (CD), and to investigate how results change among multiple outcomes across different modality. The sample included 605 twin pairs recruited from the Southern California Twin Registry. Three independent analyses were run to examine (1) how genetic and environmental influences differ across measurement instruments using Diagnostic Interview Schedule for Children (DISC-IV) and Child Behavior Checklist (CBCL), (2) how genetic and environmental influences change developmentally from age 9 to age 12, and (3) how genetic and environmental influences vary across different informants using both caregiver's and teacher's CBCL responses. Four alternative multivariate models were tested in each analysis, including a one factor common pathway model, a two factor common pathway model, a hierarchical model, and a Cholesky decomposition model. Results showed that the Cholesky model fit best for the multi-measurement data and longitudinal data, while hierarchical model fit best for the multi-informant data. Co-morbidity common to caregiver and teacher informant reports, and co-morbidity unique to each informant were both influenced largely by genetic influences. Shared environmental contributions, however, were not significant in explaining either the shared co-morbidity or co-morbidity unique to each informant.