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The Role of Social Engagement in Attachment and Bonding

A Phylogenetic Perspective

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ABSTRACT

This chapter focuses on the importance of social engagement as a mechanism that fosters the establishment of social attachment and social bonds. 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 (i.e., neuroception) and regulates visceral state to support the expression of adaptive behavior to match a neuroception of safety, danger, or life threat, (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 a unique biobehavioral state characterized by 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 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.

DEFINING SOCIAL BEHAVIOR: THE GREAT CONCEPTUAL DIVIDE

An objective of this workshop was to build bridges among researchers who study the development of attachment and bonding from both animal model and clinical population perspectives. As a primary premise of organizing this workshop, it was assumed that both cohorts share similar constructs and interests in generating knowledge related to the mechanisms of normal and atypical social behavior into clinical practice. The contrasts between the research strategies and methods of the two cohorts are forcing a re-evaluation 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 neurophy siological systems are studied with clinical populations, research designs focus on establishing correlations with the disorders and, in general, preclude the possibility of distinguishing whether 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 on human attachment is conducted primarily with y oung children and investigates normal and atypical reactions to strangers. Moreover, 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, heart rate variability).

Most research in psy chopathology accepts the validity of clinical assessment and diagnostic systems (e.g., DSM-IV) as inclusion criteria and then attempts to demonstrate that deficits in psy chological 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, constructs of social behavior and engagement are treated differently by researchers testing animal models, researchers study ing normal social behavior, researchers study ing the psychological and neurophysiological mechanisms and processes underlying a clinical diagnosis, and clinicians who diagnose and treat children with social behavior problems. 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 (1982) and the paradigmbuilding research of Ainsworth (1978). 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 (Cassidy and Shaver 1999) 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 dy ad there is an imbalance with the infant having limited abilities to move toward or away from the mother. However, in the reproductive partner dy ad, 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 due to the ability to navigate across physical distance via voluntary behavior. If social bonds were dependent upon 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 controlling the trunk and limbs, in primates and especially humans, corticobulbar pathways regulate the striated muscles of the face and head. In humans, myelination of corticobulbar pathways begins between 24 and 28 weeks gestation (Sarnat 2003) and is sufficiently developed at birth to be available to the full-term infant to signal a caregiver (e.g., vocalizations, grimace) and to engage the social (e.g., gaze, smile) and nutrient (e.g., sucking) aspects of the world. 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 (Porges 2001).

The muscles of the face and head influence both the expression and receptivity of social cues and can effectively reduce or increase social distance. 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 efferent 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 (New man 1988).