BIOGRAPHICAL SKETCH

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NAME: Elysia Poggi Davis

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POSITION TITLE: Professor of Psychology, University of Denver

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Vassar College, Poughkeepsie, NY	BA	06/1996	Psychology
University of Minnesota, Minneapolis, MN	Ph.D.	05/2002	Developmental Psychology
University of California, Irvine, CA	Post Doctoral	06/2003	Psychology

A. Personal Statement

My program of research evaluates the influence on early-life experiences on neurodevelopment. I have led an NIH funded research program that has contributed to an important shift in our understanding of the role of the prenatal environment in fetal neurodevelopment and the ensuing consequences for child and adolescent mental health. Building on this research program, Project 3 evaluates a new form of early adversity, fragmentation or unpredictability of maternal signals. During the past 4 years of the Center funding period, we have demonstrated that early-life exposure to fragmented maternal signals during the prenatal and postnatal periods exerts long-term consequences on cognitive and emotional development. For example, in both rodents and humans early-life exposure to unpredictable maternal sensory signals predicts cognitive impairments, particularly on hippocampal-dependent memory tasks. The parallel between our experimental evidence that unpredictable maternal sensory signals to the pup causally impacts cognitive development in the rat and observational links between these signals and human cognitive functioning supports the argument that predictability of maternal sensory signals influences cognitive development in both species. Thus, these findings strongly suggest that predictability of maternal sensory signals is one of the processes by which maternal care regulates neurodevelopment. In the present proposal we investigate the consequences of early-life fragmentation on a novel outcome, anhedonia.

I am experienced with the methodology to assess adolescents proposed in this application. I have longitudinally assessed this cohort from the prenatal period through early adolescence. In collaboration, with this Conte Center team we have demonstrated our ability to recruit and maintain children and adolescence for long-term follow up studies. Not only do I have the skills and experience necessary to oversee the adolescent assessments, but I also have a collaborative relationship with members of the study team. The succesful collaborative relationship I maintain with the study team combined with the experience of our research team will ensure sucessful implementation of the proposed project.

B. Positions and Honors

Positions and Employment

2002-2003 Post Doctoral Research Associate, Neurobiological and behavioral implications of prenatal stress for infant health and development, University of California, Irvine

2003-2005 Research Specialist, Project Director, Neuroendocrine influences on pregnancy, birth outcome and fetal and infant development, University of California, Irvine
2005-2011 Assistant Professor, Psychiatry and Human Behavior and Pediatrics, University of California, Irvine
2011-2012 Associate Professor, Psychiatry and Human Behavior and Pediatrics, University of California, Irvine
2011-2016 Associate Professor Psychology Department, University of Denver
2012-present Associate Professor, Psychiatry and Human Behavior, University of California, Irvine
2016-present Professor Psychology Department, University of Denver

Honors

2007 Science Leadership Conference, Adventures in Advocacy: Training the Civic Scientist, hosted by the American Psychological Association
2009 Science Leadership Conference, Enhancing the Nation's Health Through Psychological Science, hosted by the American Psychological Association
2010 Council on Research, Computing, and Library Resources, Faculty Research Award, University of California, Irvine (Monetary)
2010-2014 National Institutes of Health Grant Reviewer for Special Emphasis Panel /Scientific Review Group 2010/08 ZHD1 DSR-K (LR).
2017 PROF award, University of Denver
2018-2024 Child Psychopathology and Developmental Disabilities NIH Study Section member, Center for Scientific Review
2018 Institute for Clinical and Translational Science Team Science Award

C. Contribution to Science.

1. Early Experiences and Development: It now is evident that in order to understand individual differences in health and development it is essential to consider the prenatal environment. The prospective and longitudinal studies that I have conducted provide compelling evidence that prenatal maternal stress and stress hormones alter the developing fetal nervous system with profound and lasting consequences for later child mental health and risk for psychopathology. In addition to assessment of maternal signals at specified intervals, we have characterized patterns or trajectories of maternal signals across time and confirmed that patterns and transitions during gestation or postnatal development reveal relations between predictors and outcomes that are independent, and sometimes far superior to single time points.

a. **Davis, E.P**., Stout, S.A., Molet, J., Vegetabile, B., Glynn, L.M., Sandman, C.A., Hein, K., Stern, H., Baram, T.Z. Early life exposure to unpredictable maternal sensory signals influences cognitive development: A cross-species approach. Proceedings of the National Academy of Sciences**.** 2017; 114:10390-10395. PMC5625898
b. Baram, T.Z., **Davis, E.P.**, Obenaus, A., Sandman, C.A., Small, S., Solodkin, A., Stern, H. Fragmentation and unpredictability of early-life experience in mental disorders. American Journal of Psychiatry. 2012; 169:907-15. PMC3483144
c. Sandman, C.A., Curran, M.M, **Davis, E.P.**, Glynn, L.M, & Baram, T.Z. Cortical thinning and neuropsychiatric outcomes in children exposed to prenatal adversity: a role for placental CRH? American Journal of Psychiatry. 2018; 175:471-479. PMC5930042
d. **Davis, E.P.** & Sandman, C.A. The timing of prenatal exposure to maternal cortisol and psychosocial stress is associated with human infant cognitive development. Child Development, 2010; 81:131-138. PMC2846100

2. Fetal exposure to Synthetic Glucocorticoids is associated with Neurodevelopment: Glucocorticoids are one of the most commonly administered treatments given to women in preterm labor and are administered to facilitate fetal lung maturation. This lifesaving treatment clearly benefits survival among infants born early preterm. I have conducted several NIH funded research studies illustrating that this prevalent treatment is associated with altered neurodevelopment among the over 30% of fetuses who receive this treatment and go on to deliver full term. Specifically, glucocorticoid treatment is correlated with suppressed fetal growth, HPA axis dysregulation, risk for cognitive impairments and altered brain development. Currently, I am completing a 5-year R01 that prospectively evaluates HPA and placental responses to GC treatment and links these profiles

to gestational length and infant development, thus providing new information to clinicians faced with the decision of whether or not to administer GCs to pregnant women. Further, this project will inform our understanding of the association between the maternal/placental changes associated with GC treatment and the link to infant neurodevelopment. Because in these correlational studies the effects of GCs cannot be dissociated from other factors (e.g., the experience of preterm labor), an important next step in this area is the experimental evaluation of the effects of prenatal GCs on the development of pathophysiological risk mechanisms for anxiety. The current project tests whether GCs exert a causal effect on neuro-hormonal-behavioral risk processes that underlie the development of anxiety and thus, builds on prior work to make an important advancement in our understanding of the mechanisms by which early exposures cause changes in later brain and behavioral development.

a. Edelmann, M.*, Sandman, C.A., Glynn, LM, Wing, D.A., & **Davis, E.P**. Prenatal glucocorticoid treatment is associated with diurnal cortisol regulation in term-born children. Psychoneuroendocrinology, 2016; 72:106-112. PMC5505268
b. Waffarn F. & **Davis E.P.** Effects of antenatal corticosteroids on the hypothalamic-pituitary-adrenocortical axis of the fetus and newborn: experimental findings and clinical considerations. American Journal of Obstetrics and Gynecology. 2012; 207:446-54. PMC3485443
c. **Davis, E.P.**, Sandman, C.A., Buss, C., Wing, D.A., & Head, K. Fetal glucocorticoid exposure is associated with preadolescent brain development. Biological Psychiatry. 2013; 74:647-55. PMC3985475
d. Grant, K.A.*, Sandman, C.A. Wing, D.A., Dmitrieva, J. & **Davis, E.P.** (2015). Prenatal programming of postnatal susceptibility to memory impairments: A developmental double jeopardy. Psychological Science. 2015; 26:1054-62. PMC4504787

3. Mechanisms by which Prenatal Experiences Influence Brain Development: My novel prospective longitudinal research program, evaluating maternal-child pairs from the fetal period through adolescence, has provided a new understanding of the neural mechanisms underlying fetal programing of cognitive and mental health outcomes. My research has characterized important changes in brain structure during the preadolescent period, related to both cortical thinning (structural MRI) and alterations in connectivity (DTI). Importantly, fetal experiences are associated with individual differences in neurodevelopment during pre-adolescence. For example, variations in maternal depressive symptoms during the prenatal period are associated with profound changes in child brain development that mediate the development of behavioral problems during childhood. Our recent correlational findings identify fetal exposure to glucocorticoids (both endogenous and synthetic glucocorticoid administration) as a plausible biological mechanism that may shape the trajectory of fetal neurodevelopment. My research program has illustrated that fetal exposure to excess glucocorticoids is associated the development of limbic regions that may underlie increased risk for anxiety problems. This correlational research lay the groundwork for the current investigation which will evaluate the effects of experimental manipulation of GCs on the longitudinal assessment of neural risk mechanisms, including novel measures of network connectivity, on the emergence of anxiety. The current project would give us a new level of understanding of the causal mechanisms by which early stress hormone exposure influences subsequent neurodevelopment.

a. **Davis, E.P**., Sandman, C.A., Buss, C., & Head, K. Prenatal maternal cortisol concentrations predict neurodevelopment in middle childhood. Psychoneuroendocrinology. 2017; 75:56-73. PMC5505265
b. Buss, C.*, **Davis, E.P.**, Shahbaba, B., Pruessner, J.C., Head, K. & Sandman, C.A. (2012). Is susceptibility for affective disorders programmed *in utero*? An investigation into the association between maternal cortisol over the course of pregnancy and subsequent child limbic structure volume and affective function. Proceedings of the National Academy of Sciences. NO PMCID
c. Sandman, C.A., Buss, C., Head, K & **Davis, E.P.** Fetal exposure to maternal depressive symptoms is associated with cortical thickness in late childhood. Biological Psychiatry. 2015; 77:324-34. PMC428946
d. Snyder, H.*, Hankin, B.L., Sandman, C.A., Head, K. & **Davis, E.P.** Distinct patterns of reduced prefrontal and limbic grey matter volume in childhood general and internalizing psychopathology. Clinical Psychological Science, 2017; 5:1001-1013. PMC5794221

4. Sex Differences in Fetal Programming: It is widely recognized that sex differences exist in the vulnerability to mental illness. My collaborative research has provided new insights into the role of fetal experiences in sex specific vulnerability to mental illness. The profile of development differs for male and female fetuses. Further, we have discovered sex-specific responses to adversity as early as the fetal period. For example, fetal and neonatal exposure to maternal cortisol differentially is associated with emotional and cognitive outcomes

among males and females. Results of these studies suggest that the female fetus may be more vulnerable to the consequences of gestational stress exposure for mood and anxiety problems. These studies have important implications for the understanding of sex differences in fetal programming and for the identification of factors that contribute to sex-specific vulnerability to mental disease. The present longitudinal experimental evaluation of the sex-specific consequences of GC treatment for the neurodevelopmental could provide new insight into the mechanisms contributing to sex-specific vulnerabilities to anxiety.

a. **Davis, E.P.** & Pfaff, D. Sexually dimorphic responses to early adversity: implications for affective problems and autism spectrum disorder. Psychoneuroendocrinology, 2014; 49:11-25. PMC4165713
b. Sandman, C.A., Glynn, L.M. & **Davis, E.P.** Is there a viability vulnerability trade off? Sex differences in fetal programming. Journal of Psychosomatic Research. 2013; 75:327-335. PMC3796732
c. Kim, D.J.*, **Davis, E.P**., Sandman, C.A., Sporns, O., O'Donnell, B.F., Hetrick, W.P. Prenatal maternal cortisol has sex specific associations with child brain network properties. Cortex, 2016; 27:5230-5241. PMID:27664961
d. Glynn, L.M., Howland, M.A.*, Sandman, C.A., **Davis, E.P.**, Phelan, M., Baram, T.Z., Stern, H. Prenatal maternal mood patterns predict child temperament and adolescent mental health. Journal of Affective Disorders. 2018; 228:83-90. PMID:29241049. PMC in process

5. Identifying a new Critical Period for Brain Development: Both scientific research and clinical practice has relied on the cut off of 37 gestational weeks to identify preterm infants who may be at risk for developmental impairments. My research has contributed to a shift in our understanding of the consequences of shortened gestation by illustrating that gestational length operates along a continuum and even modest variations in length of gestation among infants who are born full term (37 to 41 gestational weeks) shapes neurodevelopment. Longer gestation, among children born at term, is associated with enhanced mental development during infancy and cortical volume during childhood. Further, using cutting edge analytic tools to assess integrity of brain networks (DTI; "connectomics") we show that longer gestation, within the bounds of term delivery, is associated with improved topological organization of the preadolescent brain, characterized by the increased communication capacity of the brain network and enhanced directional strength of brain connectivity with central hub regions. Notably, these differences in network efficiency are associated with cognitive performance. These data have clear implications both for obstetrics and for clinical psychology. First, they challenge common practice of elective deliveries during the early term period. Second, they highlight the importance of evaluating and potentially intervening with infants born in the late preterm early term periods.

a. **Davis, E.P.**, Buss, C., Muftuler, T., Head, K., Hasso, A. Wing, D.A., Hobel, C. & Sandman, C.A. Children's brain development benefits from longer gestation. Frontiers in Psychology. 2011; 2:1. ecollection. PMC3111445
b. Espel, E.*, Glynn, L.M., Sandman, C.A. & **Davis, E.P.** Longer gestation among children born full term influences cognitive, and motor development. PLoS One. 2014; 9: e113758. PMC424187
c. Kim, D.J.*, **Davis, E.P.**, Sandman, C.A., Sporns, O., O'Donnell, B.F., Hetrick, W.P. Longer gestation is associated with more efficient brain network in preadolescent children. Neuroimage, 2014; 100:619-627. PMC4138264
d. Kim, D.J.*, **Davis, E.P.**, Sandman, C.A., Sporns, O., O'Donnell, B.F., Hetrick, W.P. Children's intellectual ability is associated with structural network integrity. Neuroimage, 2015; 124(Pt A):550-6. PMC4651770

Complete List of Published Work in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1-GW-eSxLpe5P/bibliography/47610820/public/?sort=date&direction=ascending

D. Research Support

Ongoing

Reducing Fetal Exposure to Maternal Depression to Improve Infant Risk Mechanisms (R01 MH 109662)
Project Agency: NIH/NIMH
Role: PI (Multiple-PI, Ben Hankin)
Funding Period: 2017-2022
The aim of this project is to evaluate the impact of a prenatal intervention to reduce maternal depression on neurodevelopmental processes in the offspring.

Prenatal Pathways for Poverty's Influence on the Brains of Two Generations (R01 HD090068)
Funding Agency: NIH/NICHD
Role: Co-Investigator
Funding Period: 2017-2022
The major goal of this project is to identify the prenatal pathways by which poverty perturbs neural outcomes of two generations - infants and their mothers.

NIMH Silvio O. Conte Center for the Study of "Fragmented Early Life Environmental and Emotional/Cognitive Vulnerabilities" (P50 MH 096889)
Funding Agency: NIH/ National Institute of Mental Health
Center PI: Tallie Z. Baram
Role: Project PI
Funding Period: 2013-2018
The aim of this center grant is to understand mechanisms by which early adversity leads to cognitive and emotional vulnerabilities during infancy, childhood and adolescence. This center will evaluate the whether fragmented maternal signals during the prentatal and postnatal periods are associated with developmental outcomes.

Preconception and Prenatal Stress: Pathways to Child Biology and Behavior (R01 HD72021)
Funding Agency: NIH/National Institute of Child Health and Human Development
PI: Chris Dunkel Schetter
Funding Period: 2013-2018
Role: Subcontract PI
The focus of this investigation is to determine the joint role of preconception and prenatal influences on child cognition, emotional regulation and stress physiology.

Mechanisms and Effects of Prenatal Maternal Affect on Pregnancy and Infant Development (R01 HD73491)
Funding Agency: NIH/National Institute of Child Health and Human Development
PI: Mary Coussons-Read
Funding Period: 2013-2018
Role: Subcontract PI
This project will evaluate mechanisms by which prenatal maternal anxiety impacts birth and developmental outcomes.

<u>Recently Completed</u>
Vulnerability to Prenatal Glucocorticoids Programs Infant Development (R01 HD065823)
Funding Agency: NIH/NICHD/NIMH
Project period 2011-2016
The aim of this investigation is to identify maternal fetal pairs who are vulnerable to the negative consequences of glucocorticoid treatment on birth outcome and infant and child development. This project involves the prospective assessment of pregnant women and their infants who received glucocorticoids prenatally. Maternal physiology is assessed before and after treatment. Infant development is evaluated during the first postnatal year with the Laboratory Temperament Assessment Battery (Lab-Tab) and the Mental Development Index (MDI) from the Bayley Scales for Infant and Toddler Development (BSID).
Role: Principal Investigator

Poverty, Chronic Stress and Neural Regulation of Maternal Mood and Parenting (R21 HD078797)
Project Agency: NIH/NICHD
PI: Pilyoung Kim
Role: Co-Investigator
Project period: 2014-2016
This project evaluates the consequences of chronic stress and poverty during the pre and postnatal periods on maternal brain and behavior.