

The Neuroarcheology of Childhood Maltreatment

The Neurodevelopmental Costs of Adverse Childhood Events

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Introduction

Childhood maltreatment has profound impact on the emotional, behavioral, cognitive, social and physical functioning of children. Developmental experiences determine the organizational and functional status of the mature brain and, therefore, adverse events can have a tremendous negative impact on the development of the brain. In turn, these neurodevelopmental effects may result in significant cost to the individual, their family, community and, ultimately, society. In essence, childhood maltreatment alters the potential of a child and, thereby, robs us all.

The present chapter will review some of those costs from a neurodevelopmental perspective. The premise is that when the core principles of neurodevelopment are understood, the costs of adverse childhood events and maltreatment become obvious. Following a brief presentation of the key concepts of neurodevelopment, two primary forms of maltreatment will be considered: (1) neglect and (2) traumatic stress. Maltreatment of children often involves both neglect and trauma; a more complete understanding of the complex neurodevelopmental impact of the combination, however, is best understood after presenting the potential effects of each separately. This chapter presents the current articulation of a neurodevelopmental perspective of childhood maltreatment originally outlined in 1994 (Perry, 1994) and further elaborated over the last five years

(Perry, Pollard, Blakley, Baker, & Vigilante. 1995) (Perry & Pollard. 1998)

This most recent articulation outlines the issue of maltreatment through the lens of developmental neurobiology and coins a descriptive phrase, "neuroarcheology," to capture the impact of adverse events on the developing brain, with the implicit suggestion that experiences leave a 'record' within the matrix of the brain. The nature and location of this record will depend upon the nature of the experience and the time in development when the event took place – much as with the archeological record of the earth. While this phrase may be simplistic to some, it conveys important conceptual principles about the nature of childhood experience which have been lacking all too often in clinical and research formulations regarding maltreatment. Not a single psychometric instrument measuring traumatic or adverse events, for example, uses time of trauma as a meaningful variable despite the fact that it may be the most important determinant of functional outcome following maltreatment.

The neuroarcheological perspective on childhood experience, therefore, simply posits that the impact of a childhood event (adverse or positive) will be a reflection of (1) the nature, intensity, pattern and duration of the event and (2) that the resulting strengths (e.g., language) or deficits (e.g., neuropsychiatric symptoms) will be in those functions mediated by the neural systems that are most rapidly organizing (i.e., in the developmental "hot zone") at the time of the experience.

Brain Organization and Function

The human brain is the remarkable organ that allows us to sense, process, perceive, store and act on information from outside and inside the body to carry out the three prime directives required for the survival of our species: (1) survive, (2) affiliate and mate and then, (3) protect and nurture dependents. In order to carry out these core and overarching responsibilities, thousands of inter-related functions have evolved. In the human brain, structure and function have co-evolved. As we have a hierarchy of increasingly complex functions related to our optimal functioning, our brain has evolved a hierarchical structural organization (see Table 1). This hierarchy starts with the lower, simpler brainstem areas and increases in complexity up through the neocortex (Figure 1). In each of these many areas of the brain are neural systems that mediate our many brain-related functions (Figure1; Table1). The 'lower' parts of the brain (brainstem and midbrain) mediate simpler regulatory functions (e.g., regulation of respiration, heart rate, blood pressure, body temperature) while more complex functions (e.g., language and abstract thinking) are mediated by the more complex neocortical structures of the human brain.

This hierarchical structure is the heart of a neuroarcheological understanding of

adverse childhood events. This structure becomes the multi-layered soil within which the fossilized evidence of maltreatment can be found – each layer organizing at a different time and each layer reflecting the experiences –good and bad - of that era in the individual's life. Key insights to understanding human functioning, then, will come from understanding neurodevelopment.

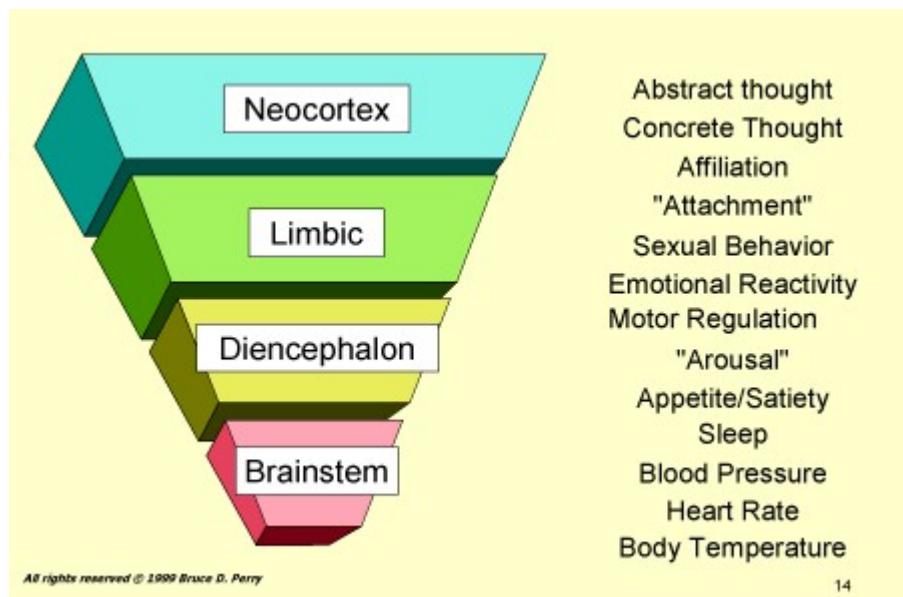


Figure 1: Hierarchical Organization of the Human Brain: The brain can be divided into four interconnected areas: brainstem, diencephalons, limbic and neocortex. The complexity of structure, cellular organization and function increases from the lower, simpler areas such as the brainstem to the most complex, the neocortex.

Neurodevelopment

Our brain's complex structure is comprised of 100 billion neurons and ten times as many glial cells – all interconnected by trillions of synaptic connections – and communicating in a non-stop, ever-changing dynamic of neurochemical activity. The brain doesn't just pop into existence. This most complex of all biological systems in the known universe is a product of neurodevelopment – a long process orchestrating billions upon billions of complex chemical transactions. It is through these chemical actions that a human being is created.

The developing child is a remarkable phenomenon of nature. In a few short

years, one single cell – the fertilized egg – becomes a walking, talking, learning, loving, and thinking being. This physical transformation is equivalent to a 6-foot tall, 200 pound man growing to the size of Connecticut in three years. In each of the billions and billions of cells in the body, a single set of genes has been expressed in millions of different combinations with precise timing. Development is a breathtaking orchestration of precision micro-construction that allows the healthy development of a human being. And the most remarkable and complex of all the organs in the human body is the human brain. In order to create the brain, a small set of pre-cursor cells must divide, move, specialize, connect and create specialized neural networks that form functional units. The key processes in neurodevelopment are summarized below.

Core Processes of Neurodevelopment

1. Neurogenesis: The brain starts as a few cells present early in the first weeks of life. From a few specialized cells in the unformed brain, come billions of nerve cells and trillions of glia. This, of course, requires that cells be "born." Neurogenesis is the birth of new neurons. The vast majority of neurogenesis takes place in utero during the second and third trimester. At birth, the vast majority of neurons, literally more than 100 billion, used for the remainder of life are present. Few neurons are born after birth, although researchers have demonstrated recently that neurogenesis can and does take place in the mature brain (Gould, Reeves, Graziano, & Gross. 1999). This is a very significant observation and may be one of the important physiological mechanisms responsible for the brain's plasticity (i.e., capacity to restore function) following injury.

Despite being present at birth, these neurons have yet to organize into completely functional systems. Many neurons need to mature themselves and undergo a set of processes that create the functional neural networks of the mature brain (Table 2).

2. Migration: Developing neurons move. Often guided by glial cells and a variety of chemical markers (e.g., cellular adhesion molecules, nerve growth factor: NGF), neurons cluster, sort, move and settle into a location in the brain that will be their final "resting" place. It is the fate of some neurons to settle in the brainstem, others in the cortex, for example. More than one half of all neurons are in the cortex. The processes of cortical cell migration and fate mapping are some of the most studied in all of developmental neuroscience (Rakic. 1981) (Rakic. 1996). It is clear that both genetic and environmental factors play important roles in determining a neuron's final location. Migration takes place primarily during the intrauterine and immediate perinatal period but continues throughout childhood and, possibly, to some degree into adult life. A host of intrauterine and perinatal insults – including infection, lack of oxygen, alcohol and various psychotropic drugs can alter migration of neurons and have profound

impact on functioning (Perry. 1988).

Table 1. A Neuroarcheological Chart of Development: Functional Organization

| Functional Division | Constituent Parts | Developmental Division | Age of Functional Maturity | Functions |
|---------------------|--|--|----------------------------|--|
| Neocortex | Cerebral cortex Frontal Lobes Temporal Lobes Parietal Lobes Occipital Lobes Corpus Callosum | Telencephalon | Puberty | Abstraction Self-image Socialization |
| | Limbic Cingulate Cortex Amygdala Hippocampus Septum | | Childhood | Affiliation Attachment Mood regulation |
| Early childhood | | Fine motor Large motor Complex state regulation (e.g., sleep, appetite) | | |
| Diencephalon | | Thalamus Hypothalamus | Diencephalon | Infancy |
| Brainstem | Midbrain Superior Colliculus Inferior Colliculus | Mesencephalon | Six months | Primary state regulation |
| | Cerebellum | Metencephalon | | Core physiological reflexes and regulatory functions |
| | Pons | Myelencephalon | Third trimester | |
| | Medulla Oblongata | | | |
| Spinal Cord | Spinal Cord | | Third trimester | |

3. Differentiation: Neurons mature. Each of the 100 billion neurons in the brain has the same set of genes, yet each neuron is expressing a unique combination of those genes to create a unique identity. Some neurons are large, with long axons; others short. Neurons can mature to use any of a hundred different neurotransmitters such as norepinephrine, dopamine, serotonin, CRF or substance P. Neurons can have dense dendritic fields receiving input from hundreds of other neurons, while other neurons can have a single linear input from one other neuron. Each of these thousands of differentiating "choices" come as a result of the pattern, intensity and timing of various microenvironmental cues which tell the neuron to turn on some genes and turn off others. Each neuron undergoes a series of "decisions" to determine their final location and specialization. These decisions, again, are a combination of genetic and microenvironmental cues. The further along in development, the more

differentiated the neuron, the more sensitive it becomes to the environmental signals. From the intrauterine period through early childhood (and to some degree beyond) neurons are very sensitive to experience-based signals, many of which are mediated by patterned neuronal activity in the neural network in which they reside. Neurons are literally designed to change in response to chemical signals. Therefore, any experience or event that alters these neurochemical or microenvironmental signals during development can change the ways in which certain neurons differentiate, thereby altering the functional capacity of the neural networks in which these neurons reside.

4. Apoptosis: Some developing neurons die. In many areas of the brain, there are more neurons born than are needed for any given function. Many of these neurons are redundant and when unable to adequately "connect" into an active neural network will die (Kuan, Roth, Flavell, & Rakic. 2000). Research in this area suggests that these neurons may play a role in the remarkable flexibility present in the human brain at birth. Depending upon the challenges of the environment and the potential needs of the individual, some neurons will survive while others will not. Again, this process appears to have genetic and environmental determinants. Neurons that make synaptic connections with others and have an adequate level of activation will survive; those cells that have little activity resorb. This is one example of a general principle of activity-dependence ("use it or lose it") that appears to be important in many neural processes related to learning, memory and development.

5. Arborization: As neurons differentiate, they send out tiny fiber-like extensions from their cell body. These dendrites become the receptive area where other neurons connect. It is in this receptive field that dozens to hundreds of other neurons are able to send neurochemical signals to the neuron. The density of these dendritic branches appears to be related to the frequency and intensity of incoming signals. When there is high activity, the dendritic network extends, essentially branching out in the same fashion as a bush may create new branches. This arborization allows the neuron to receive, process and integrate complex patterns of activity that will, in turn, determine its activity. Again, the arborization process appears to be to some degree activity-dependent. The density of the dendritic arborization appears to be related to the complexity and activity of incoming neural activity. In turn, these neural signals are often dependent upon the complexity and activity of the environment of the animal (Diamond, Law, Rhodes, et al. 1966; Greenough, Volkmar, & Juraska. 1973).

6. Synaptogenesis: Developing neurons make connections with each other. The major mechanism for neuron-to-neuron communication is 'receptor-mediated' neurotransmission that takes place at specialized connections between neurons called synapses. At the synapse, the distance between two neurons is very short. A chemical (classified as a neurotransmitter, neuromodulator or neurohormone) is released from the 'presynaptic' neuron and into the extra-cellular space (called the synaptic cleft) and binds to a specialized receptor protein in the membrane of

the 'postsynaptic' neuron. By occupying the binding site, the neurotransmitter helps change the shape of this receptor which then catalyzes a secondary set of chemical interactions inside the postsynaptic neuron that create second messengers. The second messengers such as cyclic AMP, inositol phosphate and calcium will then shift the intracellular chemical milieu which may even influence the activity of specific genes. This cascade of intracellular chemical responses allows communication from one neuron to another.

A continuous dynamic of synaptic neurotransmission regulates the activity and functional properties of the chains of neurons that allow the brain to do all of its remarkable activities. These neural connections are not random. They are guided by important genetic and environmental cues. In order for our brain to function properly, neurons, during development, need to find and connect with the "right" neurons. During the differentiation process, neurons send fiber-like projections (growth cones) out to make physical contact with other neurons. This process appears to be regulated and guided by certain growth factors and cellular adhesion molecules that attract or repel a specific growth cone to appropriate target neurons. Depending upon a given neuron's specialization, these growth cones will grow (becoming axons) and connect to the dendrites of other cells and create a synapse. During the first eight months of life there is an eight-fold increase in synaptic density while the developing neurons in the brain are "seeking" their appropriate connections (Huttenlocher. 1979) (Huttenlocher. 1994). This explosion of synaptogenesis allows the brain to have the flexibility to organize and function in with a wide range of potential. It is over the next few years, in response to patterned repetitive experiences that these neural connections will be refined and sculpted.

7. Synaptic sculpting: The synapse is a dynamic structure. With ongoing episodic release of neurotransmitter, occupation of receptors, release of growth factors, shifts of ions in and out of cells, laying down of new microtubules and other structural molecules, the synapse is continually changing. A key determinant of change in the synapse appears to be the level of presynaptic activity. When there is a consistent active process of neurotransmitter release, synaptic connections will be strengthened with actual physical changes that make the pre- and postsynaptic neurons come closer and the process of neurotransmission more efficient. When there is little activity, the synaptic connection will literally dissolve. The specific axonal branch to a given neuron will go away. Again, this powerful activity-dependent process appears to be very important for understanding learning, memory and the development. At any given moment – all throughout life – we are making and breaking synaptic connections. For the majority of life we are at equilibrium; the rate of creating new synaptic connections is equal to the rate of resorbing older, unused connections. While somewhat simplistic, it appears that the synaptic sculpting is a "use it or lose it" process. During the first eight months following birth the rate of creating new synapses far outstrips the rate of resorbing unused connections. By age one, however, and from then through early childhood, the rate of resorbing new connections is faster than the

rate of creating new synapses. By adolescence, in most cortical areas at least, this process again reaches equilibrium.

8. Myelination: Specialized glial cells wrap around axons and, thereby, create more efficient electrochemical transduction down the neuron. This allows a neural network to function more rapidly and efficiently, thereby allowing more complex functioning (e.g., walking depends upon the myelination of neurons in the spinal cord for efficient, smooth regulation of neuromotor functioning.) The process of myelination begins in the first year of life but continues in many key areas throughout childhood with a final burst of myelination in key cortical areas taking place in adolescence.

Table 2: Key Processes in Neurodevelopment

| Key Processes | Age beginning* | Greatest period of activity** | Age of equilibrium** | Other |
|---------------------------|------------------------|------------------------------------|--|---|
| <i>Neurogenesis</i> | First trimester | <i>In utero</i> | 99 % of 100 billion neurons born by birth | Evidence of hippocampal cell birth in adult life |
| <i>Migration</i> | First trimester | <i>In utero</i> through first year | Regional specific: majority of migration complete by age three | Some suggestion of migration following brain injury |
| <i>Differentiation</i> | First-second trimester | Third trimester through year one | Region specific: primary differentiation complete by age three | Continues in some fashion throughout life |
| <i>Apoptosis</i> | Third trimester | First year | Age one | Majority of programmed death complete by age three |
| <i>Arborization</i> | Third trimester | First year | Primary dendritic arborization present by age three | Very experience dependent - continued sensitivity throughout life |
| <i>Synaptogenesis</i> | Third trimester | 8 months | Region specific: with most cortical areas by age 10, other areas earlier | Continuous activity-dependent process through life |
| <i>Synaptic sculpting</i> | Birth | First four years | Region specific: cortical areas by age six | Second phase of activity during puberty |
| <i>Myelination</i> | Birth | First four years | Region specific: majority complete by 10 | Continuing important myelination through adolescence |

* This refers to the age at which approximately 10% of this specific function is taking place. In most cases, there is evidence that some of these processes have started to some degree. Almost all of these processes continue in some form throughout life, the table is designed to illustrate the

relative importance of childhood for the majority of activity in each of these processes.

***These are crude estimates based upon data from multiple sources. The major point is to demonstrate that shifting activity from neurogenesis to myelination.*

All of the neurodevelopmental processes described above are dependent upon both genetic and environmentally determined microenvironmental cues (e.g., neurotransmitters, neuromodulators, neurohormones, ions, growth factors, cellular adhesion molecules and other morphogens). Disruption of the pattern, timing or intensity of these cues can lead to abnormal neurodevelopment and profound dysfunction. The neuroarcheological perspective suggests that the specific dysfunction will depend upon the timing of the insult (e.g., was the insult *in utero* during the development of the brainstem or at age two during the active development of the cortex), the nature of the insult (e.g., is there a lack of sensory stimulation from neglect or an abnormal persisting activation of the stress response from trauma?), the pattern of the insult (i.e., is this a discreet single event, a chronic experience with a chaotic pattern or an episodic event with a regular pattern?).

While we are only beginning to understand the complexity of neurodevelopment, there are several key principles that emerge from the thousands of studies and years of focused research on these neurodevelopmental processes. These principles, as outlined below, suggest that while the structural organization and functional capabilities of the mature brain can change throughout life, the majority of the key stages of neurodevelopment take place in childhood. The core principles of neurodevelopment that support a neuroarcheological perspective of childhood adverse events are summarized below.

Core Principles of Neurodevelopment

1. Nature and nurture: For too many years, any conceptual approach to human behavior has been tainted by the nature versus nurture debate. Do genes cause human behavior or is human behavior a product of learning, education and experience? Ultimately, this debate polarizes and distracts from more complex understandings of human functioning. Genes are designed to work in an environment. Genes are expressed by microenvironmental cues, which, in turn, are influenced by the experiences of the individual. How an individual functions within an environment, then, is dependent upon the expression of a unique combination of genes available to the human species. We don't have the genes to make wings. **And** what we become depends upon how experiences shape the expression – or not - of specific genes we do have. We do have the genes to make forty sounds – and we can have the experiences that turn this genetically determined capacity into a powerful, transforming tool – language. Yet, there are many sad examples of cruel experiments of humanity, where a young child was raised in an environment deprived of language. This child, despite the genetic potential to speak and think and feel in complex humane ways, did not express

that potential fully. Genetic potential without appropriately timed experiences can remain unexpressed. Nature and nurture – we are nothing without both; we require both and we are products of both.

The influence of gene-driven processes, however, shifts during development. In the just fertilized ovum, all of the chemical processes that are driving development are very dependent upon a genetically determined sequence of molecular events. By birth, however, the brain has developed to the point where environmental cues mediated by the senses play a major role in determining how neurons will differentiate, sprout dendrites, form and maintain synaptic connections and create the final neural networks that convey functionality. By adolescence, the majority of the changes that are taking place in the brain of that child are determined by experience, not genetics. The languages, beliefs, cultural practices, and complex cognitive and emotional functioning (e.g., self esteem) by this age are primarily experience-based.

2. Sequential Developmental: The brain develops in a sequential and hierarchical fashion; organizing itself from least (brainstem) to most complex (limbic, cortical areas). These different areas develop, organize and become fully functional at different times during childhood. At birth, for example, the brainstem areas responsible for regulating cardiovascular and respiratory function must be intact for the infant to survive, and any malfunction is immediately observable. In contrast, the cortical areas responsible for abstract cognition have years before they will be 'needed' or fully functional.

This means that each brain area will have its own timetable for development. The neurodevelopmental processes described above will be most active in different brain areas at different times and will, therefore, either require (critical periods) or be sensitive to (sensitive periods) organizing experiences (and the neurotrophic cues related to these experiences). The neurons for the brainstem have to migrate, differentiate and connect, for example, before the neurons for the cortex.

The implications of this for a neuroarcheological formulation are profound. Disruptions of experience-dependent neurochemical signals during these periods may lead to major abnormalities or deficits in neurodevelopment. Disruption of critical neurodevelopmental cues can result from 1) lack of sensory experience during sensitive periods (e.g., neglect) or 2) atypical or abnormal patterns of necessary cues due to extremes of experience (e.g., traumatic stress, see below). Insults during the intrauterine period, for example, will more likely influence the rapidly organizing brainstem systems as opposed to the more slowly organizing cortical areas. The symptoms from the intrauterine disruption will alter functions mediated by the brainstem and could include sensory integration problems, hyper-reactivity, poor state regulation (e.g., sleep, feeding, self-soothing), tactile defensiveness and altered regulation of core neurophysiological functions such as respiration, cardiovascular and temperature

regulation.

This does not mean that neocortical systems are unaffected by disrupting the development of the brainstem. Indeed, one of the most important aspects of the sequential development is that important organizing signals for any given brain area or system (e.g., patterns of neural activity, neurotransmitters acting as morphogens) come from previously organized brain areas or systems. Due to the sequential development of the brain, disruptions of normal developmental processes early in life (e.g., during the perinatal period) that alter development of the brainstem or diencephalon will necessarily alter the development of limbic and cortical areas. This is so because many of the organizing cues for normal limbic and neocortical organization originate in the lower brain areas. Any developmental insult can have a cascade effect on the development of all "downstream" brain areas (and functions) that will receive input from the effected neural system.

3. Activity-dependent neurodevelopment: The brain organizes in a use-dependent fashion. As described above, many of the key processes in neurodevelopment are activity dependent. In the developing brain, undifferentiated neural systems are critically dependent upon sets of environmental and micro-environmental cues (e.g., neurotransmitters, cellular adhesion molecules, neurohormones, amino acids, ions) in order for them to appropriately organize from their undifferentiated, immature forms (Lauder. 1988; Perry. 1994) (Perry & Pollard. 1998). Lack, or disruption, of these critical cues can alter the neurodevelopmental processes of neurogenesis, migration, differentiation, synaptogenesis - all of which can contribute to malorganization and diminished functional capabilities in the specific neural system where development has been disrupted. This is the core of a neuroarcheological perspective on dysfunction related adverse childhood events (Perry. 1994) (Perry & Pollard. 1998; Perry. 1998). These molecular cues that guide development are dependent upon the experiences of the developing child. The quantity, pattern of activity and nature of these neurochemical and neurotrophic factors depends upon the presence and the nature of the total sensory experience of the child. When the child has adverse experiences – loss, threat, neglect, and injury – there can be disruptions of neurodevelopment that will result in neural organization that can lead to compromised functioning throughout life (see Neglect section, below).

A neuroarcheological perspective would predict that the dysfunction resulting from a specific adverse event is related to the disrupted (or altered) development of the neural system that is, during the adverse event, most rapidly developing. The degree of disruption is related to the rate of change in the respective neural system. The already organized and functioning neural system is less vulnerable to a developmental insult than the rapidly changing, energy-hungry and microenvironmental cue-sensitive developing system. This is so because of a principle called biological relativity. In any dynamic system, the impact of an

event or experience (disruptive or positive) is greatest on the most actively changing or dynamic parts of that system. The power of any experience, therefore, is greatest during the most rapid phases of development. Events taking place during a neural system's most active phase of organization will have more impact than events after the system has organized.

4. Windows of Opportunity/Windows of Vulnerability. The sequential development of the brain and the activity-dependence of many key aspects of neurodevelopment suggest that there must be times during development when a given developing neural system is more sensitive to experience than others (Table 3). In healthy development, that sensitivity allows the brain to rapidly and efficiently organize in response to the unique demands of a given environment to express from its broad genetic potential those characteristics which best fit that child's world. If the child speaks Japanese as opposed to English, for example, or if this child will live in the plains of Africa or the tundra of the Yukon, different genes can be expressed, different neural networks can be organized from that child's potential to best fit that family, culture and environment. We all are aware of how rapidly young children can learn language, develop new behaviors and master new tasks. The very same neurodevelopmental sensitivity that allows amazing developmental advances in response to predictable, nurturing, repetitive and enriching experiences make the developing child vulnerable to adverse experiences.

Sensitive periods are different for each brain area and neural system, and therefore, for different functions. The sequential development of the brain and the sequential unfolding of the genetic map for development mean that the sensitive periods for neural system (and the functions they mediate) will be when that system is in the developmental 'hot zone' – when that area is most actively organizing. The brainstem must organize key systems by birth; therefore, the sensitive period for those brainstem-mediated functions is during the prenatal period. The neocortex, in contrast, has systems and functions organizing throughout childhood and into adult life. The sensitive periods for these cortically mediated functions are likely to be very long.

With an understanding of the shifting vulnerability of the developing brain to experience, a neuroarcheological perspective becomes apparent. If there are disrupting adverse events during development, they will be mirrored by a matched dysfunctional development in the neural systems whose functioning the adverse experience most altered during the event. If the disruption were the absence of light during the first year of life – the systems most altered would be related to vision. If the disruption activates the stress response, the disruption will be in the neural systems mediating the stress response. The severity and chronicity of the specific dysfunction will be related to the vulnerability of the system affected. Adverse experiences influence the mature brain but in the developing brain, adverse experiences literally play a role in organizing neural systems. It is much easier to influence the functioning of a developing system

than to reorganize and alter the functioning of a developed system. Adverse childhood events, therefore, can alter the organization of developing neural systems in ways that create a lifetime of vulnerability.

Table 3: Shifting Developmental Activity across Brain Regions

| Brain Region | Age of greatest developmental activity | Age of functional maturity** | Key functions |
|---------------------|--|------------------------------|--|
| <i>Neocortex</i> | Childhood | Adult | Reasoning, problem solving, abstraction, secondary sensory integration |
| <i>Limbic</i> | Early childhood | Puberty | Memory, emotional regulation, attachment, affect regulation, primary sensory integration |
| <i>Diencephalon</i> | Infancy | Childhood | Motor control, secondary sensory processing |
| <i>Brainstem</i> | In utero | Infancy | Core physiological state regulation, primary sensory processing |

The simple and unavoidable conclusion of these neurodevelopmental principles is that the organizing, sensitive brain of an infant or young children is more malleable to experience than a mature brain. While experience may alter the behavior of an adult, experience literally provides the organizing framework for an infant and child. Because the brain is most plastic (receptive to environmental input) in early childhood, the child is most vulnerable to variance of experience during this time. In the second half of this chapter two primary forms of extreme childhood adverse experience will be discussed in context of the neuroarcheological perspective of adverse childhood events.

The Neurodevelopmental Impact of Neglect in Childhood

Neglect is the absence of critical organizing experiences at key times during development. Despite its obvious importance in understanding child maltreatment, neglect has been understudied. Indeed, deprivation of critical experiences during development may be the most destructive yet the least understood area of child maltreatment. There are several reasons for this. The most obvious is that neglect is difficult to "see." Unlike a broken bone, maldevelopment of neural systems mediating empathy, for example, resulting from emotional neglect during infancy, is not readily observable. Another important, yet poorly appreciated, aspect of neglect is the issue of timing. The needs of the child shift during development; therefore, what may be neglectful at one age is not at another. The very same experience that is essential for life at one stage of life may be of little significance or even inappropriate at another

age. We would all question the mother who held, rocked and breastfed her pubescent child. Touch, for example, is essential during infancy. The untouched newborn may literally die; in Spitz' landmark studies, the mortality rates in the institutionalized infants was near thirty percent (Spitz. 1945; Spitz. 1946). If one doesn't touch an adolescent for weeks, however, no significant adverse effects will result. Creating standardized protocols, procedures and "measures" of neglect, therefore, are significantly confounded by the shifting developmental needs and demands of childhood. Finally, neglect is understudied because it is very difficult to find large populations of humans where specific and controlled neglectful experiences have been well documented. In some cases, these cruel experiments of humanity have provided unique and promising insights (see below). In general, however, there will never be – and there never should be – the opportunity to study neglect in humans with the rigor that can be applied in animal models.

With these limitations, however, what we do know about neglect during early childhood supports a neuroarcheological view of adverse childhood experience. The earlier and more pervasive the neglect is, the more devastating the developmental problems for the child. Indeed, a chaotic, inattentive and ignorant caregiver can produce pervasive developmental delay (PDD; (Anonymous. 1994)) in a young child (Rutter, Andersen-Wood, Beckett, et al. 1999). Yet the very same inattention for the same duration if the child is ten will have very different and less severe impact than inattention during the first years of life.

There are two main sources of insight to childhood neglect. The first is the indirect but more rigorous animal studies and the second is a growing number of descriptive reports with severely neglected children.

Environmental Manipulation and Neurodevelopment: Animal Studies

Some of the most important studies in developmental neurosciences in the last century have been focusing on various aspects of experience and extreme sensory experience models. Indeed, the Nobel Prize was awarded to Hubel and Wiesel for their landmark studies on development of the visual system using sensory deprivation techniques (Hubel & Wiesel. 1963). In hundreds of other studies, extremes of sensory deprivation (Hubel & Wiesel. 1970; Greenough, Volkmar, & Juraska. 1973) or sensory enrichment (Greenough & Volkmar. 1973; Diamond, Krech, & Rosenzweig. 1964; Diamond, Law, Rhodes, et al. 1966) have been studied. These include disruptions of visual stimuli (Coleman & Riesen. 1968), environmental enrichment (Altman & Das. 1964; Cummins & Livesey. 1979), touch (Ebinger. 1974; Rutledge, Wright, & Duncan. 1974), and other factors that alter the typical experiences of development (Uno, Tarara, Else, & et.al. 1989; Plotsky & Meaney. 1993; Meaney, Aitken, van Berkal, Bhatnagar, & Sapolsky. 1988). These findings generally demonstrate that the brains of animals

reared in enriched environments are larger, more complex and functional more flexible than those raised under deprivation conditions. Diamond's work, for example, examining the relationships between experience and brain cytoarchitecture have demonstrated a relationship between density of dendritic branching and the complexity of an environment (for a good review of this and related data see (Diamond & Hopson. 1998)). Others have shown that rats raised in environmentally enriched environments have higher density of various neuronal and glial microstructures, including a 30% higher synaptic density in cortex compared to rats raised in an environmentally deprived setting (Bennett, Diamond, Krech, & Rosenzweig. 1964; Altman & Das. 1964). Animals raised in the wild have from 15 to 30% larger brain mass than their offspring who are domestically reared (Darwin. 1868; Rohrs. 1955; Rohrs & Ebinger. 1978; Rehkamper, Haase, & Frahm. 1988).

Animal studies suggest that critical periods exist during which specific sensory experience was required for optimal organization and development of the part of the brain mediating a specific function (e.g., visual input during the development of the visual cortex). While these phenomena have been examined in great detail for the primary sensory modalities in animals, few studies have examined the issues of critical or sensitive periods in humans. What evidence there is would suggest that humans tend to have longer periods of sensitivity and that the concept of critical period may not be useful in humans. It is plausible, however, that abnormal micro-environmental cues and atypical patterns of neural activity during sensitive periods in humans could result in malorganization and compromised function in a host of brain-mediated functions. Indeed, altered emotional, behavioral, cognitive, social and physical functioning has been demonstrated in humans following specific types of neglect. The majority of this information comes from the clinical rather than the experimental disciplines.

The Impact of Neglect in Early Childhood: Clinical Findings

Over the last sixty years, many case reports, case series and descriptive studies have been conducted with children neglected in early childhood. The majority of these studies have focused on institutionalized children. As early as 1833, with the famous Kaspar Hauser, feral children had been described (Heidenreich. 1834). Hauser was abandoned as a young child and raised from early childhood (likely around age two) until seventeen in a dungeon, experiencing relative sensory, emotional and cognitive neglect. His emotional, behavioral and cognitive functioning was, as one might expect, very primitive and delayed. At autopsy, Hauser's brain was noted to have a small cerebrum (cortex) with few and non-distinct cortical gyri. These findings are consistent with cortical atrophy (or underdevelopment), a condition we have reported in children following severe total global neglect in childhood (Perry & Pollard. 1997).

In the early forties, Spitz described the impact of neglectful caregiving on children in foundling homes (orphanages). Most significant, he was able to demonstrate that children raised in fostered placements with more attentive and nurturing caregiving had superior physical, emotional and cognitive outcomes (Spitz. 1945; Spitz. 1946). Some of the most powerful clinical examples of this phenomenon are related to profound neglect experiences early in life.

In a landmark report of children raised in a Lebanese orphanage, the Creche, Dennis (1973) described a series of findings supporting a neuroarcheological model of maltreatment. These children were raised in an institutional environment devoid of individual attention, cognitive stimulation, emotional affection or other enrichment. Prior to 1956 all of these children remained at the orphanage until age six, at which time they were transferred to another institution. Evaluation of these children at age 16 demonstrated a mean IQ of approximately 50. When adoption became common, children adopted prior to age 2 had a mean IQ of 100 by adolescence while children adopted between ages 2 and 6 had IQ values of approximately 80 (Dennis. 1973). This graded recovery reflected the neuroarcheological impact of neglect. A number of similar studies of children adopted from neglectful settings demonstrate this general principle. The older a child was at time of adoption, (i.e., the longer the child spent in the neglectful environment) the more pervasive and resistant to recovery were the deficits.

Money and Annecillo (1976) reported the impact of change in placement on children with psychosocial dwarfism (failure to thrive). In this preliminary study, 12 of 16 children removed from neglectful homes recorded remarkable increases in IQ and other aspects of emotional and behavioral functioning. Furthermore, they reported that the longer the child was out of the abusive home the higher the increase in IQ. In some cases IQ increased by 55 points (Money & Annecillo. 1976).

A more recent report on a group of 111 Romanian orphans (Rutter & English and Romanian Adoptees study team. 1998; Rutter, Andersen-Wood, Beckett, et al. 1999) adopted prior to age two from very emotionally and physically depriving institutional settings demonstrate similar findings. Approximately one half of the children were adopted prior to age six months and the other half between six months and 2 years old. At the time of adoption, these children had significant delays. Four years after being placed in stable and enriching environments, these children were re-evaluated. While both groups improved, the group adopted at a younger age had a significantly greater improvement in all domains.

These observations are consistent with the experiences of our clinic research group working with maltreated children. Over the last ten-year we have worked with more than 1000 children neglected in some fashion. We have recorded increases in IQ of over 40 points in more than 60 children following removal from neglectful environments and placed in consistent, predictable, nurturing, safe and

enriching placements (Perry et al., in preparation). In addition, in a study of more than 200 children under the age of 6 removed from parental care following abuse and neglect we demonstrated significant developmental delays in more than 85% of the children. The severity of these developmental problems increased with age, suggesting, again, that the longer the child was in the adverse environment - the earlier and more pervasive the neglect - the more indelible and pervasive the deficits.

The impact of deprivation can be approximated by sensory chaos. Indeed, sensory deprivation is much less clinically significant than sensory chaos. The vast majority of children suffering from neglect do so because their experiences are chaotic, dysynchronous, inconsistent and episodic rather than consistent, predictable and continuous. The organizing brain requires patterns of sensory experience to create patterns of neural activity that, in turn, play a role in guiding the various neurodevelopmental processes involved in healthy development. When experience is chaotic or sensory patterns are not consistent and predictable, the organizing systems in the brain reflect this chaos and, typically, organize in ways that result in dysregulation and dysynchronous. Imagine trying to learn a language if you only heard random words without the context, grammar and syntax of the language (i.e., the patterns of use). Even if you heard and perceived all words, you could not develop language. *Random exposure to words absent an organizing pattern leads to abnormal development of speech and language.* Our clinical group has evaluated many children capable of parroting advertising phrases from television but incapable of simple verbal communication.

This requirement for consistent, repetitive and patterned stimuli holds for all experience – cognitive, emotional, social and physical. Repetitive, patterned, consistent experience allows the brain to create an internal representation of the external world. A child growing up in the midst of chaos and unpredictability will develop neural systems and functional capabilities that reflect this disorganization.

The Impact of Neglect in Early Childhood: Neurobiological Findings

All of these reported developmental problems – language, fine and large motor delays, impulsivity, disorganized attachment, dysphoria, attention and hyperactivity, and a host of others described in these neglected children – are caused by abnormalities in the brain. Despite this obvious statement, very few studies have examined directly any aspect of neurobiology in neglected children. The reasons include a lack of capacity, until the recent past, to examine the brain in any non-invasive fashion.

Our group has examined various aspects of neurodevelopment in neglected

children (Perry & Pollard, 1997). Neglect was considered global neglect when a history of relative sensory deprivation in more than one domain was obtained (e.g., minimal exposure to language, touch and social interactions). Chaotic neglect is far more common and was considered present if history was obtained that was consistent with physical, emotional, social or cognitive neglect. When possible history was obtained from multiple sources (e.g., investigating CPS workers, family, police). The neglected children (n= 122) were divided into four groups: Global Neglect (GN; n=40); Global Neglect with Prenatal Drug Exposure (GN+PND; n=18); Chaotic Neglect (CN; n=36); Chaotic Neglect with Prenatal Drug Exposure (CN+PND; n=28). Measures of growth were compared across group and compared to standard norms developed and used in all major pediatric settings.

Dramatic differences from the norm were observed in FOC (the frontal-occipital circumference, a measure of head size and in young children a reasonable measure of brain size). In the globally neglected children the lower FOC values suggested abnormal brain growth. For these globally neglected children the group mean was below the 8th percentile. In contrast, the chaotically neglected children did not demonstrate this marked group difference in FOC. Furthermore in cases where MRI or CT scans were available, neuroradiologists interpreted 11 of 17 scans as abnormal from the children with global neglect (64.7 %) and only 3 of 26 scans abnormal from the children with chaotic neglect (11.5 %). The majority of the readings were "enlarged ventricles" or "cortical atrophy." While the actual size of the brain in chaotically neglected children did not appear to be different from norms, it is reasonable to hypothesize that organizational abnormalities exist and that with function MRI studies these abnormalities will be more readily detected.

These findings strongly suggest that when early life neglect is characterized by decreased sensory input (e.g., relative poverty of words, touch and social interactions) there will be a similar effect on human brain growth as in other mammalian species. The human cortex grows in size, develops complexity, makes synaptic connections and modifies as a function of the quality and quantity of sensory experience. Lack of type and quantity of sensory-motor and cognitive experiences lead to underdevelopment of the cortex – in rats, non-human primates and humans.

Studies from other groups are beginning to report similar altered neurodevelopment in neglected children. In the study of Romanian orphans described above, the 38 % had FOC values below the third percentile (greater than 2 SD from the norm) at the time of adoption. In the group adopted after six months, fewer than 3 % and the group adopted after six months 13 % had persistently low FOCs four years later (Rutter & English and Romanian Adoptees study team, 1998; O'Connor, Rutter, & English and Romanian Adoptees study team, 2000). Strathearn (Strathearn et al., submitted) has followed extremely low birth weight infants and shown that when these infants end up in neglectful

homes they have a significantly smaller head circumference at 2 and 4 years, but not at birth. This is despite having no significant difference in other growth parameters. Finally in a related population, maltreated children and adolescents with post-traumatic stress disorder (PTSD), De Bellis and colleagues found that subject children have significantly smaller intracranial and cerebral volumes than matched controls on MRI scan. Brain volume in these children correlated "robustly and positively" with the age of onset of PTSD trauma, and negatively with the duration of abuse, suggesting that traumatic childhood experiences may adversely affect brain development. Specific brain areas were affected differentially, in reflection of their importance in the stress response, further support of a neuroarcheological formulation of adverse childhood experience (De Bellis, Keshavan, Clark, et al. 1999).

While deprivations and lack of specific sensory experiences are common in the maltreated child, the traumatized child experiences developmental insults related to discrete patterns of over-activation of neurochemical cues. Rather than a deprivation of sensory stimuli, the traumatized child experiences over-activation of important neural systems during sensitive periods of development.

The Neurodevelopmental Impact of Traumatic Stress in Childhood

Each year in United States more than five million children are exposed to some form of extreme traumatic stressor. These traumatic events include natural disasters (e.g., tornadoes, floods, hurricanes), motor vehicle accidents, life threatening illness and associated painful medical procedures (e.g., severe burns, cancer), physical abuse, sexual assault, witnessing domestic or community violence, kidnapping and sudden death of a parent, among others (Pfefferbaum. 1997; Anonymous. 1998). These events, posing an actual or perceived threat to the individual, activate a stress response. During the traumatic event, the child's brain mediates the adaptive response. Brainstem and diencephalic stress-mediating neural systems are activated. These systems include the hypothalamic-pituitary-adrenal (HPA) axis, central nervous system (CNS) noradrenergic (NA), dopaminergic (DA) systems and associated CNS and peripheral systems that provide the adaptive emotional, behavioral, cognitive and physiological changes necessary for survival (Perry. 1994; Perry & Pollard. 1998).

Individual neurobiological responses during traumatic stress are heterogeneous (Perry, Pollard, Blakley, Baker, & Vigilante. 1995). The specific nature of a child's responses to a given traumatic event may vary with the nature, duration and the pattern of traumatic stressor and the child's constitutional characteristics (e.g., genetic predisposition, age, gender, history of previous stress exposure, presence of attenuating factors such as supportive caregivers). Whatever the individual response, however, the extreme nature of the external threat is

matched by an extreme and persisting internal activation of the neurophysiological systems mediating the stress response and their associated functions (Perry, Pollard, Blakley, Baker, & Vigilante. 1995; Perry & Pollard. 1998).

As described above, neural systems respond to prolonged, repetitive activation by altering their neurochemical and sometimes, microarchitectural (e.g., synaptic sculpting) organization and functioning. This is no different for the neural systems mediating the stress response. Following any traumatic event children will likely experience some persisting emotional, behavioral, cognitive and physiological signs and symptoms related to the, sometimes temporary, shifts in the activity of these neural systems originating in the brainstem and diencephalon. In general, the longer the activation of the stress-response systems (i.e., the more intense and prolonged the traumatic event), the more likely there will be a 'use-dependent' change in these neural systems (for review see (Perry & Pollard. 1998)). In some cases, then, the stress-response systems do not return to the pre-event homeostasis. In these cases, the signs and symptoms become so severe, persisting and disruptive that they reach the level of a clinical disorder (Perry. 1998). In a new context and in the absence of any true external threat, the abnormal persistence of a once adaptive response becomes maladaptive.

Post traumatic stress-related clinical syndromes

Post traumatic stress disorder (PTSD) is a clinical syndrome that may develop following extreme traumatic stress (DSM IV) (Anonymous. 1994). Like all other DSM IV diagnoses, it is likely that heterogeneous pathophysiologies underlie the cluster of diagnostic signs and symptoms labeled PTSD. There are six diagnostic criteria for PTSD: 1) extreme traumatic stress accompanied by intense fear, horror or disorganized behavior; 2) persistent re-experiencing of the traumatic event such as repetitive play or recurring intrusive thoughts; 3) avoidance of cues associated with the trauma or emotional numbing; 4) persistent physiological hyper-reactivity or arousal; 5) signs and symptoms present for more than one month following the traumatic event and 6) clinically significant disturbance in functioning.

Posttraumatic stress disorder has been studied primarily in adult populations, most commonly combat veterans and victims of sexual assault. Despite high numbers of traumatized children, the clinical phenomenology, treatment and neurophysiological correlates of childhood PTSD remain under studied. The clinical phenomenology of trauma-related neuropsychiatric sequelae is poorly characterized (Terr. 1991; Mulder, Fergusson, Beautrais, & Joyce. 1998). Most of the studies of PTSD have been following single discreet trauma (e.g., a shooting). The least characterized populations are very young children and

children with multiple or chronic traumatic events.

Clinical presentations

If during development, this stress response apparatus are required to be persistently active, the stress response apparatus in the central nervous system will develop in response to constant threat. These stress-response neural systems (and all functions they mediate – including sympathetic-parasympathetic tone, level of vigilance, regulation of mood, attention and sleep) will be poorly regulated, often overactive and hypersensitive. It is highly adaptive for a child growing up in a violent, chaotic environment to be hypersensitive to external stimuli, to be hypervigilant, and to be in a persistent stress-response state. It is important to realize that children exposed to traumatic stress during development literally organize their neural systems to adapt to this kind of environment. In contrast, an adult with no previous traumatic stress can develop PTSD. The cardiovascular reactivity and physiological hypersensitivity that the adult develops, however, is cue specific. This means that they will demonstrate increased heart rate, startle response and other neurophysiological symptoms when exposed to a cue from the original trauma (e.g., the Vietnam vet hearing a helicopter). In contrast, young children will develop a generalized physiological hyper-reactivity and hypersensitivity to all cues that activate the stress response apparatus. This generalized change results when the traumatic stress literally provides the organizing cues for their developing stress response neurobiology (Perry. 1999).

Clinically, this is very easily seen in children who are exposed to chronic neurodevelopmental trauma. These children are frequently diagnosed as having attention deficit disorder (ADD-H) with hyperactivity (Haddad & Garralda. 1992). This is somewhat misleading, however. These children are hypervigilant; they do not have a core abnormality of their capacity to attend to a given task. These children have behavioral impulsivity, and cognitive distortions all of which result from a use-dependent organization of the brain (Perry, Pollard, Blakley, Baker, & Vigilante. 1995). During development, these children spent so much time in a low-level state of fear (mediated by brainstem and diencephalic areas) that they consistently were focusing on non-verbal but not verbal cues. In our clinical population, children raised in chronically traumatic environments demonstrate a prominent V-P split on IQ testing (n = 108; WISC Verbal = 8. 2; WISC Performance = 10.4, Perry et al., in preparation). Often these children are labeled as learning disabled. We have seen these V-P splits in children in the juvenile justice system, child protective system and in the specialized clinical populations referred to our ChildTrauma clinic.

These children are also characterized by persisting physiological hyperarousal and hyperactivity (Perry, Pollard, Baker, Sturges, Vigilante, & Blakley. 1995; Perry. 1994; Perry. 2000). These children are observed to have increased muscle tone, frequently a low grade increase in temperature, an increased startle

response, profound sleep disturbances, affect regulation problems and anxiety (Kaufman. 1991; Ornitz & Pynoos. 1989; Perry. 2000). In addition, our studies indicate that a significant portion of these children have abnormalities in cardiovascular regulation (Perry, Pollard, Baker, Sturges, Vigilante, & Blakley. 1995; Perry. 2000). All of these symptoms are the result of a use-dependent organization of the brain stem nuclei involved in the stress response apparatus.

Children with PTSD may present with a combination of problems including impulsivity, distractibility and attention problems (due to hypervigilance), dysphoria, emotional numbing, social avoidance, dissociation, sleep problems, aggressive (often re-enactment) play, school failure and regressed or delayed development. In most studies examining the development of PTSD following a given traumatic experience, twice as many children suffer from significant post-traumatic signs or symptoms (PTSS) but lack all of the criteria necessary for the diagnosis of PTSD (Friedrich. 1998). In these cases, the clinician may identify the trauma-related symptom as being part of another neuropsychiatric syndrome.

The clinician is often unaware of ongoing traumatic stressors (e.g., domestic or community violence) or the family makes no association between the present symptoms and past events (e.g., car accident, death of a relative, exposure to violence) and may provide no relevant history to aid the clinician in the differential. As a result, PTSD is frequently misdiagnosed and PTSS are under recognized. Children with PTSD as a primary diagnosis are often labeled with Attention Deficit Disorder with Hyperactivity (ADHD), major depression, oppositional-defiant disorder, conduct disorder, separation anxiety or specific phobia. Ackerman and colleagues examined the prevalence of PTSD and other neuropsychiatric disorders in 204 abused children (ages 7 to 13) (Ackerman, Newton, McPherson, Jones, & Dykman. 1998). Thirty four percent of these children met criteria for PTSD. Over fifty percent of the children in this study suffering both physical and sexual abuse had PTSD. Using structured diagnostic interview, the majority of these children met diagnostic criteria for three or more Axis I diagnoses in addition to PTSD. Indeed, only 6 of 204 children met criteria for only PTSD. The broad co-morbidity reported in this study echoes previous studies.

Incidence and prevalence

Children exposed to various traumatic events have much higher incidence (from 15 to 90+ %) and prevalence rates than the general population (Pfefferbaum. 1997). Furthermore, the younger a child is the more vulnerable they appear to be for the development of trauma-related symptoms. The percentage of children developing PTSD following a traumatic event is significantly higher than the percentage of adults developing PTSD following a similar traumatic stress. Several studies published in 1998 confirm previous reports of high prevalence rates for PTSD in child and adolescent populations. Thirty five percent of a sample of adolescents diagnosed with cancer met criteria for lifetime PTSD

(Pelcovitz, Kaplan, Goldenberg, Mandel, Lehane, & Guarrera. 1994); 15 % of children surviving cancer had moderate to severe PTSS (Stuber, Kazak, Meeske, et al. 1997); 93 % of a sample of children witnessing domestic violence had PTSD (Kilpatrick & Williams. 1998); over 80 % of the Kuwaiti children exposed to the violence of the Gulf Crisis had PTSS (Hadi & Llabre. 1998); 73 % of juvenile male rape victims develop PTSD (Ruchkin, Eisemann, & Hagglof. 1998); 34 % of a sample of children experiencing sexual or physical abuse and 58 % of children experiencing both physical and sexual abuse all met criteria for PTSD (Ackerman, Newton, McPherson, Jones, & Dykman. 1998). In all of these studies, clinically significant symptoms, though not full PTSD, were observed in essentially all of the children or adolescents following the traumatic experiences.

Vulnerability and resilience

Not all children exposed to traumatic events develop PTSD. A major research focus has been identifying factors (mediating factors) that are associated with increased (vulnerability) or decreased (resilience) risk for developing PTSD following exposure to traumatic stress (Kilpatrick & Williams. 1998). Factors previously demonstrated to be related to risk can be summarized in these broad categories: 1) characteristics of the child (e.g., subjective perception of threat to life or limb, history of previous traumatic exposures, coping style, general level of anxiety, gender, age); 2) characteristics of the event (e.g., nature of the event, direct physical harm, proximity to threat, pattern and duration); 3) characteristics of family/social system (e.g., supportive, calm, nurturing vs. chaotic, distant, absent, anxious) (Briggs & Joyce. 1997; Stuber, Kazak, Meeske, et al. 1997; Winje & Ulvik. 1998). Each of these mediating factors can be related to the degree to which they either prolong or attenuate the child's stress-response activation resulting from the traumatic experience. Factors that increase stress-related reactivity (e.g., family chaos) will make children more vulnerable while factors that provide structure, predictability, nurturing and sense of safety will decrease vulnerability. Persistently activated stress-response neurophysiology in the dependent, fearful child will predispose to a 'use-dependent' changes in the neural systems mediated the stress response, thereby resulting in post-traumatic stress symptoms (see Table 4).

Table 4. Post-traumatic Stress Disorder: Risk and Attenuating Factors

| | <i>Event</i> | <i>Individual</i> | <i>Family and Social</i> |
|---|--|--|---|
| Increase Risk <i>(Prolong the intensity or duration of the acute stress response)</i> | 5. Multiple or repeated event (e.g., domestic violence or physical abuse) 6. Physical injury to child 7. Involves physical injury or death to loved one, particularly mother 8. Dismembered or disfigured bodies seen 9. Destroys home, school or community 10. Disrupts community infrastructure (e.g., earthquake) 11. Perpetrator is family member 12. Long duration (e.g., flood) | 13. Female 14. Age (Younger more vulnerable) 15. Subjective perception of physical harm 16. History of previous exposure to trauma 17. No cultural or religious anchors 18. No shared experience with peers (experiential isolation) 19. Low IQ 20. Pre-existing neuropsychiatric disorder (especially anxiety related) | 21. Trauma directly impacts caregivers 22. Anxiety in primary caregivers 23. Continuing threat and disruption to family 24. Chaotic, overwhelmed family 25. Physical isolation 26. Distant caregiving 27. Absent caregivers |
| Decrease Risk <i>(Decrease intensity or duration of the acute stress response)</i> | 28. Single event 29. Perpetrator is stranger 30. No disruption of family or community structure 31. Short duration (e.g., tornado) | 32. Cognitively capable of understanding abstract concepts 33. Healthy coping skills 34. Educated about normative post-traumatic responses 35. Immediate post-traumatic interventions 36. Strong ties to cultural or religious belief system | 37. Intact, nurturing family supports 38. Non-traumatized caregivers 39. Caregivers educated about normative post-traumatic responses 40. Strong family beliefs 41. Mature and attuned parenting skills |

Long-term costs of childhood trauma

PTSD is a chronic disorder. Untreated, PTSS and PTSD remit at a very low rate. Indeed the residual emotional, behavioral, cognitive and social sequelae of childhood trauma persist and appear to contribute to a host of neuropsychiatric problems throughout life (Fergusson & Horwood. 1998) including attachment problems (Bell & Belicki. 1998; Alexander, Anderson, Brand, Schaeffer, Grelling, & Kretz. 1998), eating disorders (Rorty & Yager. 1996), depression (Winje & Ulvik. 1998; Fergusson & Horwood. 1998), suicidal behavior (Molnar, Shade, Kral, Booth, & Watters. 1998), anxiety (Fergusson & Horwood. 1998), alcoholism (Fergusson & Horwood. 1998; Epstein, Saunders, Kilpatrick, & Resnick. 1998), violent behavior (O'Keefe. 1995), mood disorders (Kaufman. 1991) and, of course, PTSD (Ford & Kidd. 1998; Schaaf & McCanne. 1998).

Childhood trauma impacts other aspects of physical health throughout life, as well (Hertzman & Wiens. 1996; Orr, Lasko, Metzger, Berry, Ahern, & Pitman. 1998; Felitti, Anda, Nordenberg, et al. 1998). Adults victimized by sexual abuse in childhood are more likely to have difficulty in childbirth, a variety of gastrointestinal and gynecological disorders and other somatic problems such as chronic pain, headaches and fatigue (Rhodes & Hutchinson. 1994). The Adverse Childhood Experiences study (Felitti, Anda, Nordenberg, et al. 1998) examined exposure to seven categories of adverse events during childhood (e.g., sexual abuse, physical abuse, witnessing domestic violence: events associated with increase risk for PTSD). This study found a graded relationship between the number of adverse events in childhood and the adult health and disease outcomes examined (e.g., heart disease, cancer, chronic lung disease, and various risk behaviors). With four or more adverse childhood events, the risk for various medical conditions increased 4- to 12-fold. Clearly studies of this sort will help clarify the true costs of childhood maltreatment.

Summary and Future Directions

The remarkable property of the human brain, unlike any other animal species, is that it has the capacity to take the accumulated experience of thousands of previous generations and absorb it within one lifetime. This capability is endowed by the design of our neural systems. Neurons and neural systems are designed to change in response to microenvironmental events. In turn, our experiences influence the pattern and nature of these microenvironmental signals, allowing neural systems to create a biological record of our lives. The brain, then, becomes an historical organ. In its organization and functioning are memorialized our accumulated, synthesized and transformed experiences. And there is no greater period of sensitivity to experience than when the brain is developing. Indeed, as described above, the neuroarcheological record of maltreatment has pervasive and chronic impact on the child. An event that lasts a few months in infancy can rob a child's potential for a lifetime. The true costs of childhood maltreatment will never be appreciated, and can never be avoided, until clinicians, researchers and policy makers become aware of the core concepts of neurodevelopment and the neuroarcheology of child maltreatment.

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