ADOPTION EDUCATION LLC

EFFECTS OF STRESS IN EARLY LIFE

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TO ACCESS THE QUIZ:
After reading this course, please sign back on to www.adopteducation.com. Go to the table of contents and click on the last page (#9). Click the NEXT arrow at the bottom of the page to begin question 1 of the quiz.

A note from the AE staff: *This course contains some of the most technical and medical terminology of all our courses. The lay person will, nonetheless, be able to glean general information about the effects of stress from reading this material through to the end of the course. The reader is advised to discuss confusing or incomprehensible material with their pediatrician, social worker or other adoption or medical professional.*

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EFFECTS OF STRESS IN EARLY LIFE

INTRODUCTION

International adoptees encounter stress both prenatally and postnatally (see table below). Most are products of unwanted pregnancies. After birth, many suffer from abandonment, institutionalization, malnutrition, recurrent illnesses, environmental deprivation, and/or emotional neglect. Some are physically or sexually abused, or witness distressing events. These stressful experiences in early life may permanently alter brain physiology and lead to long-lasting physiological, psychological, and behavioral changes. Early stress may have life-long adverse effects on growth, development, health, behavior, learning, and memory. This section discusses how stress influences these areas, clinically and neurobiologically.

Table – Sources of stress for institutionalized children

Prenatal exposure to maternal depression
Abandonment
Environmental deprivation
Malnutrition
Recurrent illnesses
Removal from familiar peer groups and caregivers
Physical neglect
Emotional neglect
Physical abuse
Sexual abuse

PRENATAL AND EARLY POSTNATAL STRESS

Prenatal stress affects behavior and development of the offspring. Monkeys born to mothers stressed during pregnancy (e.g. exposed to unpredictable noises in a dark room) show decreased motor and exploratory behaviors, reduced cognitive ability, and delayed mastery of object permanence. Similarly, children whose mothers were stressed during pregnancy have less optimal outcomes, including problems with emotional regulation, motor delays, and intrauterine growth retardation.

Prenatal exposure to excess glucocorticoids (*a hormone that predominantly affects the metabolism of carbohydrates and, to a lesser extent, fats and proteins*) may harm the developing brain. Offspring of prenatally stressed rat or monkey mothers have increased basal levels of plasma ACTH (*adrenocorticotropic hormone, stimulates the adrenal cortex*), increased ACTH in response to stressful situations, and decreased ability to adapt to ongoing stress. Human infants born to mothers with prenatal depression tend to be less active, less socially responsive, and fussier than unexposed peers, possibly via the same pathway.

The possibility of “multigenerational effects” of stress has been suggested by animal studies. Infants prenatally exposed to stress have small heads; infants with small heads have abnormal stress responses. Non-optimal birth conditions (microcephaly, +/- low Apgar scores) are associated with abnormal stress responses for at least the first six months of life.

Maternal behaviors after birth also contribute to stress. Young animals and human infants have increased plasma cortisol levels and altered CRF levels (*corticotropin-releasing factor is a hormone released by the hypothalamus when faced with a fight-or-flight response which causes a chain of bodily reactions that allow an individual to concentrate on the situation*) and receptor number after maternal separation; these biochemical changes may persist until adulthood. Interestingly, the presence of a familiar social group and stable environment (as may occur in institutional care) reduces the endocrine effects of maternal separation. The presence of younger animals ("peer therapists") also helped monkeys maintain normal psychosocial behaviors after maternal loss. The animals exhibit signs of despair, however, if removed from their peer group.

The infant experiences maternal depression as separation. Accordingly, infants of depressed mothers tend to be withdrawn, less active, and show altered sleep patterns, difficulty sustaining attention, poor mastery motivation, and limited responsivity to facial expressions. In addition, these infants have elevated norepinephrine (*hormone secreted by the adrenal gland as a reaction to the "fight-or-flight response" that raises blood pressure and acts to stimulate muscle contraction*) and cortisol (*the body's natural stress-fighting and anti-inflammatory hormone*) levels, EEG changes compatible with depression, and reduced vagal tone (*the heart rate is relatively steady with low variability in the respiratory cycle*). Cognitive and language delays at 1, 4, and 12 years of age are more common in children born to depressed mothers than control children. These children have more emotional or behavioral disorders, including problems in self-regulation, behavior, and academic achievement. Massage therapy improves weight gain, emotionality, sociability, soothability, and mental and motor scores and decreases stress hormones in infants of depressed mothers.

GROWTH AND STRESS

Growth and stress are interrelated. Poor nutrition is itself stressful. Children with acute marasmus or kwashiorkor (*severe form of malnutrition caused by inadequate intake of protein and calories, and it usually occurs in the first year of life, resulting in wasting and growth retardation*) have elevated free cortisol levels. Stunted children have higher cortisol levels, higher heart rates, fewer vocalizations, more inhibitions, and less attentive behavior than their well-grown peers in response to physical and psychological stressors. Their cortisol levels correlate with nutritional status. Thus, prolonged cortisol exposure may mediate the heightened physiologic activation and poor cognitive performance common among poorly nourished children.

Independent of malnutrition, stress itself directly affects growth. Prolonged exposure to stress inhibits secretion of growth hormone and other growth factors; children with anxiety disorders tend to be shorter adults because of reduced growth hormone secretion. The poor growth exhibited by many institutionalized children likely reflects both inadequate nutrition and other stressful environmental factors.

STRESS AND THE IMMUNE SYSTEM

Stress also alters immune function. Stressed individuals tend to have leukocytosis (*an abnormally high number of white blood cells, in the blood*) that are mostly neutrophils (*a type of white blood cell that plays a major role in the body's defense against bacteria, viruses, and fungi*), reduced circulating T cells (*a white blood cell responsible for the body's immunity*), altered immunoglobulin levels, and diminished mitogen proliferative responses (*affecting cell division*) and natural killer cell activity. Chronic stress may also reduce antibody responses after immunization and have other long-term effects on health.

STRESS AND ATTACHMENT

Tactile contact or the sense of touch, is the strongest determinant of attachment among primates; young individuals deprived of tactile contact may develop autistic-like behaviors. Early tactile deprivation reduces glucocorticoid (*hormones produced by the adrenal gland which regulate protein, carbohydrate and fat metabolism*) binding sites in the hippocampus (*a part of the brain that is important for learning and memory*) and frontal cortex (*portion of the brain involved with reasoning, planning, abstract thought and other complex cognitive functions in addition to motor function*). Thus, hypothalamic-pituitary-adrenal (HPA)-axis hormones mediate responses to tactile contact and subsequent attachment. Newborn rats are hyporesponsive to HPA activation. If removed from its mother, however, the young rat's HPA-axis becomes very reactive and may remain so throughout life. This hyperreactivity is tempered if some elements of maternal interaction are maintained (stroking, licking, feeding). Thus maternal behaviors buffer HPA-axis reactivity.

In human children, diurnal patterns of cortisol production are established around 12 weeks of age. The ability to modulate stress reactivity emerges between four and six months of age. Institutionalized children living in difficult circumstances in Romania lack diurnal (*active in the daytime*) variation in cortisol production. Cortisol levels in institutionalized children in some settings correlate inversely to performance on the mental and motor scales of the Bayley test and directly with length of institutionalization. Among institutionalized children in France and Hungary, cortisol levels correlated with group size and ratio of children to caregivers.

Studies in noninstitutionalized children also support the notion that caregivers influence cortisol levels in young children. For example, stressful situations (such as immunization) induce higher levels of cortisol if the child is with an unfamiliar caregiver or is poorly attached. Similarly, cortisol levels in 9-month-olds left with an unfamiliar babysitter relate to the demeanor of the babysitter – children left with friendly, engaging sitters have lower levels than those left with withdrawn, unfriendly sitters.

Other studies suggest that attachment security and "fearfulness" relate to cortisol reactivity. For example, insecurely attached children have higher heart rates and cortisol concentrations, hyperactive HPA-axis and autonomic function, and increased limbic-hypothalamic arousal. Somewhat counterintuitively, children placed in unfamiliar situations who become quiet, stop playing, do not cry and appear to sleep have the highest elevations of cortisol, while those who demand attention (climb into lap, bring toys, cry, fret, or tantrum) have lower levels. Sensitive, responsive, and secure caretaking thus buffers cortisol elevations in infants and young children. In contrast, depressed or withdrawn caregivers augment stress responses in young children, possibly increasing the later risk of emotional or behavioral disorders.

STRESS AND MENTAL ILLNESS: POST-TRAUMATIC STRESS DISORDER AND DEPRESSION

Early childhood trauma, such as parental loss, physical or sexual abuse, or neglect, increases the risk of later mental disorders, including post-traumatic stress disorder (PTSD) and depression. Post-traumatic stress disorder is mediated in part via HPA-axis dysfunction. It is characterized by "intrusive re-experiencing," autonomic hyperarousal, reduced responsiveness, intense emotional reactions, sleep problems, learning difficulties, memory disturbances, dissociation, aggression against self and others, and psychosomatic reactions. Many of these symptoms are signs of an overactive amygdale which is located in the brain's medial temporal lobe and is believed to play a key role in the emotions.

Acutely, patients feel as though the trauma were recurring. They experience sleep disturbances, increased vigilance, exaggerated startle response, and generalized anxiety or agitation. Chronically, children exhibit detachment, restricted range of affect, dissociative episodes, sadness, somatization (*psychological needs expressed as physical symptoms*), and feelings of hopelessness. Many of these characteristics have been described in institutionalized and post-institutionalized children. This condition is underrecognized in post-adoption. Sometimes PTSD is complicated by the coexistence of mood, psychotic or other anxiety disorders, suicidal ideation, learning disabilities, or attentional difficulties.

The age at which trauma occurs is critical. Traumatic experiences in infancy may permanently alter HPA-axis responsivity. For example, rats removed from their mothers in infancy exhibit increased ACTH and corticosterone to a variety of stressors in adulthood. Similarly, compared to non-abused controls, women with a history of physical or sexual abuse as children had elevated ACTH, cortisol, and heart rate when performing mental arithmetic in a laboratory setting. Macaques raised in insecure conditions have elevated concentrations of CRF and reduced concentrations of CRF and reduced concentrations of cortisol in cerebrospinal fluid, the same findings as in humans with PTSD.

One theory is that patients with PTSD develop hypothalamic and pituitary hypersensitivity to circulating levels of cortisol. As might be expected, patients with PTSD have marked hippocampal (*the part of the brain that is important for learning and memory*) shrinkage (detectable by magnetic resonance imagine) and declarative memory deficits. Furthermore, patients with PTSD have elevated catecholamines (*class of hormones secreted by the adrenal glands and includes norepinephrine and dopamine*), independent of changes in cortisol. In a recent study of 80 children adopted from Romania in the Netherlands, 20% fulfilled clinical criteria for PTSD five years after adoption.

Major depression in adults is also linked to increased HPA-axis reactivity, elevated levels of cortisol, and relative insensitivity to cortisol feedback. Thus, childhood trauma may result in neurobiological changes that extend into adulthood, including alterations in autonomic and catecholamine activity, disregulation of HPA-axis function, increase in brain glucocorticoid receptor numbers, decrease in cortisol responsiveness, and reduction in volume of the hippocampus.

STRESS AND BEHAVIOR

Stress in early life may have long-lasting effects on behavior. Concentration, distractibility, executive function, and emotional regulation may be adversely influenced by early exposure to stress. Children who experience stress in early life in the absence of secure attachments may lose their ability to modulate impulsivity and the intensity of their feelings. The manifestations may range from learning abilities to aggression against self or others. Children who have received inadequate early care may have higher rates of personality disorders, marital discord and disruptions, and difficulty with their own parenting abilities. Previously abused or maltreated children have increased likelihood of depressive and anxiety disorders, eating disorders, suicide attempts, substance abuse and addiction, criminal behavior, and other interpersonal difficulties.

The societal cost of neglect during critical periods of infant development is an increase in violence, crime, mental health disorders, unproductive adults, and unstable family situations. Early exposure to stress may be ameliorated by a positive relationship with a competent adult, skill at learning and problem solving, engaging personality, competence and perceived efficacy by self or society, high IQ score, positive school experience, mastery of motivation, and previous successful coping experiences and, later, marriage to a supportive, nondeviant partner.

SUMMARY

Stress in early life thus has far-reaching effects on brain biochemistry and behavior, even into adult life. It should be assumed that internationally adopted children have experienced some degree of stress prior to placement with their adoptive families. Although a loving and supportive adoptive home may overcome some of the effects of stress in early life, some children may require specific treatment for prior stress. Vulnerable personality, genetic susceptibility, age at time of trauma, type and severity of trauma, and associated experiences all contribute to the outcome of children who have experienced stress in early life.

Key Points for Internationally Adopted Children

- Most international adoptees are exposed to stress pre-and postnatally.
- This exposure may have long-lasting effects on growth, the immune system, attachment, mental illness, and behavior.
- Genetic susceptibility and many other factors contribute to these responses.

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