BIOGRAPHICAL SKETCH

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NAME: Glynn, Laura M.

eRA COMMONS USER NAME (credential, e.g., agency login): lauraglynn

POSITION TITLE: Professor of Psychology

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
University of California, Davis, CA	B.A.	1993	Economics/Psychology
University of California, San Diego, CA	M.A.	1994	Experimental Psychology
University of California, San Diego, CA	Ph.D.	1998	Experimental Psychology
University of California, Irvine, CA	Post-Doctoral	2002	Psychiatry

A. Personal Statement

I am Professor of Psychology and Faculty Fellow in the Office of Research at Chapman University in Orange, California. I also maintain a second appointment in the Department of Psychiatry at the University of California, Irvine. My research focuses on the interface of psychology and biology within the context of reproduction. Specifically, my interests include: the role of psychological and biological stress in adverse birth phenotype (preterm birth and restricted fetal growth), the way in which reproductive history shapes women's health and development, and the role of prenatal and early life experience in fetal, child and adolescent development. I have relevant knowledge and expertise to offer as a Project Leader for this Project 2 in the following ways: First, because of my involvement as a PI or Co-I in nine NIH-funded longitudinal studies of mothers and their offspring, I have extensive experience in designing and conducting longitudinal studies of mothers and children. Second, as Co-Leader of Project 2, in the current funding period, I oversaw the recruitment and assessment of a new prenatal longitudinal cohort of 250 women followed for 18 months in my lab at Chapman University. These are the mother-child pairs whom we propose to follow into early childhood in this Center continuation.

B. Positions and Employment

1998-2002	Post-Doctoral Researcher, Department of Psychiatry & Human Behavior, University of California, Irvine, CA
2002-2008	Assistant Professor In-Residence, Department of Psychiatry & Human Behavior, University of California, Irvine, CA
2008-2014	Associate Professor of Psychology, Crean College of Health and Behavioral Sciences, Chapman, University, Orange, CA
2008- present	Research Scientist, Department of Psychiatry & Human Behavior, University of California, Irvine, CA
2011-2017	Chair of Psychology, Crean College of Health and Behavioral Sciences, Chapman University, Orange, CA

2014- present Professor of Psychology, Crean College of Health and Behavioral Sciences, Chapman University, Orange, CA

2017- present Faculty Fellow, Office of Research, Chapman University, Orange, CA

Awards and Honors

2014 Distinguished Research Award, Chapman University

2016- present Director, Center of Excellence in Biopsychosocial Approaches to Health, Chapman University, Orange, CA

2016 Wang-Fradkin Senior Professorship, Chapman University

2017- present Faculty Fellow, Office of Research, Chapman University, Orange, CA

2017 Elected Member of the Academy of Behavioral Medicine Research

C. Contributions to Science (Student or post-doc first authors are indicated by underscore)

1. Developmental Origins of Health and Disease: New Insights from Sex Differences and Patterns of Inputs

The introduction of the Developmental Origins of Health and Disease model (DoHaD) spurred a paradigm shift in medical, psychological and developmental perspectives on lifespan health and disease. Early in this movement, my colleagues and I began developing and following large cohorts of mothers and their offspring beginning during the prenatal period. Working with these longitudinal cohorts, I have been involved in the publication of empirical papers examining the influences of biological and psychological predictors on a range of outcomes in fetuses, infants, children and adolescents including emotion regulation, cognitive development, HPA-axis functioning, brain development, and risk for obesity. Our work in this domain has included two notable recent refinements or extensions: 1. In a reanalysis and review of the moderating effects of sex on our fetal programming findings (cited below, 2013), we make the novel proposal that although for males, exposures to early adversity threaten their viability, females use a variety of strategies to adjust to these exposures by altering their developmental trajectories. However, this flexibility among females comes with the price of increased vulnerability expressed later in development. 2. A primary aim of the UCI Conte Center is to test the overarching hypothesis that fragmented and unpredictable maternal and environmental signals shape the developing brain. The essential scientific premise of this work is that cognitive and emotional brain functions involve coordinated activities of brain circuits that integrate cellular, synaptic and network signaling. Unpredictable patterns of signals to the developing organism may drive aberrant circuit maturation that can promote cognitive and emotional dysfunction. In recent publications, we provide support for this hypothesis with correlational data in humans substantiated by data from translational non-human animal models, which allow experimental manipulation and targeted examination of neurological mechanisms.

1a. Sandman CA, **Glynn LM** & Davis EP (2013). Is there a viability-vulnerability tradeoff? Sex differences in fetal programming. *J Psychosom Res, 75,* 327-335. PMC3796732

1b. Davis EP, Stout SA, Molet J, Vegetabile B, **Glynn LM**, Sandman CA, Hein K, Stern S and Baram TZ (2017). Early life exposure to unpredictable maternal sensory signals influences cognitive development: A cross-species approach. *Proc Natl Acad Sci USA, 39,* 10390-10395. PMC5625898

1c. **Glynn LM**, Howland MA, Sandman CA, Davis EP, Phelan M, Baram TZ & Stern HS (2018). Prenatal maternal mood patterns predict child temperament and adolescent mental health. *J Affect Disord*, *228*, 83-90. PMID: 29241049. PMC in process

1d. Sandman CA, Curran MM, Davis EP, **Glynn LM**, Head K & Baram TZ (2018). Cortical thinning and neuropsychiatric outcomes in children exposed to prenatal adversity: a role for placental CRH? *Am J Psychiatry*. PMC5930042

2. Maternal Programming: Conceptualizing Pregnancy as a Sensitive Period of Development in the Human Female Lifespan

Existing research indicates that both puberty and menopause are associated with changes in women's brain structure and function. However, the magnitude of physiological alterations that accompany pregnancy and parturition are far more extreme and prolonged than either of these other two events in the lifespan of the human female. Further, compelling animal models demonstrate that the changes in brain and behavior associated with reproduction persist across the lifespan of the rodent. Despite this, relatively is known about the influences of reproduction on the brain and behavior of the human female. One of the primary goals of my research program has been to understand these influences and to provide support for the perspective that the perinatal period represents a sensitive period of neurological development for the human female. My work has advanced this goal in two domains. First, I documented that as human gestation progresses psychological responses to stress are increasingly attenuated and that these normative changes may serve a protective function for both mother and fetus. Second, in largest prospective longitudinal study conducted to date, I examined changes in cognitive function among women across pregnancy and into the early postpartum period. Results from this cohort revealed several important findings including support for women's anecdotal reports that their cognitive function is compromised (which also was consistent with previous small cross-sectional studies), that the effects of pregnancy on memory function are cumulative and that the diminished memory function observed during pregnancy persists into the postpartum period. I also have demonstrated clear associations between memory function and prenatal endocrine profiles (estradiol and cortisol), elucidating plausible underlying mechanisms. Most recently, I have shown that prenatal endocrine profiles (HPA, placental and reproductive hormones), which play a critical role in the onset and maintenance of maternal behaviors, and also likely play a role in postpartum mental health (see below), are remarkably stable within women across successive pregnancies (r's range from .41 to .75).

2a. **Glynn LM,** Wadhwa PD, Dunkel-Schetter C, Chicz-DeMet A & Sandman CA (2001). When stress happens matters: Effects of earthquake timing on stress responsivity in pregnancy. *Am J Obstet Gynecol*, *184*, 637-642. PMID:11262465

2b. **Glynn LM** (2010). Giving birth to a new brain: hormone exposures of pregnancy influence human memory. *Psychoneuroendocrinology, 35,* 1148-1155. PMID:20304563

2c. Fox M, Sandman CA, Davis EP & **Glynn LM** (2015). Intra-individual consistency in endocrine profiles across successive pregnancies. *J Clin Endocrinol Metab*, 100, 4637-4647. PMC4667165

2d. **Glynn LM**, Davis EP, Sandman CA & Goldberg WA (2016).Gestational hormone profiles predict human maternal behavior at 1-year postpartum. *Horm Behav,* 85, 19-25. PMC5929113

3. Postpartum Depression: Risk and Resilience

Among women in the US who give birth each year, between 10 and 15% will experience postpartum depression, and less than half will be identified and treated. The devastating consequences of this condition for both mother and child are pervasive and persisting. My work on this issue has advanced understanding of the role of endocrine risk factors in postpartum depression, specifically placental and hypothalamic–pituitary–adrenal axis hormones. A second contribution of this work has been to identify resources that may confer resilience or protection from postpartum depression including prenatal social support and breastfeeding.

3a. Hahn-Holbrook J, Dunkel Schetter C, Haselton MG & **Glynn LM** (2013). Does breastfeeding offer protection against maternal depressive symptomatology? A prospective study from pregnancy to 2 years after birth. *Arch Women's Ment Health, 16,* 411-422. PMC3818091

3b. **Glynn LM** & Sandman CA (2014). Evaluation of the association between placental corticotrophin-releasing hormone and postpartum depressive symptoms. *Psychosomatic Med, 76,* 355-62. PMID: 24915294

3c. Fox M, Sandman CA, Davis EP & **Glynn LM** (2018). A longitudinal study of women's depressive symptom profiles during and after the postpartum phase. *Depress Anxiety*. PMC5889323

3d. **Glynn LM**, Howland MA & Fox M (in press). Maternal programming: application of a developmental psychopathology perspective. *Development & Psychopathology*.

4. Influences of Human Milk on Child Development

It has long been recognized that breastfeeding is associated with enhanced health and development across a wide range of domains. However, relatively little is known about how human milk exerts these salutary influences. Human milk contains an array of biologically active hormones, including glucocorticoids (GCs), hormones that have been central in my work examining perinatal developmental effects on both mother and offspring. These observations lead me to publish the first papers in humans to show associations between milk cortisol and infant development. In two separate cohorts, I documented that infants exposed to higher levels of milk cortisol were more likely to exhibit fearful and reactive temperament, risk factors for later development of internalizing disorders. New findings from my lab, also document the role that human milk cortisol may play in regulating body mass index trajectories over the first two years of life, providing a provocative potential biological explanation for the protective effects of breastfeeding on obesity. These studies are among the very first in humans to show that maternal biological influences on offspring phenotype do not terminate at birth, but rather that developmental trajectories continue to be shaped through biological messages contained in milk.

4a. **Glynn LM**, Davis EP, Dunkel Schetter C, Chicz-DeMet A, Hobel CJ & Sandman CA (2007). Postnatal maternal cortisol levels predict temperament in healthy breastfed infants. *Early Hum Dev, 83,* 675-681. PMID: 17336002

4b. Grey KR, Davis EP, Sandman CA & **Glynn LM** (2013). Human milk cortisol is associated with infant temperament. *Psychoneuroendocrinology, 38,* 1178-1185. PMC4777694

4c. Hahn-Holbrook J, Le TB, Chung A, Davis EP & **Glynn LM** (2016). Cortisol in human milk predicts child BMI. *Obesity, 24,* 2471-2474. PMC5400496

5. Understanding Adverse Birth Outcomes: Advancing Biopsychosocial Perspectives

For almost two decades, I have worked to advance the understanding of premature birth, the leading cause of death in non-anomalous infants in the US. Rates of preterm birth have not declined significantly over the past few decades, and in more than 50% of the cases, the underlying cause of the early delivery is not known. Historically, the field has been limited by a lack of prospective longitudinal studies and a failure to bring more complex interdisciplinary approaches to this problem. My interdisciplinary work in this area addresses these issues by examining the interplay between psychology, biology, health behaviors, social relationships and cultural contexts to further understand the etiology of preterm birth.

5a. **Glynn LM**, Dunkel Schetter C, Chicz-DeMet A, Hobel CJ & Sandman CA (2007). Ethnic differences in adrenocorticotropic hormone, cortisol and corticotropin-releasing hormone during pregnancy. *Peptides*, *28*, 1155-1161. PMID: 17537545

5b. Dunkel Schetter C & **Glynn LM** (2011). Stress in pregnancy: empirical evidence and theoretical issues to guide interdisciplinary research. In R. Contrada & A. Baum, Eds., The Handbook of Stress Science: Biology, Psychology and Health.

5c. Okun ML, Dunkel Schetter C & **Glynn LM** (2011). Poor sleep quality is associated with preterm birth. *Sleep 34,* 1493-1498. PMC3198204

5d. Hilmert CJ, Dominguez TP, Dunkel Schetter C, Srinivas SK, **Glynn LM**, Hobel CJ & Sandman CA (2014). Lifetime racism and blood pressure changes during pregnancy: implications for fetal growth. *Health Psychology, 33,* 43-51. PMID: 23379383

Complete List of Published Work in My Bibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1bMTrrIeXzHAI/bibliograpahy/47896522/public/?sort=date&direction=ascending

D. Research Support

Ongoing Support

P50 MH096889 (Baram) 06/13-05/19
NIH/NIMH
Silvio O. Conte Center on Brain Programming in Adolescent Vulnerabilities
The purpose of this center grant is to determine the pathways through which maternal signals before and after birth increase vulnerability to cognitive and emotional problems.
Role: Project Leader

K01 DK105110 (Fox) 06/15-05/20
NIH/NIDDK
Effects of Acculturation on Gestational Biology in Mexican-American Pregnant Women
The purpose of this training grant is to explore how gestational biology may mediate between migration and intergenerational cascades of poor health.
Role: Co-Mentor

K99 MD012615 (Nguyen) 04/18-03/23
NIH/NIMHD
Place-Level Discrimination and Birth Outcomes
The purpose of this project is to develop a new indicator of discriminatory attitudes from social media data and to use hate crimes data and measures of explicit and implicit racial bias to examine the impact of discrimination on birth outcomes and racial/ethnic disparities in birth outcomes.
Role: Consultant

Completed Research Support (from last 3 years)

R01 HD065823 (Davis) 08/10-07/16
NIH/NICHD
Vulnerability to Prenatal Glucocorticoids Programs Infant Development
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 development.
Role: Co-Investigator