Social Engagement and Attachment

A Phylogenetic Perspective

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ABSTRACT: **This article focuses on the importance of social engagement and the behavioral and neurophysiological mechanisms that allow individuals to reduce psychological and physical distance. A model of social engagement derived from the Polyvagal Theory is presented. The model emphasizes phylogeny as an organizing principle and includes the following points: (1) there are well-defined neural circuits to support social engagement behaviors and the defensive strategies of fight, flight, and freeze; (2) these neural circuits form a phylogenetically organized hierarchy; (3) without being dependent on conscious awareness, the nervous system evaluates risk in the environment and regulates the expression of adaptive behavior to match the neuroception of a safe, dangerous, or life-threatening environment; (4) social engagement behaviors and the benefits of the physiological states associated with social support require a neuroception of safety; (5) social behaviors associated with nursing, reproduction, and the formation of strong pair bonds require immobilization without fear; and (6) immobilization without fear is mediated by a co-opting of the neural circuit regulating defensive freezing behaviors through the involvement of oxytocin, a neuropeptide in mammals involved in the formation of social bonds. The model provides a phylogenetic interpretation of the neural mechanisms mediating the behavioral and physiological features associated with stress and several psychiatric disorders.**

KEYWORDS: attachment; vagus; autonomic nervous system; oxytocin; evolution; Polyvagal Theory

A PHYLOGENETIC PERSPECTIVE

As the scientific knowledge of neuroanatomy and neurophysiology expands, there is a growing interest in the role neural processes play in the development of normal social behavior and in the expression of the atypical social behaviors that may provide the roots of mental illness in children. Recent advances in neuroscience have enabled researchers to study nervous system function and structure in the intact living individual. Now neuronal function can be studied, and the structural hypotheses derived from animal models and postmortem histology can be challenged and explained. These new methods of assaying neural structure and function, coupled

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with the breakthroughs in molecular genetics, are providing new tools and models, which can be integrated with existing strategies that effectively monitor dynamic neural function by time sampling neuroendocrine and autonomic parameters.

DEFINING SOCIAL BEHAVIOR: THE GREAT CONCEPTUAL DIVIDE

An objective of this conference is to build bridges among researchers who study the development of social behavior with both animal models and clinical populations. It was assumed, as a primary premise of organizing this conference, that both cohorts share the same objective of generating knowledge related to the mechanisms of normal and atypical social behavior that could be translated into clinical practice. The contrasts between the research strategies and methods of the two cohorts are forcing a reevaluation of this assumption.

Animal models often emphasize the role of a specific neural system, neurotransmitter, neuropeptide, hormone, or brain structure as a regulator of social behavior. In contrast, clinical research often focuses on studying aberrant psychological processes in clinical populations. When neurophysiological systems are studied with clinical populations, the research designs focus on establishing correlations with the disorders and, in general, preclude the possibility of distinguishing whether the physiological correlates are causes or effects of the disorder.

Although the two research strategies often use similar terms, the terms may reflect different domains of social behavior. Animal models tend to focus on the establishment of pair bonds and generate paradigms to evaluate the strength of these bonds. In contrast, research with children, investigating normal and atypical social behavior, tends to focus on the behaviors that reduce social and physical distance between individuals. For example, the terminology associated with measuring and defining social behavior differs when contrasting the compromised social engagement strategies expressed by an institutionalized child with the ability to establish pair bonds by a vole.

A final perplexing part of the conceptual divide relates to the translation of neuroscience principles and research findings into clinical practice. The clinician is the third limb of this triad. Paradoxically, although the link between social behavior and mental illness in children emerged directly from clinical observations, the features and dimensions of social behavior studied in both animal models and in laboratory studies of normal and atypical children often deviates from the features that clinicians use to define the pathology. Clinical researchers who conduct studies of social behavior are interested in either how outlier behaviors overlap with features of clinical diagnoses or how behavioral, psychological, and physiological parameters differentiate the clinical population from normal subjects. Often the parameters of interest or, at least, those that distinguish the clinical group from normal subjects focus on processes that do not have an obvious relation to the behaviors observed in clinical settings or used to define the pathology (e.g., Cortisol).

Most research in psychopathology accepts the validity of clinical assessment and diagnostic systems (e.g., DSM-IV) as inclusion criteria and then attempts to demonstrate that deficits in psychological processes and/or atypical neurophysiological response patterns underlie the disorder. The research on processes and mechanisms, whether obtained from clinical populations or by studying animal models assumed

to express behaviors similar to the clinical populations, does not easily enter the clinical realm and inform clinical assessment. Similarly, other than global diagnoses and quantitative information from standardized assessment instruments, little information from clinical observations regarding the specific features of behavior that have triggered the clinician's concern easily enters the research environment. Thus, the construct of social behavior is treated differently by researchers testing animal models, researchers studying normal social behavior, researchers studying the psychological and neurophysiological mechanisms and processes underlying a clinical diagnosis, and clinicians who diagnose and treat social behavior problems in children. Missing in this mix of metaphors, worldviews, paradigms, and diagnostic models, is a shared agenda to translate research findings into practice (i.e., assessment and treatment) and to use clinical information to inform the theoretical models being tested.

SOCIAL BEHAVIOR AND ATTACHMENT

Several researchers who study the development of social behavior in children have focused on the construct of attachment. Several of these researchers conduct studies derived from the observations of Bowlby[1] and the paradigm building research of Amsworth *et al*[2] Much of the current research on human attachment is based on the Ainsworth typology, which applies a paradigm assessing infant responses to separation. Clinicians and researchers in developmental psychopathology assume that the Ainsworth classification system and recent derivatives[3] will provide insights into the psychological mechanisms of specific disorders. In fact, diagnostic categories now include disorders such as "reactive attachment disorder" (RAD).

The traditional attachment schema derived from the Bowlby theory constitutes only a small part of social behavior. Moreover, traditional attachment theory by focusing on mother-infant relations does not include other putative attachment behaviors that are observed in the enduring bonds between peers, siblings, and mates. Missing from the traditional attachment theories is an articulation of the mechanisms mediating engagement between the individuals bonding or forming attachments.

SOCIAL ENGAGEMENT: THE PREAMBLE OF A SOCIAL BOND

To develop a social bond, individuals have to be in close proximity. This is true for the models focusing on both mother-infant attachment and the strong bonds associated with social monogamy. Both models test the strength and features of the relationship through separation paradigms. There are, of course, major differences between the contexts in which mother-infant attachment and the social bonds of reproductive partners are established and tested. One specific difference is the contrast in mobility between the mother-infant and reproductive partner dyads. In the mother-infant dyad there is an imbalance with the infant having limited abilities to move toward or away from the mother. However, in the reproductive partner dyad, there is a balance between the behavioral repertoires of the two adults.

Although proximity is critical to the establishment of social bonds, proximity is totally caused by the ability to navigate across physical distance via voluntary be-

havior. If social bonds were dependent on voluntary motor behaviors, then the newborn infant would be greatly disadvantaged because the neural regulation of the spinal motor pathways are immature at birth and take several years to fully develop. However, in mammals not all muscles are driven by corticospinal pathways. Unlike the striated muscles of trunk and limbs, corticobulbar pathways regulate the striated muscles of the face and head. The corticobulbar pathways are sufficiently developed at birth to be available to the full-term infant to signal caregiver (e.g., vocalizations, grimace) and to engage the social (e.g., gaze, smile) and nutrient (e.g., sucking) aspects of the world. These motor pathways originate in the brainstem and regulate muscles through the branches of five cranial nerves (V, VII, IX, X, XI). Thus, the neural regulation of muscles that provide important elements of social cueing are available to facilitate the social interaction with the caregiver and function collectively as an integrated social engagement system[4]

The muscles of the face and head influence both the expression and receptivity of social cues and can effectively reduce or increase social distance. Behaviorally this is observed as facial expressions, eye gaze, vocalizations, and head orientation. Neural regulation of these muscles can reduce social distance by making eye contact, expressing prosody in voice, displaying contingent facial expressions, and modulating the middle ear muscles to improve the extraction of human voice from background sounds. Alternatively, by reducing the muscle tone to these muscles, the eyelids droop, prosody is lost, positive and contingent facial expressions are diminished, the ability to extract human voice from background sounds is compromised, and the awareness of the social engagement behaviors of others may be lost. Thus, the neural regulation of the striated muscles of the face and head function both as an active social engagement system that reduces psychological distance and as a filter that can influence the perception of the engagement behaviors of others.

Special visceral efferent pathways mediate the neural regulation of the striated muscles of the face and head. Special visceral pathways emerge from three nuclei in the brainstem (nucleus of the trigeminal nerve, nucleus of the facial nerve, and nucleus ambiguus) and provide motor pathways that are contained within five cranial nerves (i.e., trigeminal, facial, hypoglossal, vagus, accessory). These pathways regulate structures that evolved from the ancient gill arches. From both clinical and research perspectives, the striated muscles of the face and head provide potent information regarding the behavioral dimensions used to express as well as to evaluate the strength of attachment or the stress to the social bond. For example, facial expressivity and prosody of vocalizations have been used as clinical indicators as well as quantifiable responses of separation distress.[5]

THE SOCIAL ENGAGEMENT SYSTEM: PHYLOGENIC ORIGINS OF BEHAVIORAL AND AUTONOMIC COMPONENTS

The phylogenic origin of the behaviors associated with the social engagement system is intertwined with the phylogeny of the autonomic nervous system. As the striated muscles, via special visceral efferent pathways, evolved into a behavioral system that regulated social engagement behaviors, there was a profound shift in neural regulation of the autonomic nervous system. Phylogenetically, these changes in both somatomotor and visceromotor regulation are observed in the transition from

reptiles to mammals. As the muscles of the face and head evolved into an ingestion (i.e., nursing) and social engagement system, a new component of the autonomic nervous system (i.e., a myelinated vagus) evolved that was regulated by a brainstem nucleus, which was also involved in the regulation of the striated muscles of the face and head (i.e., nucleus ambiguus). This convergence of neural mechanisms resulted in an integrated social engagement system with a synergism between behavioral and visceral features of social engagement. Thus, activation of the somatomotor component would trigger visceral changes that would support social engagement, while modulation of visceral state would either promote or impede social engagement behaviors. For example, stimulation of visceral states that would promote mobilization (i.e., fight or flight behaviors) would impede the ability to express social engagement behaviors, whereas increased activity through the myelinated vagus would promote the social engagement behaviors associated with a calm visceral state. Thus, we can infer the specific neural mechanisms related to the effectiveness that feeding and rocking have on promoting calm behavioral and visceral states. Specifically, both the ingestive behaviors associated with feeding and the passive rocking of an infant promote calmness by influencing the myelinated vagus. Feeding activates the muscles of mastication via trigeminal efferent pathways, which, in turn, provide afferent feedback input to the nucleus ambiguus (i.e., the source nucleus of the myelinated vagus). Rocking provides an efficient and direct influence on the vagus by stimulating vagal afferent pathways via the baroreceptors. Moreover, activation of the social engagement system dampens the neural circuits including the limbic structures that support fight, flight, or freeze behaviors.

POLYVAGAL THEORY: THREE NEURAL CIRCUITS REGULATING REACTIVITY

Evolutionary forces have molded both human physiology and behavior. Via evolutionary processes, the mammalian nervous system has emerged with specific neural and behavioral features that react to challenge to maintain visceral homeostasis. These reactions change physiological state and, in mammals, limit sensory awareness, motor behaviors, and cognitive activity. To survive, mammals must determine friend from foe, evaluate whether the environment is safe, and communicate with their social unit. These survival-related behaviors are associated with specific neurobehavioral states that limit the extent to which a mammal can be physically approached and whether the mammal can communicate or establish new coalitions.

Through stages of phytogeny, mammals and especially primates have evolved a functional neural organization that regulates visceral state to support social behavior. The Polyvagal Theory[4,6-8] emphasizes the phylogenetic origins of brain structures that regulate social and defensive behaviors, domains compromised in individuals with autism and several psychiatric disorders. The Polyvagal Theory proposes that the evolution of the mammalian autonomic nervous system provides the neurophysiological substrates for the emotional experiences and affective processes that are major components of social behavior. The theory proposes that physiological state limits the range of behavior and psychological experience. In this context, the evolution of the nervous system determines the range of emotional expression, quality of communication, and the ability to regulate bodily and behavioral state. The Poly-

vagal Theory links the evolution of the autonomic nervous system to affective experience, emotional expression, facial gestures, vocal communication, and contingent social behavior. Thus, the theory provides a plausible explanation of several social, emotional, and communication behaviors and disorders.

The polyvagal construct emphasizes the neurophysiological and neuroanatomical distinction between two branches of the vagus and proposes that each branch supports different adaptive behavioral strategies. The Polyvagal Theory articulates three phylogenetic stages of the development of the mammalian autonomic nervous system. Each state is associated with a distinct autonomic subsystem that is retained in mammals. These autonomic subsystems are phylogenetically ordered and behaviorally linked to social communication (e.g., facial expression, vocalization, listening), mobilization (e.g., fight-flight behaviors), and immobilization (e.g., feigning death, vasovagal syncope, and behavioral shutdown). The social communication system (i.e., Social Engagement System, see below) is dependent on the myelinated vagus, which serves to foster calm behavioral states by inhibiting the sympathetic influences to the heart and dampening the HPA axis.[9] The mobilization system is dependent on the functioning of the sympathetic nervous system. The most phylogenetically primitive component, the immobilization system, is dependent on the unmyelinated or "vegetative" vagus, which is shared with most vertebrates. With increased neural complexity due to phylogenetic development, the organism's behavioral and affective repertoire is enriched. The theory emphasizes the functional aspect of neural control of both the striated muscles of the face and the smooth muscles of the viscera, because their functions rely on common brainstem structures.

The Social Engagement System

The Polyvagal Theory provides an explicit neurobiological model of how difficulties in spontaneous social behavior are linked to both facial expressivity and the regulation of visceral state, and, alternatively, how social behavior may serve as a regulator of physiological activity. The theory proposes a possible mechanism to explain how these difficulties might form a core domain of several psychiatric profiles. Relevant to this focus on psychiatric disorders are the specific deficits associated with several diagnoses in both the somatomotor (e.g., poor gaze, low facial affect, lack of prosody, difficulties in mastication) and visceromotor (difficulties in autonomic regulation resulting in cardiopulmonary and digestive problems) of the social engagement system. For example, clinicians and researchers have documented these deficits in individuals with autistic spectrum disorders. Deficits in the social engagement system would compromise spontaneous social behavior and social awareness and affect expressivity, prosody, and language development. In contrast, interventions that improve the neural regulation of the social engagement system hypothetically would enhance spontaneous social behavior, state and affect regulation, reduce stereotypical behaviors, and improve language skills.

Embryologically, components of several cranial nerves known as special visceral efferent pathways develop together to form the neural substrate of a social engagement system.[8] This system, as illustrated in FIGURE 1, provides the neural structures involved in social and emotional behaviors. The social engagement system has a control component in the cortex (i.e., upper motor neurons) that regulates brainstem nuclei (i.e., lower motor neurons) to control eyelid opening (e.g., looking), facial

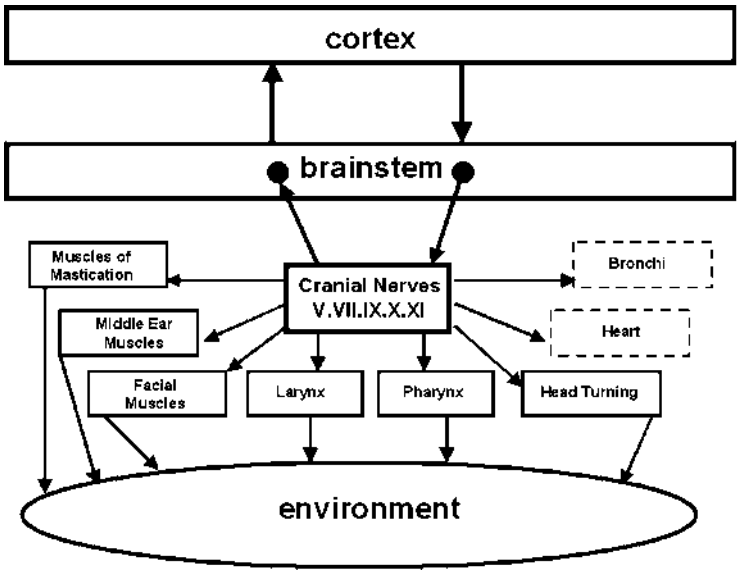

FIGURE 1. The Social Engagement System. Social communication is determined by the cortical regulation of medullary nuclei via corticobulbar pathways. The Social Engagement System consists of a somatomotor component (i.e., special visceral efferent pathways that regulate the muscles of the head and face) and a visceromotor component (i.e., the myelinated vagus that regulates the heart and bronchi). Solid blocks indicate the somatomotor component. *Dashed blocks* indicate the visceromotor component.

muscles (e.g., emotional expression), middle ear muscles (e.g., extracting human voice from background noise), muscle of mastication (e.g., ingestion), laryngeal and pharyngeal muscles (e.g., vocalization and language), and head-turning muscles (e.g., social gesture and orientation). Collectively, these muscles function as filters that limit social stimuli (e.g., observing facial features and listening to human voice) and determinants of engagement with the social environment. The neural control of these muscles determines social experiences. In addition, the source nuclei (i.e., lower motor neurons) of these nerves, which are located in the brainstem, communicate directly with an inhibitory neural system that slows heart rate, lowers blood pressure, and actively reduces arousal to promote calm states consistent with the metabolic demands of growth and restoration of our neurophysiological systems. Direct corticobulbar pathways reflect the influence of frontal areas of the cortex (i.e., upper motor neurons) on the regulation of this system. Moreover, afferent feedback through the vagus to medullary areas (e.g., nucleus of the solitary tract) influences forebrain areas that are assumed to be involved in several psychiatric disorders. In addition, the anatomical structures involved in the social engagement system have neurophysiological interactions with the HPA axis, the neuropeptides of oxytocin and vasopressin, and the immune system.[4]

The study of comparative anatomy, evolutionary biology, and embryology may provide important hints regarding the functional relation between the neural control of facial muscles and emergent psychological experiences and behavior. The nerves that control the muscles of the face and head share several common features. Pathways from five cranial nerves control the muscles of the face and head. Collectively, these pathways are labeled as special visceral efferent. Special visceral efferent nerves innervate striated muscles, which regulate the structures derived during embryology from the ancient gill arches.[10] The special visceral efferent pathways regulate the muscles of mastication (e.g., ingestion), muscles of the middle ear (e.g., listening to human voice), muscles of the face (e.g., emotional expression), muscles of larynx and pharynx (e.g., prosody and intonation), and muscles controlling head tilt and turning (e.g., gesture). In fact, the neural pathway that raises the eyelids also tenses the stapedius muscle in the middle ear, which facilitates hearing human voice. Thus, the neural mechanisms for making eye contact are shared with those needed to listen to human voice. As a cluster, the difficulties in gaze, extraction of human voice, facial expression, head gesture, and prosody are common features of individuals with autism.

Disorders of the Social Engagement System: Maladaptive or Adaptive Behavioral Strategies?

Individuals with several psychiatric and behavioral disorders have difficulties in establishing and maintaining relations. Several clinical diagnostic categories include features associated with difficulties both in expressing social behavior and in reading social cues (i.e., social awareness). These features are observed in individuals with a variety of primary psychiatric diagnoses including autism, social anxiety, posttraumatic stress disorder, and RAD.

Although a compromised social engagement system results in "maladaptive" social behavior, do these asocial behavioral strategies have "adaptive" features? The phylogeny of the vertebrate autonomic nervous system serves as a guide (i.e., the Polyvagal Theory) to understand these adaptive features. Phylogenetically, the vertebrate autonomic nervous system follows three general stages of development. Each stage supports a different category of behavior with only the phylogenetically most recent innovation (i.e., the myelinated vagus) supporting social engagement behaviors. Because the neural regulation of the myelinated vagus is integrated into the social engagement system, when the social engagement system is compromised the effects are both behavioral and autonomic. The resultant changes in autonomic state support a range of adaptive defensive behaviors. Specifically, the compromised social engagement system is associated, neurophysiologically, with a change in autonomic regulation characterized by a reduction in the influence of the myelinated vagus (i.e., ventral vagal complex including nucleus ambiguus) on the heart. The removal of the regulatory influence of the ventral vagal complex on the heart potentiates the expression of the two phylogenetically older neural systems (i.e., sympathetic nervous system, dorsal vagal complex including dorsal nucleus of the vagus). These two older neural systems foster mobilization behaviors of fight and flight via the sympathetic nervous system or immobilization behaviors of death feigning, freezing, and behavioral shut down via the dorsal vagal complex.

Neuroception: A Nervous System Evaluation of Risk

When individuals meet, what determines the biobehavioral sequence and consequence of their initial interactions? What contextual features and neural mechanisms trigger whether an individual expresses prosocial engagement or the specific defensive behaviors of fight, flight, or freeze? Regardless of the model of attachment or its dependence on cognitive, affective, behavioral, or biological constructs, the critical features that determine the valence of the interaction are related to perceived safety. Thus, the perception of safety is the turning point in the development of relationships for most mammals. The perception of safety determines whether the behavior will be prosocial (i.e., social engagement) or defensive. If the context and the other individual are perceived as safe, then the candidates for the social bond may inhibit the adaptive primitive neurobiological reactions of defense to allow the expression of social engagement. The three stages of the Polyvagal Theory articulate the neural systems that are available for social engagement and the defensive behaviors of fight, flight, and freeze. However, how are the adaptive neurobiological systems for defense functionally subdued to insure that attachment and the formation of social bonds will be the products of appropriate social engagement?

Before a social bond can occur, both individuals have to perceive each other as safe. What mediates the individual's ability to engage? Why would an infant look and coo at a caregiver, but gaze avert and cry as a stranger approached? Why would a gentle embrace be experienced as pleasurable when expressed by a lover and be experienced as assault when expressed by a stranger? Mammals have adaptive neurobehavioral systems for both defensive and social engagement behaviors. However, what enables engagement behaviors to occur, while disenabling the mechanisms of defense? The Polyvagal Theory with its focus on the phylogeny of the vertebrate autonomic nervous system provides a perspective to identify and to understand the plausible mechanisms that enable mammals to functionally switch between positive social engagement and defensive behavioral strategies. To effectively switch from defensive to social engagement strategies, the mammalian nervous system needs to perform two important processes: (1) to assess risk, and (2) if the environment is perceived as safe, to inhibit the more primitive limbic structures that control fight, flight, or freeze behaviors.

The nervous system, through the processing of sensory information from the environment, continuously evaluates risk. Because the neural evaluation of risk does not require conscious awareness, the term *neuroception* is introduced to emphasize the neural circuits that function as a safety-threat detection system capable of distinguishing among situations that are safe, dangerous, or life threatening. Because of the phylogenetic heritage of mammals, neuroception can operate without cognitive awareness via relatively primitive mechanisms that are dependent on subcortical structures (e.g., limbic). As a product of evolution, new neural systems evolved in mammals that involved cortical regulation of subcortical structures and, in many instances, co-opted the defense functions of the primitive structures to support other functions including those related to reproductive behavior and pair bonding.[8]

Based on the relative risk of the environment, both social engagement and defense behaviors may be interpreted as either adaptive or maladaptive. For example, the inhibition of defense systems by the social engagement system would be adaptive and appropriate only in a safe environment. From a clinical perspective, it would

be the inability to inhibit defense systems in safe environments (e.g., anxiety disorders, RAD) or the inability to activate defense systems in risk environments (e.g., Williams Syndrome) that might contribute to the defining features of psychopathology. Thus, an invalid neuroception of safety or danger might contribute to maladaptive physiological reactivity and the expression of the defensive behaviors associated with specific psychiatric disorders.

There is a common feature between the invalid neuroception that identifies risk when no risk is there and McEwen's concept of "allostatic load."[11] The physiological reaction to a valid risk, although metabolically costly, is adaptive. Thus, the increased metabolic activity necessary to support the mobilization behaviors of fight and flight are adaptive in the short term, but costly to the organism if maintained. The duration of the response is an important feature that distinguishes between adaptive and maladaptive reactions. The complex mammalian nervous system evolved with a great dependence on oxygen and, unlike the reptile, can survive only for short periods without oxygen. Thus, breath holding for mammals is adaptive only for short periods. In contrast, apnea is adaptive for reptiles, who because of their limited needs for oxygen can inhibit breathing for long periods, whereas apnea is potentially lethal for mammals.[12] Similarly, temporal features, in part, determine the construct of allostatic load. McEwen describes chronic stress or allostatic state as a physiological response that, although having adaptive functions in the short term, can be damaging if used for long periods when it is no longer needed (i.e., invalid neuroception). This cost of adaptation or "maladaptation" McEwen refers to as "allostatic load."

Safety Trumps Fear

In safe environments, autonomic state is adaptively regulated to dampen sympathetic activation and to protect the oxygen-dependent central nervous system from the metabolically conservative reactions of the dorsal vagal complex. However, how does the nervous system know when the environment is safe, dangerous, or life threatening and what neural mechanisms evaluate risk in the environment?

New technologies, such as functional magnetic resonance imaging, have identified specific neural structures that are involved in detecting risk. The temporal lobe is of particular interest in expanding the construct of neuroception and in identifying neural mechanisms that modulate the expression of adaptive defensive behaviors and autonomic states. Functional imaging techniques document that areas of the temporal cortex, fusiform gyrus (FG), and superior temporal sulcus (STS) are involved in detecting features such as movements, vocalizations, and faces, which contribute to an individual being perceived as safe or trustworthy.[13,14] Slight changes in these stimuli can pose threat or signal endearment. Connectivity between these areas of the temporal cortex and the amygdala suggests a top-down control in the processing of facial features that could actively inhibit activity of the structures involved in the expression of defensive strategies.[15]

Neuroanatomical and neurophysiological research with animals provides additional information regarding the modulation and inhibition of defensive behaviors via well-defined connections between the amygdala and the periacqueductal gray (PAG). The PAG is a heterogenous midbrain structure that consists of gray matter surrounding the cerebral aqueduct that connects the third and fourth ventricles. Studies have identified areas of the PAG that are organized to regulate flight, fight, or

freeze behaviors and the autonomic states that support these behaviors.[16] Stimulating rostrally within the lateral and dorsolateral PAG produces confrontational defensive behaviors (i.e., fight), while stimulating caudally within the lateral PAG and dorsolateral PAG produces escape behaviors (i.e., flight). Autonomic shifts such as increases in heart rate and blood pressure parallel these behaviors. In contrast, stimulation in the region of the PAG ventrolateral to the aqueduct (vlPAG) evokes a passive reaction of immobility, a decrease in blood pressure, and a slowing of heart rate. Interestingly, excitation of the vlPAG evokes an opioid-mediated analgesia that might adaptively raise pain thresholds. In addition, there is evidence of a functional connection between the central nucleus of the amygdala and the vlPAG that modulates both antmociception and immobilization.[17] Consistent with the Polyvagal Theory, the vlPAG communicates with dorsal vagal complex, whereas the 1PAG and dlPAG communicate with the sympathetic nervous system.

In the absence of threat, inhibitory projections from the FG and STS to the amygdala would be available to actively inhibit the limbic defense systems. This inhibition would provide an opportunity for social behavior to occur. Thus, the appearance of a friend or mate would subdue the limbic activation with the biobehavioral consequences of allowing proximity, physical contact, and other social engagement behaviors. In contrast, during situations in which the appraisal of risk is high, the amygdala and various areas of the PAG are activated. The amygdala and PAG only share connections through the central nucleus.[18]

The detection of safety subdues the adaptive defensive systems dependent on limbic structures. Thus, providing a plausible model of how a neural detection of environmental risk (i.e., neuroception) would modulate behavior and physiological state to support adaptive behaviors in response to safe, dangerous, and life-threatening environments. Conceptually, the process of detecting safety is inclusive of the detection of risk. Thus, the neural circuits that mediate the more primitive defense systems have through the processes of evolution been co-opted to support the social behavior necessary for mammalian survival. These behaviors include social engagement and the behaviors associated with social bonding (e.g., reproductive behaviors and nursing).

Co-opting the Immobilization Defense System for Reproductive Behaviors, Nursing, and the Formation of Social Bonds

Immobilization as a defense system is phylogenetically old and is associated with reduced metabolic demands and increased pain threshold. In reptiles, because of their limited need for oxygen, immobilization is a very effective defense strategy. In contrast, because mammals have a great need for oxygen, the inhibition of movement coupled with a shift in autonomic state to support the immobilization behavior (i.e., apnea and bradycardia) can be lethal. [19]>[20] However, several aspects of mammalian social behavior require immobilization, but immobilization without fear. Immobilization without fear is accomplished by co-opting the structures that regulate immobilization and pain thresholds to serve a broad range of social needs including reproduction, nursing, and pair-bonding. By focusing on the area of the PAG that coordinates freezing behavior, we can see how a primitive immobilization defense system has been modified through evolution to serve the intimate social needs of mammals. In addition, when we study the vlPAG we find that it is rich in receptors

for oxytocin, a neuropeptide associated with partuition, nursing, and the establishment of pair bonds.[21,22]

Overlapping with the area of the PAG that organizes immobility (i.e., vlPAG) are areas that when stimulated produce lordosis and kyphosis. The lordosis reflex is a hormone-dependent behavior displayed by female rodents and other mammalian species during mating. In most mammals, lordosis involves the female immobilizing in a crouching posture with her hind end available to the male for copulation. Neural tracing studies have demonstrated that the vlPAG is part of the neural circuit involved in regulating lordosis.[23] Kyphosis is an upright crouching posture that is accompanied by inhibition of limb movements. This posture is stimulated by nipple attachment and provides an opportunity for the dam to feed simultaneously a large litter. When dams initiate a nursing bout, behavioral state shifts immediately from high activity to immobility[24] When the caudal portion of the vlPAG is lesioned there are important consequences: (1) kyphotic nursing decreases, (2) litter weight gains decrease, and (3) the lesioned rats are more aggressive and more frequently attack strange males.[25]

Test of the Model

The processes of attachment and the formation of social bonds require appropriate social engagement strategies. In the preceding sections, elements of a preliminary model that links social engagement to attachment and the formation of social bonds are presented. The model is expanded from the Polyvagal Theory and emphasizes the following points: (1) there are well-defined neural circuits to support social engagement behaviors and the defensive strategies of fight, flight, and freeze; (2) without being dependent on conscious awareness the nervous system evaluates risk in the environment and regulates the expression of adaptive behavior to match the neuroception of a safe, dangerous, or life-threatening environment; (3) social engagement behaviors and the benefits of the physiological states associated with social support require a neuroception of safety; (4) social behaviors associated with nursing, reproduction, and the formation of strong pair bonds require immobilization without fear; and (5) immobilization without fear is mediated by a co-opting of the neural circuit regulating defensive freezing behaviors through the involvement of oxytocin, a neuropeptide involved in the formation of social bonds.[26,27]

FIGURES 2, 3, and 4 illustrate the role that neuroception plays in determining the neural circuits recruited to regulate social engagement, fight, flight, and freeze behaviors. Each figure illustrates a different environment context (i.e., safe, dangerous, life threat). FIGURE 2 illustrates the assumed neural circuits involved in promoting social engagement behaviors in a safe context. The detection of safe or trustworthy features derived from face, voice, and movement activate a neural circuit that projects from the temporal cortex (i.e., fusiform gyrus, superior temporal sulcus) to the central nucleus of the amygdala to inhibit defensive limbic functions (see FIGS. 3 and 4). This circuit disenables the limbic defense systems that organize and regulate fight, flight, and freeze behaviors and enables the corticobulbar pathways that regulate the social engagement behaviors (see FIG. 1). FIGURE 3 illustrates the neural circuits involved in a response to a neuroception of danger. In response to danger, the limbic defense circuits function to adaptively protect the individual. The specificity of the defense strategy, whether confrontational or avoidant (i.e., fight or

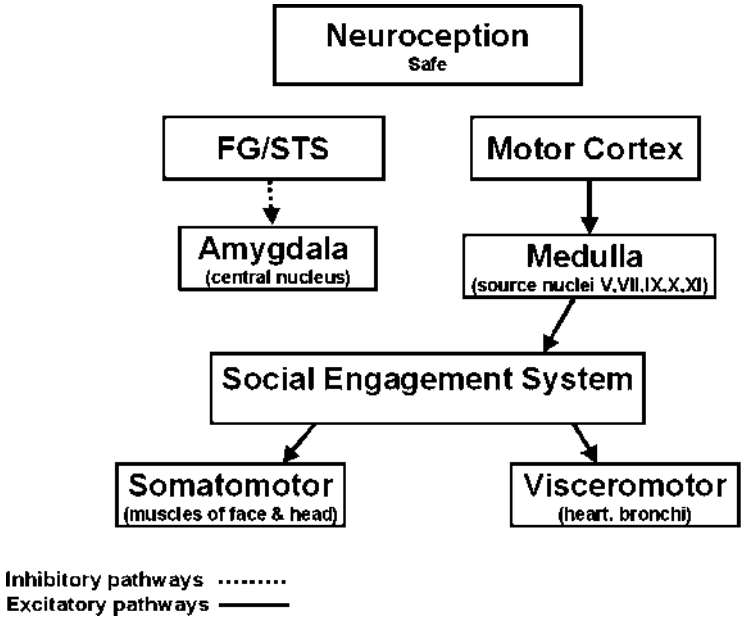

FIGURE 2. Neural structures and pathways involved in a neuroception of safety.

flight), is regulated by the PAG. To support these mobilization behaviors, the sympathetic nervous system is activated and dominates autonomic state. FIGURE 4 illustrates the neural circuits involved in response to life threat. In response to life threat, the mammalian nervous system promotes immobilization or freezing behavior. Freezing, as a defense strategy, is coordinated by the PAG. To inhibit metabolic activity during immobilization, autonomic state is under the control of the dorsal vagal complex. As proposed by the Polyvagal Theory, the autonomic reactions during each adaptive behavioral strategy is hierarchically organized after the phylogeny of both the changes in the vertebrate autonomic nervous system and changes in the behavioral repertoire from immobilization to mobilization to social engagement.

The ability to evaluate whether the environment is safe or if a person is trustworthy is difficult for individuals with a variety of psychiatric diagnoses. Current research suggests that the areas in the temporal cortex (i.e., FG, STS), which are assumed to inhibit limbic defense reactions, are not activated in clinical populations that have difficulties with social engagement behaviors (e.g., autism, schizophrenia). Moreover, individuals with other psychiatric disorders such as anxiety disorders and depression, which have as diagnostic features compromised social behavior, have difficulties in regulating visceral state (e.g., lower vagal regulation of the heart) and supporting social engagement behaviors (e.g., reduced facial expressiveness and motor control of the striated muscles of the face and head). Thus, from a theoretical perspective, a potential root of several psychiatric disorders might be linked to an in-

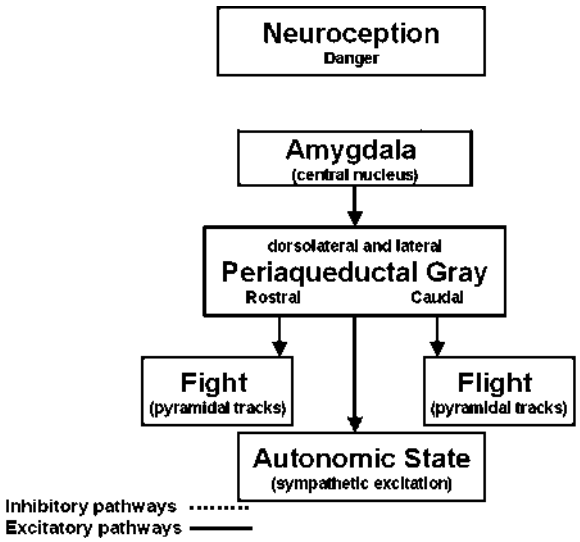

FIGURE 3. Neural structures and pathways involved in a neuroception of danger.

ability to detect safety in the environment and trustworthiness from interactions and, thus, the inability to express appropriate social engagement behaviors.

The study of attachment disorders such as RAD provides an intriguing test of the critical role of neuroception in mediating appropriate attachment and social behavior. RAD is described in both the DSM-IV (American Psychiatric Association, 1994) and the ICD-10 (World Health Organization, 1992) psychiatric diagnostic manuals. RAD comprises two clinical patterns (i.e., inhibited and uninhibited subtypes). The inhibited subtype is characterized by an emotionally withdrawn, unresponsive pattern in which there is an absence of attachment behaviors. The disinhibited subtype is characterized by indiscriminate attachment, which often is directed at strangers. These patterns have been described in institutionalized and maltreated children.[28] From a neuroception perspective, in both subtypes, the evaluation of the risk in the environment is not accurate.

Recent research on the outcomes of children raised in institutions in Romania has stimulated interest in RAD and in developing intervention strategies to remediate these devastating disturbances in social development. If an accurate neuroception of the environment is necessary for normal social behavior, then what features in the environment might potentiate normal social development? A recent study of Romanian toddlers[29] provides insight into the process. In this study, indices of RAD were evaluated in children as a function of the number of different caregivers. Two groups of institutionalized children were evaluated and contrasted with children who were never institutionalized. One group consisted of the standard institution unit in which 20 different caregivers worked rotating shifts with approximately 3 caregivers for 30 children on each shift. A second group consisted of a pilot unit in which the number of children were reduced to approximately 10, and the pool of caregivers was re-

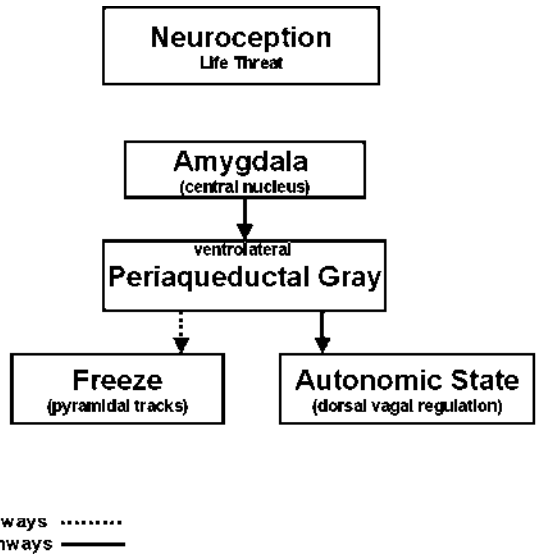

FIGURE 4. Neural structures and pathways involved in a neuroception of life threat.

duced to 4. If neuroception of safety is necessary to promote appropriate social behavior, then familiarity of caregiver would be critical. By having familiar caregivers, the child's detection of the caregiver's face, voice, and movements (the features that define a safe and trustworthy person) should trigger the inhibitory pathways to disenable the limbic defense system and foster the behaviors expressed by the social engagement system. In support of this model, the study demonstrated a monotonic relation between the number of different caregivers that a child had contact with and the indices of RAD. On all measures, the standard unit children were more likely to have higher indices of RAD, and on some measures the pilot group did not differ from the children who were never institutionalized. Thus, once we understand the contextual and social features that inhibit the neural circuits that mediate defensive behavioral strategies, we can optimize the developmental consequences of the neural circuits that promotes social engagement behaviors.

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