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CONFERENCE ABSTRACTS

Nelson Adams¹

Variability in Alcohol Preference in Maudsley Reactive Inbred Male Rats

¹Winston-Salem State University

Grant Support: NIH-NIGMS-MBRS-08040

Address: Social Sciences, 601 Martin Luther King Jr. Drive, Winston-Salem State University, Winston-Salem, NC 27110 USA, Tel: 336-750-2626, FAX: 336-750-2647, email adamsn@wssu.edu

Maudsley Reactive (MR) inbred rats, selected for high defecation in an open field, relative to Maudsley Nonreactive (MNR) rats, have been compared for alcohol preference (AP) many times. Maudsley rats from the North American Harrington derivation (MR/Har) exhibit high AP under varying conditions (N. Adams and D. A. Blizard, 2002, *Behav. Genet.* 32, 277-299). Whereas MR/Har females often show high AP across conditions, and MNRA/Har rats exhibit uniformly low-moderate AP, MR/Har males' AP ranges from avoidance of ethanol (E) to quite high AP across experimental conditions. One experimental variation that markedly alters MR males' AP is prior exposure to E. One day or multiple days of exposure to 10% E as the sole source of fluid results in more than a doubling of AP in subsequent 2-bottle choice tests in MR males. Subsequent studies showed that adult MR males exposed to 10% E for as little as 12 hrs will later display higher AP than E-naïve controls. Recently, we varied the amount of 10% E to either 4 or 8 ml on the day before 2-bottle choice between 10% E and water. Rats were either deprived (D) of water for 21 hrs or non-deprived (ND) at the time of E exposure. Results showed that D rats had reliably higher AP than ND rats; the latter group did not differ from E-naïve controls. D rats consumed their 4 or 8 ml of 10% E faster than did ND rats. These results suggest that significant changes in AP can be triggered by as little as 4 ml 10% E; because only D rats exhibited this change, it suggests that a critical blood E level may be necessary for this change in subsequent AP to occur. Furthermore, these results suggest that MR males might serve to illuminate gene-environment interactions for the emergence of moderate to high AP. Finally, MR males might provide a genetic substrate for examining the relationship between AP and gene expression.

Fazil Aliev^{1,2}, John Kramer³, Victor Hesselbrock⁴, Laura Bierut¹, Alison Goate¹, Jen C. Wang¹, Anthony Hinrichs¹, John Rice¹, Sarah Bertelsen¹, Sam Kuperman³, Marc Schuckit⁵, John Nurnberger, Jr.⁶, Howard Edenberg⁶, Bernice Porjesz⁷, Henri Beglieter⁷, Danielle M. Dick¹

Association of CHRM2 with IQ

¹ Washington University, St. Louis, MO,

² Ankara University, Ankara, Turkey

³ University of Iowa, Iowa City, Iowa

⁴ University of Connecticut School of Medicine, Farmington, CT

⁵ University of California at San Diego, San Diego, CA

⁶ Indiana University, Indianapolis, IN

⁷ SUNY Health Science Center at Brooklyn, Brooklyn, NY

Address: Department of Psychiatry, Box 8134 CID, 660 South Euclid Ave., Washington University in St. Louis, St. Louis, MO 63108 USA, Tel: 314-2862218, FAX: 314-2862213, aliev@matlock.wustl.edu

The cholinergic neurotransmitter system is thought to be involved in many aspects of memory, attention, and higher cognition. In the Collaborative Study on the Genetics of Alcoholism (COGA) sample, we have previously reported linkage and association to the cholinergic muscarinic 2 receptor gene (CHRM2) on chromosome 7 with evoked EEG oscillations (Jones et al., 2004 I believe this needs to be full citation), providing evidence that this gene may be involved in human brain dynamics and cognition. In addition, there is preliminary evidence of association with IQ scores based on a small number of polymorphisms genotyped in CHRM2 in Minnesotan (Comings reference) and Dutch (Gosso, van Belzen et al. 2006, in press) samples. In the COGA sample, we have extensively genotyped SNPs across the CHRM2 gene. Data on 876 individuals with IQ and genotypic data are available for genetic analyses. Using family-based association analyses, we find evidence of association with multiple SNPs across CHRM2 and Performance IQ, as measured by the WAIS-R. These results remain significant after taking into account gender, alcohol dependence and depression diagnoses in the sample.

Juko Ando¹, Chizuru Shikishima², Yutaro Sugimoto³, Ryo Takemura³, Kai Hiraishi⁴, Mitsuhiro Okada¹

Syllogism and intelligence: g (genetic factor) of g (general intelligence) revisited.

¹Faculty of Letters, Keio University, Minato-ku, Tokyo, Japan

²Department of Sociology, Graduate School of Keio University, Tokyo, Japan

³Department of Humanities, Graduate School of Keio University, Tokyo, Japan

⁴The University of Tokyo, Tokyo, Japan

Grant Support: Grant of Keio University

Address: Faculty of Letters, 2-15-45 Mita , Keio University, Minato-ku, Tokyo, 108-8345 Japan, Tel: 81 3 3453 4511, email: juko@msa.biglobe.ne.jp

Whether intelligence is regulated by a single general factor (g) or by multiple cognitive functions is one of the central issues in psychology. The present paper shows that deductive reasoning processes measured by syllogism tasks a general factor of intelligence phenotypically and genetically. We developed a set of syllogistic reasoning inventory, the BAROCO which consisted of five different formats; 1) abstract (All A are B/ All B are C=> All A are C) 2) graphical (the same logic types as “abstract” presented by Euler circles) 3) contentual (All of John’s friends are Paul’s friends./All of Paul’s friends are german). 4) belief congruent (All cats are mammals./All mammals are animals) 5) belief incongruent (All cats are Barbaras./All Barbaras are reptiles.). 166 pairs of MZ and 53 pairs of DZ (mean age=24.9) tool the BAROCO and Kyodai-Nx Intelligence test battery which consists of twelve subtests and gives two major factors – verbal and spatial. The BAROCO tasks showed a high inner consistency (Cronbach’s alpha=.96) and substantial phenotypic correlation with IQ (.61). Multivariate genetic analysis revealed that a common pathway model fits best for the BAROCO, verbal IQ and spatial IQ. Genetic contribution to a common factor was about 80%. The implication to the genetic structure of intelligence will be discussed.

Juko Ando¹, Shinji Yamagata² Yusuke Takahashi², Kouken Ozaki³, Ryoko Nakajima⁴, Kouichi Nonaka⁵, Noriko Kato⁶, Syuichi Ooki⁷

Genetic influences on development of weight, length, chest and head circumference in infancy

¹Faculty of Letters, Keio University, Minato-ku, Tokyo, Japan

²Department of Cognitive and Behavioral Science, The University of Tokyo, Tokyo, Japan

³Waseda University, Chuo-ku Tokyo, Japan

⁴Department of Sociology, Graduate School of Keio University, Tokyo, Japan

⁵Wako University, Tokyo, Japan

⁶National Institute of Public Health

⁷Ishikawa Prefectural Nursing University

Grant Support: Japan Science and Technology Agency

Address: Faculty of Letters, 2-15-45 Mita, Keio University, Minato-ku, Tokyo, 108-8345 Japan, Tel: 81 3 3453 4511, email: juko@msa.biglobe.ne.jp

One thousand pairs of infant twins entered the new longitudinal study for “Brain Science and Education”, called Tokyo Twin Cohort Project (ToTCoP). The current study reports developmental trajectory of their weight, body length (height), chest circumference, and head circumference in terms of quantitative genetic approach at birth, 3 month and 9 month of age. All four physique measures show substantial additive genetic and shared environmental effects as well as nonshared environment at birth. Most of shared environmental contribution are explained by gestation age and decrease to zero at 9 months of age. Additive genetic contributions increase drastically from 3 to 9 months of age (up to 70-80%). Multivariate analysis suggests that development of head circumference is regulated by its specific genetic factor different from other physique measures. Latent growth curve model is applied to these developmental data.

Raven L. Astrom¹, Sally J. Wadsworth¹, John C. DeFries¹

Etiology of stability of reading difficulties: Preliminary analysis of follow-up data from participants in the Colorado Learning Disabilities Research Center

¹Institute for Behavioral Genetics, University of Colorado, Boulder CO

Grant Support: Supported by NICHD Center Grant HD-27802, NIDCD Grant DC-05190

Address: Institute for Behavioral Genetics, 1480 30th Street, University of Colorado, Boulder CO 80309, USA, Tel: 303-735-6179, FAX: 303-492-8063, email: raven.astrom@colorado.edu

Results obtained from longitudinal studies indicate that reading deficits are generally stable (e.g., P. Satz, S.L. Buka, L.P. Lipsitt and L. Seidman, 1998, in B.K. Shapiro, P.J. Accardo and A.J. Capute, eds., Reading disability: A view of the spectrum. Timonium, MD: York Press). However, little is known about the etiology of this stability. The primary objective of the present study is to provide a preliminary assessment of genetic and environmental influences on the stability of reading difficulties. Data were analyzed from a sample of 50 twin pairs (16 MZ pairs and 34 DZ pairs) in which at least one member of each pair was classified as reading-disabled in the Colorado Learning Disabilities Research Center (CLDRC) and on whom follow-up data were available. The twins were tested at two time points (average age of 10.3 years at initial assessment and 16.1 years at follow-up). A composite measure of reading (PIAT Reading Recognition, Reading Comprehension and Spelling) was found to be highly stable, with a stability correlation of .81. For each time point, the data were subjected to univariate DeFries-Fulker multiple regression analysis (DeFries and Fulker, 1985, Beh. Genet. 15, 467-473). Estimates of the heritability of the group deficit (h^2_g) were .78 ($\pm .23$) at initial assessment and .79 ($\pm .42$) at follow-up. When these data were fitted to a bivariate extension of the basic DF model (J. G. Light and J.C. DeFries, 1995, J. Learn. Disabil, 28, 96-106.), bivariate heritability was estimated at .71, indicating that common genetic influences account for approximately 88% of the phenotypic correlation between reading measures at the two time points in this preliminary sample.

Eligible for Thompson Award

David E. Bard¹

Exploring a common etiology for alcohol and cigarette consumption and initiation: A behavior genetic analysis of liabilities using a nationally representative sample of kinship pairs.

¹University of Oklahoma

Address: Psychology, University of Oklahoma, Oklahoma City, Oklahoma, 73118 USA, Tel: (405) 271-5700, email: david-bard@ouhsc.edu

Univariate and multivariate behavior genetic models of alcohol and cigarette initiation and consumption were fit to responses from adolescents of the National Longitudinal Survey of Youth (NLSY). This nationally representative sample produced results consistent with prior behavior genetic research on both substances (e.g., Madden et al., 1999; Koopmans et al., 1999; Stallings et al., 1999). Univariate models revealed significant genetic effects for alcohol ($h^2 = .34$) and cigarette ($h^2 = .31$) consumption as well as cigarette initiation ($h^2 = .31$). They also revealed evidence of strong environmental effects on cigarette ($c^2 = .05$, $e^2 = .64$) and alcohol ($c^2 = .14$, $e^2 = .86$) initiation. There was no evidence of shared-environmental variance in either consumption measure but each suggested strong nonshared influences. This implies that both consumption and initiation are predominantly influenced by environmental sources of variation, but that consumption and smoking initiation are at least partially under the control of genes and biology. In the multivariate situation, extensions to the Heath et al. (2002) multivariate ordinal liability models were considered by simultaneously estimating random effect discrete-time survival analysis of age at initiation and Poisson-distributed counts of alcoholic drink and number of cigarettes. A single liability model, which was unanimously favored over an independent liabilities model, did reveal small to moderate common factors. Generally speaking, however, results supported the investigation of both substances and initiation and consumption separately, as substantial ACE unique effects were present in all MV models. Lastly, estimates of the effects from our study were consistently lower than those present in most previous designs. This is most likely attributable to the larger variety of genetic relatedness existing in our kinship, as opposed twin-only, sample.

Eligible for Thompson Award

David E. Bard¹, Joseph Lee Rodgers¹

Use of discrete-time survival analysis for modeling multivariate ACE models of fertility precursors from the children of the NLSY.

¹University of Oklahoma

Address: Psychology, University of Oklahoma, Oklahoma City, Oklahoma, 73118 USA, Tel: (405) 271-5700, email: david-bard@ouhsc.edu

Substantial evidence now exists that variables measuring or correlated with fertility outcomes have a heritable component. In this study, we define a series of age-sequenced fertility precursors and fit a multivariate ACE model to responses from the children (now adolescents and young adults) born to mothers of the original National Longitudinal Survey of Youth (NLSY) cohort. Three age-related precursors were considered: age at 1st menstruation, 1st dating experience, and 1st sexual intercourse. Univariate and multivariate models were in general agreement indicating strong heritability for each precursor, little to no shared environmental influences, and small to moderate nonshared influences. Genetic components in the MV model accounted for 47%, 71%, and 54% of the precursor variations, respectively. Methodologically, this study also explored the use of MV random effect discrete-time survival analyses of the precursor data. These models also incorporated an additional precursor (age at 1st marriage) and a fertility outcome (age at 1st childbirth). Results from these 5-variable discrete-time survival models are compared to biased effects from models that excluded censored cases.

Timothy C. Bates¹, Anne Castles², Michelle Luciano³, Margaret J. Wright³, Max Coltheart⁴,
Nicolas G. Martin³

Genetic bases of normal reading

¹ University of Edinburgh

² University of Melbourne

³ Queensland Institute of Medical Research

⁴ Macquarie University

Grant Support: NHMRC Australia

Address: Psychology, 7 George Square, University of Edinburgh, Edinburgh, Scotland, EH8 9JZ, UK, Tel: 44 131 651 1945, email: tim.bates@ed.ac.uk

A sib-pair linkage analysis for reading and spelling is presented based on reading and spelling phenotypes assessed in 403 unselected families of twins aged between 12 and 25 years. The analyses supported seven of the eleven candidate linkages identified earlier in dyslexic samples, with two more approaching replication level. Novel linkages at chromosomes 4p15 and 17p13 were also suggested. The results indicate that that normal variance in reading is controlled by genes which overlap mostly or perhaps entirely with those responsible for severe dyslexia, i.e., that reading and spelling form a genetic continuum from high normal performance through clinical levels of impairment. Linkages supported include 2q22.3, 3p12-q13 (DYX5), 6q11.2 (DYX4), 7q32, 15q21.1 (DYX1), 18p21 (DYX6), and Xq27.3 (DYX9). Weaker support was found for linkage at 1p34-36 (DYX8) and 2p15-16 (DYX3), with little evidence was found for linkages at 6p23-21.3 (DYX2) and 11p15.5 (DYX7). Two novel linkages at 4p15.33-16.1 and 17p13.3 received suggestive genome-wide support. For linkages at 2q22.3, 6q11.2, 7q32, 18p21, and Xq27, the present data represent the first independent replication.

Beben Benyamin¹, Ian J. Deary², Peter M. Visscher³

Precision and bias of a mixture distribution model to analyse twin data when zygosity is unknown: Simulations and application to IQ phenotypes on a large sample of twin pairs

¹Institute of Evolutionary Biology, University of Edinburgh, UK

²Department of Psychology, University of Edinburgh, UK

³Genetic Epidemiology, Queensland Institute of Medical Research, Australia

Address: Genetic Epidemiology, 300 Herston Road, Queensland Institute of Medical Research, Brisbane, QLD, 4029, Australia, Tel: +61 7 3362 0169, FAX: +61 7 3362 0101, email:

bebenB@qimr.edu.au

The classification of twin pairs based on zygosity into monozygotic (MZ) or dizygotic (DZ) twins is the basis of most twin analyses. When zygosity information is unavailable, a normal finite mixture distribution model can be used to estimate components of variation for continuous traits. The main assumption of this model is that the observed phenotypes on a twin pair are bivariate normally distributed. Any deviation from normality, in particular kurtosis, could produce biased estimates. Using computer simulations and analyses of a wide range of cognitive measures from the U.K. Twins' Early Development Study (TEDS), where zygosity is known, properties of the mixture distribution model were assessed. Simulation results showed that, if normality assumptions were satisfied and the sample size was large (e.g., 2,000 pairs), then the mixture distribution model was unbiased and gave a standard deviation of the difference between heritability estimates from known and unknown zygosity in the range of 0.02 to 0.20.

Unexpectedly, the estimates of heritability of 10 variables from TEDS using the mixture distribution were consistently larger than those from the conventional (known zygosity) model. This discrepancy was due to violation of the bivariate normality assumption. A leptokurtic distribution of pair difference was observed for all traits (except non verbal ability scores of MZ twins), even when the univariate distribution of the trait was close to normality. From an independent sample of Australian twins, the heritability estimates for IQ variables were larger for the mixture model in 6 out of 8 traits, consistent with the observed kurtosis of pair differences. This novel finding of widespread kurtosis of the pair difference may suggest that the usual assumptions of quantitative trait analysis in twin studies may be incorrect and need revisiting.

Eligible for Thompson Award

Rebecca S. Betjemann¹, Erik G. Willcutt², Richard K. Olson², Janice M. Keenan³, John C. DeFries¹, & Sally J. Wadsworth¹

A Preliminary Investigation of the Genetic Etiology of Reading Comprehension over Time

¹Institute for Behavioral Genetics, University of Colorado, Boulder CO

²Department of Psychology, University of Colorado, Boulder, CO

³Department of Psychology, University of Denver, Denver, CO

Grant Support: NICHD Center Grant HD-27802; NIDCD Grant DC-05190; NIDA Research Training Grant 5 T32 DA017637

Address: Institute for Behavioral Genetics, University of Colorado, Boulder, CO 80309-0447 USA, Tel: 303-725-8877, email: betjeman@colorado.edu

Although much research has been done on the genetics of word reading and its component processes (J.Gayán & R.K.Olson, 2003, *J. of Exp. Child Psych*, 84,97-123), very little has been done to investigate the genetics of reading comprehension. One recent study (J.M.Keenan, R.S.Betjemann, S.J.Wadsworth, J.C.DeFries, & R.K.Olson, 2006, *J. of Research in Reading*, 29,75-91) found significant heritability of reading comprehension ($h^2g = .51$), and a genetic correlation with word recognition of .85, but also found significant independent genetic variance for comprehension. Here, we use longitudinal data to investigate heritability of reading comprehension developmentally and determine if its relation with decoding changes over time. Analyses were conducted using data from a preliminary sample of 48 pairs of MZ twins and 75 DZ pairs, tested at two time points (mean ages 10.5 & 16). The PIAT reading comprehension measure is included from both time points, and decoding and phonological coding measures are included from time 1. Univariate results indicated heritabilities of .58 and .50 for reading comprehension at times T1 and T2, respectively. A Cholesky decomposition of these PIAT comprehension scores at T1 and T2 found that their genetic influences could be accounted for by a single genetic factor, with a genetic correlation between time points of 1.0. Further Cholesky models indicated that word recognition had a genetic correlation of .85 with T1 comprehension, and .88 with T2 comprehension. Phonological coding had genetic correlations with T1 and T2 comprehension of 1.0 and .70. Three of these models suggested additional independent genetic influences on comprehension (path coefficients .33-.49), but with this sample size, they did not reach significance. These results indicate that decoding abilities share significant genetic influences with reading comprehension even years later, but also suggest possible independent genetic influences on comprehension at both time points.

David A. Blizard¹

Sweet and bitter taste of ethanol in C57BL/6J and DBA2/J mouse strains

¹Center for Developmental and Health Genetics, The Pennsylvania State University, University Park PA 16802

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Address: Center for Dev Health Genetics, 201 Res Bldg D, Penn State University, University Park, PA 16802, USA, Tel: 814-865-3429, FAX: 814-863-4768, email: dab22@psu.edu

Studies of inbred strains of rats and mice have suggested a positive association between strain variations in sweet taste and ethanol intake. However, strain associations by themselves are insufficient to support a functional link between taste and ethanol intake. In genetically heterogeneous rats, taste aversions conditioned to sucrose and also to quinine generalize to ethanol (G.J. Lawrence and S.W. Kiefer, 1987, *Chemical Senses*, 12: 591-599). Accordingly, I used conditioned taste aversion to explore the sweet and bitter taste of ethanol and ability to detect sucrose, quinine and ethanol in C57BL/6J (B6) and DBA/2J (D2) mouse strains that are frequently used in alcohol research. Consistent with previous work, the present study showed that C57BL/6J mice generalized taste aversions from sucrose and quinine solutions to 10% ethanol and, reciprocally, aversions to 10% ethanol generalized to each of these solutions presented separately. Only quinine generalized to ethanol in the DBA/2J strain while reciprocal aversions from ethanol did not. Thus, considering these two gustatory qualities, 10% ethanol tastes both sweet and bitter to B6 mice but only bitter to D2. Ethanol detection thresholds did not differ between the strains. The strain-dependent gustatory profiles for ethanol may make an important contribution to the understanding of the undoubtedly complex mechanisms influencing high ethanol preference of B6 and pronounced ethanol avoidance of D2 mice.

David A. Blizard¹, Arimantas Lionikas¹, Jennifer E. Foreman¹, Frank Johannes¹, David J. Vandenberg¹, , George P. Vogler¹, Gerald E. McClearn¹

Transitivity of genetic architecture

¹Center for Developmental and Health Genetics, Penn State University, University Park, PA 16802

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Address: Center for Dev Health Genetics, 201 Res Bldg D, Penn State University, University Park, PA 16802, USA, Tel: 814-865-3429, FAX: 814-863-4768, email: dab22@psu.edu

Contextual genetics (see abstract by G.E. McClearn) provides a framework for the discussion of a variety of experimental findings, which illustrate the transitivity of genetic effects on diverse phenotypes. We will present results from several research projects conducted at the Center for Developmental and Health Genetics at Penn State that demonstrate such transitivity on phenotypes varying from behavioral (alcohol preference, hypnotic dose sensitivity) to physiological (systolic blood-pressure) and anatomic (muscle weight and muscle attachment anomalies). Evidence from our QTL-oriented studies suggests dependence of the genetic architecture of these phenotypes on one or more of the following attributes: age, stress, test order, etc. In some cases, the presence or absence of a QTL in test groups assayed under different conditions may simply reflect the fact that the statistical power inherent in an experimental design is inadequate, rather than intrinsic differences between test conditions. However, the recognition that many genetic effects may be context-specific introduces a new dynamic into the study of genetic architecture, which may be helpful in exploring mechanisms.

Dorret I. Boomsma¹, Gonneke Willemsen¹, Marlies de Lange¹, Stephanie van den Berg¹,
Jacqueline M. Vink¹

Variation in age at menarche and oral contraceptive use in Dutch women

¹Dept Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands)

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904-61-193, NWO/SPI 56-464-14192

Address: Biological Psychology, Vrije Universiteit, Amsterdam, NH, 1081 BT, The Netherlands,
Tel: 31-20-598 8787, email: dorret@psy.vu.nl

In 2002/3 we collected survey data on age at menarche and oral contraceptive use in women from Dutch twin families. Probandes were mono- and dizygotic twins. Their mothers, sisters, and their sisters-in-law (if the twin had a male cotwin) were also included in the data collection. Data on age at menarche were collected in 5656 women; data on oral contraceptive use in 5711 women.

Familial resemblance in age at menarche is influenced by additive and non-additive genetic factors. There is substantial familial resemblance for lifetime oral contraceptive use is mainly influenced by common environment.

Josh B. Bricker¹, Michael C. Stallings¹, Robin P. Corley¹, Brett C. Haberstick¹, Andy Smolen¹, Gary Stetler¹, Susan E. Young¹, John K. Hewitt¹, John C. DeFries¹

Association between age of sexual initiation and dopamine-encoding gene not replicated in combined twin and adoption samples

¹Institute for Behavioral Genetics, University of Colorado, Boulder CO 80309

Grant Support: HD010333, HD036773, DA011015, DA05131

Address: Institute for Behavioral Genetics, University of Colorado, Boulder, CO, 80309-0447, USA, Tel: 303 492 7362, email: jbricker@colorado.edu

Genetic influences on the age of first sexual initiation (AFSI) are suggested by heritability estimates in the range of 28% to 72% (J. B. Bricker, M. C. Stallings, R. P. Corley, S. J. Wadsworth, A. Bryan, D. S. Timberlake, J. K. Hewitt, A. Caspi, S. M. Hofer, S. A. Rhea & J. C. DeFries. Behavior Genetics, in press), and an association between early pubertal development and early AFSI (Rowe, D. C., 2002, *Evol. Hum. Beh.* 23(5), 365-372). It is also likely that individual differences in pubertal development and changes in hormonal levels affecting AFSI are genetically influenced (Halpern, C. T., Udry, J. R., Campbell, B. & Suchindran, C. (1993). *Psychosom Med* 55: 436-447; Mustanski, B. S., Viken, R. J., Kaprio, J., Pulkkinen, L., & Rose, R. J. (2004). *Devel Psychol* 40(6): 1188-1198). One previous study (Miller, W. B., Pasta, D. J., MacMurray, J., Chiu, C., Wu, H., & Comings, D. E. (1999). *J Biosoc Sci* 31: 43-54) has reported a significant association between a polymorphism of the DRD2 gene and AFSI. The present study attempted to replicate and extend these findings by performing candidate gene association analyses to examine whether functional polymorphisms of the dopamine transporter gene (DAT1) and the receptor-encoding genes (DRD2 and DRD4) are related to AFSI in a combined sample of twins and adopted and nonadopted siblings. In a sample of 2,823 individuals with genotype data, allele frequencies at each locus were in Hardy-Weinberg equilibrium. In a subset of 1523 individuals with genotype and AFSI information, a between-family association test found no association between those who initiated sex early (defined as before age 16) versus later and genotype at each locus. Among 738 individuals who reported an age of onset, there were no mean differences by genotype at any of the three loci. Results of within-family analyses, which are robust to population stratification, will also be presented.

Eligible for Thompson Award

Ulla Broms¹, Pamela A.F. Madden², Andrew C. Heath², Michele L. Pergadia², Saul Shiffman³, Jaakko Kaprio^{1,4}

The Nicotine Dependence Syndrome Scale in Finnish Smokers - Genetic Architecture of Nicotine Dependence

¹ Department of Public Health, University of Helsinki, Finland

² Washington University School of Medicine, St. Louis, USA

³ Department of Psychology, University of Pittsburgh, USA

⁴ Department of Mental Health and Alcohol Research, National Public Health Institute, Helsinki, Finland

Grant Support: Doctoral Programs of Public Health, University of Helsinki, Finland; The GenomEUtwin project (European Union Contract No. QLG2-CT-2002-01254); Data collection was supported by a NIH grant DA12854 to P.A.F. Madden

Address: Department of Public Health, P.O. Box 41, Mannerheimintie 172, University of Helsinki, 00014 Helsinki, Finland, Tel.: 358 9 19127572, FAX: 358 9 19127570, ulla.broms@helsinki.fi

The Nicotine Dependence Syndrome Scale (NDSS) is a new multidimensional measure of nicotine dependence. We examined the effect of genetic and environmental factors and sex-limitation on nicotine dependence, as measured by NDSS and compared it to FTND. In a family study of cigarette smoking, adult twin pairs concordant for smoking from the Finnish Twin Cohort Study, and their siblings and parents were interviewed for nicotine dependence. Subjects filled out a questionnaire with the NDSS scale (31 items) soon after the interview. We carried out analyses on 1370 smokers. The NDSS-T score (a summary measure of dependence) correlated highly with the FTND score ($r=0.64$). In exploratory factor analysis we derived three factors, named drive/priority, stereotypy/continuity and tolerance. The heritability of nicotine dependence was analyzed by using quantitative genetic methods based on linear structural modeling (Mx-statistical package). We identified 291 pairs of smokers with the NDSS T-score (65 MZ, 129 SSDZ and 97 OSDZ pairs). Additive genetic variance was 0.32 (95% CI 0.12, 0.48) and non-shared environmental variance 0.68 (95% CI 0.52, 0.88) for NDSS T-score. Genetic sex-limitation modeling showed no differences in the genetic architecture of NDSS T-score, second or third factors between men and women (no evidence for sex-specific genetic effects). The best model for second stereotypy/continuity ($h^2=0.44$, 95% CI 0.21, 0.61) and for third tolerance factor ($h^2=0.39$, 95% CI 0.18, 0.56) was an AE-model. For the first drive/priority factor the best fitting model for men was a CE-model (common environment was 0.22, 95% CI 0.05-0.37) and for women an E-model. In comparison, the FTND genetic model fitted an AE model ($h^2=0.40$, 95% CI 0.23, 0.55). Genetic modeling showed no differences in the genetic architecture of NDSS between men and women; the overall heritability estimate was modest (0.32), but some NDSS subscales showed higher heritabilities.

Eligible for Thompson Award

Jacob P. Buchanan¹, Matt McGue¹, Margaret Keyes¹, Irene Elkins¹, William G. Iacono¹

Characterization of shared environmental influence on adolescent behavior: Evidence from the Sibling Interaction and Behavior Study

¹Department of Psychology, University of Minnesota, Minneapolis MN 55455

Grant Support: Supported by USPHS grant #AA11886; USPHS grant #MH066140

Address: Psychology, University of Minnesota, Minneapolis, MN 55414, USA, Tel: 612-379-3317, email: bucha057@umn.edu

The Sibling Interaction and Behavior Study (SIBS) is a longitudinal study of 408 adoptive and 209 non-adoptive families, each consisting of a pair of adolescent siblings and their parents. The family members, particularly the siblings, complete a day-long intake assessment that covers a wide variety of behavioral indicators. The aim of the current study is to test for significant shared environmental effects in a selection of representative measures from SIBS. Previous research that suggests minimal shared environmental effects on psychological development has been based primarily on designs utilizing twins reared together (Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P., 2000, Behavioral Genetics (Fourth ed.), New York: Worth Publishers) or the comparison of twins reared apart to twins reared together (Bouchard, T. J. J., Lykken, D. T., McGue, M., Segal, N., & Tellegen, A., 1990, Science, 250, 223-228), which are indirect measures. Use of the SIBS data set provides instead a direct estimate of shared environmental effects by using the correlation between adopted siblings, who are reared together, though not genetically related. The magnitude of any shared environmental effects in psychological measures is likely to vary with the behavioral domain of interest. Consequently, the current study investigates a sample of indicators from each of four key domains of adolescent functioning: Problem Behavior (including substance use and delinquency), Personality, Mental Health (including internalizing and externalizing psychopathology), and Academics (including IQ and school achievement). We also assess whether the sibling correlations are moderated by sibling differences in gender, age, and ethnicity, which may indicate the relative influence of parental and sibling mechanisms in determining shared environmental effects.

Eligible for Thompson Award

Ross Buck¹, Benson Ginsburg¹

Communicative Genes, Units of Selection

¹University of Connecticut

Address: Communication Sciences, University of Connecticut, Storrs, CT, 06269-1085, USA,
Tel.: 860-486-4494, email: ross.buck@uconn.edu

We consider how the evolution of communication has been viewed from a gene-centric point of view, and particularly Krebs and Dawkins' (1984) view of communication as manipulation and mind-reading. We suggest that the atomistic view of genes presupposed by this view is restrictive and erroneous. Rather, all systems of communicating elements--including genes--inherently involve relational phenomena beginning at the level of the dyad. We argue that dyad-level communicative relationships involving genes are active, germ-line replicators that persist across evolutionary time, and that they are aspects of the genotype that influence the phenotype: the communication observed between genes. Dyadically-related genes can be in the same cell, in different cells, or in different organisms. Dyad-level communicative relationships are measurable via research designs assessing communication between given elements in a system relative to their communication *vis-à-vis* other elements (i.e., round-robin designs). We consider how genes, which are communicative systems in themselves, function within communicative systems inside cells, between cells inside organisms, and between organisms. We then define and distinguish three levels of communication--spontaneous communication, voluntary expression initiation, and voluntary expression formation--and outline brain mechanisms associated with each. Finally, we discuss implications for the understanding of empathy, rapport, intuition, charisma, and altruism. We present a general perspective on “selfish”; or individualistic and “cooperative”; or prosocial genetic influences on behavior, suggesting that communicative genes underlie the evolution of biocomplexity, and that kin selection and reciprocity are mechanisms for restricting prosocial impulses to kin and allies.

S. Alexandra Burt¹

Gene-environment correlations in antisocial behavior: Peer selection and friendship

¹Department of Psychology, Michigan State University, East Lansing MI

Grant Support: Supported in part by a grant from the Intramural Grants Program, Michigan State University, #04-IRGP-232

Address: 105A Psychology Bldg., Michigan State University, East Lansing, MI, 48824 USA,
Tel: 517-432-5602, email: burts@msu.edu

To date, the empirical evidence (R. Plomin, J.C. DeFries, & Loehlin, 1977, *Psychological Bulletin*, 84, 309-322) supporting the presence of active and evocative gene-environment correlations (rGE) during peer selection has been largely circumstantial. A recent study of 115 men (S.A. Burt, submitted for publication) sought to more explicitly examine rGE within peer selection. Participants completed behavioral measures, gave DNA, and then interacted in small groups. They then provided individual rankings of the other group members. Social Relations Modeling (SRM) was used to analyze the data. SRM is a two-way random effects model that partitions the variance in sociometric rankings into actor effects (i.e., the general tendency to like others) and partner effects (i.e., the general tendency to be liked by others). Analyses revealed that men higher in impulsivity and rule-breaking behaviors were more popular/better liked by others, independently of how much they tended to like others. Furthermore, a gene linked to rule-breaking behaviors (i.e., 5HT2A -G1438A) was also linked to these partner effects. In contrast, actor effects were not linked to participants' behavior or genes of risk. Together, such findings suggest that evocative rGE processes are particularly salient to initial peer selection while active processes are less important. However, it remains unclear whether and how the processes observed during initial encounters map onto actual friendships. The current study sought to do just this. We are thus collecting additional data in which participants first complete the above experiment and then recruit their three closest friends to provide DNA and complete personality and psychopathology measures. We will use these data to evaluate whether participants are similar, both phenotypically and genetically, to their friends, and whether these associations vary with participants' sociometric status.

Tanya M. M. Button¹, Soo Hyun Rhee¹, Susan E. Young¹, Robin P. Corley¹, Michael C. Stallings¹, John K. Hewitt¹

Elucidating the relationship between affiliation with delinquent peers and polysubstance dependence vulnerability

¹ Institute for Behavioral Genetics, University of Colorado, Boulder, Colorado, United States of America

Grant Support: DA011015; HD010333; MH43899

Address: Tanya Maria May Button, Institute for Behavioral Genetics, UCB 447, University of Colorado, Boulder, Colorado, 80309-0447, USA, Tel: 303-735-3152, FAX: 303-492-8063, email: Tanya.Button@Colorado.edu

There is evidence that affiliation with delinquent peers is associated with a number of externalizing outcomes in adolescents, particularly own peer delinquency (E. Simonoff, J. Elander, J. Holmshaw, A. Pickles, R. Murray, and M. Rutter, 2004, *Br. J. Psychiatry*, 184, 118-127), and substance use problems (R.H. Aseltine, Jr., 1995, *J of Health and Social Behav.* 36, 103-121; D.M. Fergusson, N.R. Swain-Campbell, & L.J. Horwood, 2002, *J. Ab. Child Psychol.* 30, 419-430). However, it is difficult to determine the nature of this relationship. Peers may influence one another's behavior, or people may select delinquent peers because of some underlying genetic predisposition that is correlated with their own behavior. Moreover, the true nature of the association likely arises from a combination of the two. The current study investigates the extent to which affiliation with delinquent peers is associated with polysubstance dependence vulnerability, and the extent to which the association between the two results from a common genetic propensity for both. In a sample of 1209 adolescent twin pairs (587 MZ and 622 DZ) we used a Cholesky Decomposition Model to partition the variance and covariance of polysubstance dependence vulnerability and affiliation with delinquent peers to determine the extent to which genetic and environmental influences contributed to their covariation. Results indicate that genes shared and non-shared environmental factors all make important contributions to the association between delinquent peer affiliations and substance dependence vulnerability in adolescents.

Andrew Canastar¹, Stephen C. Maxson²

Intersexual Aggression: Effect OF Male Strain, Intermale Aggressive Experience, and Estrous Cycling

¹Developmental Psychobiology Research Group, Department of Psychiatry, University of Colorado at Denver and Health Sciences Center, Aurora, CO

²Biobehavioral Sciences Graduate Program, Department of Psychology, University of Connecticut, Storrs, CT

Grant Support: Inbred Mouse Fund; Research Foundation of The University of Connecticut

Address: Psychiatry, RC-1N, P18-8101, M/S 8344, 12800 E. 19th Ave., U. Colorado Denver Health Science Center, Aurora CO 80010, USA, Tel:(303) 724-4404, FAX: (303) 724-4425, email: andrew.canastar@uchsc.edu

Data from human literature has identified individual differences in the expression of both intermale and intersexual aggression. Males that engage in both classes of aggression have been labeled 'panviolent'. Nonhuman animal studies have extended this data by demonstrating genetic and experiential influences on the development and expression of individual differences in panviolence. If these factors strongly shared a common neural substrate then prior aggressive experience and strain-associated aggressiveness would have similar main and interactive effects on aggressiveness toward cycling females. The results supported subtle interactive effects of prior intermale aggressive experience on aggressive and mating behavior toward cycling females. Previously reported strain differences on intersexual aggression were detected here as main effects of strain on aggressive and mating behavior toward cycling females. As with a previous study, female cycling influenced both aggressive and mating behavior toward cycling females, but showed the opposite pattern. Overall, the data supports independent effects of strain and prior aggressive experience on intersexual aggression. For mating behavior, prior aggressive experience improved performance on some behaviors in a strain-specific manner.

Sonia Chawla¹, Kimberly J. Saudino¹

Genetic and Environmental Influences on Tester-Rated Verbal and Non-Verbal Cognitive Ability in Two-Year Olds

¹ Boston University

Grant Support: NIMH Grant MH062375

Address: Psychology, Boston University, Boston, MA 02215 USA, Tel: 617-353-1102, FAX: 617-353-6922, email: sonia@bu.edu

Parent reports have shown modest bivariate heritabilities, large influences of shared environment, and small effects of unique environment on the overlap between verbal and non-verbal cognition at 24 months (Price, Eley, Dale, Stevenson, Saudino, & Plomin, 2000, *Child Dev.*, 71, 948-959). However, parent reports may not be the most ideal way to measure cognitive development in twins. Having the same person rate both twins on each measure may artificially inflate cross-twin similarity and the covariance between measures. Consequently, it is necessary to examine the genetic and environmental overlap in non-verbal and verbal cognition using tester ratings in which different raters test co-twins. The current study uses 24-month-old twins from the Boston University Twin Project, an ongoing study of activity level and related behaviors in 2- and 3-year-old twins. Verbal and non-verbal cognition were assessed using the language and cognitive facets on the Bayley Scales of Infant Development-II Mental Development Index (Bayley, N., 1993, *Bayley Scales of Infant Development (2nd Ed)*. San Antonio: The Psychological Corporation). Consistent with previous studies, both verbal and non-verbal cognitive development showed significant heritabilities and shared and non-shared environmentalities. Our data also estimate high genetic ($r_g=.77$) and shared environment correlations ($r_c=1.00$), and a non-shared environment correlation of .38 between verbal and non-verbal cognition. However, unique genetic factors still account for 41% of the variance in non-verbal cognition, though this was not found to be statistically significant. The bivariate heritability was .49, an estimate much higher than what has been observed with parent ratings, and the bivariate shared-environment estimate was much lower at .35. Consequently, when different raters test co-twins within pairs, the genetic influence on the phenotypic correlation is much higher and the influence of shared environment decreases.

Eligible for Thompson Award

Jeff Davis¹

Genetic and Social Mechanisms in Poverty Traps

¹Department of Sociology, California State University, Long Beach

Address: Sociology, 1250 Bellflower Blvd., California State University, Long Beach, Long Beach, CA, 90840 USA, Tel: (562) 985-4601, email: jdavis@csulb.edu

Persistent poverty shows two characteristics which are beyond the explanatory capacity of current social science theories. One is the substantial variation in socioeconomic behaviors among the poor. The other is behavioral epidemics associated with poverty such as early pregnancy and violence. In this paper, I develop a theoretical model of persistent poverty based upon research on the genetics of life history traits. I develop several hypotheses of the relationships between poverty and life history.

Geert DeVries¹

Development and Function of Sex Differences in Vasopressin / Vasotocin Innervation

¹Department of Psychology, University of Massachusetts, Amherst, MA 01003

Address: Department of Psychology, University of Massachusetts, Amherst, MA 01003-7720,
email: gjd@cns.umass.edu

Vasopressin (AVP) neurons in the bed nucleus of the stria terminalis and amygdala and vasotocin (AVT) neurons in homologous areas in non-mammalian vertebrates show some of the most consistently found sex differences, with males having more cells and denser projections than females. Comparative research has made this one of the best understood sex differences in terms of development and behavioral significance. Converging evidence suggests that this difference is based on a phenotypic decision made early in development. Differential rates of cell birth cannot be a factor, as AVP cells are born before the gonads start secreting steroids. Differential rates of cell death are also unlikely as the difference persists in mice with null mutations in the *Bax* gene. This mutation thwarts neuronal cell death, thereby eliminating neural sex differences that depend on differential rates of cell death. Differentiation of phenotype offers the best explanation. The sexually dimorphic AVP cells form part of a larger set of galanin expressing neurons that do themselves not differ in number. In rats, higher levels of testosterone during development in males appears to entice more neurons to co-express AVP. Despite the similarities, what triggers sexual differentiation of AVP/AVT systems varies dramatically across vertebrates. For example, estradiol, a testosterone metabolite, masculinizes this system in rats, but feminizes it in Japanese quails. In addition, sex chromosomes influence the differentiation of this system independently of gonadal hormones, a mechanisms that must be different in species where sex determination does not depend on sex chromosomes. Apparently, nature consistently finds a way of maintaining the difference, suggesting that its function is important enough to conserve it among vertebrates. The case will be made that this sex difference causes as well as prevents differences in behavior.

Danielle M Dick¹, Jen C. Wang¹, Dan Kaplan¹, Jevon Plunkett¹, Sarah Bertelsen¹, Elianna L. Goldstein¹, John P. Budde¹, Anthony Hinrichs¹, Laura Jean Bierut¹, Alison Goate¹, Howard J. Edenberg², John Nurnberger, Jr.², Victor Hesselbrock³, Sam Kuperman⁴, Marc Schuckit⁵, Jay Tischfield⁶, Bernice Porjesz⁷, Henri Begleiter⁷, Richard Rose⁸, Emma Nyman⁹, Anu-Maria Loukola⁹, Leena Peltonen^{9,10}, Jaakko Kaprio^{9,10}

The Role of Dopamine Receptor Genes in Alcohol-Related Phenotypes

¹Washington University, St. Louis

²Indiana University School of Medicine, Indianapolis, Indiana

³University of Connecticut

⁴University of Iowa

⁵University of California, San Diego

⁶Rutgers University

⁷SUNY Health Science Center

⁸Psychology Dept., Indiana University, Bloomington, IN

⁹National Public Health Institute, Finland

¹⁰University of Helsinki, Finland

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Address: Psychiatry & Psychology, Washington University, St. Louis, St. Louis, MO 63110 USA, Tel: 314-286-2297, FAX: 314-286-2213, email: dickd@wustl.edu

The dopamine receptor genes are considered likely candidates for involvement in alcohol-related phenotypes based on the role of dopamine in reward behavior. The dopamine D2 receptor gene DRD2 has been studied most extensively in relation to alcohol dependence, with mixed evidence for its involvement. Several of the other dopamine receptor genes (e.g., DRD3 and DRD4) have been associated with behavioral phenotypes related to alcohol use, such as impulsivity and novelty-seeking. In addition, it has been suggested that DRD2 may be involved more broadly in a variety of disorders characterized by impulsive, addictive, or compulsive behavior, and that inconsistency in the literature may be related to phenotypic definition. Interestingly, the genetic polymorphism that has been most widely analyzed in the DRD2 literature, TaqIA, has recently been discovered to reside in a neighboring kinase gene, ankyrin repeat and kinase domain containing 1 (ANKK1), located 10 kb downstream from the DRD2 gene. To more carefully analyze the evidence for association across this region, the Collaborative Study on the Genetics of Alcoholism (COGA) group genotyped SNPs across the DRD2 and ANKK1 genes and conducted family-based tests of association with alcohol dependence and related phenotypes. We find evidence for association with multiple SNPs (but not Taq1A) in ANKK1 with alcohol dependence, but no evidence of association with SNPs in DRD2. In addition, as part of the longitudinal FinnTwin16 study, we have genotyped SNPs in each of the dopamine receptor genes and tested for association with drinking patterns and related phenotypes at ages 16, 17, 18.5, and in the mid 20s. We also find no evidence of association between DRD2 and drinking patterns in the FinnTwin16 sample, though we find preliminary evidence of association with DRD4. This presentation will explore the role of the dopamine receptor genes in alcohol use and related behavioral phenotypes using data from these two samples.

Lisabeth F. DiLalla¹, Paula Y. Mullineaux¹

Genetic Influences Are Stronger for Aggressive Than Prosocial Behaviors in Preschool Twins

¹Southern Illinois University School of Medicine

Grant Support: SIU ORDA grant, SIU School of Medicine grant

Address: Family and Community Medicine, Mail Code 6503, Southern Illinois University School of Medicine, Carbondale, IL 62901 USA, Tel: 6184531855, email: ldilalla@siu.edu

A number of studies over the past decade have demonstrated genetic influence on childhood aggression, yet information about aggressive behaviors in very young preschoolers is sparse, and little is known about genetic influences on early prosocial behaviors. Early temperament has been shown to relate to externalizing problems, but it is not clear whether this is related to early prosocial behaviors. Thus, this study examined genetic and temperamental influences on aggressive and prosocial behaviors in preschoolers. Twins from the Southern Illinois Twins and Siblings Study (L. F. DiLalla, 2002, *Twin Research*, 5, 468-471) were rated during a peer play paradigm where one twin and one same-age, same-sex unfamiliar peer played freely for 20 minutes. Parents also rated the children on a measure of temperament. Results showed that aggressive behaviors show evidence of significant genetic influence ($h^2 = .67$), but prosocial behaviors do not appear to be genetically mediated ($h^2 = .00$). Aggressive behaviors during the peer play were correlated with parent ratings of Intensity and Activity, and prosocial behaviors were correlated with parent ratings of Approachability. Approachability and Activity showed evidence of significant heritable influence ($h^2 =$ approximately $.80$), whereas Intensity showed weaker genetic influence ($h^2 = .22$). Thus, aggression appears to have genetic influence even as early as 5 years of age, but prosocial behaviors do not. Although temperament is related to prosocial behaviors, there must be stronger environmental influences that account for the majority of the variance in prosocial behaviors at this young age.

Brian M. D’Onofrio¹, Wendy S. Slutske², Eric Turkheimer³, Robert E. Emery³, K. Paige Harden³, Andrew C. Heath⁴, Pamela A.F. Madden⁴, Nicholas G. Martin⁵

The Intergenerational Transmission of Childhood Conduct Problems: A Children of Twins Study

¹Department of Psychological and Brain Sciences, Indiana University

²Department of Psychological Sciences, University of Missouri

³Psychology Department, University of Virginia

⁴School of Medicine, Washington University

⁵Queensland Institute of Medical Research, Queensland, Australia

Grant Support: NIH grants AA07535 and AA000264; William T. Grant Foundation; National Alliance for Research on Schizophrenia and Depression

Address: Dept. of Psychological and Brain Science, 1101 E. 10th St., Indiana University, Bloomington, IN 47405 USA, Tel: 812-856-0843, email: bmdonofr@indiana.edu

The familial nature of conduct problems (CPs) has been well documented, and numerous behavior genetic studies have shown that genetic factors influence these behaviors. However, few genetically informed studies have explored the processes through which parental CPs influence offspring CPs. Parental CPs may have a direct causal influence on offspring CPs, but parents and offspring may also share common genetic or environmental factors that increase the liability in both generations. The current project utilized the Children of Twins (CoT) Design to delineate the genetic and environmental processes responsible for the intergenerational transmission of childhood CPs. The research used a high-risk sample of twins and their young adult offspring from the Australian Twin Registry, but the analyses were weighted to produce population-based parameter estimates. The magnitude of the intergenerational association was significant for all offspring, although stronger for male offspring. Genetically informed analyses indicated that the intergenerational transmission of CPs was not due to causal processes for female offspring; a common genetic liability accounted for the intergenerational relations. In contrast, the intergenerational transmission of CPs for male offspring was mediated by environmental variables specifically related to parental CD. The results cannot be accounted for by assortative mating, measures of psychopathology in both parents, or by greater levels of contact in identical versus fraternal twin families.

C.M. Drazinic¹, B.A. Pletcher², H. Zheng¹, M.W. State^{1,3}

Breakpoint Mapping Using Microarrays and FISH in an 18p Monosomy Case with Psychosis, Leukodystrophy, and Dysmorphology

¹Child Study Center, Yale University, New Haven, CT

²Center for Human and Molecular Genetics, UMDNJ-New Jersey Medical School, Newark, NJ

³Department of Genetics, Yale University, New Haven, CT

Grant Support: NARSAD (National Alliance for Research On Schizophrenia and Depression)

Address: Psychiatry, Genetics, 263 Farmington Avenue, Univ. Connecticut Health Center, Farmington, CT 06030-2103, USA, Tel: 860-679-3316, FAX: 860-679-407, email: drazinicc@psychiatry.uhc.edu

Background: Microarrays provide an efficient way to map breakpoints in patients with chromosomal copy number abnormalities. In a recent report, microarrays were used to map 18p11.23 monosomy and 5p14.1 trisomy breakpoints in a female with dysmorphic features, short stature, mental retardation, leukodystrophy, and psychosis (Drazinic et al., 2005). Here we describe a second female with isolated 18p monosomy, who presents with the same characteristics.

Methods: The patient's genomic DNA was analyzed using a GeneChip Human 50K XbaI 240 Array (Affymetrix), consisting of 58,960 single nucleotide polymorphism (SNP) probes with an average spacing of 46.5 kilobases. Fluorescence in situ hybridization (FISH) confirmed the deletion, using probes from bacterial artificial chromosomes (BAC's) of an RP-11 library.

Results: Based on the 50K SNP array and confirmatory FISH studies, the entire p arm of one chromosome 18 was deleted up to the centromeric band 18p11.1, beginning at 15.4 Mb (May 2004 freeze; <http://genome.ucsc.edu>). FLAIR MRI brain imaging in this patient revealed multiple, diffuse, nonenhancing white matter lesions.

Conclusions: While microarrays can be used to rapidly approximate the breakpoints in patients with unbalanced chromosomal abnormalities, a second method such as FISH must be used to confirm the data. Although case reports and linkage analyses have implicated 18p in patients with psychosis, additional studies are needed to determine the relative contributions of 18p candidate genes to the psychosis phenotype. Nonprogressive leukodystrophy may be a new feature of the 18p monosomy syndrome, but its relationship to the psychosis phenotype in these two cases is unclear.

Susan K. Fenstermacher¹, Kimberly J. Saudino¹

Multivariate heritability for imitation, cognitive ability and task orientation at age two

¹ Boston University

Grant Support: Supported by NIMH Grant MH062375

Address: Psychology, 64 Cummington Street, Boston University, Boston, MA 02215 USA, Tel: (617) 358-2922, email: skf73@bu.edu

Imitative performance has been shown to be related to both cognitive ability and attentional variables. However, it is not known to what extent this relation is due to overlapping genetic and environmental factors. Elicited imitation, cognitive ability, and task orientation were obtained from a sample of 205 twin pairs (MZ = 91, DZ = 114). Elicited imitation was assessed using three multi-step imitation tasks (R. Barr and H. Hayne, 1999, *Child Development* 70, 1067-1081; M. Carpenter, J. Call, and M. Tomasello, 2002, *Child Development* 73, 1431-1441) derived from prior research. Cognitive ability was measured using via a Mental Development Index (MDI) score from the Bayley Scales of Infant Development (N. Bayley, 1993, *Bayley Scales of Infant Development*, 2nd Ed; San Antonio, TX: The Psychological Corporation) Task orientation, an observer-rated temperament variable assessing attention, was derived from the Bayley Infant Behavior Record. Each of these measures has demonstrated moderate to high heritability in prior analyses. Though data collection is ongoing, analyses of the currently available data found significant intercorrelations between overall imitation scores and both cognitive performance ($r = .35, p < .01$) and task orientation ($r = -.37, p < .01$). Subsequent Cholesky decomposition of the total covariance matrix found significant genetic covariance between MDI and imitation, but not between imitation and task orientation. Significant shared environmental covariance was found between both imitation and MDI and imitation and task orientation, while some nonshared environmental covariance was found between task orientation and imitation. Thus it appears that the relationship between imitation and task orientation is due solely to overlapping environmental factors. Although not significant, approximately 50% of the genetic variance contributing to imitative performance was found to be unique from genetic effects on general cognitive ability.

Eligible for Thompson Award

Angela Friend¹, John C. DeFries², Sally J. Wadsworth², Richard K. Olson¹

Developmental differences in the genetic etiology of reading and spelling disabilities

¹Department of Psychology, University of Colorado, Boulder, CO

²Institute for Behavioral Genetics, University of Colorado, Boulder CO

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Address: Psychology, Department of Psychology, Campus Box 345, University of Colorado at Boulder, University of Colorado, Boulder, CO 80309, USA, Tel: 303 735-6595, FAX: 303 735-6595, email: angela.friend@colorado.edu

Previous twin studies have suggested a possible developmental dissociation between genetic influences on word reading and spelling deficits as a function of age, wherein genetic influence declined across age for word recognition, and increased for spelling recognition (DeFries, J. C., Alarcon, M., & Olson, R. K., 1997, in C. Hulme & M. Snowling (Eds.), *Dyslexia: Biology, cognition, and intervention*, London, England, and Wadsworth, S. J., Gillis, J.J., DeFries, J. C. & Fulker, D.W., 1989, *Irish Journal of Psychology*, 10, 509-520). We followed up these earlier results by fitting a DeFries-Fulker (DF) regression model to several new measures of word recognition, spelling recognition, and spelling production in a larger sample of twins from the Colorado Learning Disabilities Research Center. The younger group included pairs aged 8.1-11.5 years and the older group included pairs aged 11.5 – 20.2 years, with probands being selected for reading and/or spelling deficits 1.5 SD below the mean for a normal-range control-twin sample. An extension of the basic DF model was used to test the significance of age-group differences in genetic influences on group deficits in reading and spelling. In addition, we fit an individual-differences model in Mx to probands and controls, constraining the genetic factor loadings to be equal across age groups for the reading and spelling measures to test if there are comparable age differences for group deficits and for individual differences across the normal range.

Eligible for Thompson Award: Yes

Jeffrey R. Gagne¹, Kimberly J. Saudino¹

A multivariate twin analysis of inhibitory control and behavior problems at 24 months of age

¹Boston University

Grant Support: National Research Service Award (NIMH #1 F31 MH076353-01) awarded to the first author. Elizabeth Munsterberg Koppitz Child Psychology Graduate Fellowship from the American Psychological Foundation (American Psychological Association) awarded to the first author. NIH Grant MH-062375 awarded to the second author.

Address: Department of Psychology, 64 Cummington Street, Boston University, Boston, MA 02215, USA, Tel.: 617-358-1364, FAX: 617-353-3699, gagnej@bu.edu

Inhibitory control (IC) is an individual differences variable involving the self-regulation of responses to excitatory stimuli under some form of instruction or expectation. In middle childhood, low levels of IC are associated with higher levels of non-clinical behavior problems. However, this association has not been examined in early childhood. Are children who are unable to control and inhibit their behavior also exhibiting higher levels of problem behavior in toddlerhood? What factors contribute to individual differences in IC and behavior problems? This study explores associations between IC and behavior problems at 24 months of age, as well as genetic and environmental influences on both.

Participants included 100 MZ and 121 DZ twin pairs at 24 months of age. IC was assessed using the Toddler Behavior Assessment Questionnaire (TBAQ; H. H. Goldsmith, 1996, *Child Development*, 67, 218-235), and behavior problems were assessed with the Child Behavior Checklist for Ages 1 1/2-5 (CBCL; Achenbach, T. M., & Rescorla, L. A., 2000, Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families).

Correlations between parent-rated IC, externalizing behavior problems, and ADHD were significant at 24 months of age. MZ correlations for the three variables exceed DZ correlations, suggesting the presence of genetic influences for all behaviors. A multivariate analysis was conducted using a Cholesky decomposition model. Results indicate that genetic variation accounts for the majority of the variance in parent-rated IC (49%), externalizing behavior problems (42%), and ADHD (45%). Shared environmental influences explain between 25 and 40% of the variance in the three behaviors, and nonshared environment accounted for the remaining variance. Genetic correlations between IC, externalizing problems, and ADHD ranged from .50-.78, suggesting that genetic influences on the three behaviors overlap. Implications will be discussed.

Eligible for Thompson Award

Michael J Galsworthy¹, Anton Rau¹, Frieder Neuhausser-Wespy¹, Caroline Blanchard², Robert Blanchard², Hans-Peter Lipp²

Monitoring activity, aggression and stress in group-housed mice without experimenter interference or coding

¹ Div. Neuroanat. & Behav., Dept. Anatomy, Uni. Zurich, Zurich, Switzerland

² Dept. Psychology, Uni. Hawaii, Honolulu, Hawaii, USA

Grant Support: This work was supported by a NARSAD (USA) Young Investigator Award to Mike Galsworthy and by the NCCR (Swiss) Neural Plasticity and Repair.

Address: Department of Anatomy, University of Zurich, Zurich, Zurich, CH-8057, Switzerland,
Tel: 0041 1 635 5359, email: mike_galsworthy@yahoo.co.uk

By utilizing the combined technologies of micro-transponders and in-cage antennae to continually monitor individual mice, we have developed real-time recording systems to assess aspects of dominance and chronic stress and depression in group-housed mice. We employ two arenas; the Intellicage (IC) and an automated version of the Visible Burrow System (aVBS) in order to explore differing aspects of social hierarchies and their consequences. The IC involves monitoring water/liquid access and presenting cognitive tests, whereas the aVBS is a system of tubes and cages which can record aggressive chasings or amicable pairings within the arena. Data from pilot studies show clear hierarchies in groups of seven males (outbred from C57Bl/6, DBA/2, C3H and NZB; all Jackson). Weight drops, bite-marks on tails and lower activity are all significantly associated by Spearman's correlations, indicating clusters of symptoms of stress in some individuals. We also demonstrate the ability of the recording system to identify incidences of chasing and other such computed measures, allowing the complex social interactions of individuals to be recorded over long periods without recourse to experimenter effort or error.

Jody M. Ganiban¹

Accounting for genetic contributions to links between parent negativity and child outcomes

¹George Washington University

Grant Support: Supported by NIMH grant 5R01MH43373; NIMH grant 5R01MH48825

Address: Psychology, George Washington University, Washington, DC 20052 USA, Tel: 202 994-7571, FAX: 202 994-1602, ganiban@gwu.edu

Previous reports from the Nonshared Adolescent Development Project (NEAD) detected genetic contributions to associations between parental negativity and children's internalizing and externalizing behavior across adolescence (D. Reiss, J.M. Neiderhiser, E.M Hetherington, and R. Plomin (2000). *The Relationship Code*, Harvard University Press, Cambridge MA). The current study explored whether children's negative emotionality could account for these genetic effects.

NEAD included 2-parent nondivorced and step families with 2 adolescent children (average age difference = 1.6 yrs; child age range 10-18 years). Time 1 included 720 families with 5 sibling types: monozygotic (MZ; N=93) and dizygotic (DZ; N= 99) twins, and full siblings from nondivorced families (FI; N=95); full (FS; N=182), half (HS; N=109), and genetically unrelated (US; N=130) siblings from stepfamilies. At time 2, NEAD consisted of 395 families: 63 MZ, 75 DZ, 58 FI, 95 FS, 60 HS, and 44 US pairs. At both times mothers and fathers completed parenting surveys and temperament ratings for each child.

Parent negativity was associated with child internalizing and externalizing behaviors at times 1 and 2 (r 's = .39 - .63). Genetic influences accounted for 50% to 60% of the covariance between parenting and child behavior (range for mothers = 34% to 63%; range for fathers = 44% to 83%). At time 1 children's negative emotionality accounted for all of the genetic covariance between parenting and internalizing behavior for mothers and fathers; but at time 2 negative emotionality accounted for half of the genetic covariance. At both times, negative emotionality accounted for about half of the genetic covariance between parenting and externalizing behavior. These findings support an evocative developmental model in which children's genetically influenced characteristics affect the parenting they receive and their outcomes.

Aude Gérard-Desplanches¹, Silvia Stefanini², Gene Fisch³, Stefano Vicari⁴, Virginia Volterra⁵, Christine Deruelle⁶, Michèle Carlier⁷

Laterality in persons with genetic disorders and intellectual impairment: Is manual inconsistency linked to cross hand-foot preference?

¹Centre de Recherche Psych (EA327), University of Provence, Aix en Provence, France

²Department of Neuroscience, University of Parma, Parma, Italy

³Yeshiva University, and CUNY/Lehman College, New York, USA

⁴Institute of Research Children Hospital Bambino Gesù, Santa Marinella, and LUMSA University, Rome, Italy

⁵Institute of Cognitive Science and Technologies, National Research Council (CNR), Rome, Italy

⁶Institut de Neurosciences Cognitives de la Méditerranée, CNRS, Marseille, France

⁷LPC, UMR 6146 CNRS University of Provence, and University Institute of France, France

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Department of Development and Difference, 29 Avenue Robert Schuman, University of Provence, Aix en Provence Cedex 1, FRANCE, 13621, Tel: 04 42 93 39 99, FAX: ,04 42 38 91 70, email: aude.gerard2@wanadoo.fr

Carlier and al., (Behavior Genetics, in press), assessed manual laterality in 45 persons with trisomy 21 (T21) and 34 persons with Williams-Beuren syndrome (WBS). They were compared to typically developing children (TD). Lateral preferences (hand, foot) was observed with a 18 item task administered two times. More left-handers were found in the group with T21 compared to the other two groups. Inconsistent laterality (hand switch from one trial to another for the same item) was higher in the two groups with genetic diseases than in the TD group. In a recent paper (Gérard-Desplanches and al., submitted), these data were completed. Our sample was comprised of all the participants included in the preceding study plus 152 participants. In all, the groups included 62 persons with T21, 39 with WBS and 184 TD. Two independent age sub-groups were formed: 7 to 10 years old and 11 to 34 years old. No significant differences were observed between the age sub-groups. Differences in IQ levels did not correlate with differences in laterality scores. Manual and foot inconsistencies characterized both genetic groups. Cross hand-foot preference inconsistency (i.e. a preference for one hand and the opposite foot, measured at the first trial) was lower in the TD group than in the other two groups. Is manual inconsistency linked to cross hand-foot preference? Yes in the group with WBS participants which switched their hand from one trial to another at least one time exhibited more frequently cross hand-foot inconsistency. We conclude that atypical laterality is not a unique pattern as frequently described in the literature.

Eligible for Thompson Award

Nathan A Gillespie¹, Michael C Neale¹, Carol A Prescott², Kristen Jacobson³, Kenneth S Kendler¹

Is risk for marijuana initiation, abuse and dependence mediated by individual differences in substance availability and peer group deviancy?

¹Virginia Commonwealth University

²University of Southern California

³University of Chicago

Grant Support: NHMRC Sidney Sax Postdoctoral Fellowship; DA-11287 (NIH); MH/AA/DA-49492 (NIH); MH-01458 (NIH); AA-00236 (NIH)

Address: Department of Psychiatry, 800 East Leigh Street, Biotech 1, Suite 101, VIPBG, Richmond, VA 23219 USA, Tel.: 804 502 1662, email: ngillespie@vcu.edu

Individual differences in drug availability and peer group deviancy have been proposed as mechanisms leading to marijuana initiation, abuse and dependence. We investigated the genetic and environmental sources of variation in marijuana availability and peer group deviancy, and then examined the extent to which genetic and environmental variations in risk for marijuana initiation and diagnoses of abuse and dependence are mediated by individual differences in availability and peer group deviancy. In a study of adult male twins aged 24 to 62 yrs from the Virginia Twin Registry, five retrospective assessments (8-11, 12-14, 15-17, 18-21, and 22-25 yrs) of marijuana availability and peer group deviancy were collected, as well as lifetime DSM-IV diagnoses of marijuana abuse and/or dependence from 1796 adult twins. Between 8 and 25 yrs, most of the variation in familial aggregation for peer group deviancy was best explained by additive genetic effects (28%-45%). However, between 15 and 17 yrs, shared environment explained 16% of the variance, while the remaining variance was attributable to unique environmental effects. For marijuana availability, shared environmental effects peaked between 12 and 17 yrs, but then began to decline in favour of additive genetic variance. Across the five time points, the correlation between drug availability and peer group deviancy increased (0.39 to 0.61). Correlations between marijuana initiation and drug availability were modest (0.26 to 0.29). Between 8 and 17 yrs, correlations between marijuana initiation and peer group deviancy increased from 0.17 to 0.39, and were largest between 18 and 25 yrs (0.40 to 0.45). In addition to decomposing the sources of covariance between these putative predictors, we will determine using logistical regression and structural equation modelling how much of the liability in marijuana initiation, abuse and dependence is mediated by drug availability and self reports of deviant peer groups.

Elena D. Gindina¹, Sergei B. Malykh¹, Marina M. Lobaskova²

Genetic influence on parent-reported competence and behavior/emotional problems in Russian adolescent twin sample

¹Laboratory of Developmental Behavior Genetics, Psychological Institute of Russian Academy of Education, Moscow, Russian Federation

²Department of Psychology, Udmurt State University, Izhevsk, Russian Federation

Address: Developmental behavior genetics, Mokhovaya street, 9 V, Psychological Institute of RAE, Moscow, 125009, Russia, Tel.: +7(495)7687658, email: elena@pirao.ru

Emotional and behavior problems in adolescence are a strong prognostic indicator for poor adult mental health. Thus, information about its etiology is needed. Most research with adolescent children was conducted on European and American samples. We investigated genetic influences on competence and emotional and behavior problems in a sample of 245 Russian twin pairs aged 10-17 years using parent-reported data (the Child Behavior Checklist). Structural equation modeling procedure was used to estimate parameters for a full model that contains effects from sex-specific additive genes, shared environment, and nonshared environment. The shared environment in the full model was replaced with nonadditive genetic factors for some scales when indicated. Variation in school competence, withdrawn behavior, somatic complains, anxiety/depression, social problems, attention problems/hyperactivity and delinquent behavior was influenced by genetic factors and experiences specific to each child. Variation in activity and aggression was explained by common environmental factors and experiences specific to each child. Variation in social and total competence was influenced by genetic factors, shared environmental influences and experiences specific to each child. Significant quantitative sex differences in the degree of environmental influences on variation in social and school competence as well as attention problems/hyperactivity were identified.

Suzanne K. Haddad¹, Jenae M. Neiderhiser¹, Erica Spotts¹, Jody Ganiban¹, Paul Lichtenstein², David Reiss¹

Depression and Internally Directed Aggression: Genetic and Environmental Contributions

¹ George Washington University

² Karolinska Institute

Grant Support: Grant R01MH54610 from the National Institute of Mental Health; The Samuels Foundation Fellowship for Psychoanalytic Research (Sponsored by the Washington Psychoanalytic Society and George Washington University Department of Psychiatry and Behavioral Sciences)

Address: Center for Family Research, 2300 K street NW, Warwick Building Suite 313, George Washington University, Washington, DC, 20037 USA, Tel: 202 994 4669, email:

Suzannekerin@yahoo.com

This study is an effort to examine Freud's theory of depression as aggression directed toward the self (Freud, S., 1930. Civilization and its Discontents, J. Strachey, Trans., New York: W.W. Norton & Company Inc.) A previous study indicated that nonshared environmental and genetic influences contributed, in near equal amounts, to the association between internally directed aggression and depression for both men and women. In order to better understand the nature of the relationship between depression and internally directed aggression we will examine the potential nonshared environmental influences (i.e., parenting and marriage) on the association. Using data from the Twin/Offspring Study in Sweden (TOSS), a sample of 909 pairs of adult twins, their partners and one adolescent child, we will examine if and how the twin's perception of criticism (from parents retrospectively and spouse) is related to the relationship between depressive symptoms and internally directed aggression. Preliminary analyses indicate that the perception of critical parenting is not related to the association between depressive symptoms and internally directed aggression while perceived criticism from one's spouse is. Further analysis will be conducted to determine the common and unique genetic and environmental contributions to the association between perception of criticism from spouse, depression and internally directed aggression using a trivariate Cholesky model.

Eligible for Thompson Award

David Haig¹

Evolution, Genomic Imprinting, and Social Behavior

¹Department of Organismic and Evolutionary Biology, Harvard University, Cambridge, MA

Address: Department of Organismic and Evolutionary Biology, Harvard University, Cambridge MA 02138, email: dhaig@oeb.harvard.edu

An individual's relatives can be classified as symmetric kin (with equal probabilities of carrying copies of the individual's maternally and paternally derived genes) and asymmetric kin (with unequal probabilities). Inclusive fitness theory has traditionally dealt with the problem of asymmetric kin by employing a coefficient of average relatedness (on the implicit assumption that maternally and paternally derived alleles are constrained to have the same effects). However, if this assumption is relaxed, asymmetries of kinship create the possibility of internal conflicts within individuals over the performance of social behaviors (broadly defined), because behaviors that increase an individual's matrilineal inclusive fitness may differ from behaviors that increase an individual's patrilineal inclusive fitness. Such conflicts provide a plausible explanation for the evolution of genomic imprinting (gene expression that differs when a gene is maternally and paternally derived). Two factors that can give rise to the kinds of relatedness asymmetries that favor genomic imprinting are multiple paternity of a female's offspring, which favors paternally expressed genes in fetuses that extract more resources from mothers, and sex-biased dispersal, which causes group members to have different degrees of matrilineal and patrilineal kinship and may result in an internal conflict over the relative benefits of selfish and altruistic acts. The increased relatedness among sisters in haplodiploids (and for X-linked loci) is due solely to increased patrilineal relatedness.

Zeena Harakeh¹, Jenae M. Neiderhiser², Erica L. Spotts², Robert Plomin³, E. Mavis Hetherington, Rutger C. M. E. Engels, Ron H. J. Scholte, & David Reiss²

Peers and Young Adults Smoking: Univariate and Multivariate Behavioral Genetic Analyses

¹ Radboud University Nijmegen

²Center for Family Research, George Washington University, Washington, DC

³SGDP Centre, Institute of Psychiatry, London

Address: Behavioural Science Institute, Montessorilaan 3, Radboud University Nijmegen, Nijmegen, Gelderland, 6525 HR, The Netherlands, Tel.: 31 24 3612980, email: z.harakeh@pwo.ru.nl

This present study investigated the genetic and environmental influences on the association between adolescents' peer characteristics (i.e., peer college orientation, and peer delinquency & substance use) and smoking in young adulthood. We used longitudinal data of the Nonshared Environment and Adolescent Development (NEAD) Study. Parents' reports on adolescents' peer characteristics and self-reports on smoking in young adulthood were collected. Univariate and bivariate behavioral genetic analyses were conducted. Findings showed that genetic and nonshared environmental influences contributed to peer college orientation as well as smoking status. Genetic, shared and nonshared environmental influences contributed to peer delinquency and substance use. Further, genetic and nonshared environmental influences contributed to the association between adolescents' peer college orientation and smoking in young adulthood. Genetic and shared environmental influences contributed to the association between adolescents' peer delinquency and substance use, and smoking in young adulthood. In conclusion, the present study showed that both individuals' environment and genes explain why adolescents are engaged with certain peers during adolescence, why individuals smoke, and how peers might have a long-term influence on young adults' smoking.

Julie Aitken Harris¹, Philip A. Vernon¹, Kerry L. Jang²

Rated Personality and Measured Intelligence in Young Twin Children

¹The University of Western Ontario

²University of British Columbia

Address: Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, Tel: 519-661-2111 ext 84699, email: jharris@uwo.ca,

Phenotypic, genetic, and environmental correlations between measured intelligence and caregiver-provided ratings of personality were examined in a sample of 4- to 6-year-old twin children (N = 680 individuals). Personality ratings were factor analyzed and five factors were extracted, labelled agreeableness, extraversion, neuroticism, conscientiousness, and psychoticism. Univariate genetic analyses conducted on the same-sex pairs (101 MZ pairs and 132 same-sex DZ pairs) demonstrated that all of the personality factors had heritable components (range = 31% to 86%). Performance and full-scale intelligence were also found to have heritable components, but verbal intelligence was better explained by environmental factors. At the phenotypic level, agreeableness and conscientiousness correlated positively with intelligence and neuroticism and psychoticism correlated negatively with intelligence. Multivariate genetic analyses revealed that many of the observed phenotypic correlations could be explained by common genetic factors.

Julie Aitken Harris¹, Philip A. Vernon¹, Andrew M. Johnson¹, Kerry L. Jang²

The Genetics of Values

¹The University of Western Ontario

²University of British Columbia

Address: Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, Tel: 519-661-2111 ext 84699, email: jharris@uwo.ca,

The heritability of self-report personal value factors was assessed in the present study. Adult participants (N = 258 sibling pairs) completed a self-report values questionnaire. Six value factors were extracted and were labelled: spiritual versus material happiness (e.g., inner harmony versus pleasure), nationalism (e.g., world at peace), work (e.g., ambitious), religiosity (e.g., salvation), home versus adventure (e.g., family security versus excitement), and people versus things (e.g., helpful versus logical). Genetic analyses were conducted on the factors from a subsample of twins (74 monozygotic (MZ) female pairs, 19 MZ male pairs, 41 dizygotic (DZ) female pairs, and 9 DZ male pairs). Each of the value factors was found to have a genetic component with values ranging from 17% for the religiosity factor to 50% for the nationalism factor.

Julie Aitken Harris¹, Philip A. Vernon¹, Andrew M. Johnson¹, Kerry L. Jang²

Vocational Interests and Personality: Phenotypic and Genetic Relationships

¹The University of Western Ontario

²University of British Columbia

Address: Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, Tel: 519-661-2111 ext 84699, email: jharris@uwo.ca,

Relationships between personality and vocational interest factors were examined at the phenotypic and genetic levels. Twins and siblings (N = 516) completed self-report personality and vocational interest scales. Following factor analyses of each scale, five personality and six vocational interest factors were extracted. At the phenotypic level, correlations between personality and vocational interests ranged from zero to .33. Heritability estimates of the scales showed that genetic components accounted for zero to 56% of the variance for the vocational interest factors and 44% to 65% for the personality factors. Genetic correlations between the two areas ranged from zero to .50. The results suggest that personality is related to some vocational interest dimensions and that some of these observed relationships have a common genetic basis.

Sara A. Hart¹, Stephen A. Petrill¹

Univariate analyses of the growth of reading outcome measures

¹The Pennsylvania State University

Grant Support: Supported by NICHD HD38075

Address: Biobehavioral Health, 101 Amy Gardner House, The Pennsylvania State University, University Park, PA 16802, USA, Tel: (814) 865-1717, email: sah323@psu.edu

This study examined the etiology of the growth of reading over one year using the Western Reserve Reading Project, a sample of 350 same-sex MZ and DZ twins from Ohio. Reading outcomes were measured using the Woodcock Reading Mastery Tests subtests of Word ID, Word Attack and Passage Comprehension (R. Woodcock, 1987, Woodcock Reading Mastery Tests – Revised, American Guidance Service, Circle Pines, MN). All twins were in kindergarten or first grade in wave 1, and were assessed one year later in wave 2 (wave 1 age M = 6.0yrs, wave 2 age M = 7.2yrs). Difference scores were calculated by subtracting wave 2 by wave 1 scores, and then standardized for age and sex. Intraclass correlations for all difference scores suggested a significant genetic component, but also shared environmental variance (Word ID, $r_{MZ} = .81$, $r_{DZ} = .58$; Word Attack, $r_{MZ} = .74$, $r_{DZ} = .29$; and Passage Comprehension, $r_{MZ} = .64$, $r_{DZ} = .16$). Univariate genetic analyses were also conducted on the difference scores for each outcome. Results for Word ID suggested that genetic and shared environmental influences were significant when accounting for the difference between the second and first wave of measurement ($h^2 = .38$, $c^2 = .40$). Results when examining the difference between wave 2 and wave 1 Word Attack scores showed evidence that the genetic influence was significant, but the shared environmental effect was not ($h^2 = .69$, $c^2 = .00$). Similarly, the difference between wave 2 and wave 1 Passage Comprehension scores suggested strong genetic influence ($h^2 = .63$), with shared environmental effects accounting for no variance ($c^2 = .00$). Overall, the results suggest that genetics, and to a lesser extent shared environment, play a significant role in the growth of ability for reading outcomes when children first begin formal reading instruction.

Eligible for Thompson Award

Jesse L. Hawke¹, Michael C. Stallings¹, Sally J. Wadsworth¹, John C. DeFries¹

Combined analysis of reading performance data from reading-disabled and control twin pairs using the Pearson-Aitken selection formula

¹Institute for Behavioral Genetics, University of Colorado, Boulder CO 80309

Grant Support: NICHD Center Grant HD-27802
NICHD Training Grant HD-7289

Address:

Psychology, 447 UCB, Institute for Behavioral Genetics, Boulder, Colorado, 80309-0447 USA,
Tel: (303)735-6179, FAX: (303)492-8063, email: hawkej@colorado.edu

Selected twin samples provide greater analytical power than unselected samples, particularly in investigations of behaviors with relatively low prevalence in the general population. DeFries and Fulker (J.C. DeFries and D.W. Fulker, 1985, *Behav. Genet.*, 15, 467-473) proposed a regression-based method (DF analysis) for the analysis of selected twin data that has proven to be very effective in univariate and bivariate applications. However, it is not easily extended to the multivariate case. In the current study we jointly analyzed reading-performance data from selected and control twins tested in the Colorado Learning Disabilities Research Center, taking advantage of the Pearson-Aitken (P-A) selection formula (A.C. Aitken, 1934, *Proc. of the Edinburgh Math. Soc. B*, 4, 106-110) to model the effects of selection on mean and covariance structures (e.g., C.J. Hopfer, M.C. Stallings, J.K. Hewitt, and T.J. Crowley, 2003, *J. AM. Acad. Child Adolesc. Psychiat.*, 42, 834-841). Specifically, we fitted a univariate ACE model using this formula to data from selected twin pairs (MZ = 199; DZSS = 130; DZOS = 101) in which at least one member of each pair had reading difficulties, as well as to data from our control sample (MZ = 274; DZSS = 180; DZOS = 136) simultaneously. Estimates of heritability ($h^2 = 0.58$; CI = 0.50-0.67), shared environmental influences ($c^2 = 0.23$; CI = 0.15-0.31), and non-shared environmental influences ($e^2 = 0.19$; CI = 0.17-0.21) obtained from the ACE model using the P-A formula were very similar to estimates obtained from an augmented DF analysis fitted to data from only the selected twin pairs and also to data from only control twin pairs. However, results indicated that this approach provided greater power than the DF analysis. Because the P-A formula may be readily generalized to the multivariate case, our next step will be to fit this model simultaneously to data from multiple reading-related variables from our selected and control sample populations.

Eligible for Thompson Award

Yoon-Mi Hur¹

Genetic etiology of hostility in male and female adolescents: A South Korean twin study

¹Medical Research Center, Seoul National University, Seoul, South Korea

Address: Medical Research Center, 28 Yongon-dong, Seoul National University, Seoul, Jongno-gu, 110-744, South Korea, Tel: 82 2 741 6180, email: ymhur@neuroimage.snu.ac.kr

Hostility has received increasing attention because it has been shown to be a major risk factor for coronary heart disease. Although twin studies have documented moderate heritability of hostility, the majority of twin research conducted so far has involved male subjects. Using same-sex and opposite-sex twins in South Korea, the present investigation explored a differential genetic etiology of hostility in boys and girls. Over 800 twin pairs aged between 14 and 22 yrs completed a hostility scale through a mail survey. The hostility scale measures persisting disposition to experience anger and hostility and the tendency to behave in a hostile manner. A Sex-limitation model was applied to the data.

Yoon-Mi Hur¹, Juko Ando¹, Dorret I. Boomsma², Belinda Cornes³,
Jaakko Kaprio⁴, Michelle Luciano³, Hiroko Maekawa⁵, Nick Martin³, Ryoko Nakajima⁶, Syuichi
Ooki⁷, Zengchang Pang⁸, Richard Rose⁹

**A comparison of twin BMI data from Australia, China, Finland, Japan, the Netherlands,
and South Korea: Are genetic and environmental variations in BMI similar in Caucasians
and East Asians?**

¹Department of Education, Keio University, Tokyo, Japan

²Department of Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands

³Queensland Institute of Medical Research, Brisbane, Australia

⁴Department of Public Health, University of Helsinki, Helsinki, Finland

⁵Kanazawa Gakuin University, Ishikawa, Japan

⁶Keio University Graduate School of Human Relations, Tokyo, Japan

⁷Department of Health Science, Ishikawa Prefectural Nursing University, Ishikawa, Japan

⁸Qingdao Municipal Centers for Disease Control and Prevention, Qingdao, Japan

⁹Department of Psychology, Indiana University, Indiana, USA

Address: Medical Research Center, 28 Yongon-dong, Seoul National University, Seoul, Jongno-
gu, 110-744, South Korea, Tel: 82 2 741 6180, email: ymhur@neuroimage.snu.ac.kr

There have been secular increases in the prevalence of obesity and related diseases throughout most of the industrialized world. A previous study (Hur, Luciano, Martin, Boomsma, Iacono, McGue, Shin, Jun, Ooki, Beijsterveldt, and Han, 2005, *Twin Res Hum Genet.* 8, 638-648) showed that the variance of birthweight was larger in Caucasians than in East Asians and that this difference was largely attributable to a greater shared environmental variance of birthweight in Caucasians than in East Asians. Because birth weight has been documented to be a significant predictor of later body mass index (BMI) (Pietilainen, Kaprio, Rasanen, Winter, Rissanen, and Rose, 2001, *Am J Epidemiol.* 154, 21-29; Whitfield, Treloar, Zhu, and Martin, 2001, *Twin Res.* 4, 365-370), in the present study, we compared genetic and environmental variances of BMI between Caucasians and East Asians using preadolescent and adolescent twin data from Australia, China, Finland, Japan, the Netherlands, and South Korea. We explored two specific questions in the present analyses. First, are the phenotypic, genetic, and environmental variances of BMI comparable in Caucasians and East Asians in both preadolescents and adolescents? Secondly, is the pattern of sex difference in genetic and environmental influences on BMI similar between Caucasians and East Asians? The results are discussed in the context of the differences in genetic, family environmental, and psychosocial risk factors in development of obesity and obesity-related diseases in Caucasian and East Asian countries.

Yoon-Mi Hur¹, Jong Yeol Kim², Siwoo Lee², Hwayong Park², Yoosik Yoon²

Are your hands warm or cold?: A behavioral genetic analysis on the basis of a South Korean twin sample

¹Medical Research Center, Seoul National University, Seoul, South Korea

²Korea Institute of Oriental Medicine, Daejeon, South Korea

Address: Medical Research Center, 28 Yongon-dong, Seoul National University, Seoul, Jongno-gu, 110-744, South Korea, Tel: 82 2 741 6180, email: ymhur@neuroimage.snu.ac.kr

There are individual differences in the temperature of the peripheral parts of the body such as hands or feet. As part of the mail survey, over 800 pairs of South Korean twins answered for a single item “Are your hands typically cold or warm?” Genetic influences on “coldness” in hands were explored using sex-limitation models. Although the genetic mechanism that explains individual variations in “coldness” in hands is largely unknown, we speculate that genes that affect blood circulation may be involved in “coldness” in hands.

Yoon-Mi Hur¹, Jung Lee²

The role of neuroticism in obsessive-compulsive disorder: A South Korean twin study

¹Medical Research Center, Seoul National University, Seoul, South Korea

²Seoul South Korea

Address: Medical Research Center, 28 Yongon-dong, Seoul National University, Seoul, Jongno-gu, 110-744, South Korea, Tel: 82 2 741 6180, email: ymhur@neuroimage.snu.ac.kr

Neuroticism has been shown to be significantly related to obsessive-compulsive disorder. The major goal of the present study is to determine the nature of the relationships between neuroticism and various dimensions of obsessive-compulsive disorder. Over 800 pairs of twins aged between 14 and 22 yrs completed a Korean version of the Maudsley Obsessive-Compulsive disorder Inventory (MOCI) and the Neuroticism scale of Eysenck Personality Inventory through a mail survey. Thirty items of the MOCI were factor analyzed to extract different dimensions of obsessive-compulsive disorder. Multivariate model-fitting analyses were conducted on the relationships between the MOCI factors and neuroticism. The results suggest that neuroticism may be an endophenotype of obsessive-compulsive disorder.

Daniel E. Irons¹, Matt McGue^{1,3}, William G. Iacono¹, S.A. Burt²,
Bob Krueger¹, William S. Oetting³,

A Dopamine Receptor D4 Polymorphism, Attention Deficit Hyperactivity Disorder, and Disinhibitory Psychopathology

¹Department of Psychology, University of Minnesota, Minneapolis MN

²Department of Psychology, Michigan State University

³Institute of Human Genetics, University of Minnesota, Minneapolis MN

Grant Support: NIH Grant AA11886; NIH Grant MH066140

Address: Psychology, 1041A 29th Ave SE, University of Minnesota, Minneapolis MN 55414
USA, Tel: 612-481-2133, email: iron0012@umn.edu

An association has been sporadically observed between the 7-repeat allele of a 48 bp Variable Number Tandem Repeat (VNTR) polymorphism in the third exon of the dopamine receptor gene DRD4 and both Attention Deficit Hyperactivity Disorder (ADHD) diagnosis (SV Faraone, AE Doyle, E Mick, and J Biederman, 2001, *Am. Jo. Psychiatry*, 158, 1052-1057) and indicators of the novelty seeking personality traits thought to be linked to ADHD (GJ LaHoste, JM Swanson, SB Wigal, C Glabe, T Wigal, N King, and JL Kennedy, 1996, *Molecular Psychiatry*, 1, 121-124). This study examines participants in the Minnesota Twin Family Study (MTFS) for DRD4 polymorphism associated differences in both ADHD diagnosis and measures of behavioral disinhibition.

Eligible for Thompson Award

Jean-Marc Jallon¹

Evolution of *Drosophila* Populations

¹NAMC, UPS-ORSAY, 91405 ORSAY FRANCE

Address: IBAIC, BAT 446, UNIV Paris XI, ORSAY, F-91405, FRANCE, Tel: 33 1 69157461,
FAX: 33 1 69157726, email jmjallon@club-internet.fr

Hydrocarbon pheromones have been involved in *D. melanogaster* courtship. The polymorphism of these chemical signals is multiple and a number of structural genes have been identified. Marked changes have accompanied migrations of ancestral populations from western central Africa. We will explain how effects of temperature variations and sexual selection might have modified their chemical signatures and mate choices and produced the derived chemical phenotypes.

Patrick Jern¹, Pekka Santtila¹, N. Kenneth Sandnabba¹

Genetic and Environmental Effects on Rapid and Delayed Ejaculation

¹Department of Psychology, Åbo Akademi University

Grant Support: The Academy of Finland; The Åbo Akademi Foundation

Address: Department of Psychology, Brunnsgratan 8 B 25, Åbo Akademi University, Åbo, 20500, Finland, Tel: 358 22 5144 04, email: pjern@abo.fi

The prevalence estimates of rapid ejaculation (RE) vary widely, ranging from 4-30% (e.g. Simons & Carey, 2001, *Arch. of Sexual Behavior* 30, 177-219). Definitional differences are the primary reasons for this variability, and biological as well as psychosocial models have been used to describe the etiology of RE. According to Waldinger (2005, *J Sexual Medicine* 2(4), 492-497), there is normal biological variation in ejaculatory latency in men. Hence, a small subgroup in any random sample of men is expected to have RE whereas another subgroup is expected to have delayed ejaculation (DE), with the majority falling in between these two extremes. The aim of the present study was to investigate to what extent genetic and environmental factors influence RE and DE.

A sample of Finnish male twins was selected for the study (80 MZ and 97 DZ pairs). Participants responded to a self-report questionnaire with questions tapping different aspects of ejaculatory function (e.g. number of thrusts, time elapsed from penetration to ejaculation, measures taken to quicken or delay intercourse). The questions were adapted from a questionnaire developed by Grenier and Byers (1995, *Arch. of Sexual Behavior* 24(4), 447-472). After an exploratory factor analysis, a two-factor (with one factor measuring RE and the other DE) model was identified and subjected to a confirmatory factor analysis, which in turn revealed a good fit for the model.

Next, twin model fitting using Mx was performed revealing that both RE and DE were influenced by familial factors, but in different ways. RE was significantly influenced by genes, whereas DE seemed to be more influenced by shared environmental factors. Neither additive genetic nor shared environmental effects could, however, be excluded for RE or DE.

Andrew M. Johnson¹, Julie Aitken Harris¹, Jon Fleming², Kerry L. Jang²

A multivariate genetic investigation of the relationship between omnibus personality and sleep quality

¹The University of Western Ontario

²University of British Columbia

Address: Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, Tel: 519-661-2111 ext 84699, email: jharris@uwo.ca,

The present study was designed to examine the phenotypic, genetic, and environmental correlations between personality and sleep quality. In this study, 117 monozygotic (MZ) adult twin pairs (26 male and 91 female) and 79 dizygotic (DZ) adult twin pairs (17 male and 62 female) completed a self-report personality questionnaire measuring three facets of personality: psychoticism, extraversion, and neuroticism, and sleep scale designed to measure quality and patterns of sleep on seven dimensions: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction. Three basic sleep descriptors were also assessed: time spent sleeping, typical time going to sleep, and typical time the person awakes. Six of the seven sleep quality dimensions demonstrated significant heritability (use of sleeping medications was not significantly heritable), as did all three of the personality dimensions. Although time at which an individual typically awakens was best fit by a model of environmental determination, both of the other basic sleep descriptors showed significant genetic determination. The pattern of genetic correlations suggests that habitual sleep efficiency and daytime dysfunction are predicted by genetic determinants that are similar to those that produce individual differences in neuroticism and psychoticism. These preliminary results suggest interesting directions for future research on the amelioration of sleep disturbances, and may provide some insight into individual sleeping habits.

Jaakko Kaprio¹, Danielle Dick², Anja Huizink³, Richard J Rose⁴

Cannabis use among Finnish adolescents and young adults

¹University of Helsinki, Helsinki, Finland

²Washington University in St. Louis, St. Louis, Missouri, USA

³Erasmus Medical Center, Rotterdam, The Netherlands

⁴Indiana University, Bloomington, IN

Grant Support: NIAAA AA-09203 and R37 AA-12502) to RJR; Academy of Finland grants 100499, 204690, 205585) to JK.

Address: Department of Public Health, PO Box 41, Mannerheimintie 172, University of Helsinki, Helsinki 14, Finland, Tel: 358919127595, FAX: 358919127600, email: jaakko.kaprio@helsinki.fi

The genetic epidemiology of cannabis use, abuse and dependence implicates both shared environmental effects and genetic influences. Most studies have been conducted in Anglo-Saxon societies. In Finland use of cannabis was rare before a modest, gradual increase began during the 1990s, until a plateau was reached around 2000.

In the FinnTwin12 study, subjects were surveyed by questionnaire immediately after their 14th birthday; cannabis use was rare and no genetic modeling was possible. The third wave of the same study was carried out in 2000-2005, when the twins were aged 17 years and 13% had any use with cannabis. Among 1905 MZ and DZ pairs, pairwise tetrachoric correlations for any use were high in male (0.89) and female (0.87) monozygotic pairs, while correlations were somewhat lower in male dizygotic pairs (0.71), female dizygotic pairs (0.70), and male-female dizygotic pairs (0.52). The estimate of additive genetic variance was 0.53 (95% CI 0.32 to 0.75), with an estimate for shared environmental effects of 0.34 (95% .15 to .52) with no sex differences.

In the FinnTwin16 study of Finnish twins born 1975-1979, the fourth wave of data assessment was at average age 24 years during 2000-2002. 22% of subjects had used cannabis. SSAGA-based interviews of a subset of FinnTwin16 subjects (N=602 twins) indicated a high reliability of the history of any use of cannabis ($Kappa=0.85$) from the questionnaire. Also 90% of those that had used any illicit drugs were also cannabis users. Among 2005 MZ and DZ pairs, tetrachoric correlations for any use were high in male and female monozygotic pairs (both $r=0.81$), while correlations were lower in male dizygotic pairs (0.64), female dizygotic pairs (0.49), and male-female dizygotic pairs (0.33). Modeling suggested that additive genetic effects (42% of variance) and shared environmental effects (40%) are required in men, but only genetic effects (82%) in women, with the remainder accounted for by unshared environment.

Yong-Kyu Kim¹, Jayne Kelly

Effects of Social Experience on Aggressive Behavior in *Drosophila*

¹University of Georgia

Address: Genetics, University of Georgia, Athens, GA 30602, USA, Tel: (706) 542-1448, email: yongkyu@uga.edu

Aggression is an adaptive behavior that serves in the acquisition or defense of food resources, or access to mates in nature, and this behavior is observed in many animal species, including humans. Aggressive behavior is heritable and affected by both genes and environment. This behavior has been reported in several *Drosophila* species (Dow, M. and Schilcher, F., 1975, *Nature* 254, 511-512; Jacobs, M., 1978, *Behav. Genet.* 8, 487-502; Hoffmann, A., 1987, *Anim. Behav.* 35, 807-818; Papaj, D. and Messing, R., 1998, *Behaviour* 135, 1013-1030; Boake, C. et al., 1997, *PNAS* 94, 12442-12445; Lee, G. and Hall, J., 2000, *Behav. Genet.* 30, 263-275; Chen, S. et al., 2002, *PNAS* 99, 5664-5668; Nilsen, S. et al., 2004, *PNAS* 101, 12342-12347). We observed the effect of preadult social experience on aggressive behavior of *D. melanogaster* and *D. pseudoobscura*. Individual eggs were removed and placed on the surface of a standard cornmeal medium in a small glass vial, and raised until the imago emerged. Control flies were reared in groups (N=20) during development. Upon emergence, virgin flies were collected, marked individually with acrylic paint for identification, and aged for 5 days. Pairs of flies of the same sex were observed in mating chambers for 30 minutes in three different combinations: isolated vs. isolated, socialized vs. socialized, and isolated vs. socialized. Seven behaviors were scored during observation: chasing, wing vibration, wing threat, fencing, boxing, holding, and lunging. Current data show that there are quantitative differences in each of the behaviors between species. Preadult experience during development significantly affects aggressive behavior of *Drosophila*: the isolated flies display more aggressive behavior than the socialized flies for both *D. melanogaster* and *D. pseudoobscura*. In addition, brain size is influenced by social experience. The flies reared in isolation have significantly smaller brains, especially mushroom bodies, than the controls.

Julia Kim-Cohen¹

MAOA by maltreatment GxE in young children

¹Yale University

Grant Support: National Institute of Mental Health (MH45070 & MH49414); UK Medical Research Council (G9806489 & G0100527); ESRC-SCOPIC Network

Address: Psychology, 2 Hillhouse Ave, PO Box 208205, Yale University, New Haven, CT 06520, USA, Tel: (203) 432 7581, email: julia.kim-cohen@yale.edu

Previous research on adults has shown that a functional polymorphism in the promoter region of the monoamine oxidase (MAOA) gene moderates the impact of childhood maltreatment on risk for developing antisocial behavior (Caspi et al., 2002, *Science*). Thus far, attempts to replicate this finding have been mixed. The current study (i) presents new data investigating this finding in a sample of 975 7-year-old boys, and (ii) evaluates the extant data by conducting a meta-analysis of published findings. We replicated the original finding by showing that the MAOA polymorphism moderates the development of psychopathology after exposure to physical abuse, we extended the finding to childhood closer in time to the maltreatment experience, and we ruled-out the possibility of a spurious finding by accounting for passive and evocative gene-environment correlation. Moreover, meta-analysis demonstrated that across studies, the association between maltreatment and mental health problems is significantly stronger in the group of males with the genotype conferring low vs. high MAOA activity. These findings provide the strongest evidence to date suggesting that the MAOA genotype influences vulnerability to environmental stress, and that this biological process can be initiated early in life.

Marc Lalande¹

Association of imprinted non-coding RNAs with the Angelman and Prader-Willi syndromes.

¹Department of Genetics & Developmental Biology, University of Connecticut School of Medicine, Farmington, CT

Address: Genetics and Developmental Biology, 263 Farmington Avenue, University of Connecticut Health Center, Farmington, CT, 06030-3310 USA, Tel: 860 679 8349, email: lalande@uchc.edu

Human chromosome 15q11-q13, a region that is subject to genomic imprinting, encompasses the Angelman syndrome (AS) and Prader Willi syndrome (PWS) loci. The clinical manifestations of AS include microcephaly, severe mental retardation, 'puppet-like' ataxic gait with jerky arm movements, seizures, EEG abnormalities, hyperactivity and bouts of inappropriate laughter. PWS is characterized by hypotonia and failure to thrive in infancy, small hands and feet, hypogonadism, variable mental retardation, obsessive-compulsive behavior, and marked obesity resulting from hyperphagia. The majority of both AS and PWS cases (60-70%) are caused by de novo deletion of chromosome 15q11-q13. In the case of AS, the deletion is inherited from the mother whereas the deletion is of paternal origin in PWS. Several genes and non-coding RNAs that display exclusive paternal expression have been identified in the 15q11-q13 region. The paternally expressed transcripts include three classes of small nucleolar (sno) RNAs and their putative role in PWS is being intensively investigated and discussed. AS is associated with a failure to inherit a normal active maternal copy of the gene encoding ubiquitin protein ligase E3A (UBE3A). UBE3A is transcribed predominantly from the maternal allele in brain but is expressed from both alleles in most other tissues. Silencing of the paternal UBE3A allele in brain appears to be mediated in cis by a large non-coding antisense transcript (UBE3A-ATS) that is expressed exclusively from the paternal allele. My laboratory is investigating how loss of UBE3A affects neuronal function and how the interaction between UBE3A and UBE3A-ATS results in brain-specific imprinting.

Jeffrey M. Lessem¹, Robin P. Corley¹, Marissa A. Ehringer¹, Brett C. Haberstick¹, Kenneth S. Krauter¹, Isabel R. Schlaepfer¹, Michael C. Stallings¹

The Effects of Genotyping Error on Case-Control Association Using SNPs and Haplotypes

¹Institute for Behavioral Genetics, University of Colorado, Boulder

Grant Support: P01-HD31921; DA011015; EY012562; DA015522; AA015366

Address: Institute for Behavioral Genetics, 447 UCB, University of Colorado, Boulder, CO 80309-0447 USA, Tel: 303-492-2843, email: jeff.lessem@colorado.edu

Simulations are used to examine the effects of genotyping error on association in a case-control based study. SNPs forming haplotypes are simulated based on observed SNP and haplotype frequencies in PKC-gamma, and the alpha-4 and beta-2 subunits of the nicotinic receptor for both Colorado and national US samples. Multiple error rates are simulated, based on the empirical error rates observed using pre-amplified DNA on Affymetrix GeneChip Custom SNP arrays. Most individuals who are genotyped show a low genotyping error rate, of 0.01% or lower, but some individuals show a markedly higher error rate of 0.02% or greater. Reduction in power is examined with the errors compared to a perfect sample.

Chaucer C.H. Lin, M.D., Ph.D.¹, Wei J. Chen, M.D., Sc.D.²

Analysis of Vertical Transmission of Schizotypy: A Study of Taiwanese Juvenile Twins and Their Parents

¹Department of Psychiatry, Tzu Chi General Hospital and University, Hualien, Taiwan

²Institute of Epidemiology, School of Public Health, National Taiwan University, Taipei, Taiwan

Address: Psychiatry, Tzu Chi General Hospital and University, Taiwan, Hualien, 970, R.O.C.,
Tel: 886-3-8230381, email: chaucer@mail.tcu.edu

Background: The schizotypal traits measured by Schizotypal Personality Questionnaire (SPQ) are potential vulnerability markers of schizophrenia. Genetic and specific environmental factor were noted to have substantial effects on them and heritabilities of SPQ scores had been determined. This study intends to investigate whether there are genetic and cultural transmissions on these traits.

Methods: The study subjects were 232 pairs of twins recruited from junior high schools in Taipei City and their parents. Subjects completed the SPQ. Structural equation modeling applying mixed genetic and cultural transmission models using the Mx program were done and estimates of effects, including genetic, cultural transmission, and assortative mating, were determined.

Results: The correlations between schizotypal measurements were 0.1-0.45 in fathers and their sons, and were 0.01-0.20 in mothers and their daughters. According to the best-fitted models, approximately 58% of the variances in schizotypy were accounted by genetic factors in male. In women, the variances in schizotypy were not significantly accounted by genetic factors. Parents influence their offsprings' schizotypy mainly through genetic factors. Assortative mating between couples and sibling interaction were noted for schizotypy.

Conclusions: The lack of resemblance between family members of opposite sex suggests that different genetic factors influence schizotypy in men and women. Schizotypy is moderately heritable in the men but not women. The vertical transmission of schizotypy from parents to offspring is mainly genetical. The effect of vertical cultural transmission on schizotypy is minimal and, if any, is evenly from both parents.

Ashley Mackie¹, Philip A. Vernon¹, Rod A. Martin¹, Leah D. Sheppard¹,

A behavioral genetic investigation of sense of humor

¹Department of Psychology, University of Western Ontario

Grant Support: Social Sciences and Humanities Research Council of Canada

Address: Department of Psychology, University of Western Ontario, London, Ontario, N6A 5C2, Canada, Tel: 519-661-3682, FAX: 519-661-3961, email: vernon@uwo.ca

In this presentation we report the first ever behavioral genetic (BG) investigation of sense of humor as measured by the Humor Styles Questionnaire (HSQ). Two hundred pairs of adult twins completed the HSQ, which is a 32-item measure tapping four different ways people use humor in their daily lives. Two of these four dimensions of humor are potentially beneficial to psychological well-being while the other two are potentially detrimental. The two positive dimensions are Affiliative humor (e.g., saying funny things, telling jokes, amusing people) and Self-Enhancing humor (e.g., maintaining a humorous perspective even in the face of stress). The two negative dimensions are Aggressive humor (e.g., using humor to criticize or manipulate others via sarcasm, ridicule, or derision) and Self-Defeating humor (e.g., attempting to amuse others by doing or saying funny things at the expense of oneself). Preliminary univariate BG analyses reveal that an AE model provides the best fit to all four of the HSQ scales, with h^2 values between .40-.60. Participants in this study also completed the NEO-PI-R as a measure of the Big 5 personality traits and phenotypic, genetic, and environmental correlations between the HSQ scales and the Big 5 will also be reported.

Eligible for Thompson Award

Gerald E. McClearn¹

Contextual Genetics

¹Department of Biobehavioral Health, Center for Developmental and Health Genetics, The Pennsylvania State University, University Park, PA 16802

Grant Support: AG14731 from National Institute on Aging,
AA08125 from the National Institute on Alcohol and Alcohol Abuse

Address: Center for Dev. and Health Genetics, Pennsylvania State University, University Park, PA 16802, email: gm1@psu.edu

The dominant epistemological orientation in recent research on heredity has been reductionist, and it has led to profound advances in understanding of the nature of genes and of their function. A major reductionist strategy is the use of experimental procedures, and a prototypic application is the examination of the phenotypic consequence (the dependent variable) of varying the genotype at a single locus (the independent variable) while other genetic loci and the environmental circumstances under which the observations are made are controlled to certain values. In the case of complex phenotypes (as are many behavioral phenotypes) the causal inputs to the determinant system can be multitudinous and the mediating processes can be highly interactive. In such circumstances, the restrictions on the controlled variables reduce the extent to which the outcomes of the research can be regarded as generalizable beyond its narrow constraints. Characterizing the dynamics of the total system requires research strategies that explore gene function in broader contexts of “residual” genes and environment

Vivia V. McCutcheon¹, Andrew C. Heath¹, Elliot C. Nelson¹, Kathleen K. Bucholz¹, Pamela A.F. Madden¹, Nicholas G. Martin²

Associations of Trauma and Genetic Effects with Drinking Motives in a Female Twin Sample

¹Department of Psychiatry, Washington University, St. Louis, MO

²Queensland Institute of Medical Research, Queensland, Australia

Grant Support: NIAAA Grants AA07728, AA10240, T32AA07580

Address: Psychiatry, Washington University, St. Louis, MO, 63110, USA, Tel: (314) 286-2219, email: vmccutcheon@wustl.edu

This analysis uses a female twin sample to examine the associations of traumatic experience and genetic influences with drinking motives. The sample comprises female twins born in Missouri between 1975 and 1987. Responses from 2238 twins who reported having drunk any alcohol and for whom there were complete data on trauma and drinking motive items were used in this analysis. Factor analysis of 20 items about reasons for drinking derived four factors analogous to a four-factor model tested by Cooper (Cooper, M, 1994, *Psychological Assessment*, 6(2), 117-128). Trauma classes characterized by different types and frequencies of reported traumatic events were defined using latent class analysis. Genetic effects were modeled as the interaction between zygosity and cotwin endorsement of drinking motives. Drinking motives were regressed on trauma classes and genetic effects.

Four trauma classes provided the best fit to the data, with each successive class having a higher mean number of traumatic events. Class 1 was characterized by no or low probability of trauma endorsement, Class 2 by endorsement of nonassaultive events (e.g., accident, disaster), and Classes 3 and 4 were characterized by a high probability of endorsement of childhood physical and sexual abuse.

Drinking to cope was the only motive with which trauma and genetic effects ($b=.17$, 95% CI=.06, .29) were independently associated, and the only motive with a positive association with trauma class (Class 4, $b=2.26$, 95% CI=.58, 3.94). There was evidence for genetic influence on drinking for positive social rewards ($b=.13$, 95% CI=.03, .23), and Class 3 membership had a negative association with drinking for social rewards ($b=-1.25$, 95% CI=-2.17, -.36). Trauma had no association with drinking to enhance positive mood or drinking to avoid social rejection, nor was there evidence of genetic influence on these motives.

Matt McGue^{1,2}, Kaare Christensen²

The Relationship of Social Support and Activity with Late-Life Functioning: A Cotwin Control Study

¹Department of Psychology, University of Minnesota

²Department of Epidemiology, Southern Denmark University

Grant Support: Supported by U.S. National Institute on Aging (P01-AG08761) and the Danish National Research Foundation

Address: Psychology, University of Minnesota, Minneapolis, Minnesota, 55455, USA, Tel: 1.612.625.8305, FAX: 1.612.626.2079, email: mcgue001@umn.edu

Gerontologists are increasingly interested in exploring models of gene-environment interplay in late-life functioning. The Longitudinal Study of Aging Danish Twins (LSADT) began in 1995 with the assessment of all twins born and living in Denmark age 75 years and older. Follow-up assessments of surviving LSADT participants were undertaken every 2 years through 2005, with additional twins who had aged into the catchment age were added to the study. Self-report measures of both social support and social activity are included in the LSADT assessment, and both are significantly correlated with diverse measures of late-life functioning including depression symptomatology, cognitive ability and physical functioning. We sought to characterize the nature of the relationships of social support and activity with late-life functioning and changes in late-life functioning using a cotwin control design.

Michael B. Miller¹

GenetSim: Software for Simulation of Familial Data in Genetics and Epidemiology

¹Division of Epidemiology, School of Public Health, University of Minnesota, 55454

Grant Support: NIH Grant 5R01-HL09609-12; NIH Grant 1R01-AG021917-01A1

Address: Epidemiology, 1300 S 2nd St, Suite 300, University of Minnesota, Minneapolis, MN 55454 USA, Tel: 612-625-7836, FAX: 612-624-0315, mbmiller@taxa.epi.umn.edu

GenetSim provides flexible simulations of family data within an easy-to-use, high-level programming language. GenetSim was developed first within the MATLAB-like environment of the free software package Octave (Eaton, 1997), but it is being ported to the R statistical language. Except for memory limits imposed by hardware, GenetSim has no limit on pedigree sizes or structures (these can be imported from LINKAGE-format files and can include MZ twins) or number of families, no limit on number of marker or trait loci, no limit on number of chromosomes (nonhuman diploid species can easily be modeled). Genetic transmission is modeled by first generating the locations of recombination events (according to nearly any multilocus-feasible model - Haldane, Sturt, etc., or a user-specified model), and then performing gene dropping according to the given recombination pattern. Thus, it is possible to simulate trait loci, store recombination information, and later add any number of markers to selected families. GenetSim can simulate multiple QTLs with pleiotropic effects, multivariate polygenic background and any number of environmental factors, age effects, sex effects, epistasis and variable expression. Traits can be quantitative or one can use penetrance functions and/or liability threshold models for affection-status traits. We used GenetSim to produce data for the Genetic Analysis Workshop (GAW15) this year. GenetSim is freely available under the GNU General Public License at <http://taxa.epi.umn.edu/genetsim/>.

Paula Y. Mullineaux¹, Lisabeth F. DiLalla²

Maternal and Child Characteristics in Mother-Twin Interactions: Evidence of Evocate Genotype-Environment Correlation

¹Department of Psychology, Southern Illinois University, Carbondale, IL 62901

²Department of Family and Community Medicine, School of Medicine, Southern Illinois University, Carbondale, IL 62901

Address: Psychology, Department of Psychology, Mail code 6502, Southern Illinois University, Carbondale, IL 62901 USA, Tel: 618-453-5206, email: paulam@siu.edu

Previous research has indicated that mothers treat their MZ twins more similarly by age 2 ½ (H. Lytton, 1977, *Developmental Psychology*, 28, 1006-1017) but not at earlier ages (L. F. DiLalla & E.G. Bishop, 1996, *Behavior Genetics*, 26, 535-562). These results suggest that this similar maternal treatment is not apparent in early infancy. To investigate at what age this difference in maternal treatment begins, mother-twin interactions from the longitudinal Twin Infant Project (TIP) were used to compare maternal behaviors towards their MZ and DZ twins at ages 7, 9, 14, 24, and 36 months. Maternal behaviors such as sensitivity, respect for autonomy, quality of instruction, warmth, and the overall cooperation observed between the mother and the children during the interaction were compared for MZ and DZ twins. No significant differences were found for the maternal behaviors when comparing treatment of MZ and DZ twins at ages 7, 9, 14, and 24 months. At 36 months, mothers treated their MZ twins significantly more similar ($z = 2.04, p < .05$) than their DZ twins. Also at 24 months, the overall cooperation during the interaction was significantly more similar ($z = 2.27, p < .05$) for MZ twins at 24 months. These results suggest that MZ twins, due to their greater degree of genetic relatedness, are evoking more similar sensitive maternal treatment than DZ twins are by age 3 and thus suggests an evocative genotype-environment correlation. Additionally, microanalytic analyses will be conducted to determine if MZ twins engage in more similar behaviors during these interactions and if these behaviors are eliciting more similar maternal responses.

Eligible for Thompson Award

Ryoko Nakajima¹, Satoshi Sasaki², Yutaka Ono³, Juko Ando⁴

BMI and energy intake in Japanese young adult twin pairs

¹Graduate School of Human Relations, Keio University, Tokyo, Japan

²Project Leader of Scientific Evaluation of Dietary Reference Intakes, National Institute of Health and Nutrition, Tokyo, Japan.

³Health Center, Keio University, Kanagawa, Japan.

⁴Faculty of Letters, Keio University, Tokyo, Japan.

Address: Graduate School of Human Relations, 1-26-23 #308 Yaguchi, Keio University, Ohtaku, Tokyo, 146-0093 Japan, Tel: 81-90-5782-5731, email: rio_ryoko@yahoo.com

Many studies have reported that genetic factors explain more than 50% of the variance in body mass index(BMI). Most of them are results from European countries, Australia, and the US, and there is little report on Japanese population. Ooki has reported that genetic factors (additive and non-additive) and unique environmental factor account for 55%, 35%, and 10% of the variation in BMI at the age of 8 (S.Ooki and M.Yokoyama, 2003, Shoni Hoken Kenkyu, 62, 324-330). In our study, we investigated heritability of BMI in the Japanese young adult population. Secondly, there are also reports of genetic influences on energy intake (ex. M.S.Benton, S.S.Rha, M.C.Neal, and D.B.Allison, 1999, Behav. Genet. 29, 145-154). We used a brief-type self-administered diet history questionnaire(BDHQ): a short version of a validated self-administered diet history questionnaire (S.Sasaki, R.Yanagibori, and K.Amano, 1998, J Epidemiol. 8, 203-215), to estimate the amount of energy intake in the Japanese twin samples, and examined the heritability on the intake. The data were gathered from 121 monozygotic(MZ) and 52 dizygotic(DZ) twin pairs in the Keio Twin Project. Mean age was 25.08 (sd 4.38). Intra-class correlations for BMI were higher in MZ($r=.705$) than in DZ($r=.095$). ADE model was the best-fit model compared to the other models (ACE, AE, CE, E models); however, influence of additive genetic factor was found to be 0%, while influences of non-additive genetic factor and the unique environment were 76% and 24% respectively, on the variation in BMI. As for energy intake, intra-class correlations were higher in MZ($r=.304$) than DZ($r=.224$). Additive genetic factor explained 32% of the variation in energy intake. The rest of the variation was explained by unique environmental factor. We also examined the bivariate model-fitting for BMI and energy intake. Phenotypic correlation between BMI and energy intake ($r=0.13$) was all explained by genetic factors.

Jenae M. Neiderhiser¹, Zeena Harakeh²

Predictors of young adult substance initiation and use: The role of family and individual factors

¹George Washington University

²Radboud University Nijmegen

Grant Support: R01MH43373; R01MH48825; William T. Grant Found; R01MH59014; R01MH065563

Address: Dept of Psychiatry and Beh Sciences, George Washington University, Washington, DC 20037 USA, Tel:202.994.2212, FAX: 202.994.4812, cfrjmn@gwumc.edu

Parent, child, contextual and other family factors predict initiation and continued substance use. A recent review found different patterns of genetic and environmental influences for initiation and continued use of substances (CJ Hopfer et al. 2003. J Am Acad Child Adolesc Psychiatry, 42, 710-719). In this study genetic and environmental influences on adolescent parenting, drug initiation and continued use during young adulthood will be examined, as well as the role of family and individual factors. Data from the NEAD project, consisting of sibling pairs from 720 two-parent families, were used. Siblings are same-sex (48% female) and within 4 years of age (age 10-18 at T1). The T3 assessment occurred 10-13 years later (N=414 families). NEAD is comprised of six sibling types: MZ and DZ twins and full siblings in nondivorced families and full, half and step-siblings in stepfamilies. Measurement included parent and child reports at T1 and 2 and self-reports at T3. At T3 we examined drug, tobacco and alcohol initiation and continued use. Preliminary analyses support previous findings of shared environmental influences on initiation. Correlations between parenting and substance use were moderate and similar for mothers and fathers. Only correlations between parental negativity and monitoring and substance initiation could be explored further. Results from sibling correlations suggest different patterns of genetic and environmental influences with nonshared environmental and genetic influences for parental negativity and substance initiation and genetic and shared environmental influences for parental monitoring and substance initiation. These findings are consistent with previous reports that have found more shared environmental influences on monitoring. Additional analyses will incorporate child and family factors to better understand the processes involved.

Jason L. Pagan¹, Candice Holliday¹, Richard J. Rose², Richard J. Viken², Lea Pulkkinen³, Jaakko Kaprio⁴, Danielle M. Dick¹

The Role of Peers in Substance Use Initiation In Early Adolescence

¹Washington University in St. Louis

²Indiana University

³University of Helsinki

⁴University of Jyväskylä

Grant Support: These analyses are supported by AA015416 to DMD. Data collection for the Finnish Twin studies have been supported by the National Institute of Alcoholism and Alcohol Abuse (grants AA-12502, AA- 00145, and AA-09203 to RJR). Supplementary funding from the Academy of Finland the Finnish Centre of Excellence Programme (to LP and JK).

Address: Psychology, One Brookings Drive, Campus Box 1125, Washington University, St. Louis, MO, 63130, USA, Tel.: 314-935-8453, email: jl pagan@artsci.wustl.edu

There is a well-documented relationship between substance use by adolescents and use by their peers (Barnow et al., 2002; Bahr et al., 1995; Sieving et al., 2000); but there is some disagreement about whether this relationship arises through selection and/or socialization processes. Using data from a population-based longitudinal twin study, FinnTwin12 (FT12), we are exploring the processes by which peer substance use is related to early substance use in adolescents. FT12 consists of five birth cohorts of twins ascertained through Finland's Population Registry Center. Baseline data were collected on 2,742 twin families when the twins were 12 years old, and follow-up data were collected on 2,355 twin pairs two years later. At age 14, 36% and 43% of twins reported experimentation with alcohol and tobacco products, respectively; 57% of twins reported having friends who used alcohol, 60% reported friends who smoked cigarettes, and 21% reported friends with drug use. Consistent with previous studies, adolescents were significantly more likely to report alcohol initiation if they had friends who drank, smoked, or used drugs. Interestingly, gender of the adolescent, and gender of the peers appeared to moderate this relationship. Peer substance use was more strongly related to early initiation for female adolescents, and when the peer group included opposite sex friends. Genetically informative models indicate that the correlation between self and peer alcohol use is due, in part, to shared genetic factors, suggesting gene-environment correlation, whereby the adolescents' predispositions are involved in the selection of peers with similar attributes. We are currently fitting models to examine whether peer substance use behavior further moderates the factors influencing twins' substance use after controlling for these selective processes to enhance our understanding of peers' behaviors on substance use in early adolescents.

Eligible for Thompson Award: Yes

Jevon Plunkett¹, Leah Flury Wetherill², Xiaoling Xuei², Alison Goate¹, Victor Hesselbrock³, Marc Schuckit⁴, Raymond Crowe⁵, Howard J. Edenberg², Tatiana Foroud², Danielle M. Dick¹

Association between GABRA1 and Drinking Behaviors in the Collaborative Study on the Genetics of Alcoholism Sample

¹Washington University, St. Louis

²Indiana University School of Medicine, Indianapolis, Indiana

³University of Connecticut

⁴University of California, San Diego

⁵University of Iowa

Grant Support: COGA is supported by NIH Grant U10AA08401.

Address: Psychiatry, 6331 Southwood Ave #2E, Washington University, St. Louis, MO, 63105, USA, Tel: 314-452-3568, email: jevon.plunkett@gmail.com

A wealth of literature supports the role of gamma-aminobutyric acid (GABA) in neurobiological pathways contributing to alcohol dependence and related phenotypes. Human chromosome 5q contains a cluster of GABAA receptor genes, GABRA1, GABRA6, GABRB2, and GABRG2, that have been proposed as candidate genes for alcohol dependence. Animal studies, in which drinking behaviors, rather than alcohol dependence per se, are studied, have consistently tied rodent homologs to these genes; however, human studies have produced mixed results. Family-based association analyses previously conducted in the Collaborative Study on the Genetics of Alcoholism (COGA) sample yielded no evidence of association with DSMIV alcohol dependence and these genes (Dick DM, Edenberg HJ, Xuei X, Goate A, Hesselbrock V, Schuckit M, Crowe R, Foroud T. 2005, Am J Med Genet B Neuropsychiatr Genet. 132(1), 24-8). As a follow up to that study, we examined several alcohol-related behaviors in the COGA sample: (1) a broader definition of alcohol dependence, including DSMIII-R symptoms and Feighner criteria (referred to as COGA alcohol dependence) (2) withdrawal; (3) history of alcohol-induced blackouts; (4) level of response to alcohol; (5) age of onset of regular drinking; (6) age at first drunkenness. Family-based association tests were conducted, using multiple single nucleotide polymorphisms (SNPs) in each of the four GABAA receptor genes on chromosome 5q. In GABRA1, we found evidence of association with several of the drinking behavior phenotypes, including COGA alcohol dependence, history of blackouts, age at first drunkenness, and level of response to alcohol. We did not find consistent evidence of association with the remaining genes and any of the phenotypes. These analyses suggest that efforts to characterize genetic contributions to alcohol dependence may benefit by examining alcohol-related behaviors in addition to clinical alcohol dependence diagnoses.

Evelien A.P. Poelen¹, Rutger C.M.E. Engels¹, Dorret I. Boomsma², Ron H.J. Scholte¹, Gonneke Willemsen²

Alcohol Use in Dutch Adolescent and Young Adult Twins as a Function of Common Friends

¹Radboud University Nijmegen, the Netherlands

²Vrije Universiteit Amsterdam, the Netherlands)

Address: Behavioural Science Institute, Montessorilaan 3, Radboud University, Nijmegen, 6525 HE, The Netherlands, Tel: 0031 24 3615767, email: e.poelen@pwo.ru.nl

Drinking behavior of friends is assumed to play an important role in alcohol use of adolescents and young adults; young people who drink are more likely to affiliate with drinking peers, and contacts with drinking peers affect subsequent individual drinking over time. Twin research shows that drinking is partly predicted by genetic factors; monozygotic twins are more similar in their alcohol use than dizygotic twins. We used data of the Netherlands Twin Register to assess whether the extent to which twins shared their friends was related to the extent to which the twins were similar in their alcohol use. Twins reported on the friends they shared, the alcohol use of these friends and their own alcohol use. We used data of 1733 twin pairs in the age range of 12 to 26 years. Monozygotic twin pairs more often reported to share their friends than dizygotic twins (35% versus 8%). Furthermore, twins with common friends were more similar in alcohol use than twins with separate friends, and these effects were stronger for monozygotic than for dizygotic twins. When twins had separate friends and those friends were different in their alcohol use, the twins also differed in their alcohol use. This study shows the importance of alcohol use among friends for the drinking behavior of young people.

Evelien A. P. Poelen¹, Dorret I. Boomsma², Rutger C. M. E. Engels¹, Ron H. J. Scholte¹,
Gonneke Willemsen², Jan F. J. van Leeuwe¹

The Relative Contribution of Genes and Environment to Alcohol Use in Adolescents and Young Adults

¹ Radboud Universiteit Nijmegen, the Netherlands

² Vrije Universiteit Amsterdam, the Netherlands

Address: Behavioural Science Institute, Montessorilaan 3, Radboud University, Nijmegen, 6525 HE, The Netherlands, Tel.: 0031 24 3615767, email: e.poelen@pwo.ru.nl

Drinking habits and determinants of drinking can change rapidly during the transition from adolescence into young adulthood. Studies of the genetic contribution to variation in alcohol use found that the largest part in the variance of alcohol use in early and mid adolescence is explained by shared and unique environmental factors, while in late adolescence genetic factors become more important. Most previous research focused on a very restricted age range which did not allow for a comparison of the results for different age groups. The present study assesses the relative contribution of genes and environment to alcohol use among adolescents and young adults. As sex differences in alcohol use are well known, we also examined whether these relative influences differed between males and females. Data of the Netherlands Twin Register were available for 688 twin pairs in the age of 13-15 years, 744 twin pairs in the age of 16-18 years, 752 twin pairs in the age of 19-21 years and 569 twin pairs in the age of 22-24 years. Structural equation modeling took place in Mplus. There were no significant sex differences, except for the younger adolescents. Genetic factors explained more of the variance in alcohol use in older adolescents ($a^2 .77$) and young adults ($a^2 .67$ for 19-21- year olds and $.69$ for 22-24-year olds) than in younger adolescents ($a^2 .00$ for males and $.53$ for females). In the younger adolescents, shared environmental factors explained 81% of the variance in alcohol use in males in the age of 13 to 15 years and 25% of the variance in alcohol use in females, while they did not contribute to the variance of alcohol use in older adolescents and young adults. Unique environmental effects increased with age. These results confirm that genetic factors become increasingly important as adolescents grow older.

Thomas S. Price¹, Sara R. Jaffee²

Mediating And Moderating Effects Of Measured Family Environments In The Classical Twin Design

¹Institute for Translational Medicine and Therapeutics, U. Penn.

²Department of Psychology, U. Penn.

Address: ITMAT, School of Medicine, Room 807, BRB II/III, 421 Curie Boulevard, University of Pennsylvania, Philadelphia, PA 19104 USA, Tel: +1 215 898 0489, FAX: +1 215 573 9004, tom@spirit.gcrc.upenn.edu

Family-wide environments – environmental variables that are measured at the family level, such as SES – can have causal influence on phenotypes measured in the children belonging to that family. There can also be non-causal associations arising from passive and active gene-environment correlations, as well as moderating effects of the family environment on genetic influences on the phenotype. It has recently been shown that structural equation modeling and regression methods previously used for testing hypotheses of environmental mediation and moderation in twin data are inadequate for the purpose (Turkheimer E, et al. *Child Dev.* 2005;76(6):1217-33. Purcell S & Koenen KC, *Behav Genet.* 2005;35(4):491-8). We demonstrate that alternative statistical methods that model random rather than fixed effects of the measured family environment can accurately estimate the relevant parameters provided that the moderating effects of the environment are sufficiently large. We conclude that the classical twin study provides a useful alternative to extended family designs for investigating the effects of family-wide environments.

Adam S. Raefski¹, Michael J. O'Neill¹

Identification of Three Imprinted X-linked genes: Xlr3b, Xlr4b and Xlr4c in a Mouse Model for Turner's Syndrome

¹University of Connecticut

Grant Support: US National Institute of Neurological Disease and Stroke

Address: Molecular and Cell Biology, University of Connecticut, 354 Mansfield Road, U-2131, Storrs, Connecticut 06269-2131 USA, Tel: (860)486-3580, email: adam.raefski@uconn.edu

To date, approximately 80 imprinted genes have been identified, primarily in humans and mice[1]. These imprinted genes have all been found on autosomal chromosomes, and evidence of imprinted genes on the sex chromosomes has been elusive. Many of the genes that have been identified as showing imprinted patterns of expression are directly involved in fetal growth and development. Several autosomally inherited human diseases showing growth abnormalities are known to involve disruptions of normal mono-allelic expression of imprinted genes. In addition to growth abnormalities, mutations in imprinted loci frequently lead to neurobehavioral disorders. Skuse and colleagues hypothesized that certain neurobehavioral attributes of Turner's syndrome (resulting from X chromosome monosomy) involve imprinted genes on the X chromosome[2]. For individuals with Turner's syndrome there is a higher propensity for social communicative defects if the single X is maternally derived than if it is of paternal origin. An X-linked imprinted locus (loci) may also explain the greater propensity for autism and related disorders like Asperger's syndrome in males than females. We have utilized a mouse model for Turner Syndrome (X chromosome monosomy) to identify novel imprinted genes localized to the mammalian X chromosome. An initial microarray screen of 39,Xm and 39,Xp P0 whole brain samples lead to the discovery of the X-linked imprinted gene Xlr3b. Further analysis of this locus lead to the discovery of two more related imprinted genes Xlr4b and Xlr4c. While all three of these genes are paternally silenced they exhibit independence in imprinting regulation across developmental stages and tissue/brain subregions.

Chandra A. Reynolds¹

Genotype-environment interplay and cognitive aging

¹Psychology Dept., University of California, Riverside CA

Grant Support: R01-AG17561; R01-AG04563; R01-AG10175; MacArthur Foundation Research Network on Successful Aging; Swedish Council for Social Research (97:0147:1B)

Address: Psychology, UC Riverside, Riverside, CA, 92521, USA, Tel: 951-827-2430, email: chandra.reynolds@ucr.edu

The interplay of genetic and environmental markers on cognitive performance in the second half of the lifespan has been essentially unexplored. Nonshared environmental variance increases with age for most cognitive traits, which may indicate the presence of unaccounted genotype-environment interaction. We tested the presence of GxE interaction on latent growth parameters fitted to longitudinal cognitive profiles in twins from the Swedish Adoption/Twin Study of Aging (SATSA). A comparison of within pair variance ratios of MZ twins pairs stratified by candidate gene variants implicated in cognitive aging or dementia indicted the possibility of 'variability' genes for cognitive performance, including verbal and spatial abilities. Identification of possible markers of the nonshared environment (e.g., differences in social support, life events) that may interact with particular genotypes is investigated. Understanding gene-environment interplay may assist in identifying modifiable factors important to cognitive resilience.

Sally-Ann Rhea¹, Robin P. Corley¹, Josh B. Bricker¹

Predictors and Consequences of Negative Emotionality in the Colorado Adoption Project

¹Institute for Behavioral Genetics, University of Colorado, Boulder, CO

Grant Support: HD010333; HD036773; DA011015

Address: Institute for Behavioral Genetics, CU Boulder, Boulder, CO, 80309-0447, USA, Tel: 303-492-2822, FAX: 303-492-8063, email: rhea@colorado.edu

A previous exploration of the effects of divorce on early adult romantic relationships in the Colorado Adoption (CAP) sample indicated that the personality trait Negative Emotionality (NE) in fathers and their offspring was associated with unsuccessful romantic relationships. (S.A. Rhea, J. Bricker, and R.P. Corley, 2005, Behavior Genetics, 35, 818). In the current analyses, we explored possible genetic influences on this personality variable, as well as correlations between NE and several measures of late adolescent and early adulthood adjustment. First, we evaluated mother reports of fathers' NE and found significant correlations with the father's reports in all cases (.52, $p < .00$, $n=59$; .45, $p < .00$, $n=232$; and .43, $p < .00$, $n=238$ for birth, adoptive, and control parents, respectively). We were thus able to substitute mothers' reports of NE to increase our sample size given the small number of relinquishing birth father self-reports. Our expectation that we would find significant parent-offspring correlations with child NE at age 16 for both types of birth parents but not for adopting parents was not borne out as the correlations were modest and significant only for control fathers (.13, $p < .01$, $n=425$). Sibling correlations were non-significant for both related and unrelated pairs, indicating that neither additive genetic influences nor shared environment are major contributors to this measure of NE. However, NE at age 16 was significantly correlated to several measures of stability, e.g. sociopathy in late adolescence (.33, $p < .001$, $n=841$) and partner aggression in early adulthood (.24, $p < .001$, $n=768$). We found modest but significant sibling correlations for these measures for both related and unrelated sibling pairs, indicating that shared environment is influential on these behaviors. Although the antecedents of this measure of NE are still obscure, it is clearly not merely measurement error as it predicts early adulthood outcomes.

Joseph Lee Rodgers¹, David Bard¹, Warren Miller²

The Mother-Daughter-Aunt-Niece (MDAN) Design, Applied to Cross-Generational NLSY Fertility Variables

¹University of Oklahoma

²Transnational Family Research Inst

Grant Support: NICHD Grant RO1-HD043265

Address: Department of Psychology, Professor, 455 W Lindsey, University of Oklahoma, Norman, OK, 73019, Tel: 405-325-4591, FAX: 405-325-4737, email: jroddgers@ou.edu

A new biometrical design – called the MDAN design – emerges from the complex longitudinal survey design of the National Longitudinal Survey of Youth (NLSY) data. Using the cross-generational structure available in the NLSY, we link mothers to daughters and aunts to nieces, creating an MDAN (mother-daughter-aunt-niece) design. The cross-generational data include NLSY-females who are only mothers, those who are only aunts, and those who are both mothers and aunts. Further, there is within-generational biometrical information linking NLSY-Youth females to one another as cousins, half-siblings, full-siblings, and twins; and linking NLSY-Children females to one another as cousins, half siblings, full siblings, and twins. We create linking files identifying the various within- and between-generational links, and fit preliminary biometrical models using those links. Phenotypes are fertility variables, typically measured across the two generations at approximately the same age and using identical measurement instruments. Specific measures on which we focus include self-reported age at menarche and self-reported age at first intercourse. Previous research using biometrical models have studied these phenotypes within each generation; the current research substantially extends both the empirical results and the methodological innovation by taking advantage of the ability to fit three different types of genetically- and environmentally-informed structure simultaneously.

Angelica Ronald¹, Francesca Happé¹, Robert Plomin¹

What parents, teachers and children can tell us about different autistic traits: A twin study

¹SGDP Centre, Institute of Psychiatry, London

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Address: SGDP Centre, Box P083, De Crespigny Park, Denmark Hill, Institute of Psychiatry, London, London, SE5 8AF, UK, Tel: +44 207 848 0396, FAX: +44 207 848 0866,

a.ronald@iop.kcl.ac.uk

Different informants might provide divergent information about autistic-like behaviors, an important consideration in clinical practice as well as for selecting phenotypes for molecular genetic research. However, no previous twin study has studied whether different raters tap into the same underlying phenotype for autistic traits. The objectives of this study were to assess rater agreement in the general population and at the extreme, as well as the extent to which autistic traits rated by different informants show the same causal influences. The degree to which different autistic traits are caused by the same genes and environments was also assessed. Teacher, parent, and child self-report ratings for 9-year-old twins (N>3000 pairs) were collected using the Childhood Asperger Syndrome Test in the Twins Early Development Study. Multivariate twin model fitting was carried out. Low phenotypic correlations were found between raters (.11-.33). For all raters, all autistic traits showed genetic influence but there was a degree of genetic heterogeneity between the different domains, particularly between social impairments and restricted repetitive behaviors and interests (genetic correlations = .29-.50). Between different raters, there was some degree of shared genetic and environmental effects, but also rater-specific causal influences. Autistic behaviors appear to be genetically heterogeneous, a finding that agrees with two previous studies (A. Ronald, F. Happé, and R. Plomin, 2005, *Dev. Science*, 8, 444-458; A. Ronald, F. Happé, P. Bolton, L. M. Butcher, T. S. Price, S. Wheelwright, S. Baron-Cohen, and R. Plomin, in press, *J. Am. Acad. Child. Adolesc. Psychiatry*) and which may be important to take in account when designing molecular genetic studies. These data suggest that multiple raters are needed to assess autistic traits in different contexts. Clinicians are likely to receive somewhat different phenotypic information from parents, teachers, and children themselves.

Eligible for Thompson Award

Pekka Santtila¹, N. Kenneth Sandnabba¹

Preliminary evidence for genetic effects on sexual aggression

¹Department of Psychology, Åbo Akademi University

Grant Support: The Academy of Finland, The Åbo Akademi Foundation

Address: Department of Psychology, Tehtaankatu 2, Åbo Akademi University, Turku, Finland, 20500, Finland, Tel.: 358456303636, email: pekka.santtila@abo.fi

There is extensive evidence that antisocial, including aggressive, behavior is influenced by genetic factors (S. H. Rhee, and I. D. Waldman, 2002, *Psychol. Bull.* 3, 490-529) with subtypes of aggression being differentially controlled by genes and environment (L. Ligthart, M. Bartels, R. A. Hoekstra, J. J. Hudziak, and D. I. Boomsma, 2005, *Twin. Res. Hum. Genet.* 8, 483-491). We explored the existence of genetic influences on sexual aggression in a sample of Finnish male twins. Sexual aggression was measured using a revised version of the Sexual Experiences Survey (SES; M. P. Koss, and C. J. Oros, 1982, *J. Consult. Clin. Psychol.* 50, 455-457). Of the 1312 male respondents 26% had engaged in at least one type of sexual aggression. Preliminary analyses suggested that genetic factors accounted for 31% of variance in verbal sexual aggression with the rest being accounted for by nonshared environment. Variations in physical sexual aggression were mainly influenced by nonshared environmental influences with 13% of variations attributable to genetic effects although this effect was not statistically significant. Sexual aggression was positively associated with other types of antisocial behavior with some indication of shared genetic factors being responsible for the correlation. The implications of the results for understanding sexual aggression will be discussed.

Carolyn E. Sartor¹, Michael Lynskey¹, Andrew C. Heath¹

Childhood sexual abuse and course of alcohol dependence in a female twin sample

¹Washington University School of Medicine

Grant Support: NIAAA AA56583D; NIDA DA18660; NIAAA AA09022; NIAAA AA11998

Address: Psychiatry, Campus Box 8134, 660 S. Euclid Avenue, Washington University School of Medicine, Saint Louis, MO 63110 USA, Tel: 314-747-1415, email: carolyn@matlock.wustl.edu

Childhood sexual abuse (CSA) has been associated with increased risk for developing alcohol dependence (AD) after accounting for familial liability to AD (E.C. Nelson et al., 2002, Arch Gen Psychiatry 59 139-145), but the impact of CSA on the course of AD development remains largely unknown. The current study used survival analysis to examine CSA as a potential predictor of age of first alcohol use and time from first drink to AD onset in a female twin sample, controlling for co-twin AD status. The sample consisted of 3,538 female twins (954 MZ pairs and 815 DZ pairs) from the Missouri Adolescent Female Twin Study (MOAFTS; Heath et al, 2002, Twin Research 5 107-112). Mean age of participants was 21.6 years; 87% identified as Caucasian. CSA and substance use histories were assessed using a modified version of the SSAGA-II. CSA, defined as sexual molestation or rape prior to age 16, was reported by 11% of participants; 7.3% met lifetime criteria for AD.

A logistic regression analysis controlling for AD status of the co-twin revealed elevated risk for AD among CSA-positive participants (OR=1.96; CI= 1.42-2.71). Age of first drink was 15.6 years for participants with CSA histories and 16.6 years for those without. Time from first drink to AD onset was 3.6 years for CSA positive twins and 3.8 years for CSA-negative twins. Cox proportional hazard regression analyses predicting a) age of first alcohol use and b) time from first drink to AD onset revealed non-significant hazard ratios for CSA status (HR=1.11; CI=0.97-1.26 and HR=0.93; CI=0.67-1.27, respectively) after controlling for AD status of the co-twin in both models as well as age of first drink in the AD model. Findings provide additional evidence for elevated risk of AD among survivors of CSA after controlling for familial liability to AD, but the absence of distinctions in age that alcohol is first consumed and rate of progression to AD suggest that CSA has little impact on the course of AD development.

Kimberly J. Saudino¹

Cross-situational and Context Specific Effects on Activity Level

¹Boston University, Boston, MA 02215

Grant Support: Supported by grant MH062375 from the National Institute of Mental Health.

Address: Psychology Department, 64 Cummington St., Boston University, Boston, MA, 02215, USA, Tel: 617-353-3679, email: ksaudino@bu.edu

Research exploring cross-situational and context specific genetic influences on individual differences in activity level (AL) in early childhood has suggested substantial context-specific genetic variance in addition to cross-situational genetic variance (Philips, K. & Matheny, A., 1997, *J Per Soc Psychol*, 73, 129-138; Schmitz, S., Saudino, K., Plomin, R., et al., 1996, *Child Dev*, 67, 409-422). However, these studies confound situational differences with method differences. In the present study, cross-situational and context-specific genetic effects for AL at age 2 were examined using mechanical motion recorders (acticals) to assess AL in the home, lab play, and lab test situations. Observer ratings of AL in the lab and parent ratings of AL were also obtained. Preliminary analyses based on data for 106 MZ and 125 DZ twin pairs found that for all measures and in all situations, AL was significantly heritable. Multivariate analyses of the actical data found that the same genetic factors operated across all 3 situations—there was no genetic variance specific to any one situation. There were, however, substantial shared environmental influences unique to the home situation. Similarly, there were significant nonshared environmental influences specific to each situation. Correlations between actical AL in different situations were due only to genetic influences. Similar results emerged for analyses of observer rated AL in the 2 lab situations (i.e., cross-situational genetic influences; contextual environmental influences). Multivariate analyses of different measures of AL within the same situation (e.g., actical lab and observer lab; actical home and parent ratings) produced interesting results. The actical and the observer within the lab situation tap the same genetic effects, but the actical in the home and parent ratings of AL do not. Almost all of the genetic variance for parent-rated AL (92%) was independent of that for the actical measure.

Nancy L. Segal¹, Shirley McGuire², June Havlena¹, Patricia Gill²

IQ Similarity in Virtual Twins: Developmental Trends

¹California State University, Fullerton

²University of San Francisco

Grant Support: NIMH

Address: Psychology, 800 N. State College Blvd., CSU Fullerton, Fullerton, CA, 92834, USA,
Tel: 714-278-2142, email: nsegal@fullerton.edu

Virtual twins (VTs)--same-age, unrelated siblings reared together from infancy—replicate twinship, but without the genetic relatedness. The last paper from this ongoing study (Segal & Hershberger, 2005) reported an IQ intraclass correlation of .26 ($p < .01$, $n = 113$ pairs) and a IQ subtest profile correlation of .07 ($n = 111$). New data from an increased sample confirms these findings ($n = 120$ pairs). Additional IQ data, gathered from a subset of VT pairs, has allowed longitudinal analyses of intellectual similarity. These results will eventually be complemented by IQ data gathered from a new study of Chinese MZ and DZ female twin children reared apart, due to their separate adoptions under China's One-Child Policy.

Michael J. Shanahan¹, Lance D. Erickson², Stephen Vaisey¹

Neurogenetic Polymorphisms and the Adolescent Educational Career

¹University of North Carolina, Chapel Hill

²Brigham Young University

Grant Support: NIMH

Address: Sociology, CB 3210, Hamilton Hall, UNC - Chapel Hill, Chapel Hill, NC, 27599, Tel: (919) 843 9865, email: mjshan@unc.edu

Animal and human research identifies genetic variation associated with neurotransmitters and receptors as likely candidates involved in a range of antisocial behaviors and psychopathology, including impulsivity, behavioral disinhibition, substance use, externalizing symptoms, personality attributes such as hostility, and oppositional defiant and conduct disorders. The most prominent neurotransmitter candidates are monoamines, the concentration of which in the synaptic cleft is the primary determinant of the intensity of neuronal signaling. “Low levels” of signaling are thought to covary with inability to restrain impulses, impaired learning, disregard for consequences, and insensitivity to cues for punishment. Given that such behaviors are dysfunctional in school settings, neurogenetic polymorphisms associated with monoamines should be related to educational achievement and attainment. We examine this expectation by drawing on DNA, survey, and school transcript data from the National Longitudinal Study of Adolescent Health. Results reveal that SNPs are related to school outcomes, but these relationships are highly conditioned by social context.

Chizuru Shikishima¹, Shinji Yamagata², Kai Hiraishi³, Junko Ando⁴, Yutaka Ono⁵

Parental warmth and empathy: Familial influences reconsidered

¹Graduate School of Human Relations, Keio University, Tokyo

²Department of Cognitive and Behavioral Science, University of Tokyo, Tokyo

³College of Arts and Sciences, University of Tokyo, Tokyo

⁴Faculty of Letters, Keio University, Tokyo

⁵Health Center, Keio University, Kanagawa

Address: Human Relations, 2-15-45 Mita, Graduate School of Keio University, Minato-ku, Tokyo, 108-8345, Japan, Tel: 81-3-5427-1180, email: kana-s@sa2.so-net.ne.jp

Many psychological and sociological studies have sought to discover the association between variations in parenting and children's later psychological development. Bowlby's attachment theory predicts that individuals with secure attachments should be more likely to be empathic than should individuals with insecure attachments (J.A. Bowlby, 1988, *A Secure Base: Clinical Applications of Attachment Theory*, Routledge, London). However, our bivariate genetic analyses employing 1,492 Japanese adolescent and adult twins (334 MZf, 158 MZm, 96 DZf, 48 DZm, and 84 DZo pairs) revealed that the association between parental warmth and empathy was not attributable to shared environment but to children's genetics. Hence, our result did not support the hypothesis that parenting shaped children's empathy afterwards as a main effect. However, our further analyses employing the gene-environment interaction model (S. Purcell, 2002, *Twin Res.* 5, 554-571) demonstrated that the shared environmental effect for children's later empathy was moderated by the parental warmth level. The effect of shared environment was absent when parental warmth was around the mean level, while it was drastically increased when parental warmth was very high. Taking such a moderating effect in the family into account, "environment-experience interactions" should be focused on when family influences on children's later traits are investigated.

Eligible for Thompson Award

Erica L. Spotts¹, Paul Lichtenstein², Jenae M. Neiderhiser¹

The truth about cats and dogs: Are there genetic influences on pet ownership?

¹George Washington University

²Karolinska Institute

Grant Support: This project was supported by grant RO1 MH54610 from the National Institutes of Mental Health.

Address: Center for Family Research, 2300 K Street, NW 3rd Floor, George Washington University, Washington, DC, 20037, USA, Tel: 202.994.2404, FAX: , 202.994.4812, email: cfrels@gwumc.edu

Pets are an important part of many people's lives, as substantiated an increasing amount of research. Having a pet plays a beneficial role in mental and physical health (e.g. NA Pachana et al, 2005, *International Journal of Behavioral Medicine*, 12, 103-110). Additionally, there is evidence that people choose dogs that resemble them in some way (MM Roy & NJS Christenfeld, 2004, *Psychological Science*, 15, 361-363). Along with findings from empirical research comes lore suggesting that there are "cat people" and "dog people", suggesting that pet selection is linked to underlying personality traits. To explore this issue, we used the Twin Offspring Study in Sweden, a sample drawn from the Swedish Twin Registry. It consists of 233 female-female and 350 male-male adult twin pairs and their spouses. For the current study, we asked the following research questions. Do people who own pets differ in their mental health and personality from people who do not own pets? Of people who own pets, are there mental health and personality differences according to the type of pet owned? Finally, are there genetic and environmental influences on pet ownership and on any links between pet ownership and health? Preliminary findings suggest few differences between pet and non-pet owners and few differences based on the type of pet owned. Not owning a pet is slightly influenced by genetic factors, but shared and nonshared environmental factors are the predominant influence on pet ownership. Shared and nonshared environmental influences also influence the type of pet that is owned. These findings do not support the notion of "cat people" and "dog people" as an indicator of an inherent personality trait.

Jennifer A. Ulbricht¹, Jody Ganiban¹, Jenae M. Neiderhiser²

Marital Quality as a Moderator for Genetic and Environmental Influences on Parenting

¹Department of Psychology, George Washington University, Washington, DC

²Center for Family Research, George Washington University, Washington, DC

Grant Support: William T. Grant Foundation R01MH43373 (Reiss); R01MH59014 (Neiderhiser)

Address: Psychology, 2125 G St. NW, George Washington University, Washington, DC 20037
USA Tel: 571-212-5006, email: jau5b@gwu.edu

Marital quality has been linked to child externalizing and internalizing behaviors in a number of studies, often with parenting as the mediator (Reid & Crisafulli, 1990, *J. Ab. Ch. Psy.*, 18, 105-117). However, parenting is not affected by marital conflict in all cases. Genetic influences have been found for both marital quality and parenting with more genetic influence on parenting (e.g., Reiss, Neiderhiser, Hetherington, & Plomin, 2000. *The Relationship Code*. Harvard University Press). In family systems, where common genes and environment are likely to play complex roles, it is crucial to consider both genotype-environment correlations and interactions. The goal of the present study is to examine whether marital satisfaction moderates the influence of genetic and environmental factors on parenting behavior. We hypothesize, based on family process theory and previous analyses, that estimates of genetic influence on parenting will change at different levels of marital conflict. Data for this study were from the initial wave of the Nonshared Environment in Adolescent Development study. The sample consisted of a total of 720 families with same-sex sibling pairs, ages 10-18. A range of family types was sampled, with 93 MZ, 99 DZ twin pairs, and 95 sibling pairs from non-divorced families and 182 sibling, 109 half-sibling, and 130 unrelated sibling pairs from stepfamilies. Measures of marriage and parenting were taken by self-report and observer-rated taped dyadic interactions. Children also reported on parenting. Composites for marital and parenting constructs were compiled and have been used in a number of previous studies. A model of latent GE interaction developed by Purcell (2002, *Twin Research*, 5, 572-576), will be used to explore the G x marriage effects on parenting behaviors with both marital relationship and parenting as continuous variables. In addition, genotyping is available for a subset sampled in wave three of NEAD, and will be considered for further analysis.

Markus Varjonen¹, Pekka Santtila¹, N. Kenneth Sandnabba¹

Genetic and Environmental Effects on Sexual Excitation and Sexual Inhibition in the Human Male

¹Department of Psychology, Åbo Akademi University

Address: Psychology, Arken, Biskopsgatan 3, Åbo Akademi University, Turku 20500, Finland,
Tel: 358 22 1544 03, email: mvarjone@abo.fi

The Sexual Inhibition and Sexual Excitation Scales (SIS/SES) measure the propensity for sexual inhibition and excitation in men (E. Janssen, H. Vorst, P. Finn & J. Bancroft, 2002, *The Journal of Sex Research* 39, 114-126). According to the theoretical model underlying the SIS/SES, sexual response and associated behavior in the male depends on dual control mechanisms in the brain involving the balance of one excitatory and two inhibitory systems which impinge on sexual response (J. Bancroft, 1999, *Neuroscience & Biobehavioral Reviews* 23, 763-84). The present study estimated the heritability and the environmental influences on the excitatory and inhibitory mechanisms on a population based sample of male Finnish twins (N=1289) using the classical twin study design. The twin correlations and the preliminary results from the structural equation modeling suggested a modest heritability for both the inhibitory mechanisms. Sexual excitation, in contrast, was not influenced by genetic effects and similarities in this respect between twins seemed to be caused by the common environment of the twins. The results and their implications were discussed.

Eligible for Thompson Award

Irwin D. Waldman^{1,2}, Ian R. Gizer¹, Jesen A. Fagerness², Casey L. McGrath², David C. Rowe⁴

Is COMT a Risk Factor for ADHD? Testing for Association with Multiple Markers in a Gene-Based Framework

¹Emory University, Atlanta, GA

²Psychiatric and Neurodevelopmental Genetics Unit, Center for Human Genetic Research, Massachusetts General Hospital, Boston, MA

³Late of Program in Genetics and Cell Biology, University of Arizona, Tucson, AZ

Address: Psychology, 532 N. Kilgo Circle, Emory University, Atlanta, GA 30322, USA, Tel: 404-428-3293, FAX: 404-727-0372, email: psyiw@emory.edu

Despite the importance of including multiple markers in candidate genes in association studies, much confusion persists about the best ways to implement such studies. Recently, a gene-based approach to association has been posited in which the focus is on the gene itself, rather than on its constituent markers or haplotypes, in an effort to increase replication across studies. This conceptual advance has raised several important issues regarding marker selection and the best analytic approaches to use in testing for association. We address several of these issues by reexamining the association between the catechol-o-methyl-transferase gene (COMT) and childhood ADHD. We tested the association of multiple SNPs in COMT with ADHD and its constituent diagnostic subtypes and symptom dimensions using several analytic strategies in a predominantly clinic-referred sample of children aged 4-18. All of the probands met DSM-IV criteria for ADHD, as did ~33% of their siblings. As in previous studies, we genotyped the functional val/met substitution at codon 158 of COMT, as well as an additional 24 SNPs throughout the gene and its 5' and 3' flanking regions. A recent meta-analysis of 13 association studies failed to find any association between COMT and childhood ADHD (OR = 0.99). Consistent with these findings, in our sample there was no association between ADHD and the val/met SNP (OR = 1.03). Nonetheless, preliminary analyses suggested an association between COMT and ADHD using a multivariate, multi-marker test of SNPs across the gene. Follow-up TDTs of individual SNPs suggested association of ADHD with a set of SNPs located ~3 kb 3' of the val/met SNP. These results suggest that COMT is associated with ADHD but that this is not due to the val/met SNP, thus highlighting the need to search for additional functional variants in COMT. These findings also suggest the advantages of a gene-based approach to testing for association in psychiatric genetic research more generally.

Alex Weiss¹, Timothy C. Bates¹

Heritability of subjective wellbeing in a representative sample

¹ University of Edinburgh

Address: Psychology, 7 George Square, University of Edinburgh, Edinburgh, Scotland, EH8 9JZ, UK, Tel: 44 131 651 1945, email: tim.bates@ed.ac.uk

The MIDUS project has collected a range of behavioral data from a representative US sample, including a substantial number of pairs of MZ and DZ twins. Analyses are presented on the heritability of well being in this sample, which suggest a modest (0.3) heritability, with some evidence for sex-limitation. A multivariate model including scales assessing the five major factors of personality revealed genetic correlations between well-being and personality (N in particular), and a reduced model indicated that the genetic effects on wellbeing are, within the power of these analyses, reducible to the genetics of the five factor model.

Katarina Witting¹, Pekka Santtila¹, N. Kenneth Sandnabba¹

Genetic influence on female sexual function

¹Department of Psychology, Åbo Akademi University

Address: Department of Psychology, Åbo Akademi University, Åbo, FIN-20500, Finland, Tel: +358-40-8699 555, email: katarina.witting@abo.fi

Two recent twin studies reported a genetic influence on female orgasmic function with heritability estimates ranging between 31% and 51% (K. Dawood, K. M. Kirk, J. M. Bailey, P. W. Andrews, and N. G. Martin, 2005, *Twin Research and Human Genetics*, 8, 27-33; K. M. Dunn, L. F. Cherkas, and T. D. Spector, 2005, *Biol. Lett.* 1, 260-263.). We investigated genetic influences on several domains of female sexual function, as measured by the Female Sexual Function Index (FSFI, R. Rosen, C. Brown, J. Heiman, S. Leiblum, C. Meston, R. Shabsigh, et al., 2000, *Journal of Sex and Marital Therapy*, 26, 191-208), using the classical twin study. The FSFI was part of an extensive sexuality questionnaire, sent out to a population-based sample of Finnish twins between 33-43 years of age. The genetic, shared environmental, and non-shared environmental influences were estimated using intraclass correlation coefficients followed by structural equation modeling. The response rate for the female twins was 45% resulting in 2267 respondents. According to preliminary analyses, there were small genetic influences for desire, arousal, lubrication, orgasm, and pain, but not for satisfaction. Most of the variance was due to non-shared environmental effects. The results showed that there are small but significant genetic influences on several dimensions of female sexual functioning. These results are one step further of reaching an understanding of the complexity of female sexual functioning. The age span of the respondents was quite narrow and in order to see whether the genetic influences are the same throughout adulthood further studies are needed.

David J. Vandenberg^{1,2}, Richard J. O'Connor¹, Michael D. Grant^{1,2}, Akilah L. Jefferson², George P. Vogler^{1,2}, Andrew A. Strasser¹, and Lynn T. Kozlowski¹

Evidence for Epistasis among the D2 Family of Dopamine Receptor Genes in Smoking-Related Behaviors

¹Department of Biobehavioral Health

²Center for Developmental and Health Genetics, The Pennsylvania State University, University Park, PA 16802

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Address: Center for Dev. & Health Genetics, Penn State University, University Park, PA 16802 USA, Tel: (814) 863-8430, FAX: (814) 863-8429, email: djv4@psu.edu

One mechanism for generating phenotypic variance by genetic means is through epistatic (gene-gene) interactions, whereby the phenotypic effect of allele status at one locus is dependent on allele status at a second locus. In studies of human behaviors, epistasis is often not considered in candidate gene approaches, but may represent an important avenue to understanding the underlying biology of the behavior, and perhaps explain contradictory data in the literature. Cigarette smoking, like many addictive behaviors, is related to dopamine release in several reward-related brain regions, and thus alleles of the genes that encode dopamine D2-like receptors (DRD2, DRD3, and DRD4) are candidates for contributing to these behaviors. Phenotypic information concerning smoking-related behaviors from a nationally representative sample of research volunteers was analyzed for association with polymorphisms in these genes. Quantity/frequency measures (Heaviness of Smoking and Number of Cigarettes Per Day) were associated with single genes in this family; however, phenotypes related to symptoms of withdrawal (Nervousness, Trouble Sleeping, and Trouble Concentrating) while displaying no association with single genes, were influenced by significant interactions among alleles at 2 or 3 of the genes in this family. These analyses indicate that epistatic interactions may be an important part of smoking behaviors that warrant further study.

Philip A. Vernon¹, Ashley Mackie¹, K.V. Petrides², Janice Bacher¹

A behavioral genetic study of trait emotional intelligence

¹Department of Psychology, University of Western Ontario

²Institute of Education, University of London

Grant Support: Social Sciences and Humanities Research Council of Canada

Address: Department of Psychology, University of Western Ontario, London, Ontario, N6A 5C2, Canada, Tel: 519-661-3682, FAX: 519-661-3961, email: vernon@uwo.ca

Trait emotional intelligence (trait EI) is a constellation of emotion-related self-perceptions and dispositions located at the lower levels of personality trait hierarchies. In this presentation, we report the results of the first ever behavioral genetic (BG) investigation of EI in general, and of trait EI, more specifically. Two hundred pairs of adult twins completed the Trait Emotional Intelligence Questionnaire (TEIQue), a 153-item questionnaire providing comprehensive coverage of the sampling domain of trait EI (15 subscales, 4 factors, and global score). Participants also completed the NEO PI-R as a measure of the Big 5 personality traits. Preliminary univariate BG analyses reveal that most trait EI dimensions are best fit by an AE model with h^2 values ranging between .20 to .70. The heritability of the global score is .74. Phenotypic, genetic, and environmental correlations between the trait EI dimensions and the Big 5 will also be reported. Overall, the findings are fully in line with the conceptualization of emotional intelligence as a personality trait.

Gonneke Willemsen¹, Dorret I. Boomsma¹, Eco J.C. de Geus¹, Danielle Posthuma¹

BMI and waist circumference: an expression of the same genes?

¹Department of Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands

Address: Biological Psychology, van der Boechorstraat 1, Vrije Universiteit, Amsterdam, 1081BT, The Netherlands, Tel: +31-20-5988787, FAX: +31-20-5988832, ahm.willemsen@psy.vu.nl

Body mass index (BMI) and waist circumference have both been used in studies of obesity. There is a strong association between these two measures and both measures have been shown to be determined for a large extent by genetic factors, i.e. by a set of common genes. No studies as yet have determined whether linkage analyses indeed deliver the same results for BMI as for waist circumference. We examined this question by using data from two studies of the Netherlands Twin Register in which BMI and waist circumference were measured simultaneously. Body composition data are available for more than 1089 twins and siblings, coming from 491 families and including 75 male monozygotic and 105 female monozygotic twin pairs. In addition, whole genome scan data are available. BMI and waist circumference are highly correlated ($r=.82$, $p<.001$). The pattern of twin correlations suggests equal heritability for BMI and waist circumference, with lower heritability estimates for women than for men. Using structural equation modelling we will first determine the extent to which the two body composition measures are determined by the same genes. Next, linkage analyses will be conducted to determine the location of these common genes.

Gonneke Willemsen¹, Eco J.C. de Geus¹, Dorret I. Boomsma¹

The effect of urbanisation on personality and its heritability in older Dutch twins and their family members.

¹Department of Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands

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Address: Biological Psychology, van der Boechorstraat 1, Vrije Universiteit, Amsterdam, 1081BT, The Netherlands, Tel: +31-20-5988787, FAX: +31-20-5988832, ahm.willemsen@psy.vu.nl

Between 1991 and 2002 we collected personality data in Dutch twins and their family members. From the larger data set we selected all subjects born before 1960. There were 581 (470 complete) twin pairs, 370 of their spouses and around 500 of their siblings. In addition, personality data were assessed in more than 2600 pairs of parents of younger twins. Personality measures included neuroticism, extraversion, sensation seeking, anxiety and anger. Familial resemblance in twins and siblings is influenced by genetic factors. There are low to moderate spousal correlations (e.g. around 0.1 for neuroticism, extraversion and around 0.3 for sensation seeking scales). The influence of genetic factors may be modified by social factors. We analyze the influence of urbanization level of the residential area as a moderator variable. In a recent paper we showed that this is one of the few traits that do not show heritability in younger or older subjects.

Shinji Yamagata¹, Yusuke Takahashi¹, Nobuhiko Kijima², Yutaka Ono³, Junko Ando⁴

Experience of stressful life events rather obscures genetic vulnerability of depression and anxiety in normal population: A study of gene-environment interaction.

¹Department of Cognitive and Behavioral Science, The University of Tokyo, Tokyo, Japan

²Psychological Laboratory, Keio University, Kanagawa, Japan

³Health Center, Keio University, Kanagawa, Japan

⁴Faculty of Letters, Keio University, Tokyo, Japan

Address: Cognitive and Behavioral Science, Shigemasu Laboratory, 3-8-1 Komaba, The University of Tokyo, Meguro-ku, Tokyo, 158-0093, Japan, Tel: 0426-65-3357, email: yamagata@bayes.c.u-tokyo.ac.jp

The present study examined whether the amount of stressful life events experienced moderate genetic and environmental influences on depressive and anxious mood. Data of 295 monozygotic and 132 dizygotic twin pairs who participated in Keio Twin Project was analyzed. Conventional univariate analyses revealed that experience of stressful life events, depressive and anxious mood were all explained by additive genetic and nonshared environmental influences, with heritability being .46, .28, and .29, respectively. Experience of stressful life events was only weakly correlated with depressive and anxious mood, both genetically ($r_g = .24$ and $.17$, respectively) and environmentally ($r_e = .17$ and $.16$, respectively). However, analyses of gene-environment interaction using continuous moderator variables (S. Purcell, 2002, *Twin Res*, 5, 554-571) revealed that experience of stressful life events significantly altered the genetic and environmental etiology of both depressive and anxious mood; for those who experienced more stressful life events, genetic influences on anxious mood were smaller and environmental influences on both depressive and anxious mood were larger. These results suggest that linkage/association study of depression and anxiety using normal population would benefit from selecting subpopulation who experienced few stressful life events: an opposite prediction from diathesis-stress model.

Eligible for Thompson Award

Rowe A. Young¹

VISUAL AND MOTOR BEHAVIORS INTERACT TO IDENTIFY ASPECTS OF DYSLEXIA

¹Tucson, Arizona

Address: 5853 N.Paseo Niquel, Tucson, AZ 85718 USA, Tel: 520 299-1250, FAX: 520 531-1377, email: rowey@aol.com

Dyslexia is often theorized to have a primarily phonological basis. Recent genetic and hemispheric brain studies have been interpreted as consistent with this theory. Our work suggests alternative/additional reasons for a substantial part of the dyslexia syndrome. We hypothesize an interactive lateralized visual and inverted sensory motoric contributory basis. At the 1987 BGA meeting, we suggested motor and visual lateral interactive behavior variables (not just phonological processing) were related to reading disabilities. Further refinement of the research protocol and the use of additional observations and variables that we call "Inverted Direction Processing" (IDP) and "Inconsistent Visual Dominance"(IVD), have yielded new insights into these relationships. IVD is found by checking for eye dominance using two different lateral eye/hand positions to sight a target. In our pooled sample of 1120 observations from multiple populations, we find consistent right sighting is found in only 28%, consistently left sighting is found in 12%, and mixed or inconsistent sighting dominance is found in 60%. IDP is an observable motoric response to rotational stimuli, thought to represent a dominant mirror image sensation of movement and its behavioral consequences. One sample from a 4 year Community College, identified a large number of subjects with IDP (52 out of a total of 155). In this sample, tests reveal that consistent eye dominance patterns, are significantly correlated with higher standardized reading scores. Concurrently our LD family testing and pedigree data support a familial component to IDP and IVD manifestation. Further research could assess the linkage of these IDP and IVD characteristics to genetic markers. Implications for improving reading skills in individuals with IDP and IVD are addressed.

Mo Zheng¹, Laura A. Baker¹

**The role of inattention and hyperactivity in oppositional behavior in 9-10 year old children:
A twin study**

¹Psychology Department, University of Southern California

Address: SGM 501, Psychology, USC; Los Angeles CA 90089-1061 USA; 213 821-2959;
email: zheng@usc.edu

Attention Deficit Hyperactivity Disorder (ADHD) and Oppositional Defiant Disorder (ODD) are two of the most common externalizing behavioral disorders in children and adolescents, and they have been frequently observed to co-occur. To understand the etiology of their comorbidity, the authors studied 605 9 to 10 year old twin pairs from the Southern California Twin Study (277 Monozygotic and 328 dizygotic twins). Parents' ratings of ADHD and ODD symptoms for each twin were assessed by the Diagnostic Interview Schedule for Children Version 4 (NIMH DISC-IV). A multivariate genetic model was fit to assess the heritability of each disorder and the contribution of genetic and environmental factors to their association. The results of this study suggest that the co-occurrence of ADHD and ODD is primarily due to their common genetic influence.