A New Model for Understanding and Treating Borderline Personality Disorder, Revised Edition 2023. Heron Creek Press, NY
Lee Crandall Park, M.D. and Thomas J. Park, Ph.D.

Extended Abstract (12-20-2024)

There is strong evidence that **genetic factors** underlie or contribute to the development of severe mental disorders. **Borderline Personality Disorder (BPD)** is such a severe mental disorder, characterized by major dysfunctions of **Theory of Mind (ToM)**. It is currently understood to be a **Diathesis-Stress Disorder**, meaning that inherent genetic defects, limitations or predispositions engage with adverse childhood experiences to cause the disorder. We will argue, however, that BPD is fully developmentally caused, with the genetic contribution being a natural "**Risk Factor"** that is, very paradoxically, not a flaw or a cause but instead is an inherent enhancement of the uniquely human Social Intelligence faculty for developing a Mind (ToM).

How could this seemingly preposterous theory be correct, that the neurobiological faculty for developing the human mind can also provide vulnerability to developing a severe mental disorder? Even Professor Marsha Linehan, the brilliant, empathic and religiously pious psychologist who created Dialectical Behavior Therapy (DBT) for BPD, and who recently documented her own extensive history of severe BPD (Building a Life Worth Living, A Memoir, 2020), believes that she has a general "biological predisposition" to mental illness. She has never entertained even a possibility that although her exceptional sensitivity to others and her empathic nature are quite positive characteristics, they nevertheless could also have provided a vulnerability to developing a mental disorder in a setting of the routine psychological/emotional "invalidation" that she had experienced on an everyday basis throughout her childhood and adolescence.

There are two characteristics of the neurobiological faculty for development of the mind that provide vulnerability to faulty ToM development: (1) This **Social Intelligence** faculty is **Differentially Susceptible** to the nature of the developmental social environment, such that it will develop normally in a healthy social environment but can also develop defectively in an adverse environment (**the general p-factor)**. (2) Inherent enhancement of this faculty provides exceptional potential for healthy development, but it also provides exceptional vulnerability to developmental failure in an adverse social environment.

We will argue that pre-borderline children are born with this enhanced Theory of Mind neurobiology, which provides the genetic vulnerability for the occurrence of BPD when there is a specific adverse childhood **Developmental Social Learning Environment (DSLE)**. Three considerations are at the basis for this argument. First, Borderline Personality Disorder is essentially a disorder of the working mind, which does not even exist at birth. As in the case of language, it is only an informationally empty neurobiological potential that must be activated and gradually developed by means of a lengthy, interactive social learning engagement with other humans, progressing in development on an everyday basis throughout infancy, childhood, adolescence and early adulthood. The infant

exhibits behaviors and vocal communications that are programmed for obtaining a secure base with caretakers, and gradually develops language skills that accelerate an interpersonal learning experience leading to the mature ToM ability to correctly understand self and to correctly process and understand social information about the mental states of others. In summary, the human working Mind, along with Language, must be entirely constructed by engagement in a very lengthy post-birth **Social Learning Experience***,* and therefore it is critical to carefully examine this learning experience when researching mind and language development rather than to routinely assume that defects must be inherently based. Secondly, the well-documented **Very High Sensitivity** of all pre-borderline children and borderline patients to the social environment is the behavioral expression of an enhanced neurobiological faculty for pursuing this learning experience. This inherent enhancement involves an exceptionally vulnerable level of openness and trust **(Epistemic Trust)** that would be a major learning asset in a normal, or especially in an enriched DSLE. However, this very Differentially Susceptible neurobiological enhancement initially provides the same high level of Epistemic Trust and vulnerability when the sensitive pre-borderline "orchid" child engages in this learning experience with adverse DSLE.

Thirdly, whereas examination of childhood histories of borderline patients has revealed extensive reports of intermittent childhood physical/sexual abuse, there has been minimal exploration of general levels of psychological/emotional support versus adversity/neglect in the routine DSLE, even though this environment contributes the primary source for a child's development of the mind, and even though there have been numerous reports of such everyday adverse social environments in BPD, including Marsha Linehan's childhood.

We will argue that more careful research will fully confirm that the **highly sensitive** and **exceptionally vulnerable** pre-borderline child is born with an **enhanced ToM potential**, but experiences **a routine, often invalidating, psychological/emotional communicative mismatch** with one or more caretakers who are experienced as **trusted mentors.** This invalidation is usually somewhat subtle, persisting on an everyday basis throughout the developmental years and directly interfering with and severely perverting proper development of the child's enhanced Theory of Mind. This gradually leads to BPD as a learned mental disorder, presenting a predominant level of **Epistemic Mistrust** of others and the severe, complex self/other psychopathologies that uniquely characterize BPD. The adversity may largely involve caretakers with good intentions, but who are quite uninformed and/or misguided about how to understand and engage with a highly sensitive child who may exhibit highly autonomous thinking and behaviors, rather than the caretakers having deliberate adverse motivations. This would include significant degrees of irritability about and overcontrol of the child's needy sensitivity and behaviors, involving a routine negativity towards and interference with expressed feelings and thoughts that are unfamiliar or alien to the parent's ways of thinking, thereby impeding rather than assisting in the natural development of ToM. The well-known antipathy towards the very "difficult" borderline patient held by a great many mental health professionals continues this lifelong adverse social experience.

The co-presence in borderline patients of inherently enhanced, yet developmentally impaired ToM neurobiology provides for the complex and sometimes quite paradoxical symptomatology that uniquely identifies BPD, including the high sensitivity that has become developmentally entangled to become a major participant in the psychopathology, with the initially "enhanced" sensitivity becoming the dysfunctional "hyper" sensitivity. Patients may eagerly enter treatment, seeking resolution of their chronic attachment failures, but when they begin to experience the wished for closeness and vulnerability with the therapist it can also resonate strongly with the enduring pain of the child to parent attachment failures, arousing increasing distrust and hostility. This rather abrupt change from a quite positive initial engagement to very unpleasant negativity can then disappoint and disturb the bewildered therapist who may feel he/she has been deceived. Further, although borderline patients demonstrate severe developmentally engendered impairments in mentalizing, trusting others, and understanding self and others, the inherent neurobiological enhancement for developing a mind remains, and they can at times, surprisingly, demonstrate good or even exceptional mentalization and social perceptivity **(Borderline Empathy Paradox).** Despite their severe condition this perceptivity has enabled them to occasionally outmaneuver even experienced clinicians, drawing them into strong countertransferences and boundary violations that contribute to treatment failures. This confusing interpersonal power, paradoxically involving both enhanced and defective ToM, is a key contributor to the stigma about BPD.

Theory and research focused on identifying flawed neurobiological sources for BPD continues apace despite persistent failure to actually confirm any causative inherent flaws, whereas research examining the everyday developmental social experience as possibly the essential source of BPD is essentially non-existent. This might be compared to focusing entirely on genetic vulnerabilities underlying signs of emaciation, while neglecting careful investigation of what has been actually happening in the person's daily life. A bias that interferes with effectively researching the mind and its development involves the fundamental problem that neither the patient's mind (ToM), nor what was occurring in it during development, can be directly identified by the preferred medical methods, but instead must be examined/inferred by means of an interactive engagement of the clinician's mind, over a necessary period of time, with the mind of the patient, as well as with the minds of family members and others. This necessity for engaging in a genuine relationship of meaningful duration as a "research tool" has generally been neglected as unnecessary in psychiatry, with patients often being referred to non-MDs for any relationship engagement work.

Full confirmation that Borderline Personality Disorder is usually or almost always a fully developmentally engendered disorder of Theory of Mind requires **New Research** outlined in Section F. For instance, regarding the high sensitivity characteristic, is vulnerability to parental approval/disapproval so great that even helpful corrections or suggestions can be experienced as disapproval unless carefully worded? If neurobiological anomalies are confirmed in borderline patients, are they deficits or enhancements, and are they present at birth or do

they appear after the ToM learning experience has commenced post-birth? With regard to **Prevention**, research must focus on clearer understanding of the borderline sensitivity characteristic and then developing guidelines for identifying and parenting highly sensitive children. With regard to **Treatment**, current models are very successful in resolving the acute general symptomatology. However, they persistently fail to satisfactorily resolve the self/other psychopathologies that are at the basis of the disorder, including particularly the painfully negative sense of self, the deep sense of emptiness, and the great difficulty developing trust about and enduring intimacy with others. Minimal research attention has been directed to examining the influence of the psychiatrist's verbal/emotional communications and behaviors, his/her personality characteristics such as capacity for empathy or level of narcissism, focus on or neglect of the significance of the developmental social experience, availability for urgent contact, and length of sessions, as these may relate to treatment success/failure. This is despite the likelihood that the therapist/patient relationship interaction may well be the key to a more complete recovery (just as the parent/child interaction is the key to developing the mental illness), and despite the remarkable findings by Whitehorn/Betz and others about the significance of the therapist/patient interaction for patient recovery from mental illness as far back as the 1950's. New research must also focus on patients who have the high level of **Severity** identified in the discovery populations. This is necessary because parenting is never perfect, such that we all experience a range of ToM flaws (including "Borderline Spectrum Disorders"). Much research has lacked clarity about this, resulting in blurred and contradictory findings.

Necessary treatment modifications following from this new **Theory of Mind Development Model (TOMDM)** are presented in Chapter 2. With regard to **Psychotherapy**, three modifications are presented in Chapter 2, Section B:

(1) Learning from verbal interactions with the therapist.
(2) Learning from behavioral interactions with the therapist.
(3) Identification of undeveloped Social Intelligence potentials, including possible talents, and then learning how to make good use of them.

Full text is available at: leecrandallparkmd.net/BPDmonograph.
Available free in book format by request at: lpark3@jhmi.edu. See also on ResearchGate.