Variability in alcohol preference in Maudsley reactive inbred male rats

Nelson Adams

1Winston-Salem State University. Grant Support: NIH-NIGMS-MBRS-08040, Social Sciences, 601 Martin Luther King Jr. Drive, Winston-Salem, NC 27110 USA, e-mail 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 [Adams N, Blizard DA (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 h will later display higher AP than E-naïve controls. Recently, we varied the amount of 10% E to either 4 ml or 8 ml on the day before 2-bottle choice between 10% E and water. Rats were either deprived (D) of water for 21 h or non-deprived (ND) at the time of E exposure. Results showed that D rats consumed their 4 ml 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.

Association of CHRM2 with IQ

Fazil Aliev1, 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

1Department of Psychiatry, Box 8134 CID, 660 South Euclid Ave., Washington University in St. Louis School of Medicine, St. Louis, MO 63110 USA, e-mail: aliev@matlock.wustl.edu, 2Ankara University, Ankara, Turkey, 3University of Iowa, Iowa City, Iowa, 4University of Connecticut School of Medicine, Farmington, CT, 5University of California at San Diego, San Diego, CA, 6Indiana University, Indianapolis, IN, 7SUNY Health Science Center at Brooklyn, Brooklyn, NY
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) Int J Psychophysiol 53:75–90], 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 et al (2003) Molecular Psychiatry 8:10–13] 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.

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

Juko Ando1, Shinji Yamagata2, Yusuke Takahashi2, Koken Ozaki3, Kunitake Suzuki4, Ryoko Nakajima5, Koichi Nonaka6, Noriko Kato7, Syuichi Ookï8

1Faculty of Letters, 2-15-45 Mita, Keio University, Minato-ku, Tokyo, 108-8345 Japan, e-mail: juko@msa.biglobe.ne.jp, 2Department of Cognitive and Behavioral Science, The University of Tokyo, Tokyo, Japan, 3Japan Science and Technology Agency, Tokyo, Japan, 4Tokyo Metropolitan University, Tokyo, Japan, 5Graduate School of Human Relations, Keio University, Tokyo, Japan, 6Wako University, Tokyo, Japan, 7National Institute of Public Health, Saitama, Japan, 8Ishikawa Prefectural Nursing University, Ishikawa, Japan, *Supported by Japan Science and Technology Agency (JST)

More than one thousand pairs of infant twins have entered a new longitudinal study called Tokyo Twin Cohort Project (ToTCop), a part of “Brain Science and Education” programs by JST. The present study reports quantitative genetic investigation on developmental trajectories of their weight, body length (height), chest circumference, and head circumference at birth, 3 months and 9 months of age. At birth, all the four physique measures show substantial additive genetic and shared environmental effects as well as nonshared environmental ones. Most of shared environmental contributions are explained by gestation age and decrease to zero at 9 months of age. Additive genetic contributions increase drastically from 3 months to 9 months of age for all the measures meanwhile their phenotypic variances become stable (for weight) or even decrease (for height and chest//head circumference). Multivariate genetic analysis of the latent growth curve parameters (intercept and slope/velocity) suggests that development of head circumference is regulated by its specific genetic factor independent from those for the other physique measures.

Syllogism and intelligence: g (Genetic Factor) of g (General Intelligence) revisited

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

1Faculty of Letters, 2-15-45 Mita, Keio University, Minato-ku, Tokyo, 108-8345 Japan, e-mail: juko@msa.biglobe.ne.jp, 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

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  Æ 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  = 0.96) and substantial phenotypic correlation with IQ (0.61). Factor analysis revealed that two distinctive logical types were
extracted, one showed substantial common environment and no genetic effect and the other showed substantial genetic and no shared environmental effects. 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%.

Etiology of stability of reading difficulties: preliminary analysis of follow-up data from participants in the Colorado learning disabilities research center

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

¹Institute for Behavioral Genetics, University of Colorado, Boulder, CO. Grant Support: Supported by NICHD Center Grant HD-27802, NICHD Grant DC-05190, 1480 30th Street, 80309, USA, e-mail: raven.astrom@colorado.edu

Results obtained from longitudinal studies indicate that reading deficits are generally stable [e.g., Satz P, Buka SL, Lipsitt LP, Seidman L (1998) In: Shapiro BK, Accardo PJ, Capute AJ (eds) Reading disability: a view of the spectrum. York Press, Timonium, MD). 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 0.83. For each time point, the data were subjected to univariate DeFries-Fulker multiple regression analysis [DeFries, Fulker (1985) Beh Genet 15:467–473]. Estimates of the heritability of the group deficit (h²g) were 0.75 (± 0.23) at initial assessment and 0.62 (± 0.42) at follow-up. When these data were fitted to a bivariate extension of the basic DF model [Light JG, DeFries JC (1995) J Learn Disabil 28:96–106], bivariate heritability was estimated at 0.71, indicating that common genetic influences account for approximately 86% of the phenotypic correlation between reading measures at the two time points in this preliminary sample.

Modeling age-of-onset in behavior genetic substance use research: it’s about time?

David E. Bard¹

¹University of Oklahoma, Psychology, Oklahoma City, Oklahoma, 73118 USA, e-mail: david-bard@ouhsc.edu

A brief history of age-at-onset modeling in behavior genetics was presented followed by a new discrete-time survival method for estimating ACE variance components of genetically informative age-at-onset data. The new method was framed as an adaptation of the Goldstein, [Pan, Byrner (2004)] multilevel model for event histories. Extensions of the model for multivariate outcomes were also discussed. Using this new technique, univariate and multivariate behavior genetic models of alcohol and cigarette initiation 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)]. Initiation of either substance appeared to be predominantly influenced by the environmental sources of variation, with little to no support for additive genetic influences. Despite this similarity, results supported the investigation of both initiations separately, as substantial ACE unique effects were present in the MV model. Lastly, estimates of shared-environmental effects from this study were consistently lower than those present in most previous investigations. This can likely be attributed both to the larger variety of genetic relatedness existing in this kinship, as opposed twin-only, sample, as well as the greater variability in age-at-onset measured initiation, as opposed to status indicators (e.g., used, never used). Pros and cons of this more detailed initiation phenotype were discussed in the context of past, present, and future substance use theory and research.

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

David E. Bard¹, Joseph Lee Rodgers¹

¹University of Oklahoma, Psychology, Oklahoma City, Oklahoma, 73118 USA, e-mail: 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.

Genetic bases of normal reading

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

1Psychology, 7 George Square, University of Edinburgh, Edinburgh, Scotland, EH8 9JZ, UK, e-mail: tim.bates@ed.ac.uk, 2 University of Melbourne, 3Queensland Institute of Medical Research, 4Macquarie University. Grant Support: NHMRC Australia

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 where also suggested. The results indicate 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.

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

Beben Benyamin1, 4, Ian J. Deary2, Peter M. Visscher3

1Institute of Evolutionary Biology, University of Edinburgh, UK, 2Department of Psychology, University of Edinburgh, UK, 3Genetic Epidemiology, Queensland Institute of Medical Research, Australia, 4Genetic Epidemiology, 300 Herston Road, Queensland Institute of Medical Research, Brisbane, QLD, 4029, Australia, e-mail: 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 Developments 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 model 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.
A preliminary investigation of the genetic etiology of reading comprehension over time

Rebecca S. Betjemann 1, Erik G. Willcutt 2, Richard K. Olson 2, Janice M. Keenan 3, John C. DeFries 1, Sally J. Wadsworth 1

1 Institute for Behavioral Genetics, University of Colorado, 447 UCB, Boulder, CO 80309-0447 USA, e-mail: betjemann@colorado.edu, 2 Department of Psychology, University of Colorado, Boulder, CO, 3 Department 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; NIMH Training grant T32 MH016880-25

Although much research has been done on the genetics of word reading and its component processes [Gayán J, Olson RK (2003) J Exp Child Psych 84:97–123], very little has been done to investigate the genetics of reading comprehension. One recent study [Keenan JM, Betjemann RS, Wadsworth SJ, DeFries JC, Olson RK (2006) J Res Reading 29:75–91] found significant heritability of reading comprehension ($h^2_g = 0.51$), and a genetic correlation with word recognition of 0.85, 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 59 pairs of MZ twins and 98 DZ pairs, tested at two time points (mean ages 10.2 & 16.2). The PIAT reading comprehension measure and a word decoding composite score were included from both time points. Univariate estimates of heritability for reading comprehension were 0.37 at Time 1 and 0.61 at Time 2, suggesting higher heritability at Time 2. A Cholesky decomposition of the PIAT comprehension scores and decoding scores at both time points revealed that comprehension and decoding share a substantial amount of genetic influence (genetic correlations of Time 1 decoding and Time 1 comp. = 0.83, Time 1 decoding and Time 2 comp. = 0.84, Time 1 comp. and Time 2 decoding = 0.91, Time 2 decoding and Time 2 comp. = 0.89). Despite the high genetic correlations between comprehension and decoding, there also is evidence for a unique genetic factor for comprehension independent of decoding (path coefficients of 0.35 and 0.42 for Time 1 and Time 2 comprehension, respectively). These preliminary results indicate that decoding abilities share significant genetic influences with reading comprehension even years later, but also suggest possible independent genetic influences on comprehension.

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

David A. Blizard 1

1 Center for Dev Health Genetics, 201 Res Bldg D, Penn State University, University Park, PA 16802, USA, e-mail: dab22@psu.edu. Grant Support: DC-02230 David A. Blizard; AA-014711 David A. Blizard; AA-08454 to Gerald E. McClearn.

Acknowledgements: I thank Gerald E. McClearn and Marion E. Frank for valuable comments.

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 [Lawrence GJ, Kiefer SW (1987) Chem 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.

Transitivity of genetic architecture

David A. Blizard 1, Arimantas Lionikas 1, Jennifer E. Foreman 1, Frank Johannes 1, David J. Vandenbergh 1, George P. Vogler 1, Gerald E. McClearn 1

1 Center for Dev Health Genetics, 201 Res Bldg D, Penn State University, University Park, PA 16802, USA, e-mail: dab22@psu.edu. Grant Support:
AG14731 and from the National Institute on Aging, AA08125 and AA014711 from National Institute of Alcohol Abuse and Alcoholism.

Contextual genetics (see abstract by GE 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.

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

Dorret I. Boomsma, Gonneke Willemsen, Marlies de Lange, Stephanie van den Berg, Jacqueline M. Vink

Dept Biological Psychology, Vrie Universitie, Amsterdam, NH, 1081 BT, The Netherlands, e-mail: dorret@psy.vu.nl. Grant Support: Dutch Heart Foundation/NWO-900-562-137, NWO 904-61-090, Investeringssubsidie NWO 575-25-006 and NWO 480-04-004, NWO 985-10-002, NWO-MW 904-61-193, NWO/SPI 56-464-14192

In 2002/3 we collected survey data on age at menarche and oral contraceptive use in women from Dutch twin families. Probands 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 that is mainly influenced by common environment.

Association between age of sexual initiation and dopamine-encoding genes in combined twin and adoption samples

Josh B. Bricker, Michael C. Stallings, Robin P. Corley, Brett C. Haberstick, Andrew Smolen, Gary Stetler, Susan E. Young, John K. Hewitt, John C. DeFries

Institute for Behavioral Genetics, University of Colorado, Boulder CO 80309-0447, USA, e-mail: jbricker@colorado.edu. Grant Support: HD010333, HD036773, DA011015, DA05131

Genetic influences on the age of first sexual initiation (AFSI) are suggested by heritability estimates in the range of 28% to 72% [Bricker JB, Stallings MC, Corley RP, Wadsworth SJ, Bryan A, Timberlake DS, Hewitt JK, Caspi A, Hofer SM, Rhea SA, DeFries JC (in press) Behav Genet], and an association between early pubertal development and early AFSI [Rowe DC (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 CT, Udry JR, Campbell B, Suchindran C (1993) Psychosom Med 55:436–447; Mustanski BS, Viken RJ, Kaprio J, Pulkkinen L, Rose RJ (2004) Devel Psychol 40(6):1188–1198]. One previous study [Miller WB, Pasta DJ, MacMurray J, Chiu C, Wu H, Comings DE (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. Between-family association tests indicate no association between AFSI and any of three loci, however within-family analyses conducted in QTDT [Abecasis GR, Cardon LR, Cookson WOC (2000) Am J Hum Genet 66:279–292] found a significant association between DRD2 and AFSI such that the presence of the A1 allele was associated with earlier AFSI.
The nicotine dependence syndrome scale in Finnish smokers—genetic architecture of nicotine dependence

Ulla Broms1, Pamela A. F. Madden2, Andrew C. Heath2, Michele L. Pergadia2, Saul Shiffman3, Jaakko Kaprio1,4

1Department of Public Health, P.O. Box 41, Mannerheimintie 172, University of Helsinki, 00014 Helsinki, Finland, email: ulla.broms@helsinki.fi, 2Washington University School of Medicine, St. Louis, USA, 3Department of Psychology, University of Pittsburgh, USA, 4Department of Mental Health and Alcohol Research, National Public Health Institute, Helsinki, Finland. Grant Support: Doctoral Programs Alcohol Research, National Public Health Institute, Pittsburgh, USA, 4Department of Mental Health and Alcohol Research, National Public Health Institute, 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

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 0.39 (95% CI 0.23, 0.55) 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.

Characterization of shared environmental influence on adolescent behavior: evidence from the sibling interaction and behavior study

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

1Department of Psychology, University of Minnesota, Minneapolis, MN 55414, 55455, USA, e-mail: bucha057@umn.edu. Grant Support: Supported by USPHS grant #AA11886; USPHS grant #MH066140

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 JC, McClean GE, McGuffin P (2000) Behavioral Genetics, 4th edn. Worth Publishers, New York] or the comparison of twins reared apart to twins reared together [Bouchard TJJ, Lykken DT, 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.
Communicative genes, units of selection
Ross Buck1, Benson Ginsburg1

1Communication Sciences, University of Connecticut, Storrs, CT, 06269-1085, USA, e-mail: 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—intrinsically 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.

Gene-environment correlations in antisocial behavior: peer selection and friendship
S. Alexandra Burt1

1Department of Psychology, Michigan State University, 105A Psychology Bldg., East Lansing MI, 48824 USA, e-mail: burts@msu.edu. Grant Support: Supported in part by a grant from the Intramural Grants Program, Michigan State University, #04-IRGP-232

To date, the empirical evidence [Plomin R, DeFries JC, Loehlin (1977) Psychol Bull 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.

Elucidating the relationship between affiliation with delinquent peers and polysubstance dependence vulnerability
Tanya M. M. Button1, Soo Hyun Rhee1, Susan E. Young1, Robin P. Corley1, Michael C. Stallings1, John K. Hewitt1

1Tanya Maria May Button, Institute for Behavioral Genetics, UCB 447, University of Colorado, Boulder, Colorado, 80309-0447, USA, e-mail: Tanya.Button@Colorado.edu. Grant Support: DA011015; HD010333; MH43899

There is evidence that affiliation with delinquent peers is associated with a number of externalizing outcomes.
in adolescents, particularly own peer delinquency [Simonoff E, Elander J, Holmshaw J, Pickles A, Murray R, Rutter M (2004) Br J Psychiatry 184:118–127], and substance use problems [Aseltine RH Jr (1995) J Health Social Behav 36:103–121; Fergusson DM, Swain-Campbell NR, Horwood LJ (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.

**Intersexual aggression: effect of male strain, intermale aggressive experience, and estrous cycling**

Andrew Canastar¹, Stephen C. Maxson²

¹Psychiatry, RC-1N, P18-8101, M/S 8344, 12800 E. 19th Ave., U. Colorado Denver Health Science Center, Aurora CO 80010, USA, e-mail: andrew.canastar@uchsc.edu, ²Biobehavioral Sciences Graduate Program, Department of Psychology, University of Connecticut, Storrs, CT. Grant Support: Inbred Mouse Fund; Research Foundation of The University of Connecticut

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.

**Genetic and environmental influences on tester-rated verbal and non-verbal cognitive ability in two-year olds**

Sonia Chawla¹, Kimberly J. Saudino¹

¹Psychology, Boston University, Boston, MA 02215 USA, e-mail: sonia@bu.edu. Grant Support: NIMH Grant MH062375

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 edn. The Psychological Corporation, San Antonio]. 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 (rg = 0.77) and shared environment correlations (rc = 1.00), and a non-shared environment correlation of 0.38 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 0.49, an estimate much higher than what has been observed with parent ratings, and the bivariate shared-environment estimate was much lower at 0.35. 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.

**Genetic and social mechanisms in poverty traps**
Jeff Davis

Sociology, 1250 Bellflower Blvd., California State University, Long Beach, Long Beach, CA, 90840 USA, e-mail: 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.

**Development and function of sex differences in vasopressin/vasotocin innervation**
Geert DeVries

Department of Psychology, University of Massachusetts, Amherst, MA 01003-7720, e-mail: 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 death 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.

**Evidence for genes influencing general externalizing psychopathology**
Danielle M. Dick, Fazil Aliev, Jen C. Wang, Anthony Hinrichs, Howard J. Edenberg, Tatiana Foroud, John Nurnberger, Jr., Victor Hesselbrock, Marc Schuckit, Sam Kuperman, Bernice Porjesz, Henri Begleiter, Laura Jean Bierut, Alison Goate

Departments of Psychiatry & Psychology, Box 8134, Washington University in St. Louis, St. Louis, MO 63110 USA, e-mail: dickd@wustl.edu, Indiana University School of Medicine, Indianapolis, Indiana, University of Connecticut Health Center, Farmington, CT, University of California, San Diego VA Medical Center, San Diego, CA, University of Iowa College of Medicine, Iowa City, IA, SUNY Health Science Center at Brooklyn, Brooklyn, NY. Grant Support: COGA is supported by NIH Grant U10AA08401 from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute on Drug Abuse (NIDA).

The Collaborative Study on the Genetics of Alcoholism (COGA) has made rapid progress in recent years in identifying genes involved in the predisposition toward alcohol dependence. Efforts are currently underway to better characterize the risk associated with these identified genes. Alcohol dependence (AD) is commonly comorbid with other behavioral disinhibitory phenotypes, such as illicit drug dependence (DD), antisocial personality disorder (ASPD), and conduct
disorder (CD). Furthermore, twin studies suggest that this overlap is due, in large part, to shared genes. Findings from the neurophysiological literature, in which electrophysiological endophenotypes are shared across these disorders, further support the idea that genetic factors influence a latent behavioral disinhibition trait, which is involved in the manifestation of these related clinical phenotypes (Porjesz et al. 2005). Using the wealth of data collected as part of the COGA project, we have begun to explore the extent to which genes identified as influencing alcohol dependence may also be associated with related disinhibitory disorders. Initial efforts have focused on studying two genes, GABRA2 and CHRM2, that are significantly associated with alcohol dependence in the COGA sample, and that have been replicated in independent samples. For both of these genes, we do find evidence of association with other behavioral disinhibitory disorders, including illicit DD, ASPD, and CD. In addition, both genes are associated with electrophysiological endophenotypes. Furthermore, the evidence for association with CHRM2 is strongest when considering latent factor scores comprised of AD, DD, CD, and ASPD symptoms, as well as novelty seeking and sensation-seeking scale scores. These findings suggest that these genes are involved in general behavioral disinhibition and support the idea that some genes contribute to a shared genetic susceptibility toward general externalizing psychopathology, as defined by multiple forms of substance dependence, comorbid externalizing disorders, and impulsive personality traits.

**Genetic influences are stronger for aggressive than prosocial behaviors in preschool twins**

Lisabeth F. DiLalla1, Paula Y. Mullineaux1

1Family and Community Medicine, Mail Code 6503, Southern Illinois University School of Medicine, Carbondale, IL 62901 USA, e-mail: ldilalla@siu.edu. Grant Support: SIU ORDA grant, SIU School of Medicine grant

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 [DiLalla LF (2002) Twin Res 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 min. Parents also rated the children on a measure of temperament. Results showed that aggressive behaviors show evidence of significant genetic influence (h2 = 0.67), but prosocial behaviors do not appear to be genetically mediated (h2 = 0.00). 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 (h2 = approximately 0.80), whereas Intensity showed weaker genetic influence (h2 = 0.22). 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.

**The intergenerational transmission of childhood conduct problems: a children of twins study**

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

1Department of Psychological and Brain Science, 1101 E. 10th St., Indiana University, Bloomington, IN 47405 USA, e-mail: bmdonofr@indiana.edu, 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 AA00264; William T. Grant Foundation; National Alliance for Research on Schizophrenia and Depression

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.

Breakpoint mapping using microarrays and FISH in an 18p monosomy case with psychosis, leukodystrophy, and dysmorphology

C. M. Drazinic1, 2, B. A. Pletcher2, H. Zheng1, M.W. State1,3

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), 4Psychiatry, Genetics, 263 Farmington Avenue, Univ. Connecticut Health Center, Farmington, CT 06030-2103, USA, e-mail: draziniccc@psychiatry.uchc.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 50 K 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.

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

Susan K. Fenstermacher1, Kimberly J. Saudino1

1Psychology, 64 Cummington Street, Boston University, Boston, MA 02215 USA, e-mail: skf73@bu.edu. Grant Support: Supported by NIMH Grant MH062375

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 [Barr R, Hayne H (1999) Child Development 70:1067–1081; Carpenter M, Call J, Tomasello M (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 [Bayley N (1993) Bayley Scales of Infant Development, 2nd edn. The Psychological Corporation, San Antonio, TX] 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 ($r = 0.35$, $P < 0.01$) and task orientation ($r = 0.37$, $P < 0.01$). 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.

Developmental differences in the genetic etiology of reading and spelling disabilities

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

1Department of Psychology, Campus Box 345, University of Colorado at Boulder, CO 80309, USA, e-mail: angela.friend@colorado.edu, 2Institute for Behavioral Genetics, University of Colorado, Boulder CO. Grant Support: GRANT SUPPORT: NICHD Center Grant HD-27802, and NICHD Training Grant HD-007289

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 JC, Alarcón M, Olson RK (1997) In: Hulme C, Snowling M (eds) Dyslexia: biology, cognition, and intervention, London, England; Wadsworth SJ, Gillis JJ, DeFries JC, Fulker DW (1989) Irish J Psychol 10:509–520]. We followed up these earlier studies by fitting a DeFries–Fulker (DF) regression model to two measures of word recognition and two measures of spelling in a larger sample of twins from the Colorado Learning Disabilities Research Center. Depending on the measure, the younger group (8.1–11.5 years) ranged from 73 MZ to 129 MZ pairs and 56 to 98 DZ pairs, and the older group (11.5–20.2 years) ranged from 60 MZ to 182 MZ pairs and 55 MZ to 150 DZ pairs. Probands were selected for reading and/or spelling deficits of at least 1.5 SD below the mean of the normal-range control twin sample. The heritability estimates for the group deficit in word reading among same-sex twin pairs were similar for both measures in older and younger age groups. The \( h^2_g \) estimates for a composite of the two word reading measures were 0.65 in the younger group and 0.61 in the older group. For spelling, there was a nonsignificant trend for both measures. The \( h^2_g \) estimates for a composite of the two spelling measures were 0.50 and 0.73 for the younger and older groups, respectively. Similar results were obtained when opposite-sex pairs were included in the analysis.

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

Jeffrey R. Gagne1, Kimberly J. Saudino1

1Department of Psychology, 64 Cummings Street, Boston University, Boston, MA 02215, USA, e-mail: gagnej@bu.edu. 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.

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; Goldsmith HH (1996) Child Development 67:218–235], and behavior problems were assessed with the Child Behavior Checklist for Ages 1 1/2–5 [CBCL; Achenbach TM, Rescorla LA (2000) University of Vermont, Research Center for Children, Youth, and Families, Burlington, VT). 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 0.50–0.78, suggesting that genetic influences on the three behaviors overlap. Implications will be discussed.

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

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

¹ Div. Neuroanat. & Behav., Department of Anatomy, University of Zurich, Zurich, Zurich, CH-8057, Switzerland, e-mail: mike_galsworthy@yahoo.co.uk, ²Department 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.

By utilizing the combined technologies of micro-tran-sponders 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.

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

Jody M. Ganiban¹

¹Psychology, George Washington University, Washington, DC 20052 USA, email: ganiban@gwu.edu. Grant Support: Supported by NIMH grant 5R01MH43373; NIMH grant 5R01MH48825


NEAD included 2-parent nondivorced and step families with 2 adolescent children (average age difference = 1.6 years; 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 = 0.39 – 0.63). 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.

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

Aude Gérard-Desplanches¹, ³, Christine Deruelle², Silvia Stefanini³, Gene Fisch⁴, Stefano Vicari⁵, VirginiaVolterra⁶ and Michèle Carlier⁷

¹Centre de Recherche PsyCLÉ (EA327), University of Provence, Aix en provence, France, ²Institut de Neurosciences Cognitives de la Méditerranée, CNRS, Marseille, France, ³Department of Neuroscience,
Is risk for marijuana initiation, abuse and dependence mediated by individual differences in substance availability and peer group deviancy?

Nathan A. Gillespie1, 4, Michael C Neale1, Carol A. Prescott2, Kristen Jacobson3, Kenneth S. Kendler1

1Virginia Commonwealth University, 2University of Southern California, 3University of Chicago. Grant Support: NHMRC Sidney Sax Postdoctoral Fellowship; DA-11287 (NIH); MH-01458 (NIH); AA-00236 (NIH), 4Department of Psychiatry, 800 East Leigh Street, Biotech 1, Suite 101, VIPBG, Richmond, VA 23219 USA, e-mail: 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 years to 62 years from the Virginia Twin Registry, five retrospective assessments (8–11, 12–14, 15–17, 18–21, and 22–25 years) 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 years, most of the variation in familial aggregation for peer group deviancy was best explained by additive genetic effects (28%–45%). However, between 15 years and 17 years, 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 years and 17 years, 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 years and 17 years, 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.

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

Elena D. Gindina1, 3, Sergei B. Malykh1, Marina M. Lobaskova2

1Laboratory of Developmental Behavior Genetics, Psychological Institute of Russian Academy of
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.

**Depression and internally directed aggression: genetic and environmental contributions**

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

¹Center for Family Research, 2300 K street NW, Warwick Building Suite 313, George Washington University, Washington, DC, 20037 USA, e-mail: Suzannekerin@yahoo.com, ²Karolinska 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)

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., W.W. Norton & Company Inc., New York] 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/Observatory 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.

**Evolution, genomic imprinting, and social behavior**

David Haig¹

¹Department of Organismic and Evolutionary Biology, Harvard University, Cambridge MA 02138, e-mail: 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.

Peers and young adults smoking: univariate and multivariate behavioral genetic analyses

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

1Behavioural Science Institute, Montessorilaan 3, Radboud University Nijmegen, Nijmegen, Gelderland, 6525 HR, The Netherlands, e-mail: z.harakeh@pwo.ru.nl, 2Center for Family Research, George Washington University, Washington, DC, 3SGDP Centre, Institute of Psychiatry, London

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. 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.

Rated personality and measured intelligence in young twin children

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

1Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, e-mail: jharris@uwo.ca, 2University of British Columbia

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.

The genetics of values

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

1Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, e-mail: jharris@uwo.ca, 2University of British Columbia, Vancouver, British Columbia

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 sub-sample 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.

Vocational interests and personality: phenotypic and genetic relationships

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

¹Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, e-mail: jharris@uwo.ca, ²University of British Columbia, Vancouver, British Columbia

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 0.33. 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 0.50. The results suggest that personality is related to some vocational interest dimensions and that some of these observed relationships have a common genetic basis.

Univariate analyses of the growth of reading outcome measures

Sara A. Hart¹, Stephen A. Petrill¹

¹Biobehavioral Health, 101 Amy Gardner House, The Pennsylvania State University, University Park, PA 16802, USA, e-mail: sah323@psu.edu. Grant Support: Supported by NICHD HD38075

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 (Woodcock R (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 = 6.0 years, wave 2 age M = 7.2 years). 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, rMZ = 0.81, rDZ = 0.58; Word Attack, rMZ = 0.74, rDZ = 0.29; and Passage Comprehension, rMZ = 0.64, rDZ = 0.16). 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 (h² = 0.38, c² = 0.40). 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 (h² = 0.69, c² = 0.00). Similarly, the difference between wave 2 and wave 1 Passage Comprehension scores suggested strong genetic influence (h² = 0.63), with shared environmental effects accounting for no variance (c² = 0.00). 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.

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

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

¹Psychology, 447 UCB, Institute for Behavioral Genetics, University of Colorado, Boulder, CO 80309-0447 USA, e-mail: hawkej@colorado.edu. Grant Support: NICHD Center Grant HD-27802, NICHD Training Grant HD-7289

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 [DeFries JC, Fulker DW (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 [Aitken AC (1934) Proc Edinburgh Math Soc B, 4:106–110] to model the effects of selection on mean and covariance structures [e.g., Hopfer CJ, Stallings MC, Hewitt JK, Crowley TJ (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.

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

Y.-M. Hur¹, J. Ando², M. Bartels³, C. E. M. van Beijsterveldt⁵, D. I. Boomsma⁵, B. Cornes⁴, W. G. Iacono⁵, J Kaprio⁶, H.-R Lajunen⁶, C. H. Lin⁷, M. Luciano⁴, H. Maekawa², M. McGue⁵, R. Nakajima², N. G. Martin⁴, R. Rose⁸, S. Ooki⁹

¹Seoul National University, Seoul, South Korea, ²Keio University, Tokyo, Japan, ³Vrije Universiteit, Amsterdam, The Netherlands, ⁴Queensland Institute of Medical Research, Brisbane, Australia, ⁵University of Minnesota, Twin Cities, USA, ⁶University of Helsinki, Helsinki, Finland, ⁷Tzu Chi University, Taiwan, ⁸Indiana University, Bloomington, USA, ⁹Ishikawa Prefectural Nursing University, Ishikawa, Japan

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, 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 female prepregnancy weight has been documented to be a major determinant of the fetal birthweight, in the present study, we compared genetic and environmental variances of BMI between Caucasians and East Asians using adolescent twin data from Australia, Finland, Japan, the Netherlands, South Korea, the United States, and Taiwan. The results of data analyses showed that phenotypic variances of BMI were significantly greater in Caucasians than in East Asians; and these greater variances were largely due to greater genetic variances of BMI in Caucasians than in East Asians. Shared environmental variances were minimal and equatable across Caucasians and East Asians. We speculate that the difference of frequencies of genes associated with obesity, and possible differences in the effects of puberty on body weight might be responsible for the differences in the genetic variability in BMI observed between Caucasians and East Asians. The results of the present study point out the importance of genetic differences between Caucasians and East Asians in the risk of obesity-related diseases.

**A behavioral genetic study of the coldness of hands on the basis of South Korean twins**

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

¹Seoul National University, Seoul, South Korea, ²Korea Institute of Oriental Medicine, Daejeon, South Korea

The two major goals of the present study were to explore whether the coldness of hands is a heritable trait and to investigate gender differences in genetic influences on the coldness of hands. Eight hundred and fifty-seven South Korean twin pairs completed a single item of the coldness of hands. In both males and females, MZ twins showed higher correlations than DZ twins for the coldness of hands, suggesting that genetic factors are important in individual differences in the coldness of hands. A sex-limitation model was applied to the data. Model-fitting analyses yielded three main conclusions. First, in both males and females, 39% of individual differences in the coldness of hands are attributable to genetic factors. Second, same genes and same environmental factors might be responsible for individual differences in the coldness of hands in males and females. Finally, shared environmental influences on the coldness of hands are negligible.

**Heritability of hostility in South Korean adolescent twins**

Yoon-Mi Hur¹

¹Seoul National University, Seoul, South Korea

Six hundred and forty-seven South Korean late adolescent and young adult twins completed a hostility
A sex-limitation model was applied to the data. The results showed that in both males and females about 32% of individual differences in hostility was associated with genetic factors, whereas shared environmental influences were near zero. Heritability of hostility found in the present study was consistent with those estimated from adult twin samples, suggesting that genetic influences on hostility emerge relatively early in life and persist into adulthood.

**Bivariate analysis of obsessive compulsive disorder and neuroticism in South Korean twins**

Yoon-Mi Hur¹, Jung Lee¹

¹Seoul National University, Seoul, South Korea

Six hundred and thirty-five South Korean twins completed Maudseley Obsessive-Compulsive Inventory (MOCI) and the Neuroticism scale of Eysenck Personality Questionnaire (EPQ). The phenotypic correlation of the total score of MOCI and the neuroticism scale was significant \((r = 0.44)\). A bivariate model was applied to the data. The results of model-fitting analyses showed that the relationship between MOCI and neuroticism was due to common genetic factors. The results of the present study suggest that neuroticism may be a genetic vulnerability marker for obsessive-compulsive disorder.

**A dopamine receptor D4 polymorphism, attention deficit hyperactivity disorder, and disinhibitory psychopathy**

Daniel E. Irons¹, Matt McGue¹,³, William G. Iacono¹, S. A. Burt², Bob Krueger¹, William S. Oetting³

¹Psychology, 1041A 29th Ave SE, University of Minnesota, Minneapolis MN 55414 USA, e-mail: iron0012@umn.edu, ²Department of Psychology, Michigan State University, ³Institute of Human Genetics, University of Minnesota, Minneapolis MN. Grant Support: NIH Grant AA11886; NIH Grant MH066140

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 [Faraone SV, Doyle AE, Mick E, Biederman J (2001) Am J Psychiatry 158:1052–1057] and indicators of the novelty seeking personality traits thought to be linked to ADHD [LaHoste GJ, Swanson JM, Wigal SB, Glabe C, Wigal T, King N, Kennedy JL (1996) Mol 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.

**Evolution of Drosophila populations and their chemical signals**

Jean-Marc Jallon¹,²

¹NAMC, UPS-Orsay, 91405 Orsay France, ²BAT446, UNIV Paris XI, Orsay, 91405, France, e-mail jmjallon@club-internet.fr

In Drosophila, cuticular hydrocarbons CHC play two important roles, protection against dessication and pheromone communication. As *Drosophila melanogaster* is a cosmopolitan species, it is possible to collect wild flies in many parts of the world; actually 85 populations were collected, bred in similar conditions and, after a number of generations, studied for their CHC. In central Africa, probably the craddle of the melanogaster subgroup, *D. melanogaster* populations are characterized by 5,9-heptacosadiene as major female CHC and 7-pentacosene in males, as those collected in the Tai primary forest of Ivory Coast. From these ancestral characteristics a multiple polymorphism has developed. The female ancestral CHC trait was retained in all sub-Saharan African populations but elsewhere was found only in the Caribas. Meanwhile the proportion of male major 7-pentacosene decreased gradually with the latitude of the collection site and that of shorter (Z)7 tricosene increased. Thus different selective mechanisms seem to have been involved. CHC biosynthesis studies have shown that elongation-decarboxylation was involved together with a small number of desaturation steps. Thus only a small number of enzyme activities are required, mainly desaturases, elongases and decarboxylases (Jallon and Wicker-Thomas 2003). Dallerac et al. (2000) have shown that at least two D9 desaturases are involved in the first desaturation, coded by genes Desat1 and Desat2. The former enzyme, present in both sexes of all populations, leads to (Z7) compounds while DESAT2, present only in females of ancestral populations, leads to (Z5) compounds. Then Takahashi et al. (2001) have established that 17 base pairs were missing in the promoter region of Desat2 of derived genomes. It is thus highly probable that a transposon (insertion/excision) is involved in the female major CHC change. Moreover mate choice experiments have shown that derived females are preferred by derived males - but courted as intensively by ancestral males (Haerty et al. 2001).
2002.) During migrations from Western Central Africa, ancestral populations have met colder and colder temperatures and meanwhile their major CHC became less elongated (Rouault et al. 2004). Actually several fatty acid elongase genes have been described in yeast and mouse with different chain lengths and levels of unsaturation and close to twenty homologous sequences have been found in Drosophila genome (Chertemps et al. 2005). Thus it is probable that several useful elongase enzymes coexist in \textit{D. melanogaster} which might have been exposed to different temperature selection pressures during these migrations, which might explain the change in CHC chain lengths. Actually the elongation steps are plastic as a simple shift in the breeding temperature (29/18°C) markedly decreases the proportion of (Z)7 pentacosene (Rouault et al. 2004); moreover the expression level of the first characterized elongase gene was much higher at 18°C (Chertemps et al. 2005). Such an evolutive mechanism might have been strengthened by sexual selection as derived females markedly prefer derived males (Haerty et al. 2002).

Genetic and environmental effects on rapid and delayed ejaculation

Patrick Jern\(^1\), Pekka Santtila\(^1\), N. Kenneth Sandnabba\(^1\)

\(^1\)Department of Psychology, Brunnsgatan 8 B 25, Åbo Akademi University, Åbo, 20500, Finland, e-mail: pjern@abo.fi. Grant Support: The Academy of Finland; The Åbo Akademi Foundation

The prevalence estimates of rapid ejaculation (RE) vary widely, ranging from 4% to 30% [e.g. Simons, Carey (2001) Arch Sexual Behav 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 Med 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 Sexual Behav 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.

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

Andrew M. Johnson\(^1\), Julie Aitken Harris\(^1\), Jon Fleming\(^2\), Kerry L. Jang\(^2\)

\(^1\)Administrative and Commercial Studies, The University of Western Ontario, Ontario, London, N6A5C2, Canada, e-mail: jharris@uwo.ca, \(^2\)University of British Columbia, Vancouver, British Columbia

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.

Cannabis use among Finnish adolescents and young adults

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

1Department of Public Health, PO Box 41, Mannerheimintie 172, University of Helsinki, Helsinki 14, Finland, e-mail: jaakko.kaprio@helsinki.fi,
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

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% 0.15 to 0.52) 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.

Effects of social experience on aggressive behavior in Drosophila

Yong-Kyu Kim1, Jayne Kelly1

1Genetics, University of Georgia, Athens, GA 30602, USA, e-mail: 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, 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, Messing R (1998) Behaviour 135:1013–1030; Boake C et al. (1997) PNAS 94:12442–12445; Lee G, 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 min 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.

MAOA by maltreatment GxE in young children

Julia Kim-Cohen1

1Psychology, 2 Hillhouse Ave, PO Box 208205, Yale University, New Haven, CT 06520, USA, e-mail:
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.

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

Marc Lalande

1Department of Genetics & Developmental Biology, 263 Farmington Avenue, University of Connecticut Health Center, Farmington, CT, 06030-3310 USA, e-mail: 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.

**The effects of genotyping error on case-control association using SNPs and haplotypes**

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

1Institute for Behavioral Genetics, 447 UCB, University of Colorado, Boulder, CO 80309-0447 USA, e-mail: jeff.lessem@colorado.edu. Grant Support: P01-HD31921; DA011015; EY012562; DA015522; AA015366

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.
Analysis of vertical transmission of schizotypy: a study of Taiwanese Juvenile twins and their parents

Chaucer C. H. Lin¹, Wei J. Chen²

¹Department of Psychiatry, Tzu Chi General Hospital and University, Taiwan, Hualien, 970, R.O.C., e-mail: chaucer@mail.tcu.edu, ²Institute of Epidemiology, School of Public Health, National Taiwan University, Taipei, Taiwan

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. 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. 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. 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.

A behavioral genetic investigation of sense of humor

Ashley Mackie¹, Philip A. Vernon¹, Rod A. Martin¹, Leah D. Sheppard¹, Julie Aitken Harris²

¹Department of Psychology, University of Western Ontario, London, Ontario, N6A 5C2, Canada, e-mail: vernon@uwo.ca, ²Administrative and Commercial Sciences, University of Western Ontario. Grant Support: Social Sciences and Humanities Research Council of Canada

This presentation is the first behavioral genetic (BG) investigation of sense of humor as measured by the Humor Styles Questionnaire (HSQ). Two hundred and eighty five 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 and Self-Enhancing humor. The two negative dimensions are Aggressive humor and Self-Defeating humor. Univariate BG analyses reveal that an AE model provides the best fit to the two beneficial types of humor (affiliative and self-enhancing), with $h^2$ values of 0.52 and 0.41 respectively. A CE model, however, provides the best fit to the two detrimental types of humor (aggressive and self-defeating). We are currently examining the relationship between the 5 factors of the NEO-PI-R and the 4 humor styles.

Contextual genetics

Gerald E. McClearn¹

¹Department of Biobehavioral Health, Center for Developmental and Health Genetics, Pennsylvania State University, University Park, PA 16802, e-mail: gm1@psu.edu. Grant Support: AG14731 from National Institute on Aging, AA08125 from the National Institute on Alcohol and Alcohol Abuse

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.
Associations of trauma and genetic effects with drinking motives in a female twin sample

Vivia V. McCutcheon1, 2, Andrew C. Heath1, Elliot C. Nelson1, Kathleen K. Bucholz1, Pamela A. F. Madden1

1Washington University, St. Louis, MO. Grant support: NIAAA Grants AA07728, AA10240, T32AA07580, 2Department of Psychiatry, 660 South Euclid Ave., Campus Box 8134, St. Louis, MO 63110, USA, 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 1985. Responses from 2466 twins who reported having drunk any alcohol and for whom there was 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) Psychol Assessment 6(2):117–128]. Categories for experience of trauma were defined a priori into four groups: (1) no trauma, (2) nonassaultive events only (accident, disaster, witnessing injury or killing), (3) assaultive events (rape or sexual molestation at age 18 or older, severe physical assault, being threatened with a weapon or kidnapped) with no history of childhood events, and (4) childhood events (neglect, physical or sexual abuse). The childhood trauma category was positively associated with drinking to cope ($b = 0.05$, 95% CI = 0.00, 0.10) and negatively associated with drinking for social reasons ($b = -0.05$, 95% CI = -0.09, -0.00). Assaultive trauma was negatively associated with drinking to conform ($b = -0.05$, 95% CI = -0.11, -0.00). There was evidence for genetic influence on drinking to cope ($b = 0.16$, 95% CI = 0.05, 0.26), drinking to conform ($b = 0.14$, 95% CI = 0.01, 0.26), and drinking for social reasons ($b = 0.11$, 95% CI = 0.02, 0.20). Traumatic experience in this sample is not a robust predictor of motives for drinking. Genetic influence, while modest, is a stronger predictor of drinking motives.

The relationship of social support and activity with late-life functioning: a Cotwin control study

Matt McGue1,2, Kaare Christensen2

1Department of Psychology, University of Minnesota, Minneapolis, Minnesota, 55455, USA, e-mail: mcgue001@umn.edu, 2Department of Epidemiology, Southern Denmark University. Grant Support: Supported by U.S. National Institute on Aging (P01-AG08761) and the Danish National Research Foundation

Gerontologists are increasing 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 measure 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.

GenetSim: software for simulation of familial data in genetics and epidemiology

Michael B. Miller1

1Division of Epidemiology, School of Public Health, 1300 S 2nd St, Suite 300, University of Minnesota, Minneapolis, MN 55454 USA, e-mail: mbmiller@taxa.epi.umn.edu. Grant Support: NIH Grant 5RO1-HL09609-12; NIH Grant 1R01-AG021917-01A1

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 (non-human 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/.

Maternal and child characteristics in mother–twin interactions: evidence of evocative genotype-environment correlation

Paula Y. Mullineaux1, Lisabeth F. DiLalla2

1Psychology, Department of Psychology, Mail code 6502, Southern Illinois University, Carbondale, IL 62901 USA, e-mail: paulam@siu.edu, 2Department of Family and Community Medicine, School of Medicine, Southern Illinois University, Carbondale, IL 62901

Previous research has indicated that mothers treat their MZ twins more similarly by age 2 1/2 [Lytton H (1977) Develop Psychol 28:1006–1017] but not at earlier ages [DiLalla LF, Bishop EG (1996) Behav Genet 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, 24, and 36 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.

BMI and energy intake in Japanese young adult twin pairs

Ryoko Nakajima1, Satoshi Sasaki2, Yutaka Ono3, Juko Ando4

1Graduate School of Human Relations, 1-26-23 #308 Yaguchi, Keio University, Ohta-ku, Tokyo, 146-0093 Japan, e-mail: riu_ryoko@yahoo.com, 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

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 [Ooki S, Yokoyama M (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 [e.g. Benton MS, Rha SS, Neal MC, Allison DB (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 [Sasaki S, Yanagibori R, Amano K (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 = 0.705) than in DZ (r = 0.095). 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 = 0.304) than DZ (r = 0.224). 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.

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

Jenae M. Neiderhiser\(^1\), Zeena Harakeh\(^2\)

\(^1\)Department of Psychiatry and Beh Sciences, George Washington University, Washington, DC 20037 USA, e-mail: cfrjmn@gwumc.edu, \(^2\)Radboud University Nijmegen. Grant Support: R01MH43373; R01MH48825; William T. Grant Found; R01MH59014; R01MH065563

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 [Hopfer CJ 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.

**The impact of peer substance use on early adolescence alcohol use**

Jason L. Pagan\(^1\), Candice Holliday\(^1\), Richard J. Rose\(^2\), Richard J. Viken\(^2\), Lea Pulkkinen\(^3\), Jaakko Kaprio\(^4\), Danielle M. Dick\(^1\)

\(^1\)Psychology, One Brookings Drive, Campus Box 1125, Washington University, St. Louis, MO, 63130, USA, e-mail: jlpagan@artsci.wustl.edu, \(^2\)Indiana University, \(^3\)University of Jyväskylä, \(^4\)University of Helsinki. 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)

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 available for 1,451 same-sex twin pairs. 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 indicated significant genetic and environmental influences on having substance using peers and on alcohol use at age 14 and at age 17 (after controlling age 14 use). Evidence for significant genetic influence on having peers who use substances suggests that peer selection may be involved. The association between having alcohol using peers and both concurrent and future alcohol use was due to both shared genetic and shared environmental factors. The
association between cigarette and drug using peers and concurrent and future alcohol use was mediated entirely through environmental pathways.

**Association between GABRA1 and drinking behaviors in the collaborative study on the genetics of alcoholism sample**

Jevon Plunkett¹, Leah Flury Wetherill², Xiaoling Xue², Alison Goate¹, Victor Hesselbrock³, Marc Schuckit⁴, Raymond Crowe⁵, Howard J. Edemberg², Tatiana Foroud², Danielle M. Dick¹

¹Psychiatry, 6331 Southwood Ave #2E, Washington University, St. Louis, MO, 63105, USA, e-mail: jevon.plunkett@gmail.com, 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.

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 DSMIIIR 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.

**Alcohol use in Dutch adolescent and young adult twins as a function of common friends**

Evelien A. P. Poelen¹, ³, Rutger C. M. E. Engels¹, Dorret I. Boomsma², Ron H. J. Scholte¹, Gonneke Willemsen²

¹Radboud University Nijmegen, the Netherlands, ²Vrije Universiteit Amsterdam, the Netherlands, ³Behavioural Science Institute, Montessorilaan 3, Radboud University, Nijmegen, 6525 HE, The Netherlands, e-mail: 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.

**The relative contribution of genes and environment to alcohol use in adolescents and young adults**

Evelien A. P. Poelen¹, ³, Dorret I. Boomsma², Rutger C. M. E. Engels¹, Ron H. J. Scholte¹, Gonneke Willemsen², Jan F. J. van Leeuwe¹

¹Radboud Universiteit Nijmegen, the Netherlands, ²Vrije Universiteit Amsterdam, the Netherlands,
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 ($a^2 = 0.77$) and young adults ($a^2 = 0.67$ for 19–21-year olds and $a^2 = 0.69$ for 22–24-year olds) than in younger adolescents ($a^2 = 0.00$ for males and $a^2 = 0.53$ 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.

### Identification of three imprinted X-linked genes: Xlr3b, Xlr4b and Xlr4c in a Mouse model for Turner’s syndrome

Adam S. Raefski1, Michael J. O’Neill1

1Molecular and Cell Biology, University of Connecticut, 354 Mansfield Road, U-2131, Storrs, Connecticut 06269-2131 USA, e-mail: adam.raefski@uconn.edu. Grant Support: US National Institute of Neurological Disease and Stroke

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 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 (2005) Child Dev 76(6):1217–1233. Purcell S, Koenen KC (2005) Behav Genet 35(4):491–498]. 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.

### Mediating and moderating effects of measured family environments in the classical twin design

Thomas S. Price1, Sara R. Jaffee2

1ITMAT, School of Medicine, Room 807, BRB II/III, 421 Curie Boulevard, University of Pennsylvania, Philadelphia, PA 19104 USA, e-mail: tom@spirit.gcrc.upenn.edu, 2Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA

Family-wide environments—environmental variables that are measured at the family level, such as SES—can...
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.

Genotype-environment interplay and cognitive aging

Chandra A. Reynolds1

1Psychology Department, University of California, Riverside CA, 92521, USA, e-mail: chandra.reynolds@ucr.edu. Grant Support: R01-AG17561; R01-AG04563; R01-AG10175; MacArthur Foundation Research Network on Successful Aging; Swedish Council for Social Research (97:0147:1B)

The interplay of genetic and environmental markers on cognitive performance in the second half of the life-span 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.

Predictors and consequences of negative emotionality in the Colorado adoption project

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

1Institute for Behavioral Genetics, CU Boulder, Boulder, CO, 80309-0447, USA, e-mail: rhea@colorado.edu. Grant Support: HD010333; HD036773; DA011015

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 [Rhea SA, Bricker J, Corley RP (2005) Behav Genet 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 (0.52, \( P < 0.00, n = 59 \); 0.45, \( P < 0.00, n = 232 \); and 0.43, \( P < 0.00, n = 238 \) 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 (0.13, \( P < 0.01, n = 0.425 \)). 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 (0.33, \( P < 0.001, n = 841 \)) and partner aggression in early adulthood (0.24, \( P < 0.001, n = 768 \)). 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.

The mother-daughter-aunt-niece (MDAN) design, applied to cross-generational NLSY fertility variables

Joseph Lee Rodgers1, David Bard1, Warren Miller2

1Department of Psychology, Professor, 455 W Lindsey, University of Oklahoma, Norman, OK, 73019, e-mail: jrodgers@ou.edu, 2Transnational Family Research Inst. Grant Support: NICHD Grant RO1-HD043265

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.

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

Angelica Ronald¹, Francesca Happé¹, Robert Plomin¹

¹SGDP Centre, Box P083, De Crespigny Park, Denmark Hill, Institute of Psychiatry, London, London, SE5 8AF, UK, a.ronald@iop.kcl.ac.uk. Grant Support: The Twins Early Development Study is funded by the Medical Research Council, grant G0500079

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 (0.11–0.33). 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 = 0.29–0.50). 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 [Ronald A, Happé F, Plomin R (2005) Dev Sci 8:444–458; Ronald A, Happé F, Bolton P, Butcher LM, Price TS, Wheelwright S, Baron-Cohen S, Plomin R (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.

Maternal behavior in mice: quantitative trait loci (QTL) mapping

Pierre L. Roubertoux¹, ⁴, Isabelle Le Roy², Marc Jamon¹, Michèle Carlier³

¹CNRS-UMR6196 Plasticité et physio-pathologie de la motricité, Marseille, France, ²Institut de transgénose, Orléans, France, ³Institut Universitaire de France and CNRS-UMR6146 Laboratoire de psychologie cognitive, Marseille, France. Grant support: CNRS, Université de Provence and Université de la Méditerranée, ⁴UMR 6196 – P3M, Génomique Fonctionnelle, Comportements et Pathologies, 31 Chemin Joseph-Aiguier, 13402 Marseille cedex 20, France; e-mail: rouber@dpm.cnrs-mrs.fr

The behaviors recorded during a retrieving test are reliable predictors of maternal behavior in mice. QTL mapping may pave the way for deciphering the neurochemical correlates of care to pups. A standardized retrieving test revealed marked differences among a set of 12 inbred strains of mice. The C57BL/6J and NZB/BINJ strains appeared as being highly contrasted for most of the behaviors recorded during the retrieving test. The NZB/BINJ were poorer retrievers, in general, than C57BL/6J females. The two strains were selected to generate a segre-
gating population of F2 females. QTL mapping was performed for several measures of maternal behavior with this population. The weight of the nest was significantly linked to chromosome 10. Time to retrieve the whole litter was linked to chromosomes 11 (significant linkage) and 12 and 15 (suggestive linkage). An interesting behavior was move away, defined as the number of times the female drew away from one of her pups, situated outside the nest, without their being transported. A highly significant QTL appeared on chromosome 11 (lod score: 9.78) and on chromosome 17 (significant lod score: 4.15). The QTL for three other behavior were also investigated. Regions on chromosomes 10, 11 and 15 were common to several retrieving behaviors. The genetic structure that was show by the QTL analysis was compared to the factorial structure shown by the analysis of the correlations between strains that approximate additive correlations.

**Preliminary evidence for genetic effects on sexual aggression**

Pekka Santtila¹, N. Kenneth Sandnabba¹

¹Department of Psychology, Tehtaankatu 2, Åbo Akademi University, Turku, Finland, 20500, Finland, e-mail: pekka.santtila@abo.fi. Grant Support: The Academy of Finland, The Åbo Akademi Foundation

There is extensive evidence that antisocial, including aggressive, behavior is influenced by genetic factors [Rhee SH, Waldman ID (2002) Psychol Bull 3:490–529] with subtypes of aggression being differentially controlled by genes and environment [Ligthart L, Bartels M, Hoekstra RA, Hudziak JJ, Boomsma DI (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; Koss MP, Oros C (1982) J Consult Clin Psychol 50:455–457]. Of the 1,312 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.

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

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

¹Psychiatry, Campus Box 8134, 660 S. Euclid Avenue, Washington University School of Medicine, Saint Louis, MO 63110 USA, e-mail: carolyn@matlock.wustl.edu. Grant Support: NIAAA AA56583D; NIDA DA18660; NIAAA AA09022; NIAAA AA11998

Childhood sexual abuse (CSA) has been associated with increased risk for developing alcohol dependence (AD) after accounting for familial liability to AD [Nelson EC 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 Res 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.

**Cross-situational and context specific effects on activity level**

Kimberly J. Saudino

Psychology Department, 64 Cummington St., Boston University, Boston, MA, 02215, USA, e-mail: ksaudino@bu.edu. Grant Support: Supported by grant MH062375 from the National Institute of Mental Health

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.

**IQ similarity in virtual twins: developmental trends**

Nancy L. Segal, Shirley McGuire, June Havlena, Patricia Gill

California State University, Psychology, 800 N. State College Blvd., CSU Fullerton, Fullerton, CA, 92834, USA, e-mail: nsegal@fullerton.edu, University of San Francisco. Grant Support: NIMH

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 and Hershberger, 2005) reported an IQ intraclass correlation of 0.26 ($P < 0.01$, $n = 113$ pairs) and a IQ subtest profile correlation of 0.07 ($n = 111$). 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.

**Neurogenetic polymorphisms and the adolescent educational career**

Michael J. Shanahan, Lance D. Erickson, Stephen Vaisey

University of North Carolina, Chapel Hill, Brigham Young University. Grant Support: NIMH, Sociology, CB 3210, Hamilton Hall, UNC – Chapel Hill, Chapel Hill, NC, 27599, e-mail: 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 determinants of the intensity of neuronal signaling. “Low levels” of signaling are thought to co-vary 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.

**Parental warmth and empathy: familial influences reconsidered**

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

¹Human Relations, 2-15-45 Mita, Graduate School of Keio University, Minato-ku, Tokyo, 108-8345, Japan, e-mail: kana-s@sa2.so-net.ne.jp, 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

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 [Purcell S (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.

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

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

¹Center for Family Research, 2300 K Street, NW 3rd Floor, George Washington University, Washington, DC, 20037, USA, e-mail: cfrels@gwumc.edu, ²Karolinska Institute. Grant Support: This project was supported by grant RO1 MH54610 from the National Institutes of Mental Health.

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. Pachana NA et al (2005) Int J Behav Med 12:103–110]. Additionally, there is evidence that people choose dogs that resemble them in some way [Roy MM, Christenfeld NJS (2004) Psychol Sci 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.

**Marital quality as a moderator for genetic and environmental influences on parenting**

Jennifer A. Ulbricht¹, Jody Ganiban¹, Jenae M. Neiderhiser², Tanya M. M. Button³

¹Department of Psychology,2125 G St. NW, George Washington University, Washington, DC 20037 USA, e-mail: jau5b@gwu.edu, ²Center for Family Research, George Washington University, Washington, DC, ³Institute of Behavior Genetics, Boulder, CO. Grant Support: William T. Grant Foundation R01MH43373 (Reiss); R01MH59014 (Neiderhiser)

Marital quality has been linked to child externalizing and internalizing behaviors in a number of studies, often with parenting as the mediator [Davies, Cummings
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 was to examine whether marital satisfaction moderates the influence of genetic and environmental factors on parenting behavior. 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, child report and observer-rated taped dyadic interactions. Composites for marital and parenting constructs were compiled and have been used in a number of previous studies. The model of latent GE interaction developed by Purcell [(2002) Twin Res 5:572–576], was used to explore the G x marriage effects on parenting behaviors with both marital relationship and parenting as continuous variables. The genetic and environmental influences on parenting were found to change with levels of marital conflict/satisfaction. The moderator effects also appeared qualitatively different for measures of specific marital conflict and global satisfaction. Differences in the moderation pattern were also noted between mothers and fathers.

**Genetic and environmental effects on sexual excitation and sexual inhibition in the human male**

Markus Varjonen¹, Pekka Santtila¹, N. Kenneth Sandnabba³

¹Department of Psychology, Arken, Biskopsgatan 3, Åbo Akademi University, Turku 20500, Finland, e-mail: mvarjone@abo.fi

The Sexual Inhibition and Sexual Excitation Scales (SIS/SES) measure the propensity for sexual inhibition and excitation in men [Janssen E, Vorst H, Finn P, Bancroft J (2002) J Sex Res 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 [Bancroft J (1999) Neurosci Biobehav Rev 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.

**Is COMT a risk factor for ADHD? Testing for association with multiple markers in a gene-based framework**

Irwin D. Waldman¹,², Ian R. Gizer¹, Jesen A. Fagerness², Casey L. McGrath², David C. Rowe⁴

¹Psychology, 532 N. Kilgo Circle, Emory University, Atlanta, GA 30322, USA, e-mail: psyiw@emory.edu, 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

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.
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.

Heritability of subjective wellbeing in a representative sample

Alex Weiss1, Michelle Luciano2, Timothy C. Bates1

1Psychology, 7 George Square, University of Edinburgh, Edinburgh, Scotland, EH8 9JZ, UK, e-mail: tim.bates@ed.ac.uk, 2Queensland Institute of Medical Research

Well-being is known to be heritable. Here we present the first joint analysis of well-being and personality, testing the hypothesis that the genetic basis of well-being is due to normal personality variance in Extraversion, Neuroticism, and Conscientiousness. Data from 973 twin pairs in the MIDUS project (a representative US sample) are presented, indicating modest heritability for well-being with some evidence for sex-limitation. Multivariate modeling including measures of Openness, Conscientiousness, Extraversion, Agreeableness and Neuroticism (the major Five Factor Model domains of personality), revealed genetic correlations between well-being and personality (E & N in particular), and a reduced model indicated no significant genetic effects on well-being beyond those due to personality.

Genetic influence on female sexual function

Katarina Witting1, Pekka Santtila1, N. Kenneth Sandnabba1

1Department of Psychology, Åbo Akademi University, Åbo, FIN-20500, Finland, e-mail: katarina.witting@abo.fi

Two recent twin studies reported a genetic influence on female orgastic function with heritability estimates ranging between 31% and 51% [Dawood K, Kirk KM, Bailey JM, Andrews PW, Martin NG (2005) Twin Res Human Genet 8:27–33; Dunn KM, Cherkas LF, Spector TD (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, Rosen R, Brown C, Heiman J, Leiblum S, Meston C, Shabsigh R et al. (2000) J Sex 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 years and 43 years of age. The genetic, shared environmental, and non-shared environmental influences were estimated using intraclasc 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.

Evidence for epistasis among the D2 family of dopamine receptor genes in smoking-related behaviors

David J. Vandenbergh1,2,3, Richard J. O’Connor1, Michael D. Grant1,2, Akilah L. Jefferson2, George P. Vogler1,2, Andrew A. Strasser1, and Lynn T. Kozlowski1

1Department of Biobehavioral Health, 2Center for Developmental and Health Genetics, The Pennsylvania State University, University Park, PA 16802. Grant Support: CA81639 from the National Cancer Institute and A Tobacco Settlement grant from the Pennsylvania Department of Health to D.J.V. The Department specifically disclaims responsibility for any analyses, interpretations, or conclusions, 3Center for Dev. & Health Genetics, Penn State University, University Park, PA 16802 USA, e-mail: 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.

A behavioral genetic study of trait emotional intelligence

Philip A. Vernon¹, Ashley Mackie¹, K. V. Petrides², Julie Aitken Harris³, Janice Bacher¹

¹Department of Psychology, University of Western Ontario, London, Ontario, N6A 5C2, Canada, e-mail: vernon@uwo.ca, ²Institute of Education, University of London, ³Administrative and Commercial Sciences, University of Western Ontario. Grant Support: Social Sciences and Humanities Research Council of Canada

Trait Emotional Intelligence (EI) refers to behavioral dispositions and self perceived abilities that influence how well one understands and deals with emotional information. The present study examined the genetic and environmental basis of trait EI. A sample of 152 pairs of male and female monozygotic and dizygotic twins aged 18–68 years completed the Trait Emotional Intelligence Questionnaire (TEIQue). The TEIQue is a 153-item self-report questionnaire which measures 20 dimensions of EI. Pearson correlations were computed between the TEIQue scores of MZ and DZ twins. Univariate behavior-genetic model-fitting was then employed to determine the relative contributions of genetic and environmental factors to individual differences in the development of trait EI. Results showed that all but two of the 20 EI dimensions are best fit by an AE model with $h^2$ values ranging between 0.25 and 0.55. Heritability of the total EI score is 0.33. As this was the first behavior genetic study of trait EI, future research is needed to replicate our results. We are also currently collecting NEO-PI-R data from the same sample of twins and will then conduct multivariate analyses between the TEIQue and the Big-5 personality dimensions.

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

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

¹Biological Psychology, van der Boechorststraat 1, Vrije Universiteit, Amsterdam, 1081BT, The Netherlands, e-mail: 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 ($r = 0.82$, $P < 0.001$). 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.

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

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

¹Department of Biological Psychology, van der Boechorststraat 1, Vrije Universiteit, Amsterdam, 1081BT, The Netherlands, e-mail: ahm.willemsen@psy.vu.nl. Grant Support: Dutch Heart
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.

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

Shinji Yamagata1, Yusuke Takahashi1, Nobuhiko Kijima2, Yutaka Ono3, Juko Ando4
1Cognitive and Behavioral Science, Shigemasu Laboratory, 3-8-1 Komaba, The University of Tokyo, Meguro-ku, Tokyo, 158-0093, Japan, e-mail: yamagata@bayes.c.u-tokyo.ac.jp, 2Psychological Laboratory, Keio University, Kanagawa, Japan, 3Health Center, Keio University, Kanagawa, Japan, 4Faculty of Letters, Keio University, Tokyo, Japan

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 0.46, 0.28, and 0.29, respectively. Experience of stressful life events was only weakly correlated with depressive and anxious mood, both genetically (rg = 0.24 and 0.17, respectively) and environmentally (re = 0.17 and 0.16, respectively). However, analyses of gene–environment interaction using continuous moderator variables [Purcell S (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.

Tested lateral visual and motor behavior interaction identify suspected aspects of Dyslexia

Rowe A. Young1
15853 N.Paseo Niquel, Tucson, AZ 85718 USA, e-mail: rowey@aol.com
Acknowledgements: Thanks for contributions and collaboration from: Benson Ginsburg, Dawn Bradway, Fred Kort (University of Connecticut), Jim Kaple, Zhao Chen, Guanglin Wu (University of Arizona)

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 using the YGLD (Young Ginsburg Lateral Direction Assessment), 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, the test reveals that consistent eye dominance patterns, are signifi-
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.

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

Mo Zheng\textsuperscript{1,2}, Laura A. Baker\textsuperscript{1}

\textsuperscript{1}Psychology Department, University of Southern California, SGM 501, \textsuperscript{2}Psychology, USC; Los Angeles CA 90089-1061 USA, e-mail: mzheng@usc.edu. Grant Support: NIMH #58354

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.