Kristen C. Jacobson, Ph.D. (Department of Psychiatry & Behavioral Neuroscience). Kristen Jacobson is Associate Professor in the Department of Psychiatry and Behavioral Neuroscience at The University of Chicago. Through a combination of collaborative and independent studies Dr. Jacobson has developed a program of research that sheds light on the underlying biological pathways through which social and environmental experiences "get under the skin" to affect behavior, which in turn can help to better understand the underlying causes of mental health. Likewise, her research investigating how individual differences, including individual differences in biological and genetic characteristics, moderate the effects of environmental and social factors on behavior can help to inform and design more targeted intervention and prevention programs. Dr. Jacobson is a behavioral geneticist by training, and is currently Co-Associate Director of Twin Projects at the University of Chicago. Her behavioral genetic work has focused primarily on identifying gene X environment (gXe) interactions and on using genetically informative designs to better understand the development and structure of problem behavior. In addition, Dr. Jacobson's current research uses interdisciplinary approaches to examine the joint interplay of environmental, social, psychological, and biological influences on adolescent development. Newer research interests concern the underlying neurobiology of socioemotional development, the role of oxytocin and vasopressin in sensitivity to social environments, and the influence of human-animal interaction on biology and behavior. As a behavioral scientist, Dr. Jacobson bridges the gap between biological and social sciences. Her research program is unique in that it is highly interdisciplinary, spanning fields of developmental psychology and psychiatry, behavioral genetics, sociology, anthrozoology, neuroscience, and endocrinology.

***Training*.**

A.B., Psychology (*cum laude*) and English, Cornell University, Ithaca, NY, 1990

M.S., Human Development & Family Studies, Pennsylvania State University, State College, PA, 1994

Ph.D., Human Development & Family Studies, Pennsylvania State University, State College, PA, 1999

Post Doctoral Fellow, Virginia Institute for Psychiatric & Behavioral Genetics, Virginia Commonwealth University, Richmond, VA, 1999-2000

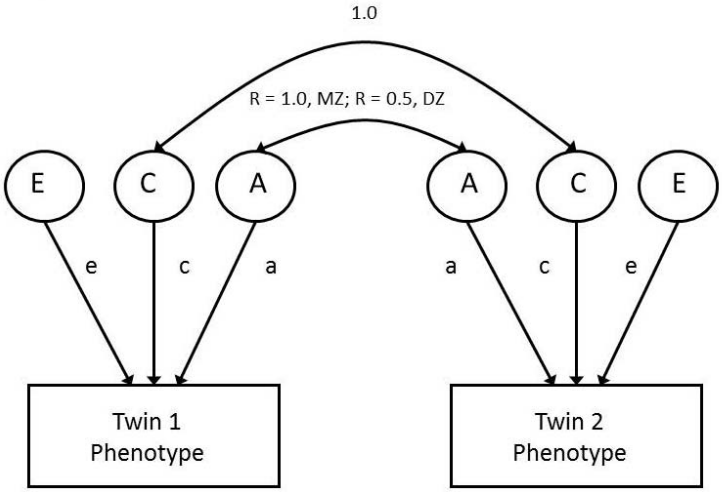
***Research Programs*.**

Behavioral Genetics

Traditionally, behavioral genetics designs rely on studies of identical (monozygotic, MZ) and fraternal (dizygotic, DZ) twins to determine the extent to which genes and

environments contribute to individual differences in behaviors or traits. ***Figure 1*** shows the standard behavioral genetic model for a single trait. Because identical twins share 100% of their segregating genes and fraternal twins share approximately 50% of their segregating genes, behavioral genetic models can be used to decompose the variation in any given phenotype into three sources: additive genetic factors (A), common shared environmental factors (C), and nonshared environmental factors (E). The proportion of individual differences in a given trait that is due to genetic factors is referred to as the heritability of a trait. Shared environmental factors include those environmental factors that serve to make individuals in a family similar to one another. Common shared environmental influences include socioeconomic status, family structure, and shared peer influences. To be considered a shared environmental influence, the environmental influence must be experienced by both individuals. Thus, shared environmental factors (C) are correlated 1.0 across twins, regardless of whether they are identical or fraternal twins. In addition, the shared environmental influence must have the same influence on behavior for both individuals. For example, the experience of parental divorce is an environmental factor that is shared by two siblings in the same family. However, if one sibling responds to the parental divorce by throwing himself into his studies, and the other sibling responds by acting out and skipping school, then parental divorce is not likely to be a shared environmental influence for the cognitive development of these two siblings. If divorce typically had dissimilar effects on siblings, it would count mostly as a nonshared environmental influence. Nonshared environmental influences are any environmental influences that serve to make individuals dissimilar. Nonshared environmental influences can occur if exposure to the environment is not shared by siblings. For example, birth order, accidents, and different peer groups are nonshared environmental influences. Likewise, as stated above, "shared" environmental factors that have different influences on behavior for individuals are considered nonshared environmental factors, as are measurement errors, which are assumed to be random and uncorrelated across twins. By definition, nonshared environmental factors do not correlate across twins.

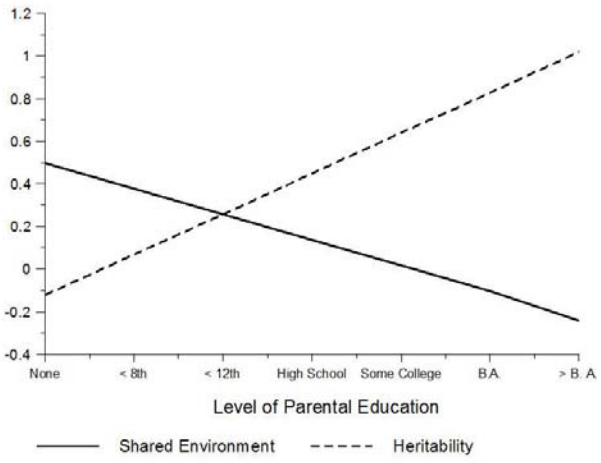
Figure 1. The Univariate ACE Model



Behavioral genetics within an ecological framework

While the importance of genetic influence on individual differences in personality, behavior, and mental and physical health is now widely accepted, Dr. Jacobson's early behavioral genetic studies challenged the notion that heritabilities are the same for

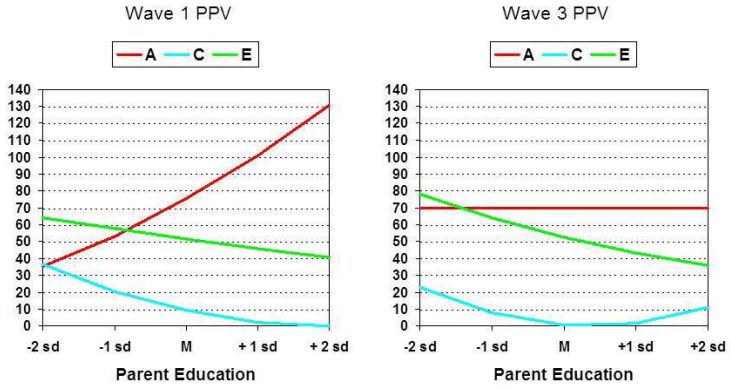
Figure 2. Gene x Environment Interaction for Verbal IQ



individuals in different ecological niches.

For example, she discovered that the importance of genetic factors on adolescent body mass index varied as a function of both gender and race/ethnicity (*Jacobson & Rowe, 1999*). Likewise, her collaborative research was the first to show that the heritability of cognitive ability varies among individuals in different socioeconomic contexts (*Rowe, Jacobson, & van den Oord, 1999*). As seen in ***Figure 2***, the heritability of verbal IQ is greater among adolescents whose parents are more highly educated. Among highly educated families, the heritability of verbal IQ was .76, and shared environmental effects were negligible. In contrast, among less well-educated families, shared environmental factors accounted for a significant proportion of variance (23%), and genetic factors accounted for only 26% of the variance. Results from this paper have been widely replicated in other samples of children and adolescents, and the socioeconomic moderation of genetic factors on cognition is considered one of the best examples of gene X environment (gXe) interactions in twin studies.

Figure 3. GxE interactions for Peabody Picture Vocabulary (PPV) in Adolescence (Wave 1) and Young Adulthood (Wave 3)



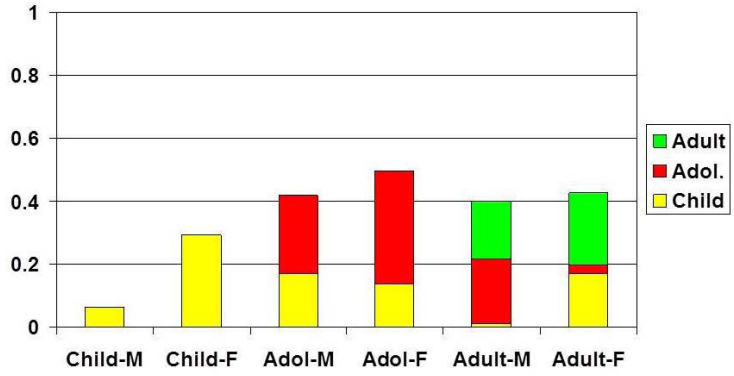
Interestingly, follow-up work examining whether the heritability of cognitive ability varies across socioeconomic context in studies of older adults has not found evidence for gXe interactions. In a study of word recognition in middle-aged male twins from the Vietnam Era Twin Registry (VETR), Dr. Jacobson found that while the importance of shared environmental factors was higher among adults from less well-educated families, there was no difference in the importance of genetic factors (*Kremen, Jacobson, et al., 2003*). A more recent study using the VETR sample also failed to find any evidence for moderated genetic or environmental influences on general cognitive ability in early adulthood (*Grant, et al., 2010*). The discrepancy in results among studies of children and adolescents versus studies of adults suggests the intriguing possibility that there may be a critical period of development during which early environmental experiences can alter genetic programming of cognition. Recently, Dr. Jacobson tested this hypothesis directly using longitudinal data from the twin/sibling sample of the National Longitudinal Study of Adolescent Health (*Jacobson &*

Vasilopoulos, under review). This study, which was a replication and extension of results reported in *Rowe et al. (1999),* found that, as predicted, parental education moderated additive genetic influences (A) on Peabody Picture Vocabulary (PPV) in adolescence (Wave 1) but not in young adulthood (Wave 3) (see ***Figure 3***). In addition, the effects of shared environmental factors (C) were markedly weaker in young adulthood, consistent with other longitudinal research.

These results suggest that the importance of childhood environmental factors on individual differences in cognitive ability decreases over time, and also that childhood environmental factors "lose" their ability to modify genetic influences on adult phenotypes. However, recent work from Dr. Jacobson's laboratory indicates that untreated hypertension suppresses genetic influence on cognitive ability among middle aged male twins (*Vasilopoulus, et al., 2012b*), indicating that the types of environmental factors that can disrupt genetic programming of cognition may vary across the lifespan.

Behavioral genetics and the development and structure of problem behavior

Figure 4. Heritability Estimates for Antisocial Behavior among Males (M) and Females (F) at Different Developmental Periods



Dr. Jacobson also uses multivariate behavioral genetic models to better understand the development and structure of problem behavior. One of her early papers was the first to report that the heritability of conduct disordered behavior has decreased over time, due primarily to an increase in the importance of shared environmental factors among twins born in more recent cohorts (*Jacobson et al., 2000*). Another paper showed that the importance of genetic factors on antisocial behavior varies as a function of both gender and age. As can be seen in ***Figure 4***, genetic influence on antisocial behavior increases from childhood to adolescence and adulthood, and the increase in heritability occurs earlier among females than males (*Jacobson et al., 2002*). These findings suggest that physiological and/or social factors associated with puberty may "turn on" genes related to antisocial behavior, a hypothesis Dr. Jacobson is currently testing in other samples.

Dr. Jacobson has further combined multiple measures of problem behaviors into single multivariate behavioral genetic models to better understand the structure of problem behavior. In a paper published in the *American Journal of Psychiatry*, she found that common genetic factors accounted for most of the comorbidity between different types of substance use and misuse among adult male twins in the Virginia Twin Registry (*Kendler, Jacobson, Prescott, & Neale, 2005*). With Laura Baker at the University of Southern California, she discovered that combing reports of childhood problem

behaviors from parents, children, and teachers results in higher heritabilities than analyses based on only a single reporter, supporting strong genetic influence on shared views of problem behavior (*Baker, Jacobson, et al., 2007*). Recent work with the Pennsylvania Twin Cohort sample has revealed that different aggressive behaviors are underpinned by two distinct etiological factors with different genetic and nonshared environmental influences (*Yeh, Coccaro, & Jacobson, 2010*).

Relatedly, multivariate behavioral genetic models including both psychosocial variables and behavioral outcomes have begun to shed light on processes of gene-environment interplay in the development of problem behaviors. For example, *Jacobson & Rowe (1999)* found that the relationship between attachment to family, attachment to school, and adolescent depressed mood is explained in large part by common genetic factors. Work done with colleagues at VCU has implicated a complex pattern of genetic and environmental influences on longitudinal relationships between peer group deviance and conduct disordered behavior (*Kendler, Jacobson, Meyer, & Eaves, 2008*) and peer group deviance and cannabis use (*Gillespie et al., 2009*).

In 2003, Dr. Jacobson received a Mentored Scientist Training Award (K01) from the National Institutes of Mental Health to examine the role of genetic and environmental influences on biological measures that may serve as endophenotypes for antisocial and related problem behaviors. She has been involved in projects investigating the underlying genetic and environmental architecture of physiological measures (e.g., *Crider et al., 2004; Tuvblad et al., 2010*) and cortisol response *(*e.g., *Franz et al.,2010*), and she has been a long-time collaborator on the Vietnam Era Twin Study of Aging (VETSA), which includes analysis of genetic and environmental influences on brain structure related to cognitive aging (e.g., *Kremen et al., 2010; Panizzon et al., 2009, 2012*). One of her recent postdoctoral trainees has used the VETSA sample to better understand relationships among physical health and cognitive function in older adults (*Vasilopoulos et al., 2012a, 2013*). Finally, Dr. Jacobson is a Co-Investigator on Dr. Emil Coccaro's NIMH-funded study, *Pathways to Aggression*, which is collecting functional magnetic resonance imaging (fMRI) data from over 200 pairs of twins from the Pennsylvania Twin Cohort to better understand how genetic factors influence socioemotional processing related to aggressive behavior.

Example Publications:

Jacobson, K.C., & Rowe, D.C. (1998). Genetic and shared environmental influences on adolescent body mass index: Interactions with race and sex. *Behavior Genetics, 28*, 265-278.

Jacobson, K.C., & Rowe, D.C. (1999). Genetic and environmental influences on the relationships between family connectedness, school connectedness, and adolescent depressed mood: Sex differences. *Developmental Psychology, 35*, 926-939.

Rowe, D.C., Jacobson, K.C., & Van den Oord, E. J. C. G. (1999). Genetic and environmental influences on vocabulary IQ: Parental education as moderator. *Child Development, 70*, 1151-1162.

Jacobson, K.C., Prescott, C.A., & Kendler, K.S. (2000). Genetic and environmental influences on juvenile antisocial behaviour assessed on two occasions. *Psychological Medicine, 30*, 1315-1325.

Jacobson, K.C., Prescott, C.A., & Kendler, K.S. (2002). Sex differences in genetic and environmental influences on antisocial behavior from childhood to adulthood. *Development and Psychopathology, 14*, 395-416.

Kendler, K.S., Jacobson, K.C., Prescott, C.A., & Neale, M.C. (2003). The specificity of genetic and environmental risk factors in men for the illicit use and abuse/dependence of cannabis, cocaine, hallucinogens, sedatives, stimulants, and opiates. *American Journal of Psychiatry, 160(4)*, 687-695.

Crider, A., Kremen, W.S., Xian, H., Jacobson, K.C., Waterman, B., Eisen, S.A., Tsuang, M.T., & Lyons, M.J. (2004). Stability, consistency, and heritability of electrodermal response lability in middle-aged Male Twins. *Psychophysiology, 51*, 501-509.

Jacobson, K.C. (2005). Genetic influence on the development of antisocial behavior. In K. Kendler & L. Eaves (Eds) *New Directions for Psychiatric Genetics* (pp. 197-232). Washington, DC: American Psychiatric Press, Incorporated.

Jacobson, K.C. (2005). Gene-environment interaction. In B.S. Everitt & D.C. Howell (Eds.) *Encyclopedia of Statistics in Behavioral Science (Wiley Series in Probability and Statistics, Volume 2, S. Purcell, Volume Editor*, pp. 698-701). Chichester, England: John Wiley & Sons, Ltd.

Kremen, W.S., Jacobson, K.C., Xian, H., Eisen, S.A., Waterman, B., Toomey, R., Neale, M.C., Tsuang, M.T., & Lyons, M.J. (2005). Heritability of Word Recognition in Middle-Aged Men Varies as a Function of Parental Education. *Behavior Genetics, 35*, 417-433.

Neale, M.C., Jacobson, K.C., & Røysamb, E. (2006). Multivariate genetic analysis of sex-limitation in human populations. *Twin Research and Human Genetics, 9*, 481-489.

Baker, L.A., Jacobson, K.C., Raine, A., Lozano, D.I., & Bezdjian, S.J. (2007). Genetic and environmental bases of childhood antisocial behavior: A multi-trait multi-method twin study. *Journal of Abnormal Psychology, 116*, 219-235.

Kendler, K.S., Jacobson, K.C., Gardner, C.O., Gillespie, N.G., Aggen, S.A., & Prescott, C.A. (2007). Creating a social world: A developmental twin study of peer group deviance. *Archives of General Psychiatry, 64*, 958-965.

Jacobson, K.C., Beseler, C.L., Su, J., Faraone, S.V., Glatt, S.J., Kremen, W.S., Lyons, M.J., & Tsuang, M.T. (2008). Ordered subsets linkage analysis of antisocial behavior in substance use disorder among participants in the Collaborative Study on the Genetics of Alcoholism. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 147B*, 1258-1269.

Kendler, K.S., Jacobson, K.C., Myers, J.M., & Eaves, L.J. (2008). A genetically informative developmental study of the relationship between conduct disorder and peer deviance in males. *Psychological Medicine, 38,* 1001-1011.

Gillespie, N.A., Neale, M.C., Jacobson, K.C., & Kendler, K.S. (2009). The role of peer group deviance in genetic and environmental pathways to cannabis use. *Addiction, 104*, 420-429.

Panizzon, M.S., Fennema-Notestine, C., Eyler, L.T., Jernigan, T.L., Prom-Wormley, E., Neale, M., Jacobson, K., Lyons, M.J., Grant, M.D., Franz, C.E., Xian, H., Tsuang, M., Fischl, B., Seidman, L., Dale, A., & Kremen, W.S. (2009). Distinct Genetic Influences on Cortical Surface Area and Cortical Thickness. *Cerebral Cortex, 19*, 2728-2735.

Kremen, W.S. & Jacobson, K.C. (2010). Introduction to the special issue on Pathways between Genes, Brain, and Behavior. *Behavior Genetics, 40(2),* 111-113.

Franz, C.E., York, T.P., Eaves, L.J., Eisen, S.A., Hauger, R., Hellhammer, D., Jacobson, K.C., Levine, S., Lupien, S., Lyons, M.J., Mendoza, S., Prom-Wormley, E., Xian, H., & Kremen, W.S (2010). Genetic and environmental contributions to basal levels and responsivity of salivary cortisol in middle-aged men. *Behavior Genetics, 40, 467-479.*

Grant, M.D., Kremen, W.S., Jacobson, K.C., Franz, C., Xian, H., Eisen, S.A., Toomey, R., & Lyons, M.J. (2010). Does parental education have a moderating effect on the genetic and environmental influences of general cognitive ability in early adulthood? *Behavior Genetics, 40, 438-446.*

*Tuvblad, C., Isen, J., Baker, L.A., Raine, A., Lozano, D., & Jacobson, K.C. (2010). The genetic and environmental etiology of sympathetic and parasympathetic arousal in children. *Behavior Genetics 40, 452-466.*

*Yeh, M.T., Coccaro, E.F., & Jacobson, K.C. (2010). Multivariate behavioral genetic analyses of aggressive behavior subtypes. *Behavior Genetics, 40,* 603-617.

Panizzon, M.S., Hauger, R., Eaves, L.J., Chen, C., Dale, A.M., Eyler, L.T., Fischl, B., Fennema-Notestine, C., Franz, C.E., Grant, M.D., Jacobson, K.C., Jak, A.J., Lyons, M.J., Mendoza, S.M., Neale, M.C., Prom-Wormley, E., Seidman, L.J., Tsuang, M.T., Xian, H., & Kremen, W.S. (2012). Genetic influences on hippocampal volume differ as a function of testosterone level in middle-aged men. *NeuroImage, 59*, 1123-1131.

*Vasilopoulos, T., Franz, C.E., Panizzon, M.S., Xian, H., Grant, M.D., Lyons, M.J., Toomey, R., Jacobson[a], K.C., & Kremen[a], W.S. (2012). Genetic architecture of the Delis-Kaplan Executive Function System Trails Making Test: Evidence for distinct genetic influence on executive functioning. *Neuropsychology, 26*, 238-250. ([a]joint senior authors).

*Vasilopoulos, T., Kremen, W.S., Kim, K., Panizzon, M.S., Stein, P.K., Xian, H., Grant, M.D., Lyons, M.J., Toomey, R., Eaves, L.J., Franz, C.E., & Jacobson, K.C. (2012). Untreated hypertension decreases heritability of cognition in late middle age. *Behavior Genetics, 42*: 107-120.

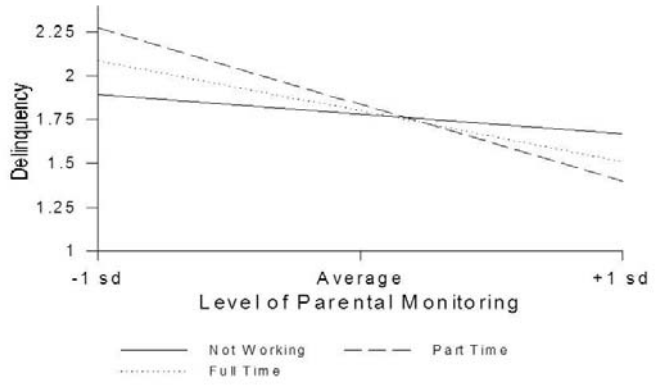
*Vasilopoulos, T., Grant, M.D., Franz, C.E., Panizzon, M.S., Xian, H., Toomey, R., Lyons, M.J., Kremen, W.S., & Jacobson, K.C. (2013). Shared and distinct genetic influences among different measures of pulmonary function. *Behavior Genetics. E-pub available 01/09/13.*

Jacobson, K.C., & *Vasilopoulos, T. (under review). Developmental changes from adolescence to young adulthood in moderating effects of parental education on heritability of vocabulary IQ. *Psychological Science.*

Multiple Levels of Influence on Adolescent Problem Behaviors

Dr. Jacobson's work is greatly influenced by the Bioecological Model developed by Dr. Urie Bronfenbrenner, whom she was privileged to work with after college. This model has guided her research examining influences on youth problem behaviors at different ecological levels, including neighborhood, school, peer, family, and individual psychosocial, biological, and genetic factors. Bronfenbrenner's multi-layered model highlights not only the importance of measuring environmental, biological, social, and genetic factors at multiple levels of analysis, but also the importance of unique person and environmental characteristics as potential moderators of the relationship between family processes and adjustment.

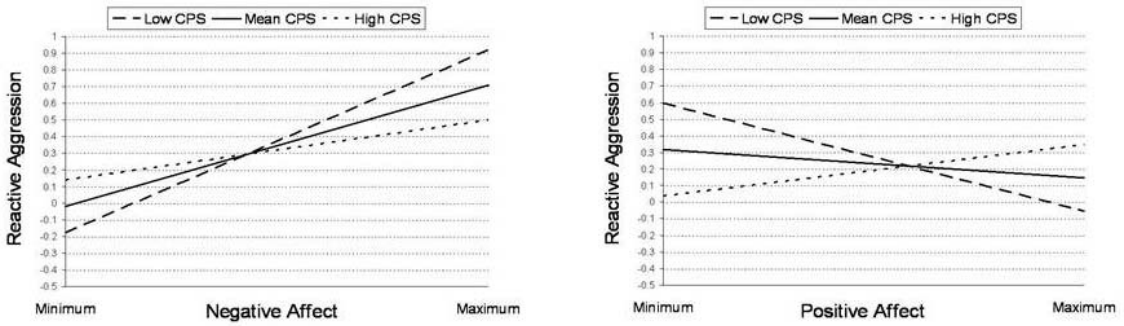
Figure 5. Moderating Effects of Maternal Employment on Relationships between Parental Monitoring and Youth Delinquency



As part of her graduate work at Penn State, Dr. Jacobson applied Bronfenbrenner's Ecological Model to examine person and context moderators of the relationship between family processes and adolescent adjustment. Part of this research focused on gender, age, and maternal work status as moderators of the relationship between parental monitoring and adolescent delinquency. She found that effective parental monitoring had a greater impact on adolescent delinquency among older adolescents compared to younger adolescents among males, but that the reverse was true for females (*Jacobson & Crockett, 2000*). She also found that parental monitoring had a stronger association with adolescent delinquency and sexual behaviors when adolescents had mothers who were employed full- or part-time, suggesting that effective parental monitoring might compensate for a lack of direct supervision (***Figure 5***).

Dr. Jacobson's interest in applying ecological models to understand individual differences in development has continued throughout her career. Recently, her lab published a paper showing that children high on psychopathy are less responsive to parental affect than children with low levels of psychopathic traits (*Yeh, et al., 2011*). As shown in ***Figure 6***, 9-10 year old children with high levels of psychopathy are less responsive to variations in *both* positive and negative parental affect than children lower on psychopathology. This novel finding suggests that psychopathy is related to overall deficits in processing of emotional stimuli, and not just to deficits in the processing of negative (aversive) stimuli. Another of her postdoctoral fellows has conducted a series of projects looking at interactions between individual and environmental characteristics on adult aggression, and found that the relationship between hostile attribution bias and aggression in adults is moderated by levels of impulsivity (*Chen et al., 2012a*) and that exposure to childhood trauma alters the relationships between measures of social information processing and aggression (*Chen et al., 2012b*).

Figure 6. Interactions between Child Psychopathic Traits (CPS) and Parental Positive and Negative Affect



From Neighborhoods to Neurons and Beyond

In 2007, Dr. Jacobson received an NIH Director's New Innovator Award to conduct a multi-phase study investigating how community- and school-level factors moderate biological and psychosocial influences on individual differences in adolescent behavior. This project generated the "Neighborhoods to Neurons and Beyond" (NNB) cohort.

In **Phase I**, Dr. Jacobson obtained in-school survey data from N=3,350 6th-8th graders across 14 public schools in the Chicago area. Individual schools were selected to maximize racial/ethnic and socioeconomic variation. 40.4% of NNB youth were in schools with high racial/ethnic variation, 34.2% in minority schools, including predominantly African American (15.0%) and predominantly Hispanic schools (19.2%), with the remaining 25.5% in predominantly Caucasian schools. Schools differed in the % of students eligible for free meal programs (a marker for SES), ranging from 7-80% (M=44.0%). Importantly, there was variation in SES among African American (34-80%) and mixed race/ethnicity (21-62%) schools, with less variation among Hispanic (65-70%) and Caucasian (7-17%) schools. Phase I also include data from an additional

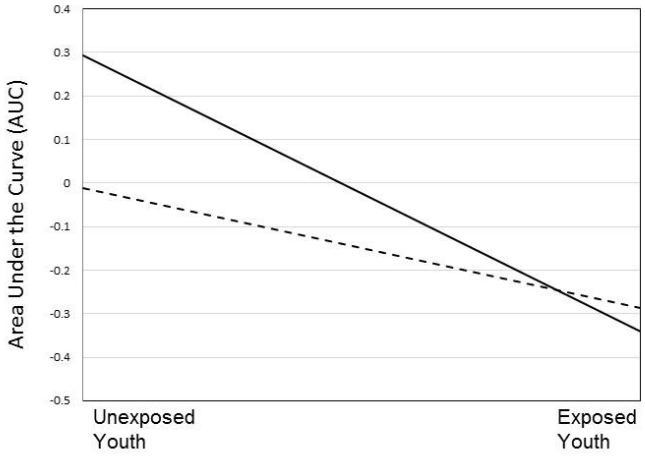
N=232 youth who were enrolled in private/Catholic schools. **Phase II** was an in-lab, follow-up, family study of N=241 youth quasi-randomly selected from the NNB cohort. Phase II obtained detailed self-report and interview data on youth behavior and individual and contextual factors, as well biological and behavioral measures described below. Data were collected from N=241 Phase I youth, their primary caregivers (77.1% biological mothers), and N=137 siblings aged 10-18.

Data collected during Phases I and II include self-report measures on risk/protective factors and youth problem behaviors. Phase II included caregiver reports of family history of psychopathology, demographic characteristics, and youth behavioral problems, and expanded the self-report measures in youth. In addition, youth in Phase II participated in a series of objective, behavioral tasks assessing decision-making (i.e., the Delay Discounting task), impulsivity (e.g., the Go/No-Go task and Balloon Analog Task), stress response (e.g., the Countdown task), and socioemotional processing (e.g., recognition of emotional FACES and affective pictures from the IAPS paradigm). Dr. Jacobson also added measures of empathy and prosocial behavior to the NNB Phase II cohort to look at both positive and negative youth outcomes, and collected plasma to use for baseline measures of oxytocin from N=265 youth and N=59 biological mothers. These samples were assayed by Dr. Sue Carter, one of the world's leading experts in the study of neuropeptides. At present, NNB is the largest sample of baseline oxytocin data in youth. In addition, a subset of N=40 NNB youth participated in an fMRI study of emotion recognition and empathy (**Phase III**) through collaboration with Dr. Jean Decety, and blood was collected from all NNB youth and their parents for use in later genetic and epigenetic analyses.

Associations between exposure to community violence and youth outcomes

Exposure to community violence is one of the strongest predictors of youth outcomes in the NNB sample. During Phase I, NNB youth indicated if they had been exposed to 3 violent events (seen someone shot/stabbed, had someone pull a weapon on them, been jumped) and the frequency at which they heard gunshots during the past month, combined into a single yes/no index of community violence exposure (CVE). 34% of the NNB cohort reported exposure to one or more of these events; percentages of youth within a given school who reported CVE ranged from 15.2-74.2%. The CVE measure was also used in the Phase II study. In both studies, exposed youth showed poorer outcomes (effect sizes range from $d = 0.25$ to $d = 0.96$), including higher rates of delinquency, aggression, depressed mood, and illegal substance use, and lower levels of prosocial behavior and empathy, even after controlling for age, gender, race/ethnicity, and socioeconomic factors. Exposed youth reported significantly ($p < .001$) higher levels of parent-child conflict ($d = 0.75$), providing support for correlations of exposure to violence across both community and family levels. In a manuscript under review (*Jacobson & Chen, under review*), youth reporting CVE ($b = -0.08$, $p<.05$) showed smaller area-under-the-curve (AUC) during the Delay Discounting task, indicating that they were more likely to devalue future rewards relative to immediate rewards. Moreover, the relationship between CVE and AUC was stronger for youth aged 14 and older (***Figure 7***), consistent with current theories on relationships between

Figure 7. Effects of Exposure to Violence on Objective Measures of Decision Making: Moderating Effects of Age

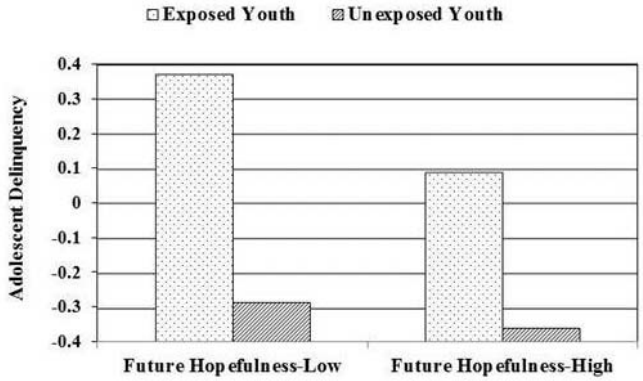


Note. The dashed line represents associations between Community Violence Exposure and AUC for Youth <14; The solid line represents associations between Community Violence Exposure and AUC for Youth 14 and older.

developmental changes in brain structure and function, decision-making, and youth risky behavior. The fact that CVE may alter youth's cognitive and emotional capabilities that underpin risky behavior is the basis for a follow-up R01 grant application submitted to NIDA (currently under review), where new longitudinal data would be collected from the NNB Phase II cohort to determine the extent to which CVE (and other risk factors) alter both behavioral and neurobiological indicators of decision-making that predict trajectories of youth sexual risk and substance use behaviors.

Given the significant negative effect of CVE on youth outcomes, a recent manuscript (*Chen, Voisin, and Jacobson, in press*) examined whether promotive factors across different ecological levels (i.e., future hopefulness, family warmth, school attachment, and neighborhood cohesion) moderated relationships between CVE and youth delinquency in N=2,980 youth from the Phase I NNB study (M_{age}=12.5; 41.1% males). After controlling for demographic factors, delinquency was positively associated with CVE and inversely associated with each of the promotive factors. When interaction effects between all promotive factors and CVE were examined simultaneously, only future hopefulness moderated the relationship between CVE and delinquency. Specifically, CVE had a weaker association with delinquency for youth reporting high levels of future hopefulness in comparison to those with low levels of future hopefulness (***Figure 8***). At the same time, promotive factors from other domains are also important. For example, a second paper from the NNB study found that family factors were especially important in reducing delinquency among impulsive youth (*Chen & Jacobson, in press*). Specifically, parental knowledge had a stronger association with decreased levels of delinquency for adolescents reporting higher levels of impulsivity, compared with youth reporting lower levels of

Figure 8. Future Hopefulness & Youth Exposure to Violence: Joint Effects on Adolescent Delinquency

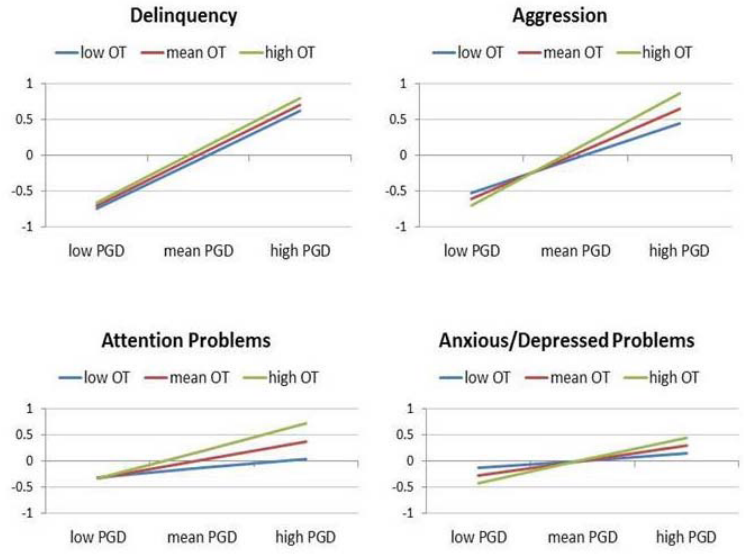


impulsivity. In addition, the inverse relationship between family warmth and delinquency was significant for adolescents with high levels of, but not for those with average or below-average levels of impulsivity.

Oxytocin as a potential biological marker of youth sensitivity to social environments

Dr. Jacobson is currently testing the novel hypothesis that the neuropeptide oxytocin (OT) may serve as a biological marker of youth sensitivity to social environments. Prior research in other labs has shown that OT administration enhances affiliative behaviors, suppresses production of cortisol during physical or psychological challenges, and reduces psychological distress. OT is also implicated in social and affective disorders. Clinical studies have shown decreased levels of OT in adult patients with depression and studies have found that trauma predicts lower levels of basal OT in adults. However, emerging evidence suggests that, under certain conditions, oxytocin may increase *maladaptive* social behaviors (e.g., aggression), indicating that the effects of oxytocin may be sensitive to environmental and social conditions. Analysis of the first N=179 baseline OT samples from Phase II NNB youth revealed that OT is unrelated to CVE or youth behavioral or emotional problems. However, OT correlates with youth reports of attachment to mothers ($r = 0.23$, $p < .05$), validating this measure of OT as a potential biological indicator of social affiliation. In addition, OT interacted with peer deviance to predict youth reports of aggression, attention problems, and anxious/depressed symptoms from the CBCL, but did not interact with peer deviance to predict delinquency. Specifically, youth with higher levels of OT and higher reports of peer deviance show the greatest levels of problem behavior (***Figure 9***), and the relationship between peer deviance and negative outcomes was stronger for youth with higher levels of OT.

Figure 9. Oxytocin (OT) as Modulator of Associations between Peer Group Deviance (PGD) and Youth Problem Behaviors



Example Publications:

Jacobson, K.C., & Crockett, L.J. (2000). Parental monitoring and adolescent adjustment: An ecological approach. *Journal of Research on Adolescence, 10*, 65-97.

*Yeh, M.T., *Chen, P., Raine, A., Baker, L.A., & Jacobson, K.C. (2011). Child psychopathic traits moderate relationships between parental affect and child aggression. *Journal of the American Academy of Child & Adolescent Psychiatry, 50*, 1054-1064.

*Chen, P., Coccaro, E.F., & Jacobson, K.C. (2012a). Hostile attributional bias, negative emotional responding, and aggression in adults: Moderating effects of gender and impulsivity. *Aggressive Behavior, 38,* 47-63.
*Chen, P., Coccaro, E.F., Lee, R., & Jacobson, K.C. (2012b). Moderating effects of childhood maltreatment on associations between social information processing and adult aggression. *Psychological Medicine*, 42, 1293-1304.
*Chen, P, & Jacobson, K.C. (2012). Developmental trajectories of substance use from early adolescence to young adulthood: Gender and racial/ethnic differences. *Journal of Adolescent Health, 50,* 154-163.
*Chen, P., & Jacobson, K.C. (in press). Impulsivity moderates environmental influences on adolescent delinquency: A comparison across family, school, and neighborhood contexts. *Journal of Abnormal Child Psychology*.
*Chen, P., Voisin, D.R., & Jacobson, K.C. (in press). Community violence exposure and adolescent delinquency: Examining a spectrum of promotive factors. *Youth & Society*.
*Chen, P, & Jacobson, K.C. (under review). Longitudinal effects of college attendance on patterns of substance use: A comparison between Caucasians and African Americans. *J of Adolescent Health*.
*Chen, P, & Jacobson, K.C. (under review). Minority youth are less susceptible than non-minority youth to risk factors associated with delinquency and depressed mood. *PLoS One*.
Jacobson, K.C., & *Chen, P. (under review). Exposure to community violence is associated with individual differences in temporal discounting in youth aged 10-18. *Child Development.*

Research on Human-Animal Interactions

Dr. Jacobson's lab is also investigating the effects of human-animal interaction (HAI) on youth biology and behavior, funded by the NICHD. In one study, detailed measures of pet ownership, attitudes towards pets, and attachment to family dogs were added to the Phase II NNB study. In this project, attitudes towards pets and attachment to family dogs are correlated with youth empathy and prosocial behavior, and that these associations with youth socioemotional development are as strong (or stronger) than the effects of positive family, school, and neighborhood characteristics (*Jacobson, forthcoming*). At the same time, the causal effects of HAI have yet to be determined. For example, a recent publication by Dr. Jacobson showed that individual differences in the frequency of playing with pets in middle aged male twins was due, in part, to genetic factors, and that the impact of shared environmental factors, which would include childhood exposure to pets, was negligible (*Jacobson et al., 2012*). Likewise, characteristics of both human *and the animal* are likely to influence attachment to family pets and the potential positive impact of HAI. A forthcoming publication by one of Dr. Jacobson's postdoctoral trainees found that attachment to family dogs varied systematically as a function of specific canine behavioral characteristics, including trainability, separation problems, excitability, and attention-seeking behaviors (*Hoffman et al., in press*).

Currently Dr. Jacobson is conducting a study of the effects of HAI on prosocial behavior and stress reactivity in N=120 young adults aged 18-25. Subjects are brought into Dr. Jacobson's lab for two separate visits. During the second visit, subjects participate in a brief interaction with a trained therapy dog. Subjects also complete several prosocial behavior paradigms, and participate in the Trier Social Stress Test, with the order of tasks counterbalanced across subjects. Plasma samples are taken before and after the HAI to determine whether interacting with a therapy dog increases oxytocin levels. The primary research hypotheses are that HAI will increase prosocial behavior and decrease

biological markers of stress responsivity (i.e., physiological measures and cortisol response), and that the positive effects of HAI will be increased among subjects with a history of positive childhood exposure to dogs.

Example Publications:

Jacobson, K.C., *Hoffman, C.L., *Vasilopoulos, T., Lyons, M.J., Kremen, W.S., & Franz, C.E. (2012). Genetic and environmental influences on frequency of play with pets among middle-aged men: A behavioral genetic analysis. *Anthrozoös, 41*, 441-456.

*Hoffman, C. *Chen, P., Serpell, J.A., & Jacobson, K.C. (in press). Do canine behavioral characteristics predict owner attachment to pet dogs? *Journal of Human Animal Interaction, 1,* 20-37.

Jacobson, K.C. (forthcoming). Effects of pets on youth outcomes: socioemotional adjustment, antisocial behavior, and emotion recognition. Chapter to appear in L. Freund, S. McCune, L. Esposito, & J. Griffin (Eds.). *Social neuroscience of human-animal interactions*. Taylor Francis Publishers.

*Hoffman, C. & Jacobson, K.C. (under review). Benefits of human-animal interaction: A growing area of research in psychology. *Journal of Human Animal Interaction.*

***Funding*.**

Current Projects:

- NIH/NICHD R03 HD070679: *Long-term benefits of child dog ownership: Effects on stress and social behavior*
  PI: Jacobson 01/01/12-12/31/13
- NIH/NICHD R03 HD066598: *They Call it Puppy Love: Epidemiology and biology of the child-dog bond*
  PI: Jacobson 07/01/10-06/30/13
- NIH/NIA R01 AG018386: *VETSA 2: A longitudinal twin study of cognitive aging*
  PI (subcontract): Jacobson 09/15/08-08/31/13
- NIH/NIMH R01 MH080109: *Understanding the pathways to aggression*
  PI: Coccaro 04/16/08-03/31/13
- University of Chicago CTSA-ITM: *Core Subsidy Award*
  PI: Jacobson 09/01/12-05/31/13

Selected Past Projects:

- NIH/OD DP2 OD003021: *From Neighborhoods to Neurons and Beyond*
  PI: Jacobson 09/30/07-08/31/12
-BRF Seed Grant: *A Pilot Study of genetic and environmental influences on amygdala and dorsal anterior cingulate cortex activation: A Twin Study of fMRI*
  PI: Jacobson 06/01/07-05/31/08
-NIH/NIA R01 AG022982: *VETSA longitudinal twin study of cortisol and aging*
  PI (subcontract): Jacobson 09/30/06-06/30/09
-NIH/NIMH R01 MH058354: *Development of conduct problems: Genetic and environmental interface*
  PI (subcontract): Jacobson 01/05/06-06/30/11
-NIH/NIMH K01 MH068484: *Genetics of vulnerability to antisocial behavior*
  PI: Jacobson 09/15/03-08/31/07