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Perinatal Programming of Neurodevelopment
Preface: Biological Embedding: Long-Term Effects of Early-Life Experiences and Approaches to Prevention Remediation

Introduction

Until recent years, what happens to an individual early in life was largely ignored because it was falsely believed that the brain and body were shaped by experiences when the child becomes able to respond rationally to the social environment. We now know in increasing detail, in both animal models and studies on our own species, that prenatal stress can have adverse effects that are manifested in prematurity or low birth weight at term, as well as in behavioral characteristics that are manifested throughout the life course. Furthermore, we now know that postnatal parental care and abuse and neglect in humans, and nest disruption and separation of infants from their mothers in animal models, play a powerful role in later mental and physical health. This volume addresses many aspects of adverse pre- and early postnatal influences on subsequent physical and mental health and this introductory overview will discuss the role of both animal and human studies and the translation and cross-talk between them in achieving a better understanding of underlying processes and mechanisms so that interventions can be developed to present or, when necessary, treat disorders that may arise. In all of this, the brain is the central organ of stress and adaptation and the “lived experiences” of an individual are an important contributor to physical and mental health outcomes and the brain represents an important target for prevention and amelioration of early-life adversity (McEwen and Getz 2013).

Towards an Understanding of Mechanisms and Consequences of Biological Embedding

Animal models have contributed enormously to our understanding of how, through the brain, the development of body and the brain are affected. This began with the “neonatal handling” studies of Levine and Denenberg (Levine et al. 1967) and led to the recent, elegant work of Meaney, Syzf and colleagues (Meaney and Szyf 2005). Epigenetic, transgenerational effects transmitted by maternal care are central to
these findings (Hackman et al. 2010). Besides the amount of maternal care, the consistency over time of that care and the exposure to novelty are also very important, not only in rodents (Akers et al. 2008; Tang et al. 2006), but also in monkey models (Parker et al. 2006). Prenatal stress impairs hippocampal development in rats, as does stress in adolescence (Isgor et al. 2004). Abusive maternal care in rodents and the surprising attachment shown by infant rats to their abusive mothers appears to involve an immature amygdala (Moriceau and Sullivan 2006), activation of which by glucocorticoids causes an aversive conditioning response to emerge. Maternal anxiety in the variable foraging demand (VFD) model in rhesus monkeys leads to chronic anxiety in the offspring, as well as signs of metabolic syndrome (Coplan et al. 2001; Kaufman et al. 2005). Nest disruption of mothers nursing mouse pups impairs development of the hippocampus and other brain systems (Rice et al. 2008).

In studies of adverse childhood experiences (ACE) in human populations, there are reports of increased inflammatory tone, not only in children, but also in young adults related to early-life abuse, that includes chronic harsh language, as well as physical and sexual abuse (Danese et al. 2009; Miller and Chen 2010). Chaos in the home is associated with development of poor self-regulatory behaviors, as well as obesity (Evans et al. 2005). It should be noted that the ACE study was carried out in a middle class population (Anda et al. 2010), indicating that poverty is not the only source of early-life stressors.

Nevertheless, low socioeconomic status (SES) does increase the likelihood of stressors in the home and neighborhood, including racial isolation, chaos, noise and ugliness as, well as toxic chemical agents, such as lead and air pollution (Chang et al. 2009; McEwen and Tucker 2011; Theall et al. 2013). Without a determination of exact causes, it has been reported that low SES children are found to be more likely to be deficient in language skills, as well as self-regulatory behaviors and also in certain types of memory that are likely to be reflections of impaired development of parasympathetic gyrus language centers, prefrontal cortical systems and temporal lobe memory systems (Farah et al. 2006; Hart 1995). Low SES is reported to correlate with smaller hippocampal volumes (Hanson et al. 2011). Lower subjective SES, an important index of objective SES, is associated with reduction in prefrontal cortical gray matter (Gianaros et al. 2007) and with increased inflammatory tone in serum along with altered white matter in the brain that is also associated with increased adiposity (Gianaros et al. 2012; Verstynen et al. 2013).

Moreover, having grown up in a lower SES environment is accompanied by greater amygdala reactivity to angry and sad faces (Gianaros et al. 2008b), which may be a predisposing factor for early cardiovascular disease (Gianaros et al. 2008a) that is known to be more prevalent at lower SES levels (Adler et al. 1993). Finally, depression is often associated with low SES, and children of depressed mothers, followed longitudinally, have shown increased amygdala volume while hippocampal volume was not affected (Lupien et al. 2011).

Yet, on the positive side, there are the “reactive alleles” also referred to as “biological sensitivity to context” that lead to beneficial outcomes and even better outcomes in nurturing environments compared to less reactive alleles, even though those same alleles can enhance adverse outcomes in a stressful early-life environ-
Regarding adverse outcomes and good and bad “environments,” the active process of adaptation to stressors (“allostasis” (McEwen and Stellar 1993; Sterling and Eyer 1988)) is adjusted via epigenetic influences to optimize the individuals adaptation to, and resulting fitness for, a particular environment, whether more or less threatening or nurturing as described in the Adaptive Calibration model (Del Giudice et al. 2011).

It is important to note that the conceptual models of allostasis and allostatic load are orthogonal to the model of Adaptive Calibration (Del Giudice et al. 2011) and provide complementary ways of understanding individual developmental trajectories and their adaptive value as well as their consequences. One lesson from these two models is that there are “trade-offs” in terms of physical and mental health that, on the one hand, may increase the likelihood of passing on one’s genes by improving coping with adversity and enhancing mental health and overall reproductive success, but, on the other hand, may impair later health, e.g., by eating of “comfort foods” (see for example (Jackson et al. 2010)).

Understanding and attempting to modify such individual health outcomes is an important component of “personalized medicine” and must be considered along with pharmacogenomics in the development of therapies (Davidson and McEwen 2012; McEwen and Getz 2013). In this connection, it should be noted that resilience means not only the ability to resist stress-induced change, but also the ability to show experience-related recovery and adaptation or compensation, for example, when an individual from a safe environment is placed into a dangerous one or vice versa. It is the plasticity of the brain and body that are keys to the amelioration of early-life adversity.

**Interventions to Ameliorate Early-Life Adversity**

What can be done to remediate the effects of chronic stress, as well the biological embedding associated with early-life adversity? Interventions may involve pharmaceutical, as well as behavioral, or “top-down,” interventions (i.e., interventions that involve integrated CNS activity, as opposed to pharmacological agents) that include cognitive-behavioral therapy, physical activity and programs that promote social support and integration and meaning and purpose in life (Carlson et al. 2009; Fried et al. 2004; Ganzel et al. 2010; McEwen and Gianaros 2011). More targeted interventions for emotional and cognitive dysfunction may arise from fundamental studies of such developmental processes as the reversal of amblyopia and other conditions by “releasing the brakes” that retard structural and functional plasticity (Bavelier et al. 2010). It should be noted that many of these interventions that are intended to promote plasticity and slow decline with age, such as physical activity and positive social interactions that give meaning and purpose, are also useful for promoting “positive health” and “eudaimonia” (Ryff and Singer 1998; Singer et al. 2005) independently of any notable disorder and within the range of normal
behavior and physiology. It should also be noted that, while complete reversal of early-life adversity may not be possible, compensatory changes in neural architecture and molecular and neurochemical processes in key brain regions such as amygdala and prefrontal cortex can be envisioned (Caldji et al. 1998). Thus it is important to explore the strategies, possibilities and limits of adult brain plasticity, as will be discussed below.

As noted above, “top down” therapy is one strategy and one example is regular physical activity, which has actions that improve prefrontal and parietal cortex blood flow and enhance executive function (Colcombe et al. 2004). Moreover, regular physical activity, consisting of walking an hour a day, 5 out of 7 days a week, increases hippocampal volume in previously sedentary adults (Erickson et al. 2011). This finding complements work showing that physically fit individuals have larger hippocampal volumes than sedentary adults of the same age-range (Erickson et al. 2009). It is also well known that regular physical activity is an effective antidepressant and protects against cardiovascular disease, diabetes and dementia (Babyak et al. 2000, Snyder et al. 2010). Moreover, intensive learning has also been shown to increase volume of the human hippocampus (Draganski et al. 2006).

Other “top down” activities include social integration and support, and finding meaning and purpose in life, and these are known to be protective against allostatic load (Seeman et al. 2002) and dementia (Boyle et al. 2010). Programs such as the Experience Corps that promote these, along with increased physical activity, have been shown to slow the decline of physical and mental health and to improve prefrontal cortical blood flow in a similar manner to regular physical activity (Carlson et al. 2009; Fried et al. 2004).

Depression and anxiety disorders are examples of a loss of resilience, in the sense that changes in brain circuitry and function, caused by the stressors that precipitate the disorder, become “locked” in to a particular state and thus need external intervention. Indeed, prolonged depression is associated with shrinkage of the hippocampus (Sheline 1996; Sheline 2003) and prefrontal cortex (Drevets et al. 1997). While there appears to be no neuronal loss, there is evidence for glial cell loss and smaller neuronal cell nuclei (Rajkowska 2000; Stockmeier et al. 2004), which is consistent with a shrinking of the dendritic tree described above after chronic stress. Indeed, a few studies indicate that pharmacological treatment may reverse the decreased hippocampal volume in unipolar (Vythilingam et al. 2004) and bipolar (Moore et al. 2000) depression, but the possible influence of concurrent cognitive-behavioral therapy in these studies is unclear.

Depression is more prevalent in individuals who have had adverse early-life experiences (Anda et al. 2010). BDNF may be a key feature of the depressive state and elevation of BDNF by diverse treatments ranging from antidepressant drugs to regular physical activity and may be a key feature of treatment (Duman and Monteggia 2006). Yet, there are other potential applications, such as the recently reported ability of fluoxetine to enhance recovery from stroke (Chollet et al. 2011). However, a key aspect of this new view (Castren and Rantamaki 2010) is that the drug is opening a “window of opportunity” that may be capitalized by a positive
behavioral intervention, e.g., behavioral therapy in the case of depression or the intensive physiotherapy to promote neuroplasticity to counteract the effects of a stroke.

This is consistent with animal model work that shows that ocular dominance imbalance from early monocular deprivation can be reversed by patterned light exposure in adulthood that can be facilitated by fluoxetine, on the one hand (Vetencourt et al. 2008) and food restriction, on the other hand (Sanacora et al. 2012), in which reducing inhibitory neuronal activity appears to play a key role (Dhabhar et al. 2012). Investigations of underlying mechanisms for the re-establishment of a new window of plasticity are focusing on the balance between excitatory and inhibitory transmission and removing molecules that put the “brakes” on such plasticity (Tanaka et al. 2001).

In this connection it is important to reiterate that successful behavioral therapy, which is tailored to individual needs, can produce volumetric changes in both prefrontal cortex in the case of chronic fatigue (de Lange et al. 2008), and in amygdala, in the case of chronic anxiety (Holzel et al. 2010). This reinforces two important messages: (i) that plasticity-facilitating treatments should be given within the framework of a positive behavioral or physical therapy intervention; and (ii) that negative experiences during the window may even make matters worse (Castren and Rantamaki 2010). In that connection, it should be noted that BDNF also has the ability to promote pathophysiology, as in seizures (Heinrich et al. 2011; Kokaia et al. 1995; Scharfman 1997).

Conclusions

Pre- and postnatal experiences have a profound and lasting effect upon physical and mental health acting via the brain and the biological embedding of positive and negative experiences. The chapters in this volume document many aspects of this in both animal models and humans, and this introductory chapter has outlined treatment strategies and their potential efficacy and limitations for ameliorating the effects of early-life adversity. However, the best solution is to prevent the adversity from happening in the first place. The Nurse-Family Partnership (http://www.nursefamilypartnership.org/) is a primary example of a program designed to educate expectant parents on optimal ways of interacting with their infants and children to promote healthy development, and the National Scientific Council on the Developing Child (http://developingchild.harvard.edu/index.php/activities/council/) provides a rich website on this topic and is working actively to bring such preventative programs into practice.
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PART I Perinatal Programming: Studies in Laboratory Animals

1 Changes Induced by Prenatal Stress in Behavior and Brain Morphology: Can They Be Prevented or Reversed?............................ 3
Marta Weinstock

2 Sleep in Prenatally Restraint Stressed Rats, a Model of Mixed Anxiety-Depressive Disorder................................. 27
Jérôme Mairesse, Gilles Van Camp, Eleonora Gatta, Jordan Marrocco, Marie-Line Reynaert, Michol Consolazione, Sara Morley-Fletcher, Ferdinando Nicoletti and Stefania Maccari

3 Hormonal Modulation of Catecholaminergic Neurotransmission in a Prenatal Stress Model................................. 45
Maria Eugenia Pallarès and Marta C. Antonelli

4 Involvement of Nitric Oxide, Neurotrophins and HPA Axis in Neurobehavioural Alterations Induced by Prenatal Stress.............. 61
Damian G. Maur, Cecilia G. Pascuan, Ana M. Genaro and Maria A. Zorrilla-Zubilete

5 Prenatal Stress and Adult Drug-Seeking Behavior: Interactions with Genes and Relation to Nondrug-Related Behavior .................. 75
Tod E. Kippin, Jaonnalee C. Campbell, Kyle Ploense, Chris P. Knight and Jared Bagley

6 A Self-Medication Hypothesis for Increased Vulnerability to Drug Abuse in Prenatally Restraint Stressed Rats .................. 101
Marie-Line Reynaert, Jordan Marrocco, Eleonora Gatta, Jérôme Mairesse, Gilles Van Camp, Francesca Fagioli, Stefania Maccari, Ferdinando Nicoletti and Sara Morley-Fletcher
7 How Postnatal Insults May Program Development: Studies in Animal Models ................................................................. 121
Carla Dalmaz, Cristie Noschang, Rachel Krolow, Charlis Rainekei and Aldo. B. Lucion

8 Perinatal Positive and Negative Influences on the Early Neurobehavioral Reflex and Motor Development .................. 149
Gabor Horvath, Dora Reglődi, Jozsef Farkas, Gyongyver Vadasz, Barbara Mammel, Timea Kvarik, Greta Bodzai, Blanka Kiss-Illés, Dorottyja Farkas, Attila Matkovits, Sridharan Manavalan, Balazs Gaszner, Andrea Tamas and Peter Kiss

9 Short- and Long-Term Consequences of Perinatal Asphyxia: Looking for Neuroprotective Strategies .......................... 169

10 Affective, Cognitive, and Motivational Processes of Maternal Care ................................................................. 199
Mariana Pereira and Annabel Ferreira

11 Role of Sensory, Social, and Hormonal Signals from the Mother on the Development of Offspring ............................. 219
Angel I. Melo

Part II Perinatal Programming: Studies in Humans

12 Retrospective Studies ........................................................................................................................................ 251
Patrícia Pelufo Silveira and Gisele Gus Manfro

13 Prenatal Stress and Its Effects on the Fetus and the Child: Possible Underlying Biological Mechanisms ...................... 269
Vivette Glover

14 Using Natural Disasters to Study Prenatal Maternal Stress in Humans ........................................................................ 285
Suzanne King and David P. Laplante

15 Early Life Influences on Cognition, Behavior, and Emotion in Humans: From Birth to Age 20 ........................................ 315
Bea R. H. Van den Bergh, Eva M. Loomans and Maarten Mennes
# Contents

**Part III  Epigenetic And Translational Studies**

16  Perinatal Programming of Neurodevelopment: Epigenetic Mechanisms and the Prenatal Shaping of the Brain ........................................... 335  
Paula A. Desplats

17  Epigenetic Mechanisms of Perinatal Programming: Translational Approaches from Rodent to Human and Back .................. 363  
Patrick O. McGowan

18  Perinatal Administration of Aromatase Inhibitors in Rodents as Animal Models of Human Male Homosexuality: Similarities and Differences ...................................................................................... 381  
Sandra Olvera-Hernández and Alonso Fernández-Guasti

**Part IV  Prevention Programs**

19  Impact of the Perinatal Environment on the Child’s Development: Implications for Prevention Policies .................. 409  
Françoise Molenat

20  Perinatal Programming Prevention Measures ........................................... 425  
A. Miguel Larguía, María Aurelia González, Néstor Alejandro Dinerstein and Constanza Soto Conti

21  Pregnancy Outcomes After a Maternity Intervention for Stressful Emotions (PROMISES): A Randomised Controlled Trial .......................... 443  
Huibert Burger, Claudi L. H. Bockting, Chantal Beijers, Tjitte Verbeek, A. Dennis Stant, Johan Ormel, Ronald P. Stolk, Peter de Jonge, Mariëlle G. van Pampus and Judith Meijer

ERRATUM .................................................................................................................. E1

Index ................................................................................................................................. 461
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