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NEUROCEPTION:

A Subconscious System for Detecting Threats and Safety

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What determines how two human beings will act toward each other when they meet? Is this initial response a product of learning from culture, family experiences, and other socialization processes? Or is the response the expression of a neurobiological process that is programmed into the very DNA of our species? If the response has a neurobiological basis, are there specific features of the other person's behavior that trigger either feelings of safety, love, and comfort or feelings of danger? Why do some children cuddle and warmly conform to embraces, yet others stiffen and pull back from the same overture? Why do some children smile and actively engage a new person, while others avert their gaze and withdraw?

Does knowledge of human biology help us to understand the triggers and mechanisms of these behaviors during normal development? If we learn how behavioral features trigger neural circuits that facilitate social behavior, will we be better able to help children with severe developmental disabilities, such as autism, improve their social behavior?

By processing information from the environment through the senses, the nervous system continually evaluates risk. I have coined the term *neuroception* to describe how neural circuits distinguish whether situations or people are safe, dangerous, or life threatening. Because of our heritage as a species, neuroception takes place in primitive parts of the brain, without our conscious awareness. The detection of a person as safe or dangerous triggers neurobiologically

at a glance

- *Neuroception* describes how neural circuits distinguish whether situations or people are safe, dangerous, or life threatening.
- Neuroception explains why a baby coos at a caregiver but cries at a stranger, or why a toddler enjoys a parent's embrace but views a hug from a stranger as an assault.
- The Polyvagal Theory describes three developmental stages of a mammal's autonomic nervous system: Immobilization, mobilization, and social communication or social engagement.
- Faulty neuroception might lie at the root of several psychiatric disorders, including autism, schizophrenia, anxiety disorders, depression, and Reactive Attachment Disorder.

determined prosocial or defensive behaviors. Even though we may not be aware of danger on a cognitive level, on a neurophysiological level, our body has already started a sequence of neural processes that would facilitate adaptive defense behaviors such as fight, flight, or freeze.

A child's (or an adult's) nervous system may detect danger or a threat to life when the child enters a new environment or meets a strange person. Cognitively, there is no reason for them to be frightened. But often, even if they understand this, their body betrays them. Sometimes this betrayal is private; only they are aware that their hearts are beating fast and contracting with such force that they start to sway. For others, the responses are more overt. They may tremble. Their faces may flush, or perspiration may pour from their hands and forehead. Still others may become pale and dizzy, and feel precipitously faint.

Humans have three principal defense strategies—fight, flight, and freeze.

This process of neuroception would explain why a baby coos at a familiar caregiver but cries at the approach of a stranger, or why a toddler enjoys a parent's gentle embrace but interprets the same gesture from a stranger as an assault. We can see the process at work when two toddlers encounter each other in a playground sandbox. They may decide that the situation and each other are safe if the sandbox is familiar territory, if their pails and shovels have roughly similar appeal, and if they (the toddlers) are about the same size. The toddlers may then express positive social engagement behaviors—in other words, they may start to play.

"Playing nice" comes naturally when our neuroception detects safety and promotes physiological states that support social behavior. However, prosocial behavior will not occur when our neuroception misreads the environmental cues and triggers physiological states that support defensive strategies. After all, "playing nice" is not appropriate or adaptive behavior in dangerous or life-threatening situations. In these situations, humans—like other mammals—react with more primitive neurobiological defense systems. To create relationships, humans must subdue these defensive reactions to engage, attach, and form lasting social bonds. Humans have adaptive neurobehavioral systems for both prosocial and defensive behaviors.

What allows engagement behaviors to occur, while disenabling the mechanisms of defense? To switch effectively from defensive to social engagement strategies, the nervous system must do two things: (1) Assess risk, and (2) if the environment looks safe, inhibit the primitive defensive reactions to fight, flee, or freeze.

By processing information from the environment through the senses, the nervous system continually evaluates risk. As evolution has proceeded, new neural systems have developed. These systems use some of the same brain structures that are involved in defense functions to support forms of social engagement. Neuroception now may encourage the development of social bonds and provide the opportunity for reproduction.

Social Engagement and Defensive Behavior: Adaptive or Maladaptive Strategies?

Social engagement and defense behaviors may be adaptive or maladaptive, depending on the level of risk that is present in the environment. From a clinical perspective, the defining features of psychopathology may include either a person's inability to *inhibit* defense systems in a safe environment or the inability to *activate* defense systems in a risky environment—or both. Only in a safe environment is it adaptive and appropriate to simultaneously inhibit defense systems and exhibit positive social engagement behavior. Faulty neuroception—that is, an inaccurate assessment of the safety or danger of a situation—might contribute to the maladaptive physiological reactivity and the expression of defensive behaviors associated with specific psychiatric disorders. In typically developing children, however, neuroception detects risk accurately. Children's cognitive awareness of risk matches their "gut response" to danger.

When our nervous system detects safety, our metabolic demands adjust. Stress responses that are associated with fight and flight, such as increases in heart rate and cortisol mediated by the sympathetic nervous system and hypothalamic-pituitary-adrenal axis, are dampened. Similarly, a neuroception of safety keeps us from entering physiological states that are characterized by massive drops in blood pressure and heart rate, fainting, and apnea—states that would support "freezing" and "shutdown" behaviors.

How does the nervous system know when the environment is safe, dangerous, or life threatening? 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. Specific areas of the brain detect and evaluate features, such as body and face movements and vocalizations that contribute to an impression of safety or trustworthiness. Researchers have identified an area in the cortex that becomes activated when we see familiar faces and hear familiar voices. This process of identifying familiar and trustworthy people and evaluating the intentions of others based on "biological movements" of face and limbs seems to be located in the temporal lobe of the cortex. If neuroception identifies a person as safe, then a neural circuit actively inhibits areas of the brain that organize the defensive strategies of fight, flight, and freeze. Slight changes in the biological movements that we see can shift a neuroception from "safe" to "dangerous." When this shift occurs, the neural systems associated with prosocial behav-

ior are disrupted and the neural systems associated with defensive strategies are triggered.

In the presence of a safe person, then, the active inhibition of the brain areas that control defense strategies provides an opportunity for social behavior to occur spontaneously. Thus, the appearance of a friend or caregiver would subdue the neural circuits in the brain that regulate defensive strategies. And as a consequence, closeness, physical contact, and other social engagement behaviors become possible. In contrast, when situations appear risky, the brain circuits that regulate defense strategies are activated. Social approaches are met with aggressive behavior or withdrawal.

Immobilization Without Fear

As we have seen, humans have three principal defense strategies—fight, flight, and freeze. We are familiar with fight and flight behaviors, but know less about the defense strategy of immobilization, or freezing. This strategy, shared with early vertebrates, is often expressed in mammals as "death feigning." In humans, we observe a behavioral shutdown, frequently accompanied by very weak muscle tone. We also observe physiological changes: Heart rate and breathing slow, and blood pressure drops.

Immobilization, or freezing, is one of our species' most ancient mechanisms of defense. Inhibiting movement slows our metabolism (reducing our need for food) and raises our pain threshold. But in addition to freezing defensively, mammals immobilize themselves for essential prosocial activities, including conception, childbirth, nursing, and the establishment of social bonds. For example, when an infant nurses, the mother has to restrain her movements. When a child is embraced, the child is functionally immobilized. Reproductive behaviors also involve a degree of immobilization. However, immobilization with fear elicits profound, potentially lethal, physiological changes (i.e., dramatic slowing of heart rate, cessation of breathing, and dropping of blood pressure). Through the process of evolution, neural circuits in the brain that were originally involved in freezing behaviors were modified to serve intimate social needs. Over time, these brain structures grew receptors for this neuropeptide. Oxytocin is released during the birth process and nursing. It is also released in the brain during activities that help establish social bonds. Thus, when we sense that our environment is safe, the release of oxytocin allows us to enjoy the comfort of an embrace without fear. But if our nervous system identifies someone as dangerous, no oxytocin is released and we struggle against the attempted embrace.

Social Engagement: The Preamble to a Social Bond

To develop a social bond, it it not enough to inhibit defense systems. People must also be physically close to each other. This is true whether they are a mother and baby forming an attachment relationship or two adults forming a social bond. There are, of course, major differences between the contexts in which mother–infant attachment and the social bonds of reproductive partners are established. Consider mobility, for example. Due to immature neural development, the baby has limited ability to move either toward or away from the mother. In contrast, two adults who may become reproductive partners are likely to have similar behavioral repertoires.

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If the creation of social bonds depended on voluntary motor behaviors, then the human newborn would be greatly disadvantaged: The neural regulation of the spinal motor pathways is immature at the time of birth and takes several years to develop fully. Fortunately, social engagement does *not* depend on how well we can regulate our limbs and move our bodies. Voluntary limb and trunk movement require neural pathways linking the cortex to spinal nerves (i.e., corticobulbar pathways). Social engagement depends, rather, on how well we can regulate the muscles of our faces and heads via pathways linking the cortex with the brain stem (i.e., corticospinal pathways). These are the muscles that give expression to our faces, allow us to gesture with our heads, put intonation into our voices, direct our gaze, and permit us to distinguish human voices from background sounds. Corticospinal pathways to spinal nerves regulate the muscles that control the trunk and limbs; corticobulbar pathways to cranial nerves regulate the muscles of the face and head. The neural pathways from the cortex to these nerves (i.e., corticobulbar) are myelinated sufficiently at birth to

allow the infant to signal a caregiver (by vocalizing or grimacing, for example) and to engage the social and nutrient aspects of the world (by gazing, smiling, and sucking, for example).

The neural regulation of the muscles of the face and head influences how someone perceives the engagement behaviors of others. More specifically, this neural regulation can reduce social distance by allowing humans (including infants) to:

- Make eye contact;
- Vocalize with an appealing inflection and rhythm;
- Display contingent facial expressions; and
- Modulate the middle-ear muscles to distinguish the human voice from background sounds more efficiently.

The detection of a person as safe or dangerous triggers neurobiologically determined prosocial or defensive behaviors.

Alternatively, when the tone of these muscles is reduced, which occurs spontaneously in response to a neuroception of danger or a life threat in the external environment (e.g., a dangerous person or situation) or the internal environment (e.g., fever, pain, or physical illness) environment:

- The eyelids droop;
- The voice loses inflection;
- Positive facial expressions dwindle;
- Awareness of the sound of the human voice becomes less acute; and
- Sensitivity to others' social engagement behaviors decreases.

It is important to remember that neuroreception of danger or a threat to life can occur with respect to the external environment (e.g., a dangerous person or situation) or the internal environment (e.g., fever, pain, or physical illness). Even flat (rather than angry) facial affect might prompt a neuroception of danger or fear and disrupt the development of normal spontaneous interactive and reciprocal social engagements. For example, the flat affect of a depressed parent or the flat affect of an ill child might trigger a transactional spiral that results in compromised emotional regulation and limited spontaneous social engagement.

Polyvagal Theory: Three Neural Circuits That Regulate Reactivity

Where do humans' intricate neurobehavioral systems for prosocial and defensive behaviors come from? As we have suggested earlier, mammals—including humans—must distinguish friend from foe, evaluate the safety of the environment, and communicate with their social unit. According to the Polyvagal Theory (see Porges, 1993, 1995, 1997, 1998, 2001), mammals—especially primates—have evolved brain structures that regulate both social and defensive behaviors. In other words, evolutionary forces have molded both human physiology and human behavior. As the vertebrate nervous system became more complex during the course of evolution, its affective and behavioral repertoire expanded. A product of this phylogenetic process is a nervous system that provides humans with the ability to express emotions, communicate, and regulate bodily and behavioral states.

The Polyvagal Theory links the evolution of the neural regulation of the heart to affective experience, emotional expression, facial gestures, vocal communication, and social behavior that is responsive to the behavior of others. The theory points out that the neural control of the heart is neuroanatomically linked to the neural control the muscles of the face and head.

The Polyvagal Theory describes three stages in the development of a mammal's autonomic nervous system. Each of the three major adaptive behavioral strategies is supported by a distinct neural circuit involving the autonomic nervous system:

1. **Immobilization**
   - Feigning death, behavioral shutdown.
   - The most primitive component, shared with most vertebrates.
   - Dependent on the oldest branch of the vagus nerve (an unmyelinated portion originating in an area of the brain stem known as the dorsal motor nucleus of the vagus).

2. **Mobilization**
   - Fight–flight behaviors.
   - Dependent on the functioning of the sympathetic nervous system, a system associated with increasing metabolic activity and increasing cardiac output (e.g., faster heart rate, greater ability of the heart to contract).

3. **Social communication or social engagement**
   - Facial expression, vocalization, listening.
   - Dependent on the myelinated vagus, which originates in an area of the brain stem known as the nucleus ambiguus. The myelinated vagus fosters calm behavioral states by inhibiting the influence of the sympathetic nervous system on the heart.

Infants, young children, and adults need appropriate social engagement strategies in order to form positive attachments and social bonds. At the University of Illinois at Chicago, we have been developing a model that links

social engagement to attachment and the formation of social bonds through the following steps:

1. Three well-defined neural circuits support social engagement behaviors, mobilization, and immobilization.
2. Independent of conscious awareness, the nervous system evaluates risk in the environment and regulates the expression of adaptive behavior to match the neuroception of an environment that is safe, dangerous, or life threatening.
3. A neuroception of safety is necessary before social engagement behaviors can occur. These behaviors are accompanied by the benefits of the physiological states associated with social support.
4. Social behaviors associated with nursing, reproduction, and the formation of strong pair bonds requires immobilization without fear.
5. Oxytocin, a neuropeptide involved in the formation of social bonds, makes immobilization without fear possible by blocking defensive freezing behaviors.

Neuroception and Mental Health Disorders

So far, we have been discussing neuroception that works. Ideally, a baby's neuroception of her environment shows her a safe place to explore. But even if her neuroception warns her—accurately—of danger from a "frightened or frightening" caregiver, the baby can take some defensive measures, even though they are likely to be ineffective and are almost certain to be psychologically costly. What happens when neuroception itself is impaired? From a theoretical perspective, faulty neuroception—that is, an inability to detect accurately whether the environment is safe or another person is trustworthy—might lie at the root of several psychiatric disorders:

- Areas in the temporal cortex that are assumed to inhibit fight, flight, or freeze reactions are not activated in people with autism or schizophrenia, who have difficulty with social engagement.
- Individuals with anxiety disorders and depression have compromised social behavior; difficulties in regulating the heart rate, as reflected in measures of vagal control of the heart; and reduced facial expressiveness.
- Maltreated and institutionalized children with Reactive Attachment Disorder tend to be either inhibited (emotionally withdrawn and unresponsive) or uninhibited (indiscriminate in their attachment behavior; Zeanah, 2000). Both types of behavior suggest faulty neuroception of the risk in the environment.

Recent research on children raised in Romanian orphanages has stimulated interest in Reactive Attachment Disorders and in finding ways to remediate the devastating disturbances in their social development. If the behavior of these children suggests faulty neuroception of risk in the environment, are there features in the environment that might help the children feel safer and then begin to move toward more normal social behavior?

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A recent study of Romanian toddlers being raised in an orphanage (Smyke, Dumitrescu, & Zeanah, 2002) illustrates the usefulness of the construct of neuroception in understanding the development of normal and atypical attachment behaviors. Researchers evaluated two groups of institutionalized children and compared them to children who had never been institutionalized. One group of institutionalized children (the standard unit) was cared for according to prevailing standards: Twenty different caregivers worked rotating shifts, with approximately 3 caregivers for 30 children on each shift. A second group of children, the pilot unit, consisted of 10 children with 4 caregivers. If we apply our concept of neuroception to this study, we would hypothesize that familiar caregivers would be essential to children's neuroception of safety—which, in turn, would be essential for the promotion of appropriate social behavior. Specifically, a child's ability to recognize a caregiver's face, voice, and movements (the features that define a safe and trustworthy person) should set in motion the process of subduing the limbic system and allowing the social engagement system to function.

The data from the Smyke et al. (2002) study supports our hypothesis. The higher the number of caregivers children had contact with, the higher the incidence of Reactive Attachment Disorder among these children. The

standard-unit children were more likely than the other two groups to have Reactive Attachment Disorder. On some indices of Reactive Attachment Disorder, the pilot-group children did not differ from the children who had never been institutionalized. These findings suggest that once we understand the contextual and social features that inhibit the neural circuits which mediate defensive behavioral strategies, we can "optimize" the development of prosocial behavior.

At the University of Illinois at Chicago, we are using a newly developed biologically based behavioral intervention based on principles derived from the Polyvagal Theory. We are testing this approach with children with autism and individuals with language and social communication problems. Our model assumes that for many children with social communication deficits, including those diagnosed with autism, the social engagement system is neuroanatomically and neurophysiologically intact. Yet these children do not engage in voluntary prosocial behaviors. To improve spontaneous social behavior, we have reasoned, an intervention must stimulate the neural circuits that regulate the muscles of the face and head. The Polyvagal Theory predicts that once the cortical regulation of the brain-stem structures involved in the social engagement are activated, social behavior and communication will spontaneously occur as the natural emergent properties of this biological system. The intervention "stimulates" and "exercises" the neural pathways involved in listening and simultaneously stimulates the function of other aspects of the social engagement system. The intervention provides acoustic stimulation that has been computer altered to systematically modulate the neural regulation of the middle-ear muscles. Theoretically, the middle-ear muscles need to be regulated during listening, and the nerves that regulate these muscles are linked to the nerves that regulate the other muscles of the face and head involved in social engagement. Preliminary results are promising. They suggest that interventions designed to improve spontaneous social behavior should: (1) ensure that the context elicits in participants a neuroception of safety that will allow the social engagement system to function; and (2) exercise the neural regulation of the social engagement system.

Conclusions

According to the Polyvagal Theory (including the concept of neuroception), our range of social behavior is limited by our human physiology, which has evolved from that of more primitive vertebrates. When we are frightened, we are dependent upon the neural circuits that evolved to provide adaptive defensive behaviors for more primitive vertebrates. These neural circuits provide physiological mechanisms that reflexively organize mobilization or immobilization behaviors before we are consciously aware of what is happening. When, on the other hand, neuroception tells us that an environment is safe and that the people in this environment are trustworthy, our mechanisms of defense are disenabled. We can then behave in ways that encourage social engagement and positive attachment.

Focusing on biologically based behaviors common to all humans allows practitioners to imagine new intervention paradigms to help children whose social behavior and attachment are compromised. We can alter the caregiving environment so that it will appear—and be—safer for children and less likely to evoke mobilization or immobilization responses. We can also intervene directly with children, exercising the neural regulation of brain stem structures, stimulating the neural regulation of the social engagement system, and encouraging positive social behavior.

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