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Modeling the Development of Borderline Personality Disorder: A Formal Theory Approach

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by

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DEDICATION

To my dad: thank you for always believing in me, especially during the times I struggled to believe in myself. I couldn't have made it this far without your endless love, support, and subpar jokes. Thank you for all that you do.

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I would like to thank the researchers that were kind enough to share their data with me to make this project possible. I would also like to acknowledge my advisor and committee members for their time and assistance throughout this journey.

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ABSTRACT

Borderline personality disorder (BPD) is a complex disorder characterized by instabilities in emotion regulation, impulse control, and interpersonal relationships. BPD affects adolescent and adult populations at similar rates, with traits often detectable in late childhood or early adolescence. Despite some progress in the understanding of BPD and its development, contemporary theories have yet to address several important questions in the literature. The present study suggests that many limitations associated with current theoretical models of BPD may be attributable to an overdependence on verbally specified, or weak theories. In most cases, weak theories are constructed using vague or imprecisely defined hypotheses that are difficult to properly evaluate or improve. This dissertation argues that advancing the understanding of BPD and its development may benefit from using more rigorous methodologies for theory development, including formal modeling approaches. To illustrate the potential utility of formal modeling in BPD research, the current study adopts principles from the intelligence literature to develop a formalized theory of BPD using the steps provided in the Theory Construction Methodology (TCM) by Borsboom and colleagues (2021). More specifically, principles from dynamic mutualism theory were adapted to create a formalized model of BPD and its development using the TCM, a step-by-step framework that can be used to construct formal models in psychology. Although formal modeling is not expected to solve the numerous theoretical challenges associated with BPD, it provides a foundation for systematically formalizing theoretical models of BPD. Additionally, such an approach may lead to important insights and advancements in the understanding and treatment of BPD.

CHAPTER ONE: INTRODUCTION

Borderline personality disorder (BPD) is a complex and often debilitating personality disorder (PD) that affects between 1-3% of the general population (Grant et al., 2008; Lenzenweger et al., 2007; Trull et al., 2010). BPD is characterized by significant economic and social burdens due to its high treatment utilization and various functional impairments (Chanen et al., 2017; Kulacaoglu & Kose, 2018). Although personality disorder diagnoses have historically been restricted to adult populations, accumulating evidence suggests that BPD is a developmental disorder that extends throughout the lifespan and can be reliably diagnosed in adolescence (Kaess et al., 2014; Miller et al., 2008; Tackett et al., 2009; Winsper et al., 2015). Available evidence suggests that BPD symptoms typically peak during adolescence and decline into adulthood; however, these traits can persist or even worsen with age for some individuals (Sharp & Wall, 2018).

The increased attention toward understanding BPD symptoms throughout the lifespan has facilitated greater acceptance and reduced stigma toward studying BPD in children and adolescents. This has fostered essential advances in the understanding of etiological mechanisms that may contribute to the onset and maintenance of BPD symptoms. Notably, these improvements have been made possible by two distinct research methodologies: the longitudinal investigation of BPD traits at younger ages and the use of multifactorial models to examine BPD and its developmental trajectory (Zanarini, 2000). Consistent with the multifactorial models used in developmental psychopathology research (Rutter & Sroufe, 2000), examining BPD through

this lens suggests that its symptoms can be understood as a product of evolving transactions across abnormal and normal biological, environmental, psychological, and social processes (Chanen & Kaess, 2012; Cicchetti & Rogosch, 2002). For example, research has posited several environmental risk factors (e.g., history of childhood trauma, sexual abuse, invalidating environment), genetic vulnerabilities (e.g., executive dysfunction, emotional vulnerability), and their interactions (e.g., biological vulnerability x invalidating environment) to influence the onset and development of BPD (e.g., Amad et al., 2014; Bradley et al., 2005; Carpenter et al., 2013; Crowell et al., 2009; Stepp et al., 2016).

Despite the significance of these studies, research has remained limited in its ability to identify factors that maintain BPD, as well as clarify whether these factors are distinct from its etiological mechanisms (Lenzenweger & Cicchetti, 2005). Although this may reflect the multifaceted and complex nature of BPD (Cartwright, 2008), prevention and intervention approaches would likely benefit from a more nuanced understanding of the developmental pathways that contribute to greater symptom severity and dysfunction, as opposed to pathways that facilitate symptom remission over time (Lenzenweger & Cicchetti, 2005). Prior research has proposed various hypotheses for identifying individuals at greater risk for severe manifestations of BPD; however, these studies often focus on broader risk factors and overlook other relevant variables, including protective factors. Indeed, a recent review of meta-analytic studies concerning risk and protective factors for BPD and other personality disorders highlighted this gap by examining 56 pertinent studies, none of which examined the role of possible protective factors (Solmi et al., 2021).

The inherent complexities of studying BPD are also arguably compounded by the existing methodologies used to develop and validate its theoretical models. In other words, the

majority of BPD theories can be characterized as "weak theories" in that they offer imprecise hypotheses and lack clarity as to how and under what conditions key variables interact (Fried, 2020a). Accordingly, while we acknowledge that no single theory, regardless of its strength or robustness, can fully elucidate the etiology or development of BPD, we believe that the process of theory formalization may still yield meaningful contributions to the scientific understanding of BPD and related disorders.

Theories in Psychology

Theories are typically defined as conceptual frameworks or models that contain existing knowledge and/or assumptions that help organize, explain, and predict various observed (or unobserved) phenomena (Fried, 2020a, 2020b). The goal of a theory is not merely to explain "raw data," but to account for robust phenomena that are thought to underlie empirical data (Haslbeck et al., 2021). In clinical psychology, the hypothetico-deductive method has been the primary approach to building theoretical framework. This entails formulating specific hypotheses derived from empirical observations, which are then tested to generate evidence that either refutes or supports the theory in question (Robinaugh et al., 2021; Ward & Haig, 1997). Under ideal circumstances, this allows for scientific evidence to accumulate based on a strong theory and its corresponding predictions, which in turn guides future research and promotes scientific advancement (Conway et al., 2020).

Related to theories are theoretical models,[1] which are often constructed alongside a broader theory to provide a localized description of a specific phenomenon and its mechanisms (Fried, 2020b). Accordingly, theoretical models serve as tools for offering simplified, systematic

[1] Of note, theoretical models should not be confused with data models, which can take on many forms (e.g., descriptive statistics, correlations, structural equation models [SEM]) and are constructed to summarize the empirical trends or patterns in empirical data (Kellen, 2019).

descriptions of a phenomenon or process and are rarely used as an *explanation* for the phenomenon (Fried, 2020a, 2020b; Smaldino, 2020). Investigating complex phenomenon may consequently require researchers to construct several models that focus on different explanatory levels (e.g., behavioral, cognitive, environmental, familial, genetic, neurological, etc.) and/or their interactions (e.g., behavioral genetics, gene-environment interactions; Hawkins-Elder & Ward, 2020). For example, the biosocial theory of BPD and its extensions attempt to understand the development of BPD symptoms (i.e., the phenomenon of interest) by describing how different elements in the target system[2] (e.g., invalidating environment, poor coping strategies) interact with other parts of the system (e.g., emotional sensitivity) to contribute to the development of BPD (Crowell et al., 2009; Linehan, 1993).

When the target system is sufficiently represented by the theoretical model, researchers can employ surrogative reasoning to use the theory itself to derive predictions about the target system and related phenomena (Robinaugh et al., 2021; Swoyer, 1991). For example, medical scientists often rely on surrogative reasoning to better understand disease outcomes in humans via mouse models. The advantage of surrogative reasoning is that it allows researchers to draw inferences that would otherwise be infeasible or unethical with traditional methods (El Skaf et al., 2024). An important prerequisite for engaging in surrogative reasoning, however, relies on the ability of researchers to infer how the target system behaves (e.g., how elements of the target system are related over time) based on the assumptions of the theory (Robinaugh et al., 2021). This process can be challenging, especially when theoretical predictions are imprecise, vague, or

[2] The term target system refers to real-world variables or features that are examined to acquire fundamental knowledge about the phenomenon of interest. Target systems can thus be understood as roadmaps for understanding how aspects of the real world and their relationships contribute to a specific phenomenon. As such, theories often seek to explain a given phenomenon by creating theoretical models that serve as a proxy for the target system (Haslbeck et al., 2022).

correspond to a relatively large number of possible outcomes (i.e., Spielraum; Meehl, 1990). Stated differently, when theoretical predictions can be supported by a wide range of empirical outcomes, deducing how elements in a target system might behave and interact to produce the phenomenon of interest can be exceedingly difficult (Grahek et al., 2021; Haslbeck et al., 2021). For example, Linehan's prominent biosocial model of BPD posits several environmental risk factors (e.g., invalidation of childhood emotions, negative reinforcement of emotion, harsh parenting, etc.) to potentially contribute to the development of BPD. However, this wide range of risk factors offers poor predictive utility as to who will actually develop these symptoms (Linehan, 1993). Furthermore, the theory's wide range of environmental risk factors implies that a positive association between BPD and any one of these vulnerabilities could be interpreted as support for the model, making theory falsification challenging (Oberauer & Lewandowsky, 2019).

Building Theories in Psychology: Current Problems and Future Directions

In recent years, increasing attention has been devoted to solving the problems that arise from the overuse of verbal theories, which often lack precision and adequate formalization. Prior to this discussion, much of the literature was focused on addressing methodological concerns that were believed to contribute to the replication failures in psychology, such as p-hacking and publication biases (Nelson et al., 2018; Shrout & Rodgers, 2018). However, many researchers have instead argued that the more pressing concern in psychological research is an impending "theory crisis" due to the overdevelopment of weak theories[3] (e.g., Eronen & Bringmann, 2021; Fried, 2020b; Haslbeck et al., 2021; Muthukrishna & Henrich, 2019; Oberauer & Lewandowsky, 2019).

[3] For the purpose of this paper, the term "weak theory" is used to refer to any theoretical framework that is based on vague assumptions and/or hypotheses using natural language.

As discussed by Fried (2020a), weak theories are usually expressed using ambiguous, verbal statements[4] that are prone to hiding crucial assumptions due to their imprecision. Without concrete, specific hypotheses, empirical findings that are generally consistent with the assumptions of a weak theory may promote a false sense of security by inadvertently masking other crucial assumptions, contradictions, or shortcomings of the theory (Guest & Martin, 2020; Smaldino & McElreath, 2016).

Weak theories are also problematic because they can be defended by incorporating post-hoc assumptions, such as hidden or unmeasured moderators or an alleged flaw in the research design (Fried, 2020a). For example, the biosocial model of BPD suggests that invalidating environments play a central role in the development of emotion dysregulation (Linehan, 1993). Under this assumption, if a longitudinal study were to reveal that children raised in invalidating environments exhibited comparable levels of emotion dysregulation to those not raised in such environments, one might propose a nebulous "third variable" to explain these unexpected results rather than interpreting them as evidence against the theory (Oberauer & Lewandowsky, 2019). While an extraneous variable may truly exist in some cases, secondary explanations that are readily identifiable and capable of reconcile inconsistent findings reduces the likelihood of a theory being falsified (Grahek et al., 2021). This practice enables alternative explanations to justify the poor predictive utility of a theory, and enables problematic theories to exist without offering an incentive for improvement (Oberauer & Lewandowsky, 2019).

[4] It is worth noting that other researchers have cautioned against arbitrarily labeling verbal theories as "weak theories," as this may deter researchers from using less formalized theories despite the value these theories may hold (DeYoung & Krueger, 2020). Likewise, verbal theories, notwithstanding their potential drawbacks, provide conceptual foundations for which new and existing empirical findings can be integrated, and are a necessary first step to creating more comprehensive formal theories (Maatman, 2021).

Formal theories and improving psychological research

In an effort to address some of the concerns associated with weak theories, as well as strengthen future theories, several researchers have advocated for the use of systematic approaches to develop and assess psychological theories (Borsboom et al., 2021; Eronen & Bringmann, 2021; Guest & Martin, 2020; Haslbeck et al., 2021; Smaldino, 2020; van Rooij & Baggio, 2020). These proposals have varied to some degree, though an overwhelming number agree on the importance of formalizing verbal theories via formal modeling or other computational methods (Borsboom et al., 2021; Devezer et al., 2020; Guest & Martin, 2020; Haslbeck et al., 2021; Navarro, 2021; Oberauer & Lewandowsky, 2019; Robinaugh et al., 2021; Smaldino, 2020; van Rooij & Baggio, 2020). For the purposes of this paper, we define formal modeling as the process by which a theory is specified using logical, mathematical, or computational expressions (Haslbeck et al., 2021; Oberauer & Lewandowsky, 2019; Robinaugh et al., 2021).

Compared to verbal theories, the construction and implementation of formal theories offers greater specificity and precision by allowing researchers to directly observe and manipulate the behavior of a target system based on theoretical assumptions (Robinaugh et al., 2021). This enables researchers to evaluate the theory based on its ability to reproduce or explain real-world phenomena (Wang et al., 2023) and sets the stage for testing alternative predictions that would otherwise be difficult through verbal explanation alone (Maatman, 2021). By expressing a theory in mathematical or computational terms, researchers are more capable of deriving theoretical predictions and evaluating what the theory can and cannot explain through simulations (Fried, 2020a). Simulating data based on the assumptions of the theory is particularly useful, as it provides information as to what the data would look like if the theory was true.

Equally, this process presents opportunities for conducting meaningful comparisons between theory-implied data and observed data on the phenomenon of interest (Fried, 2020b). Such comparisons may yield valuable insight that can help improve the overall worth of the theory (Wang et al., 2023).

Constructing formal theories in psychology

Despite the lack of consensus on the optimal approach to formalizing psychological theories, the Theory Construction Methodology (TCM), proposed by Borsboom and colleagues (2021), has gained increasing attention in the literature. The TCM provides a structured foundation for developing formal theories in psychology and consists of five steps: 1) identify relevant phenomena, 2) formulate a prototheory, 3) develop a formal model, 4) verify the adequacy of the formal model, and 5) appraise the value of the formal theory (Borsboom et al., 2021).

In contrast to traditional theory development, which is centered on the hypothetico-deductive framework, the TCM initiates the theory development process by identifying a set of relevant phenomena that are capable (and worthy) of being explained. For this phase to be successful, researchers must carefully select robust variables, which may lead to the selection of phenomena that are already well-established in the literature (Borsboom et al., 2021).

In the second phase of the TCM, a verbalized "prototheory" is established. The prototheory serves as an initial explanatory model for the phenomenon of interest by describing how the phenomenon would arise if the assumptions of the theory were true (Haig, 2014). Consequently, the objective of the prototheory is to identify the pertinent components of the larger target system and generate hypotheses as to how these components are related and cause the phenomenon to emerge (Haslbeck et al., 2021). This process is analogous to abductive

reasoning (i.e., inference to the best explanation), such that an incomplete set of observations are used to derive an explanation that "most likely" accounts for the phenomenon (Peirce, 1974). During this step, researchers may consider "borrowing" explanatory principles from existing theories or other scientific disciplines to better inform their prototheory (Borsboom et al., 2021). For example, Chow et al. (2005) adapted principles from physics to study dynamic changes in emotion regulation via a dampened oscillator model. The authors adapted this model to examine changes in college students' emotions and found that it provided a useful framework for understanding the dynamics of emotion and how emotional experiences differ across individuals.

After the prototheory is developed, the third step of the TCM involves translating the verbal theory into a formal model, such that the principles of the prototheory are expressed using logical or mathematical language (e.g., algebraic expression, differential equations, etc.). These equations should contain variables that are measurable in addition to constants (i.e., adjustable parameters) whose values are typically derived through logical proofs or data (Borsboom et al., 2021). For instance, verbal theories that contain variables characterized by nonlinear dynamics are commonly expressed using differential equations because they are capable of modeling nonlinear change (Hirsch, 1984). An advantage of this approach is that constants and other parameters in the equation can be modified later on, allowing researchers to evaluate how different parameter values influence predicted outcomes (Jaccard & Jacoby, 2019; Wilson & Collins, 2019).

In the fourth step of the TCM, the adequacy of the formal model is investigated using analytic derivations or data simulations. In other words, the formal model is tested to see whether it can generate the phenomena of interest as a matter of course (Borsboom et al., 2021; Fried, 2020a). The adequacy of the formal model is therefore validated if it produces the

phenomenon and provide a reasonable explanation for its emergence. For example, the dynamic mutualism theory of intelligence provides an alternative explanation for the observed positive correlations, or positive manifold, found amongst cognitive abilities (van der Maas et al., 2006). The authors formalized the emergence of this positive manifold in terms of statistical results that were expected to occur if their theory were true. Specifically, the authors posited that if the mutualism model was supported, then the simulation should produce robust and positive correlations among cognitive processes that result in the extraction of a strong unidimensional factor.

In the fifth and final step of the TCM, the explanatory power of the formalized theory is rigorously evaluated. This requires the strength of the theory to be assessed by considering its overall value and scientific merit, which may include examining its explanatory breadth, precision, predictive utility over existing theories, or potential for generating new insights and hypotheses. This step is of particular importance, as new theories frequently provide a reasonable explanation for a phenomenon of interest but lack parsimony and accuracy in their description (Borsboom et al., 2021).

Although no gold standard exists for assessing the overall value of a theory, measuring the explanatory breadth and precision of the theory are often viewed as critical components of theory appraisal (Borsboom et al., 2021; Haslbeck et al., 2021). Explanatory breadth refers to the number of phenomena a theory can adequately explain or predict, while explanatory precision refers to the specificity of the phenomena that are accounted for by the theory (Thagard, 1978). These concepts, albeit related, often have an inverse relationship that must be balanced. In simpler terms, a theory that seeks to explain a wider set of phenomena may end up being too general and imprecise, while a theory with too high of precision may have a limited scope that

hinders its overall usefulness. Theories that tend to be most informative strike a balance between their precision and their ability to explain a wider array of phenomena (Haslbeck et al., 2021).

In evaluating the overall value of the formal theory, the TCM stresses the importance of comparing the theory-implied data (i.e., simulated data) derived from the mathematical model to relevant empirical or observed data. If the theory-implied and empirical data models produce similar enough results, then greater certainty can be attributed to the formal theory's ability to represent the underlying target system (Robinaugh et al., 2021). However, if the theory-implied and empirical data models are largely incongruent, then the source of these discrepancies should be explored. Auxiliary assumptions[5], which are embedded in the data simulation process, are a suitable place to start, as they can be easily adjusted. If auxiliary assumptions cannot reasonably explain these discrepancies, then abductive reasoning (i.e., inference to the best explanation) may help researchers identify a sensible conclusion that can guide adaptations to the formal theory. This iterative process allows the theory to be refined until it aligns with empirical processes, resulting in a more accurate description of the underlying target system (Haslbeck et al., 2021).

Taken together, the TCM, and theory formalization more broadly, offer several advantages over traditional theory-building approaches by providing tools for better understanding the connections amongst psychological concepts, theoretical predictions, and empirical data (Eronen & Romeijn, 2020; Guest & Martin, 2020). Nonetheless, it is important to acknowledge that formal theories are not without limitations, and their utility can easily be obscured by imprecise specification or poorly defined mathematical relationships (Teufel & Fletcher, 2016). Likewise, both verbal and formal theories are equally limited when constructs of

[5] Auxiliary assumptions reflect any decision or assumption that was involved in producing the simulated data. For example, poorly selected parameter values in the computer simulation may serve as one auxiliary explanation behind incongruent empirical and theory-implied data models.

interest or psychological mechanisms are difficult to isolate, define, or measure over time (Eronen & Romeijn, 2020).

Notwithstanding these above limitations, the use of formal and/or computational models for developing and refining theories still provide unique opportunities for improving the study of various psychological phenomena. This is particularly true for phenomena that are dynamic and sufficiently complex, which can be more difficult to examine using conventional approaches (Jaccard & Jacoby, 2019). For example, Wang et al. (2023) used a formal modeling approach to investigate the core predictions of the General Escape Theory of Suicide. Through their simulations, the authors found that their formalized theory produced and explained several key phenomena in the suicide literature and offered valuable insight into the study of suicide as a complex system. We believe this logic applies to the study of BPD and other psychopathology as well, and that such an approach may advance the current landscape of BPD and help identify areas for future research.

The Present Study

The present study represents a preliminary step towards integrating formal models with BPD research. Using the steps outlined in the TCM, this study sought to propose a formalized developmental theory of BPD by adapting the core components of dynamic mutualism theory (van der Maas et al., 2006; van der Maas et al., 2017). Dynamic mutualism was first introduced as an opposing theory to the g-factor model of intelligence and has been recently adapted to better understand the development of BPD and psychopathology more broadly. However, research examining dynamic mutualism and BPD has been limited to theoretical explorations (Choate et al., 2020a) or studies using null-hypothesis significance testing to examine dynamic mutualism predictions that are verbally expressed (Choate et al., 2023). As such, this study is the

first to formalize the key assumptions of mutualism theory with respect to the development of BPD. While we recognize that formal modeling is unlikely to alleviate the numerous theoretical issues related to BPD, we argue that the theory formalization process provides an opportunity to better assess dynamic mutualism theory, identify its strengths and weaknesses, and pinpoint areas in need of further refinement and/or empirical study. Ultimately, it is our hope that this paper encourages researchers to consider formalizing other existing theories of BPD in order to better understand its etiology, pathogenesis, and heterogenous clinical features that have contributed to a poor understanding of its development (Kulacaoglu & Kose, 2018).

The structure of this paper is as follows. In Chapter Two, we provide an overview of dynamic mutualism theory with respect to intelligence and explain how its principles can be translated to understand the development of BPD. In Chapter Three, we propose a formalized model of BPD based on the core assumptions of dynamic mutualism theory and evaluate the model via computer simulations. Next, we compare the theory-implied data derived from the simulations to real-world data on BPD and evaluate the explanatory breadth and precision of the formal model. Lastly, in Chapter Six, the implications, strengths, and limitations of the formalized mutualism model of BPD are discussed. Future directions are similarly provided.

CHAPTER TWO: DYNAMIC MUTUALISM THEORY

Dynamic Mutualism in Intelligence Research

Dynamic mutualism theory was first proposed as a developmental theory of intelligence that provides an alternative explanation for the positive manifold (i.e., positive correlations) observed amongst cognitive abilities (van der Maas et al., 2006). In line with Step 1 of the TCM, the dynamic mutualism model of intelligence was proposed as a way of explaining the development of cognitive ability after identifying several key phenomena worth explaining. Specifically, the model aimed to explain the following phenomenon: a) measures of intelligence are characterized by a positive manifold, b) cognitive ability increases throughout development, and c) the heritability of intelligence increases with age.

In contrast to Spearman's *g*-factor theory, the mutualism model indicates that intellectual abilities do not arise from a latent construct, but rather from a series of mutually beneficial interactions between lower-level cognitive, biological, and environmental processes. Associations between lower-level cognitive processes are assumed to be independent in early childhood but are predicted to gradually strengthen during certain periods of development. The positive manifold of intelligence thereby emerges as a matter of course via mutualistic interactions that evolve over time (van der Maas et al., 2006; van der Maas et al., 2017).

The dynamic mutualism model of intelligence was originally derived from a pair of first-order nonlinear differential equations known as the Lotka-Volterra predator-prey model in biological systems (May, 1973; Murray, 2002). The Lotka-Volterra equations were designed to

explain the dynamics between two populations ($x1$ [prey], and $x2$ [predator]) that change as a function of one another and other environmental variables (e.g., food scarcity). This leads to oscillations in the population sizes of x_1 and x_2 over time and can be mathematically derived using the equation below (see Equation 1).

Equation 1

$$\frac{dx_1}{dt} = ax_1\left(1 - \frac{x_1}{K}\right) - Mx_2x_1,$$

$$\frac{dx_2}{dt} = (Mbx_1 - c)x_2$$

Based on principles from the Lotka-Volterra model, van der Maas et al. (2006) adapted these equations to develop the dynamic mutualism model of intelligence. The mathematical representation of the mutualism model of intelligence is presented in Equation 2.

Equation 2

$$\frac{dx_i}{dt} = a_i x_i\left(1 - \frac{x_i}{K_i}\right) + a_i \sum_{\substack{j=1 \\ j \neq i}}^{W} M_{ij}x_j x_i / K_i$$

$$K_i = c_i G_i + (1 - c_i)E_i$$

Equation 2 states that change in x of a given cognitive process i (dx_i) at each time point (t) is a product of the sum of interaction weights of each cognitive process j (captured by matrix **M**), multiplied by the growth rate of process i (a_i) times the current level of x_i divided by the carrying capacity, or asymptote, for that process (K_i). Stated differently, a_i is a random parameter that differs across subjects and determines the steepness of the logistic growth curve associated with each x_i. The mutualism model assumes a_i and x_0 (i.e., the starting value of a given x_i) are independent from the stable state of the model, implying that individual differences during initial

stages of development do not explain individual differences at later stages (van der Maas et al., 2021).

Most relevant to the mutualism model is matrix **M**, a population parameter (i.e., values are equal across subjects) that contains small, mostly positive interaction weights between all x_i processes (M_{ij}). When the system is found to near a state of equilibrium, and weights in matrix **M** are equal across individuals, M_{ij} are essentially regression weights that denote the direct influence of one cognitive ability on the other (Kan et al., 2019; van der Maas et al., 2006). K_i is the asymptote, or upper growth limit of a given x_i, and is allowed to differ across subjects. Parameters **K** are thought to capture factors that limit the maximum amount of growth for each x_i (e.g., genetic and/or environmental constraints), and as noted in Equation 2, are a linear function of both genetic (G_i) and environmental effects (E_i) with weights c and c–1, respectively (van der Maas et al., 2017). Unlike the *g*-factor theory of intelligence that suggests individuals who score similarly on intelligence tests do so because they have equivalent factor scores on *g* (van der Maas et al., 2017), mutualism theory suggests that different developmental pathways (reflected in **K**) may ultimately lead to the same level of intelligence. For instance, one individual may score above-average on measures of intelligence due to large K_i values for a specific process (e.g., memory capability, processing speed, etc.), while another individual may score higher because of a general absence of low K_i values (van der Maas et al., 2017).

When appropriately small starting values are selected for $\mathbf{x_0}$, **a**, **K**, and **M**, each x_i will generally follow a logistic growth curve until a point of equilibrium is reached (i.e., the asymptote value is greater than K_i). This pattern of growth assumes that parameters **K** are uncorrelated and $M_{ij}>0$, indicating that change is due to unique genetic and environmental influences and mostly positive interactions amongst lower-level cognitive processes (van der

Maas et al., 2006; van der Maas et al., 2017). This mathematical formulization was subsequently used as the foundation for conducting a series of simulations that investigated varying accounts of cognitive development. This was accomplished by manipulating core assumptions and parameter values of Equation 2 to accommodate various developmental scenarios that are described in greater detail below.

In the first simulation, van der Maas and colleagues (2006) set all M_{ij} to 0 and sampled parameters x_0, a_i, and K_i from an uncorrelated normal distribution with a means and standard deviations (*SDs*) set to 6 (*SD* = 0.5), 3 (*SD* = 0.5), and 0.05 (*SD* = 0.01), respectively. Given that parameters **K** were sampled from an uncorrelated distribution and the weights in matrix **M** were set to 0, a positive manifold was not expected to emerge. This assertion was supported by the resultant simulated data that found the correlations amongst cognitive abilities to be approaching zero, with the eigenvalue analysis similarly supporting a zero-factor structure.

In the second simulation, van der Maas et al. (2006) explored a common cause, or *g*-factor model of cognitive development by sampling the resource/carrying capacity parameters (**K**) from a correlated multivariate normal distribution. The interaction weights between cognitive processes were still set to zero ($M_{ij} = 0$). This implied that above-average scores on measures of intelligence were attributable to a biological factor, such as *g*, that is causal in nature. Unlike the first simulation, these adaptations produced a positive manifold with a one-factor solution representing the data well. This was considered evidence in favor of *g* and was considered the primary competitor for the mutualism model of intelligence (van der Maas et al., 2006).

In the last scenario, the dynamic mutualism model of intelligence was investigated by introducing small, mostly positive weights in matrix **M**, while sampling parameters **K** from an

uncorrelated multivariate distribution. Indeed, data simulated from the mutualism model, as delineated in Equation 2, demonstrated a robust positive manifold. This provided preliminary support for the idea that mutually beneficial interactions among cognitive processes can adequately explain the positive manifold of intelligence without involving a causal entity, such as *g* (van der Maas et al., 2006; van der Maas et al., 2017).[6]

Mutualistic Processes in Borderline Personality Disorder

Analogous to the intelligence literature, factor analytic studies on BPD have similarly revealed a positive manifold to characterize the relationship among BPD symptoms (Fossati et al., 1999). Further, these symptoms are typically found to intensify during specific developmental windows, with heritability generally increasing from adolescence to young adulthood (Bornovalova et al., 2009; Hawkins et al., 2014; Stepp, 2012). These observations align with the Step 1 of the TCM, which involves identifying empirical phenomena that are worthy of being explained. Here, the phenomenon of interest is the development of the positive manifold that underlies the nine BPD symptoms.

Historically, prevailing theories of BPD and its development have predominantly focused on understanding the interactions between environmental influences and genetic predispositions, often overlooking the possibility of inter-symptom dynamics. Dynamic mutualism theory thus provides an alternative framework for understanding the development of BPD by accounting for dynamic and potentially causal relationships among its symptoms. While some assumptions of the mutualism model of intelligence may not directly translate to BPD, its core assumptions have

[6] It is worth mentioning that because parameters in **M** are held constant across subjects, individual differences in intelligence cannot solely be attributable to mutually beneficial interactions in M_{ij}. Rather, it is small, between-person differences in the average values of **K** that are then weighted by **M** that ultimately contribute to individual differences in cognitive ability (van der Maas et al., 2006).

undergone both theoretical (Choate et al., 2020b) and empirical investigation (Choate et al., 2023) and provide an attractive framework for studying its development.

Despite these recent advancements, no study to our knowledge has formalized a dynamic mutualism model specifically for BPD. In other words, no study to date has examined whether assumptions of mutualism theory could offer a viable explanation for the positive manifold of BPD symptoms. Moreover, empirical tests of mutualism with respect to BPD or other forms of psychopathology are limited to interpretations based on imprecise predictions that were tested via null-hypothesis statistical testing (Choate et al., 2022, 2023; McElroy et al., 2018; Murray et al., 2016). However, identifying the types of evidence needed to adequately support or refute a theory can be challenging without knowing what the data would look like if the theory were true. For instance, McElroy et al. (2018) verbally translated the assumptions of dynamic mutualism into a set of statistical patterns that were expected to emerge if dynamic mutualism adequately explained the development of the p-factor. Using bifactor analysis, the authors suggested that mutualism would be supported if the general p-factor increased in strength and variance accounted for over time at the expense of the specific factors. The results of their study were inconclusive, revealing evidence for and against mutualism and highlighting the difficulties of investigating a theory with imprecise predictions. In comparison, formalized theories may offer a more precise and structured representation of the theory, help clarify its assumptions and predictions, and lead to more defined research questions that are straightforward to test (Robinaugh et al., 2021).

Developing a Prototheory of the Development of BPD

In the second step of the TCM, a working prototheory that provides a general explanation for the phenomenon of interest is developed. The prototheory is then translated into a formal

model in the third step of the TCM. In the current study, principles from the dynamic mutualism model of intelligence were extrapolated to construct a prototheory of BPD and its development (van der Maas et al., 2006; van der Maas et al., 2017).

Equivalent to the mutualism model of intelligence, the general premise of the prototheory is that BPD, at least in part, may develop as a function of mutually beneficial interactions among its specific symptoms. Although dynamic mutualism theory does not specify when mutualistic interactions should first emerge, the timing of these effects was based on available evidence indicating that BPD symptoms can be identified around 11 years of age (Bernstein et al., 1993; Zanarini et al., 2011). Furthermore, some studies have gone so far as to suggest that BPD can be reliably diagnosed in children aged 11 or older (Guilé et al., 2018).

Consistent with the general premise of mutualism theory, it was predicted that individuals who are susceptible to developing BPD will demonstrate small and even negative associations between BPD symptoms at earlier ages (i.e., ages 11-12), with these symptoms developing increasingly strong and positive associations with age. Mutualism theory also implies that BPD symptoms should demonstrate a single-factor structure, particularly later in development once mutualistic processes have begun. These predictions serve as the foundation for both the mutualism model of intelligence and the prototheory of BPD and can be mathematically expressed via differential equations (see Equation 3).

Equation 3

$$\frac{dx_i}{dt} = a_i x_i \left(1 - \frac{x_i}{K_i}\right) + a_i \sum_{\substack{j=1 \\ j \neq i}}^{W=9} M_{ij} x_j x_i / K_i \quad \ldots t = 10$$

$$K_i = c_i G_i + (1 - c_i) E_i$$

In Equation 3, the development of BPD is modeled as a system comprised of W symptoms (e.g., affective instability, unstable interpersonal relationships, etc.), which are captured in vector **x**. W is equal to nine because it reflects the nine BPD symptoms defined in the *DSM*-5. The time, or t parameter is set to 10 to serve as a proxy for ten yearly assessments capturing change in BPD from ages 11 to 20. Of note, this differs from the approach used by van der Maas et al. (2006) which extracted data at a single time point after the model reached a state of equilibrium.

Growth or change in each symptom (x_i) is influenced by other symptoms in the model in addition to the autonomous growth processes that are specific to that symptom (i.e., growth that does not depend on other symptoms in the model). The autonomous growth of each symptom is captured in the first part of the equation, where a_i denotes the growth rate and K_i denotes the maximum amount of growth, or the "carrying capacity," for that specific symptom. Carrying capacity refers to one's propensity to develop a given BPD symptom, which is shaped by different genetic and environmental predispositions. The additive representation of **K** suggests that the potential for developing BPD increases with greater environmental exposure, particularly for those who possess genetic vulnerabilities associated with BPD (Carpenter et al., 2013; Crowell et al., 2009). Once symptoms of BPD are present, they will also trend towards the individual's overall carrying capacity. For instance, some individuals may exhibit an elevated K_i for a given symptom as a consequence of specific environmental factors (e.g., poverty, parental abuse), whereas others may display a larger K_i for the same symptom due to preexisting neurobiological vulnerabilities (e.g., poor executive functioning; Winsper et al., 2016) that confer a greater risk for developing that particular symptom (Chanen & Kaess, 2012; Winsper, 2018). This implies that individuals with more genetic or environmental vulnerabilities will have

an elevated potential to develop symptoms of BPD and maintain these symptoms compared to individuals with few to no vulnerabilities.

Generally speaking, parameters **a** work in tandem with parameters **K** to shape the stability, oscillations, and overall dynamics of the model, while simultaneously influencing the strength of the mutualistic interactions between symptoms. However, because **a** and **K** are sampled independent from each other, symptoms are initially uncorrelated but become correlated with age because of developing mutualistic interactions (de Ron et al., 2023; van der Maas et al., 2006). Although van der Maas et al. (2006) do not conceptually discuss the significance of a_i apart from that it controls the steepness of the logistic growth curve for each x_i, this parameter significantly influences the initial dynamics of the model and the strength of the mutualistic interactions. With respect to BPD, a_i was conceptualized as capturing unique, external factors that conceivably influence the initial growth rate and interactions among BPD symptoms. These factors include but are not limited to: preceding internalizing and/or externalizing psychopathology, social processes (e.g., peer groups), cognitive processes (e.g., poor executive functioning), neurobiological processes (e.g., hormonal changes), and so forth (Bohus et al., 2004; Crowell et al., 2009; Stepp et al., 2016). For example, an adolescent diagnosed with attention-deficit/hyperactivity disorder (ADHD) may have an elevated a_i for the impulsivity symptom of BPD because ADHD is similarly characterized by poor impulse control. Having a larger a_i may subsequently augment the adolescent's probability of expressing the impulsivity symptom of BPD, with more accelerated growth expected at earlier stages of development.

In the second part of the equation, the mutualistic interactions between the various BPD symptoms (M_{ij}) are accounted for by matrix **M** and influence the stability, equilibrium points, and general dynamics of the model. Taken together, the mathematical model states that, at each

time point (t), change in a given BPD symptom (x_i) is a function of its own autonomous growth (explained by a_i and K_i), and a function of mutually beneficial interactions (M_{ij}) amongst other BPD symptoms (van der Maas et al., 2021; van der Maas et al., 2006; van der Maas et al., 2017). At any given time, values can be obtained for each x_i to yield a dataset of n subjects with scores across the nine BPD symptoms at time t (van der Maas et al., 2021).

It is important to note that for the formal model to be useful, it should not only explain the positive manifold underlying BPD, but also account for other key components associated with the disorder. In other words, for the formal model to be of value, it should at a minimum account for the following: a) individual differences in the susceptibility to developing BPD, b) variability in the intercorrelations among BPD symptoms, c) differences in the centrality, or importance, of certain BPD symptoms, d) variability in the onset of BPD symptoms, e) symptom decline into adulthood, and f) improvements in BPD in response to evidence-based intervention. Therefore, further changes were made to the original dynamic mutualism model in order to develop a formalized model that offered a more accurate depiction of the complex phenomena associated with BPD. These modifications are described in greater detail below.[7]

Reciprocal interactions

In the mutualism model of intelligence, the interactions captured in matrix **M** were fixed to 0.05 for all subjects and cognitive processes (van der Maas et al., 2006). However, restricting M_{ij} to be equal across individuals and symptoms seemed untenable for a model of BPD. Instead, it may be more realistic to suggest that symptoms are characterized by different M_{ij} weights,

[7] Parameter values that were modified from the original mutualism model were selected based on preliminary analyses that provided general guidelines for facilitating model convergence. For example, large values for M_{ij} were avoided, as this implies that each x_i grows without bound, which is an unreasonable scenario and would lead to problems with the model converging properly (van der Maas et al., 2006).

which also vary across individuals. Therefore, we allowed weights in matrix **M** to vary across both symptoms and subjects. This was accomplished by creating a 9x9 matrix with all M_{ij} similarly set to 0.05 and then randomly sampling a new matrix for each subject based on a mean of 0.08 and *SD* of 0.02. This provided a more realistic structure for modeling varied relationships between BPD symptoms across individuals.

Growth parameters

The growth parameters are imperative for investigating the behavior of the model under varying conditions. In the mutualism model of intelligence, parameters **a** were randomly sampled from an uncorrelated multivariate normal distribution with a mean of 6 and a *SD* of 0.50. However, these values were manipulated in various scenarios (described later) to explore whether other values may be more appropriate. Additionally, in the mutualism model of intelligence, K_i were considered random parameters (i.e., they differed across subjects) that were sampled from an uncorrelated multivariate distribution with a mean of 3 and *SD* of 0.50. This implied that genetic/environmental effects of one cognitive process were unrelated to the genetic and environmental effects of another cognitive process. However, this assumption was modified for the purposes of this study, as research suggests that the genetic and environmental contributors to BPD are likely interdependent (Nia et al., 2018). Thus, K_i for each symptom and subject were sampled from a correlated multivariate distribution (r = .25) using the same mean as the mutualism model of intelligence. Additional modifications were considered on a case-by-case basis and are discussed in greater detail in Chapter Three.

CHAPTER THREE: EVALUATING THE FORMAL MUTUALISM MODEL OF BPD

The fourth step of the TCM involves evaluating the adequacy of the prototheory and its accompanying mathematical model. This is usually accomplished by simulating data based on the mathematical model (Borsboom et al., 2021). Equation 3 provides a mathematical foundation for implementing the formalized mutualism model of BPD as a computational model, thereby allowing the theory-implied behavior to be simulated. This process aids in determining whether the theory does indeed explain the phenomena of interest, while providing an opportunity to evaluate the strengths and shortcomings of the theoretical model (Smaldino, 2017).

A preliminary simulation was first conducted based on the mathematical formulation presented in Equation 3 to establish baseline model behavior and to ensure that the initial parameter values led to model convergence. In other words, majority of the initial parameter values as discussed in van der Maas et al. (2006) were used in the first simulation. Next, a series of simulations were implemented to assess whether the model successfully explained and/or produced the following BPD features mentioned earlier: a) individual differences in the propensity to developing BPD, b) variability in the relationships amongst BPD symptoms, c) differences in the centrality, or importance, of certain BPD symptoms, d) variability in the onset of BPD symptoms, e) decreasing severity of BPD symptoms in adulthood, and f) reduction in symptoms with appropriate interventions, such as Dialectical Behavior Therapy (DBT). Each feature was systematically evaluated by adjusting select parameter values and assumptions, followed by data simulation based on these changes.

For the main model and each sub-model, 100,000 virtual samples of individuals were generated using the *deSolve* and *mutualism* packages in R-Studio (Soetaert et al., 2010). Data were simulated for all nine BPD symptoms (i.e., x_i). across ten time points, which served as a proxy for ten annual assessments across ages 11-20. Similar to van der Maas and colleagues (2006), histograms of the correlations between BPD symptoms and eigenvalues were computed after each simulation using the *psych* package in R-Studio (Revelle & Revelle, 2015).

Simulation 1: BPD Trajectories Based on the Dynamic Mutualism Model of Intelligence

The preliminary simulation generated data for 100,000 individuals using the same parameter values as the original mutualism model simulation (van der Maas et al., 2006). Specifically, M_{ij} were set to 0.05 for all symptoms, the mean of K_i was set to 3, and the mean of a_i was set to 6 with a *SD* of 0.50. The starting value of each symptom was also set to 0.05 with a *SD* of 0.01. As discussed previously, the only differences between this simulation and the original mutualism model of intelligence simulation were that parameters **K** were drawn from a multivariate *correlated* distribution instead of an uncorrelated distribution. Additionally, data were simulated across ten time points instead of one and weights in matrix **M** were allowed to vary between subjects.

The results of this simulation revealed exponential growth early in development and seemed unrealistic for BPD. The exceptionally high levels of growth were due to the large a_i values, which were subsequently modified to have a smaller mean of 2 with a *SD* of 1. This produced more reasonable levels of growth, such that BPD symptoms initially demonstrated low, and even negative correlations with each other that increased with age. To illustrate this point, the trajectories of the BPD symptom "chronic feelings of emptiness" are plotted over time using data from 50 randomly selected subjects (see Figure 1). The majority of individuals experienced

the greatest acceleration in this symptom between ages 12 and 14, with some individuals demonstrating a slower rate of growth until 18 years of age.

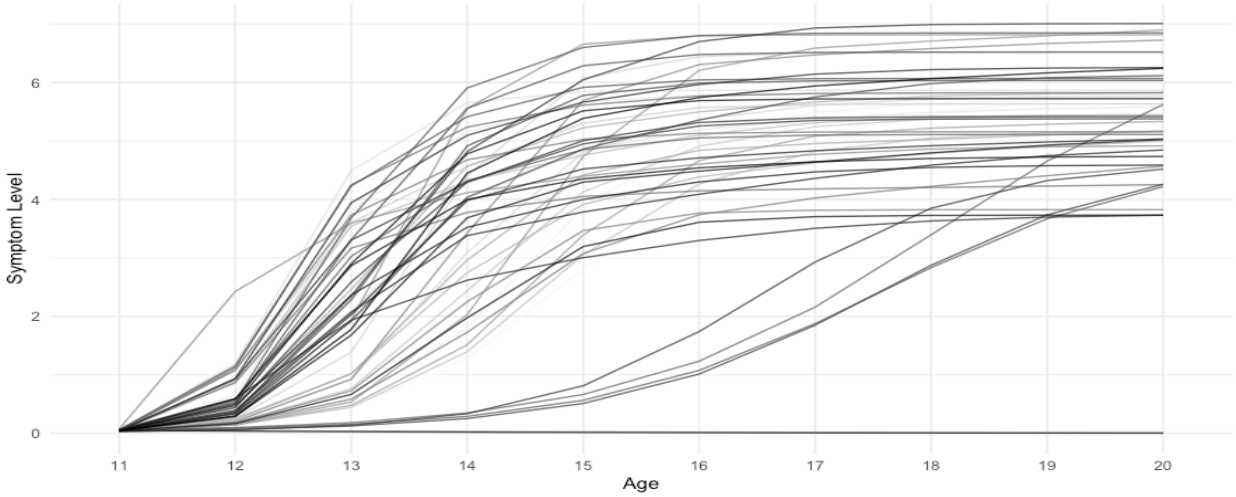

Figure 1. Change in Chronic Feelings of Emptiness.

Similar to van der Maas et al. (2006), the correlations between BPD symptoms and the resulting eigenvalues were also examined (see Figure 2). The histograms display correlations between the observed symptoms at the first, middle, and last time point (i.e., ages 11, 15, and 20). Consistent with predictions of dynamic mutualism, weaker correlations were observed between symptoms at age 11, with substantially larger correlations (r = ~0.4) found at ages 15 and 20. The results of the eigenvalue analyses also aligned with dynamic mutualism (Figure 2). At age 11, the eigenvalues indicated a zero-order structure, suggesting weak associations between symptoms. At ages 15 and 20, a dominant first eigenvalue (i.e., value that substantially exceeded one) was found, followed by considerably weaker eigenvalues. This suggested that a one-factor solution was most appropriate given the data. Taken together, this simulation demonstrated that the formalized mutualism model of BPD and its initial assumptions were capable of producing growth patterns that are consistent with dynamic mutualism theory.

In the following section, we further refined the formal model by adjusting specific parameters to determine if the model could reasonably explain and/or producing five important features associated with BPD. Updates to the model were done systematically, such that only one parameter was changed at a time for clarity.

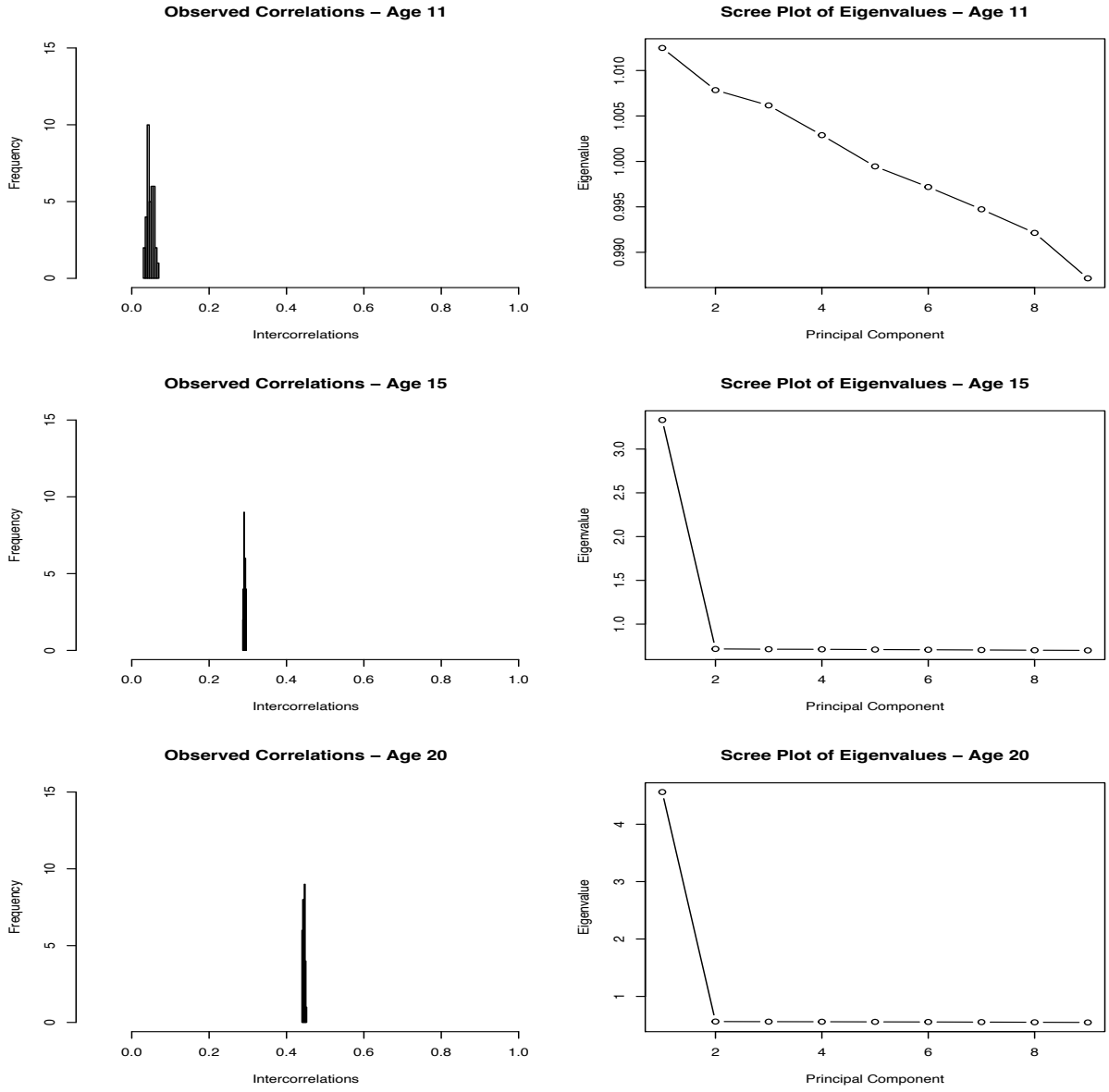

Figure 2. Observed Correlations and Eigenvalue Analysis.

Note. The correlations are very similar between processes, which is likely due to all BPD symptoms being simulated based on very similar a and K parameter values. Furthermore, although parameters M_{ij} were randomly sampled for each interaction and across subjects, specifying all pairs of weights to have the same mean is also expected to reduce variability in the observed correlations. These parameters will be important to modify in later scenarios to provide a more accurate depiction of the target system.

Simulation 2: Individual Differences in the Propensity to Developing BPD

The mutualism model of intelligence is predicated on the assumption that all individuals will undergo at least some increase in their cognitive abilities over time (van der Maas et al., 2006). However, this assumption is not applicable to BPD, as only 1-3 % of the general population will develop BPD in their lifetime (Ellison et al., 2018; Tomko et al., 2014). Moreover, a large proportion of individuals exhibiting BPD symptoms do not necessarily develop increased symptomology with age (Sharp & Wall, 2018), which differs from the development of intelligence.

The literature has highlighted a number of factors believed to increase the risk of developing BPD, with particular emphasis on trauma and/or negative life events. Childhood trauma is often viewed under the umbrella of invalidating experiences (Linehan, 1993), and is frequently studied in empirical investigations due to the strong associations found between traumatic life events and severity of BPD symptoms (Distel et al., 2011). For example, in a sample of 314 maltreated children and 285 non-maltreated children, maltreatment was associated with significantly higher BPD scores (Hecht et al., 2014). Intriguingly, some research suggests that the nature of the traumatic events do not predict differences in the onset and trajectories of BPD; rather, it is the sole presence of a traumatic event that has the most utility in predicting BPD traits (Bozzatello et al., 2020).

Developmental studies also emphasize the importance of antecedent internalizing and/or externalizing symptoms in predicting the onset and progression of BPD. A systematic review by Stepp and colleagues (2016) indicated that 84% of studies examining psychopathology as a prospective predictor of BPD found at least one disorder or broader domain (i.e., internalizing or externalizing) to significantly predict future BPD symptoms. In adulthood, internalizing and

externalizing disorders are also found to have elevated rates of co-occurrence with BPD, which may be partially due to the strikingly similar risk factors shared between BPD and other disorders (Sharp & Wall, 2018).

From a dynamic mutualism perspective, the unique genetic and environmental vulnerabilities, including the influence of preceding psychopathology, are captured within the carrying capacity, or **K** parameters. As such, it was hypothesized that individuals with a higher number of genetic and/or environmental predispositions (as denoted by a larger K_i) would have an increased risk of developing at least one BPD symptom. In contrast, individuals with smaller carrying capacities were expected to have a reduced risk of developing BPD and to display less severe symptoms. Although the following was not tested, it was further hypothesized that individuals with larger carrying capacities would be at an increased risk of developing other psychopathology, as many genetic and environmental risk factors associated with BPD are shared with other conditions (Eaton et al., 2011; Sharp & Wall, 2018).

To test whether individual differences in **K** accounted for variability in the likelihood of developing BPD, the trajectories of four random subjects were first plotted using identical parameter values and assumptions as Simulation 1. These trajectories can be found in Figure 3. Next, the simulation was updated and the means of K_i for the same four subjects were reduced from 3 to 1. Setting the mean of K_i to 1 led to estimation issues, and the means were slowly increased until the model converged. A mean of 1.80 was the lowest possible value that facilitated model convergence. The results of this simulation are displayed in Figure 4 and indicated that decreasing K_i led to substantially lower growth for all subjects.

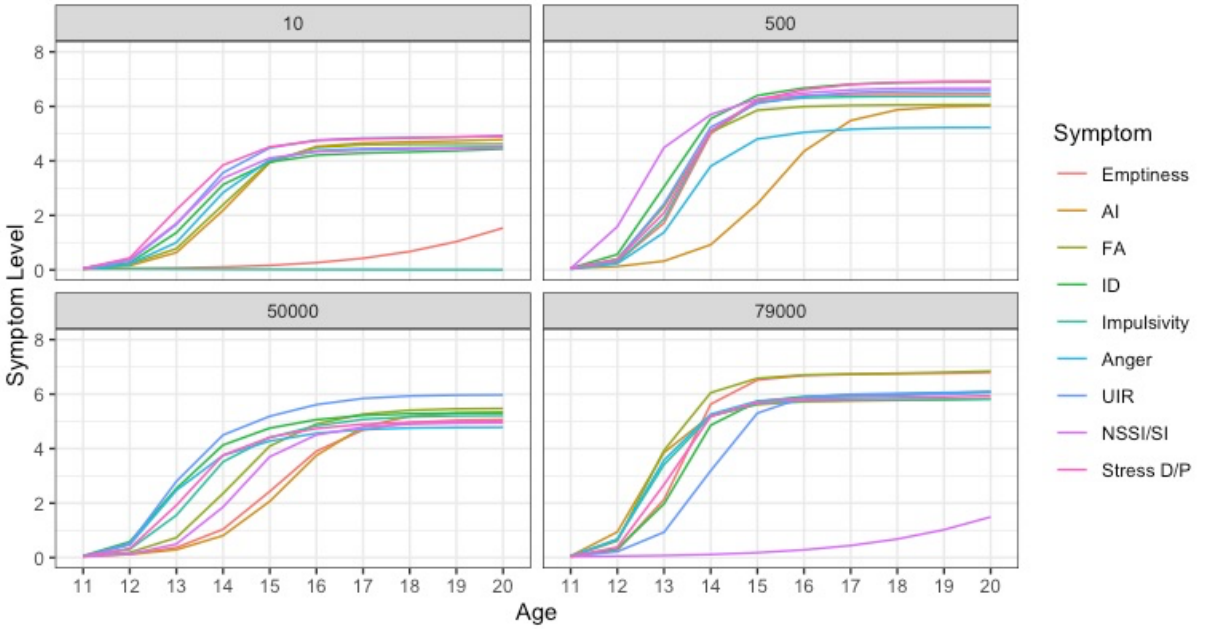

Figure 3. Symptom Trajectories of Four Random Subjects.

Note. AI = affective instability; FA = fear of abandonment; ID = identity disturbance; UIR = unstable interpersonal relationships; NSSI/SI = non-suicidal self-injury/suicidal ideation; Stress D/P = stress-induced dissociation/paranoia.

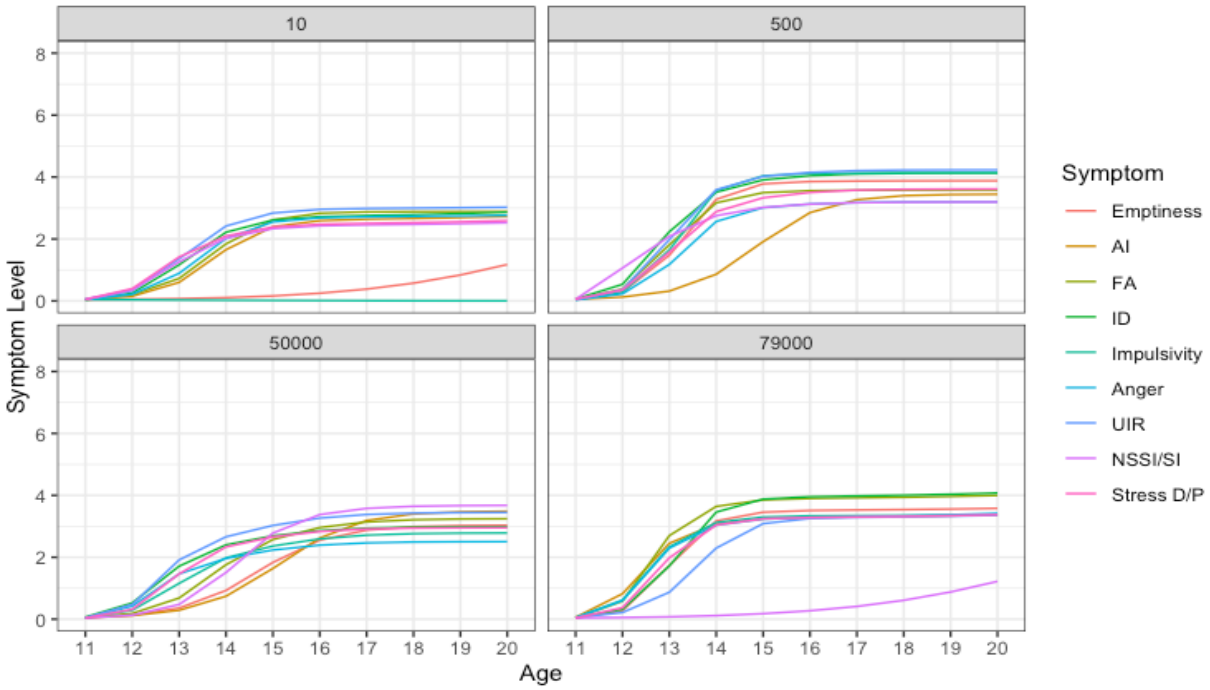

Figure 4. Data of Four Random Subjects with Modified Values for K.

Note. AI = affective instability; FA = fear of abandonment; ID = identity disturbance; UIR = unstable interpersonal relationships; NSSI/SI = non-suicidal self-injury/suicidal ideation; Stress D/P = stress-induced dissociation/paranoia.

Less variability in the individual growth trajectories of most symptoms was also observed.[8] Overall, this adaptation confirmed that parameters **K** significantly influence symptom trajectories, with larger carrying capacities leading to more severe symptomology. Although this simulation was based on the trajectories of only four subjects, similar logic can be applied to explain why some groups of individuals develop clinical-level BPD symptoms, whereas the majority develop little to no symptoms. This can be achieved by specifying two or more unique sets of growth parameters that correspond to distinct subpopulations (e.g., clinical sample, community sample).

For illustration purposes, Simulation 2 was repeated, except different parameter values were used for two distinct groups of subjects. The first 50,000 subjects represented a clinical population, while the subsequent 50,000 subjects represented individuals from a community sample. For simplicity, we utilized identical parameter values for the "clinical group" as Simulation 2 (K_i mean = 3, r = .25; a_i mean = 2, SD = 1; all M_{ij} set to 0.05, SD range: 0.01 – 0.04). Conversely, a_i and K_i for each BPD symptom in the "community group" were sampled from a multivariate normal distribution with a mean of 1 (SD = 0.50) and 0.50 (SD = 0.10), respectively. The M_{ij} weights were identical to the clinical group, though could have been adjusted if desired.[9]

The trajectories of four randomly selected subjects (two from the clinical group and two from the community group) are presented in Figure 5. The BPD trajectories for the entire subpopulations can be found in Figure 6.

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[8] The initial growth observed across symptoms is accounted for by parameters **a**, which had a mean of 2 and a SD of 1. Consequently, if a particular symptom is suspected to have minimal to no growth for a given person, the value of a_i is expected to be low, with the carrying capacity for that symptom also correspondingly low.

[9] When implementing these changes, some of the initial parameter values led to model convergence errors and were updated to more appropriate values. Of note, values were selected arbitrarily, and other values could have been used to achieve similar results.

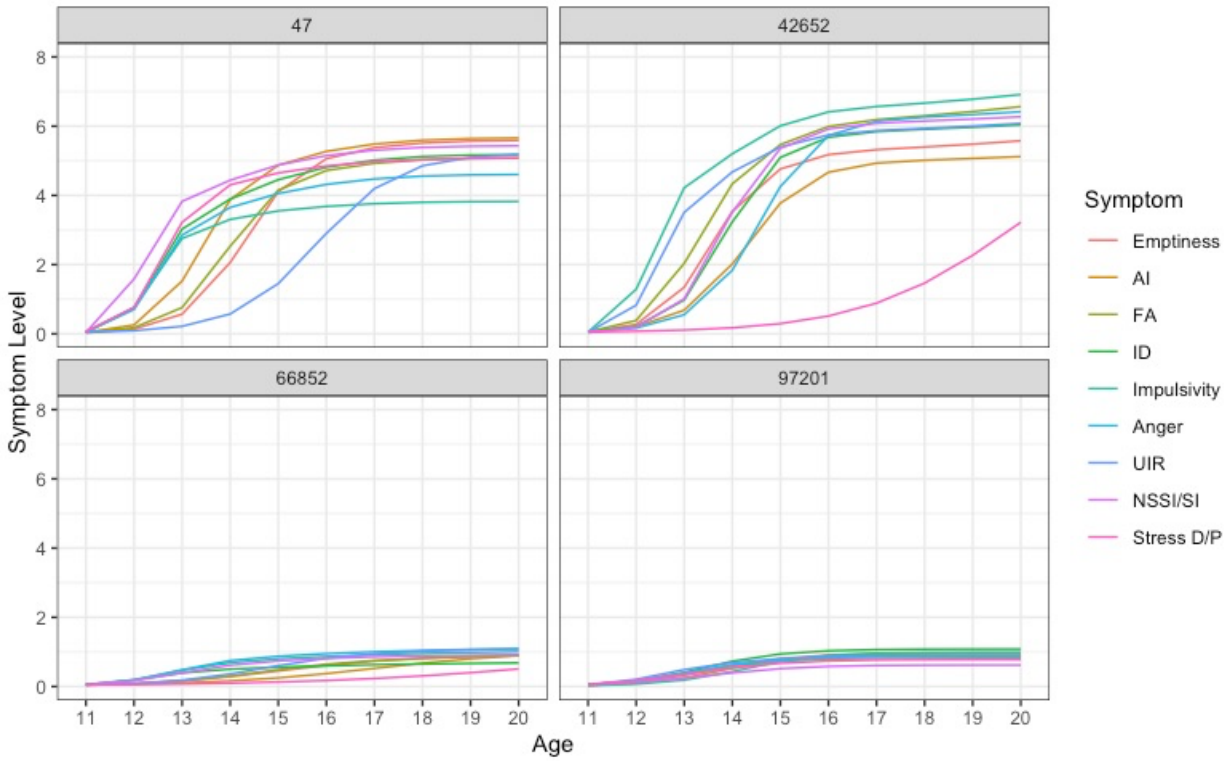

Figure 5. Trajectories of Randomly Sampled Subjects from Both Groups.

Note. AI = affective instability; FA = fear of abandonment; ID = identity disturbance; UIR = unstable interpersonal relationships; NSSI/SI = non-suicidal self-injury/suicidal ideation; Stress D/P = stress-induced dissociation and paranoia.

Overall, group differences in the development of BPD symptoms were clear. Subjects from the "community group" demonstrated extremely small levels of growth with the opposite pattern of results found for the "clinical group." The minor amount of growth that was observed for the community sample is attributable to the small a_i and K_i values, which were necessary for model convergence. However, if the model could be estimated with both growth parameters set to 0, then no growth would be expected. In comparison, the clinical sample demonstrated steeper elevations in BPD symptoms that steadily increased over time. Therefore, despite having similar M_{ij} weights in both groups, differences in parameters **K**, and to a lesser extent parameters **a**, significantly influenced the trajectories of these symptoms. As such, the formalized model appeared to successfully account for individual differences in the propensity to developing BPD by introducing greater variability in the growth parameters.

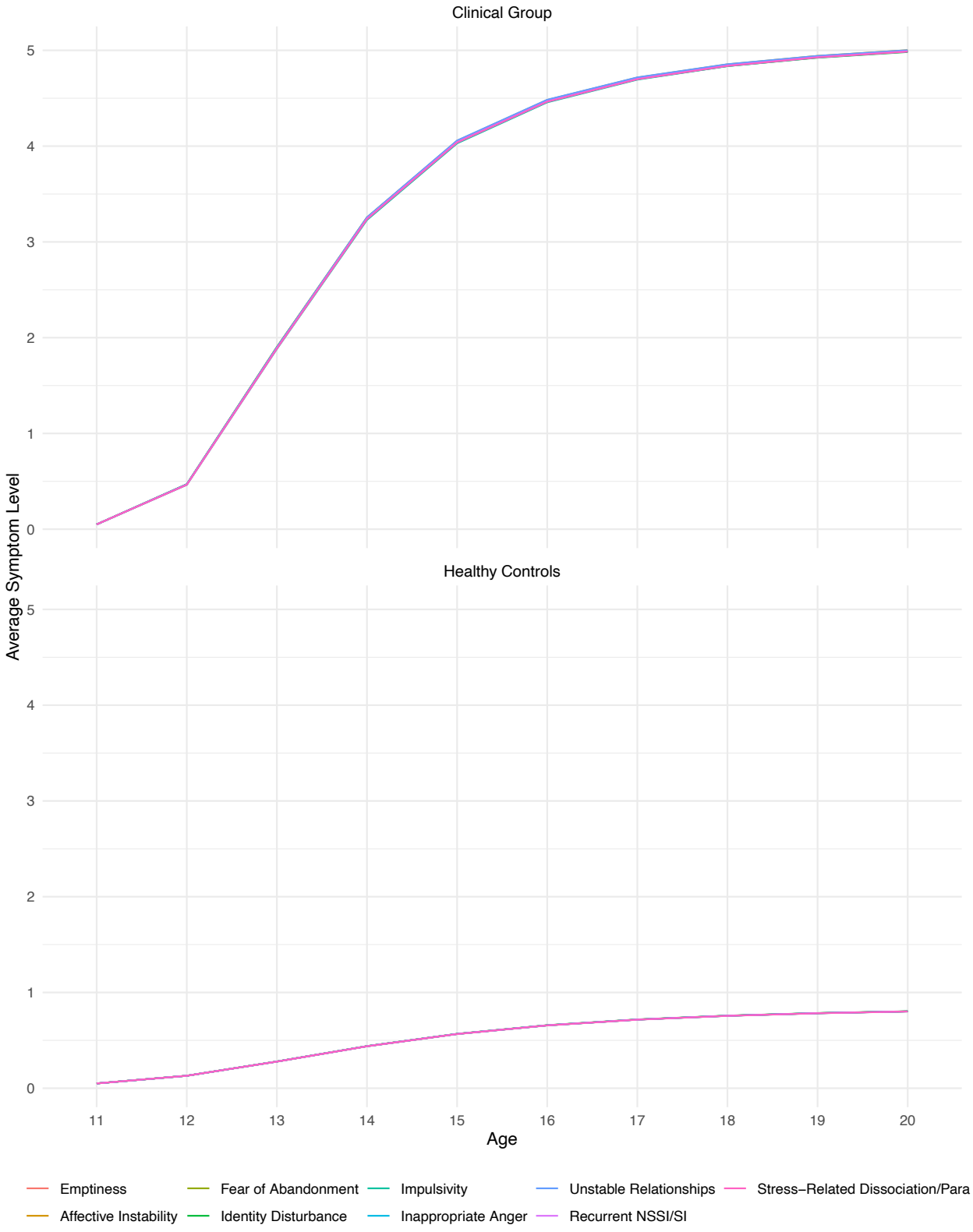

Figure 6. BPD Symptom Trajectories for Healthy and Clinical Samples.

Simulation 3: Variability Between BPD Symptoms and Differences in Centrality

The simulations conducted insofar have incorporated variability in matrix **M** via random sampling. However, this has been done under the assumption that M_{ij} weights are equivalent across symptom pairs. This implies that the M_{ij} for "affective instability" to "identity disturbance" has the same numerical value as the M_{ij} weight for "chronic feelings of emptiness" to "recurrent non-suicidal self-injury/suicidal ideation." Nonetheless, the available evidence indicates that BPD symptoms do not have equivalent influences on each other, and some symptoms may contribute more to the development and/or maintenance of other symptoms (Woods et al., 2020). Therefore, the goal of Simulation 3 was to determine if the formal model could account for asymmetrical relationships among BPD symptoms, including the possibility that some symptoms exert a greater influence on the development of other symptoms.

Affective instability is typically considered the hallmark feature of BPD and is the strongest and most consistent predictor of future BPD symptoms (Stepp et al., 2014; Tragesser et al., 2010; Tragesser et al., 2007). Affective instability is robustly correlated with impulsivity, identity problems, interpersonal difficulties, and recurrent NSSI/SI (Conklin et al., 2006; Koenigsberg et al., 2001; Peters et al., 2016), and is one of the most influential symptoms in network analytic studies (Peters et al., 2023; Richetin et al., 2017). Although generalizing the above findings to the current study is challenging due to their cross-sectional nature, they provide some support for certain BPD symptoms having a more important role in the onset, maintenance, and progression of other symptoms. Accordingly, weights in matrix **M** were changed to reflect variability in the relationships between symptoms and to acknowledge that certain symptoms may have a greater influence on the development of other symptoms than vice versa (see Table 1).

Based on the available literature,[10] affective instability, identity disturbance, and intense, inappropriate anger were considered the more influential symptoms that may promote the development or maintenance of other BPD symptoms. Affective instability was posited to be the most influential symptom based on existing theoretical models (e.g., biosocial theory) and empirical research (Linehan, 1993; Tragesser et al., 2007), and was assigned a weight of 0.15. Identity disturbance and intense, inappropriate anger were hypothesized to be the second most influential symptoms and were assigned weights of 0.10.

Symptoms that were hypothesized to have at least moderate influence on other symptoms included chronic feelings of emptiness, fear of abandonment, impulsivity, and unstable interpersonal relationships. These symptoms were assigned a weight of 0.05, except for weights related to affective instability, identity disturbance, and inappropriate anger. Weights from moderately influential symptoms to highly influential symptoms were assigned a weight of 0.02, as the most central symptoms were hypothesized to be influenced by other symptoms at a lesser rate. For example, the M_{ij} weight representing the relationship of unstable interpersonal relationships to affective instability was 0.02, while the M_{ij} for unstable interpersonal relationships to impulsivity was 0.05.

Symptoms including recurrent NSSI/SI and stress-related paranoia and dissociation were hypothesized to have little influence on the development of other symptoms and were assigned a small weight of 0.01. This decision was based on empirical evidence indicating that symptoms such as affective instability play a significant role in the development and maintenance of NSSI and suicidal behavior among individuals with BPD (Reichl & Kaess, 2021). Relatedly, stress-

[10] Due to the absence of longitudinal data documenting how BPD symptoms evolve and influence each other over time, establishing the causality or sequence of these relationships remains challenging. Thus, several M_{ij} weights were assigned a value based on theoretical rather than empirical reasoning and are meant to provide a preliminary starting point that can be adjusted if needed.

related dissociation/paranoia was hypothesized to be primarily activated by external events rather than other BPD symptoms, implying that its influence on other symptoms should be relatively weak.

Table 1. Updated Weights for Matrix M.

		E	AI	FA	ID	IMP	ANGER	UIR	NSSI/SI	D/P
		x1	x2	x3	x4	x5	x6	x7	x8	x9
E	x1	0.05	0.15	0.05	0.10	0.05	0.10	0.05	0.01	0.01
AI	x2	0.02	0.05	0.02	0.10	0.02	0.10	0.02	0.01	0.01
FA	x3	0.05	0.15	0.05	0.10	0.05	0.10	0.05	0.01	0.01
ID	x4	0.02	0.15	0.02	0.05	0.02	0.10	0.02	0.01	0.01
IMP	x5	0.05	0.15	0.05	0.10	0.05	0.10	0.05	0.01	0.01
ANGER	x6	0.02	0.15	0.02	0.10	0.02	0.05	0.02	0.01	0.01
UIR	x7	0.05	0.15	0.05	0.10	0.05	0.10	0.05	0.01	0.01
NSSI	x8	0.05	0.15	0.05	0.10	0.05	0.10	0.05	0.05	0.01
D/P	x9	0.05	0.15	0.05	0.10	0.05	0.10	0.05	0.01	0.05

Note. Weights in this table are intended to read from top to bottom. For example, the influence of affective instability to emptiness is 0.15, while the influence of emptiness to affective instability is 0.02. The diagonal values, which reflect the influence that a given symptom has on itself, were all set to 0.05, suggesting a moderate degree of self-regulation. Similar to the initial simulation, weights were randomly sampled with a standard deviation ranging between 0.02 and 0.06. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = Impulsivity in at least 2 areas; ANGER = intense, inappropriate anger; UIR = unstable interpersonal relationships; NSSI/SI = recurrent non-suicidal self-injury/suicidal ideation; D/P = stress-induced dissociation and paranoia.

Based on the values in Table 1, M_{ij} were randomly sampled for each subject with a *SD* ranging from 0.02 to 0.06. Diagonal values, which are conceptually akin to an autocorrelation or self-feedback loop, were set to 0.05 for all symptoms and subjects. This sampling procedure enabled M_{ij} to vary across individuals while ensuring that symptoms had at least some degree of self-regulation. These updated weights were then used to simulate data for a new sample of 100,000 subjects. Remaining parameters (e.g., $x0_i$, a_i, and K_i) remained identical to the preceding simulation. The results of this simulation are shown in Figure 7. Findings were surprisingly

similar to the prior simulations apart from a slight increase in variability in the growth trajectories and relationships among BPD symptoms.

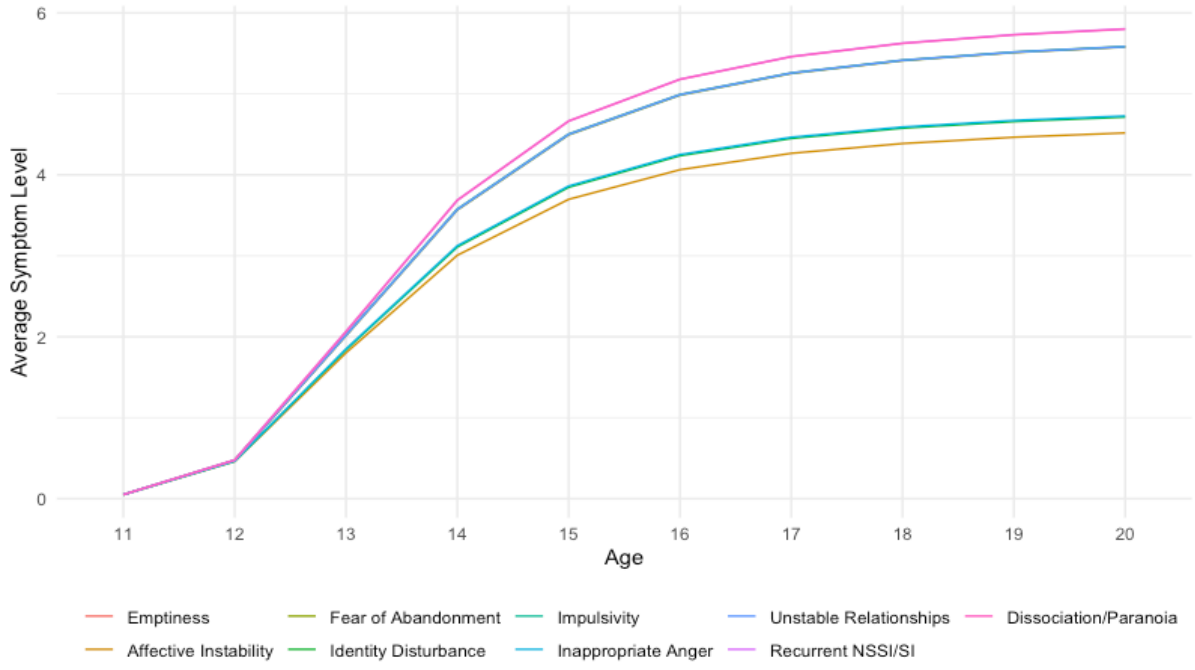

Figure 7. Average Symptom Trajectories Based on the Updated M_{ij} Weights.

To better assess differences in the symptom relationships themselves, correlation matrices at ages 11, 15, and 20 were computed from the current simulation and Simulation 1. These values were then subtracted from one another to provide a straightforward assessment of how changes in **M** impact the overall correlations between symptoms. Differences in correlations can be found in Figure 8, with negative values meaning that the simulation with the updated M_{ij} weights resulted in a larger correlation than Simulation 1. At age 11, correlations were identical, which was expected considering that no growth should have occurred. Conversely, ages 15 and 20 were found to have minor differences, particularly for correlations related to affective instability at age 15. Additionally, the strength of the correlations related to identity disturbance and intense, inappropriate anger, experienced some increases, particularly at age 15.

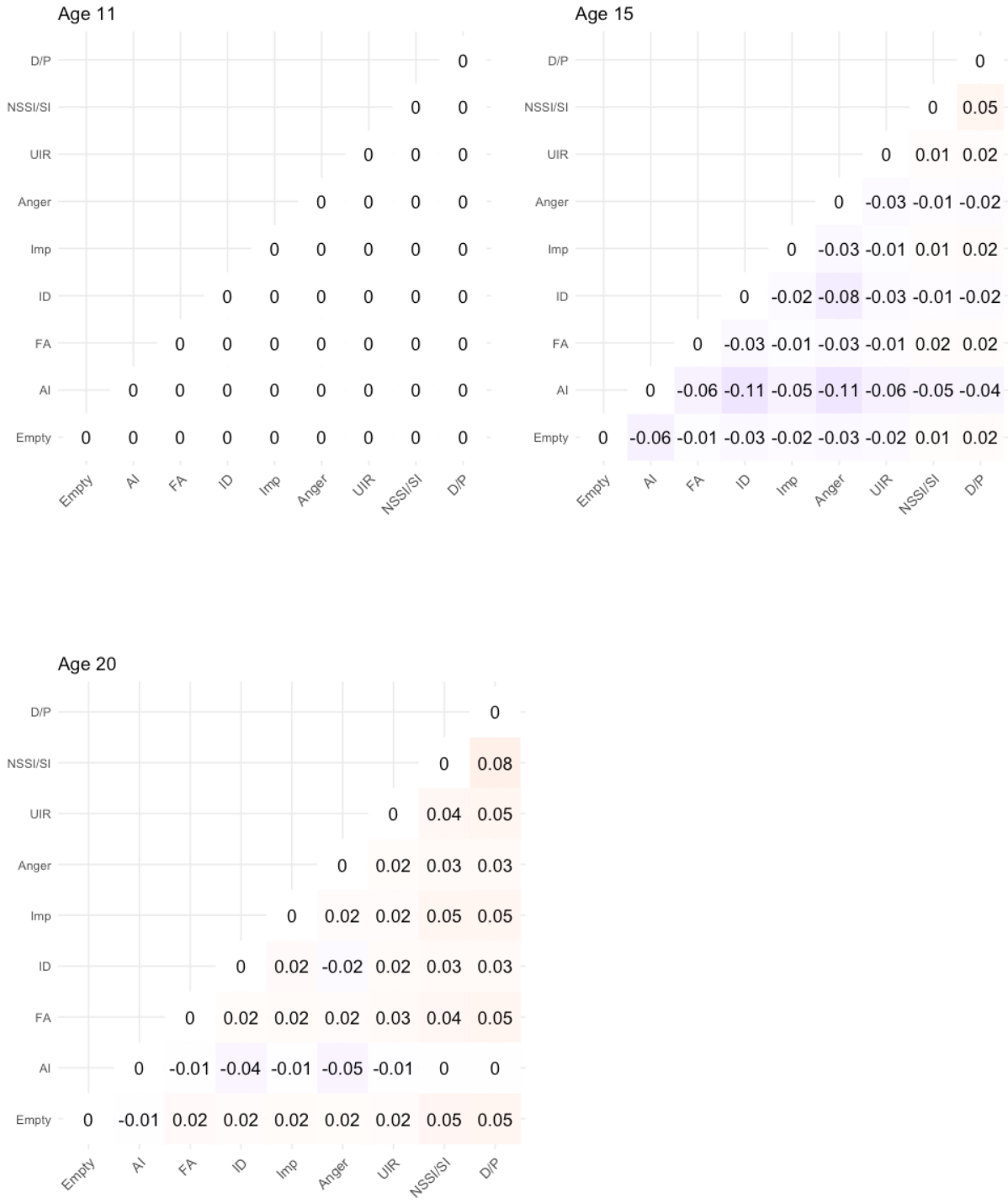

Figure 8. Difference in Correlations Between Simulations.

Note. Empty = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; Imp = Impulsivity; UIR = unstable interpersonal relationships; NSSI/SI = non-suicidal self-injury/suicidal ideation; D/P = stress-induced dissociation and paranoia.

Collectively, these findings indicated that the model could accommodate variability in the relationship between symptoms fairly well. This simulation also demonstrated that increasing variability in M_{ij} does not appear to significantly alter the symptom trajectories or their correlations, at least when examined at the aggregate level. Therefore, an important question becomes what additional changes may be warranted to increase variability between symptoms. The answer lies primarily in parameters **a**, which were originally sampled across symptoms with a mean of 2 and *SD* of 1. Although the simulation allows subjects to have different a_i values, allowing BPD symptoms to have varying a_i means fosters increased variability in the initial trajectories of symptoms, as well as the strength of their interactions. This is because a_i not only controls the initial growth rate, but also influences the synergetic properties between symptoms that are captured in matrix **M**. To demonstrate this concept, a supplemental simulation was implemented where the means of a_i ranged from 1–2.5, with the *SD* remaining at 1. Results are displayed in Figure 9 and show how allowing the a_i means to differ can lead to greater variability in the overall symptom trajectories.

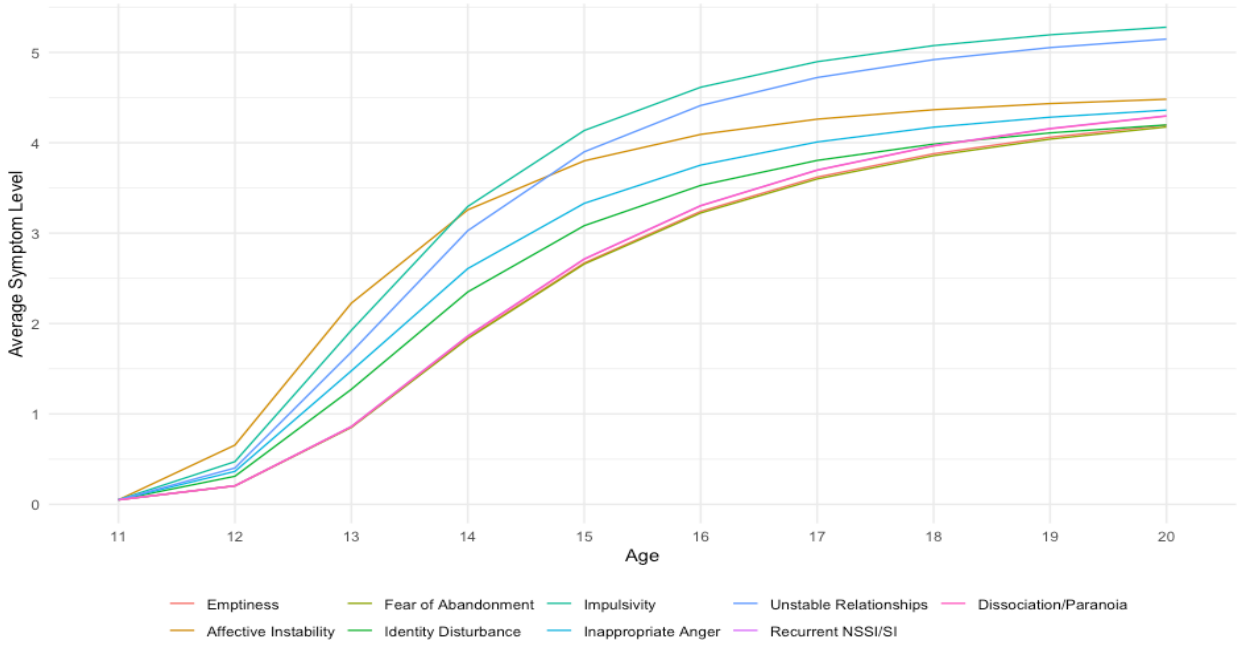

Figure 9. Changes in Growth Trajectories with Updated M_{ij} Weights.

The observed correlations and eigenvalues were also plotted to better visualize the increased variability achieved by modifying M_{ij} and a_i (see Figure 10). Comparably weak correlations were observed at the first time point (i.e., age 11), with correlations increasing in size at ages 15 and 20. The histograms suggested greater variability in the relationships between symptoms, such that the cross-sectional correlations fluctuated at a higher rate compared to previous simulations. Of note, while the means of a_i were arbitrarily selected, the means could have been weighted based on theoretical predictions. For example, more central symptoms, such as affective instability, could have been hypothesized to have a larger a_i. This implies that its initial growth is influenced to a higher extent by external processes relative to other BPD symptoms. Indeed, symptoms such as affective instability and impulsivity are often viewed as important vulnerabilities to the development of subsequent symptoms, providing indirect support for the possibility of these symptoms to have a larger a_i (Crowell et al., 2009). In other words, these symptoms may be more likely to have an earlier onset and greater acceleration compared to other symptoms.

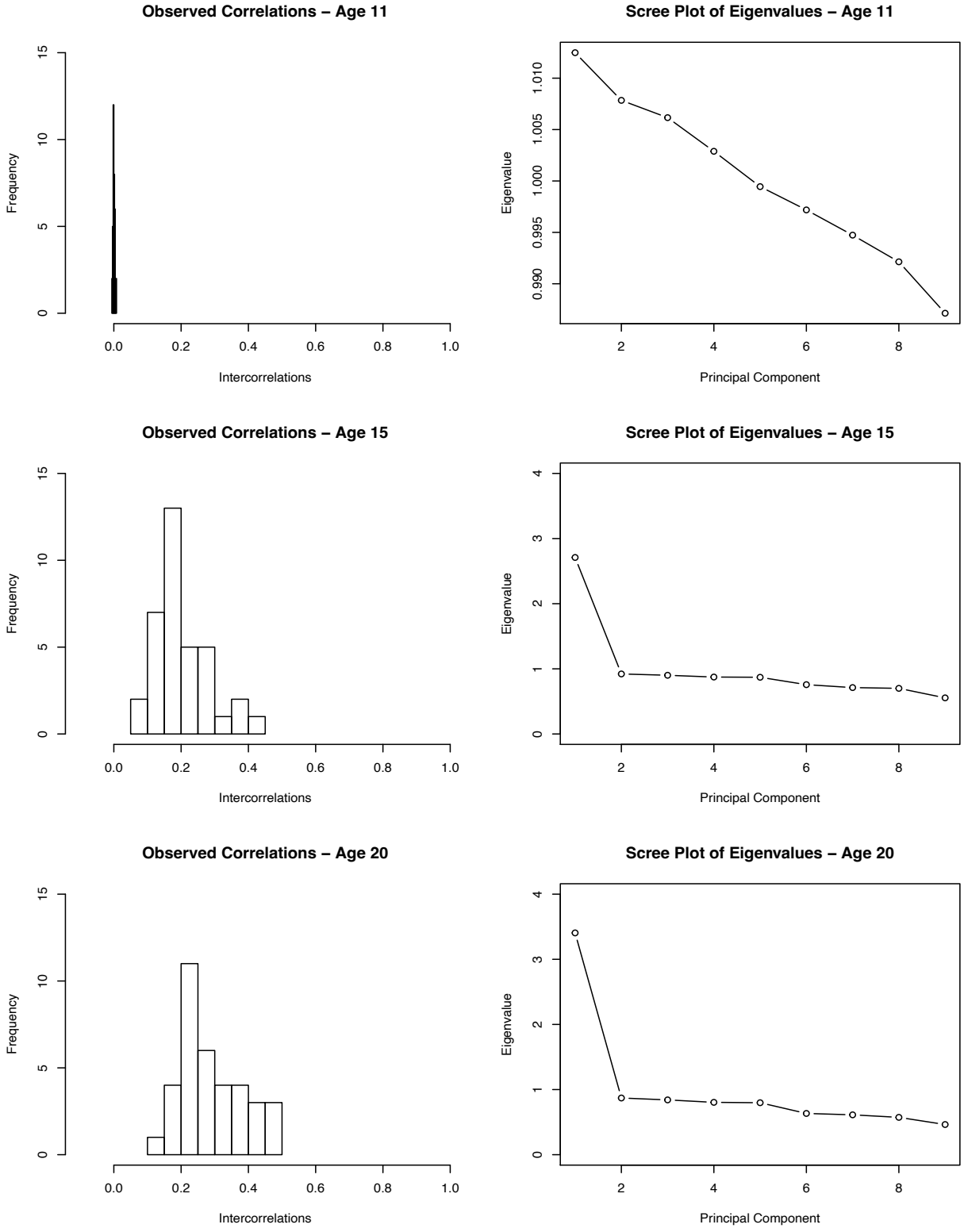

Figure 10. Observed Correlations and Eigenvalues with Updated a_i and M_{ij}.

Simulation 4: Variability in the Onset of BPD Symptoms

Despite the rise in developmental studies examining BPD at younger ages, the specific onset of these various symptoms remains unclear. Previous studies have indicated that affective instability is often one of the first symptoms to emerge (Zanarini et al., 2011); however, the lack of developmental research in this area makes it difficult to determine if symptoms meaningfully vary in their age of onset. Nonetheless, if BPD symptoms are assumed to emerge at different ages, this can be mathematically accounted for by setting the means of parameters **a** and **K** to a small number and increasing these values during the point of development in which the symptom is thought to be expressed.

To ensure this logic was mathematically replicable, Simulation 4 was conducted such that all symptoms were modeled to have fairly similar growth trajectories except for stress-induced paranoia/dissociation. The a_i, or growth rate, of stress-induced paranoia/dissociation was set to 0.50 with a *SD* of 0.10, while the means and *SDs* of remaining symptoms were set to 1.50 and 1, respectively. Next, the a_i for stress-induced paranoia/dissociation was updated at $t = 6$ (i.e., age 16) to mimic this symptom having a delayed onset relative to other BPD symptoms. Results are shown in Figure 11 and confirmed that the initial growth of stress-induced dissociation/paranoia was low relative to other symptoms. By age 16, this symptom had clear accelerations in growth that were substantially steeper compared to the other symptom trajectories. This provided some support for the model's capacity to allow symptoms to emerge at varying points in development by manipulating the growth parameters at different time periods.

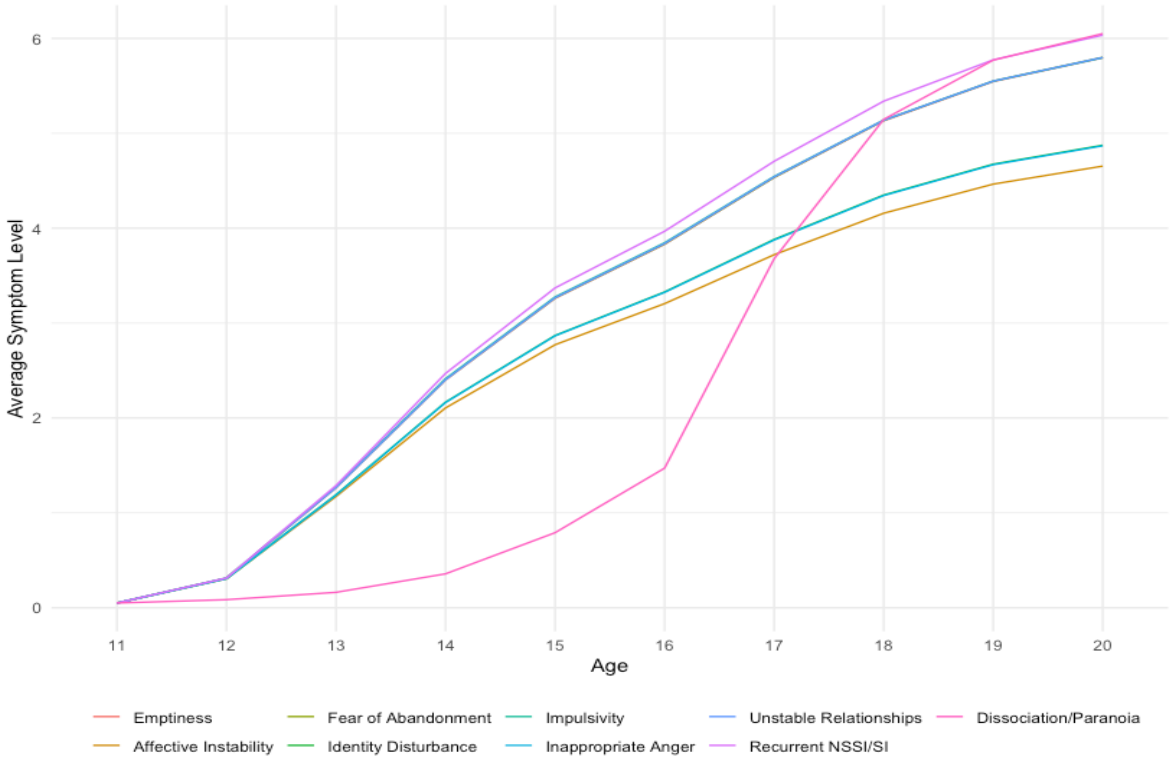

Figure 11. Manipulating the Onset of Select Symptoms.

Simulation 5: Symptom Declines in Adulthood

Research on BPD suggests that several individuals who exhibit BPD symptoms in adolescence will "age out" of these traits by early adulthood (Sharp & Rossouw, 2019). Accordingly, it is worth examining whether this type of growth could be explained by the formal mutualism model of BPD. In Simulation 5, this possibility was tested by manipulating select parameter values at different time points. To increase overall variability between symptoms, the mean of a_i ranged from 0.50 to 2.50, K_i were sampled from a multivariate correlated distribution (r = .25) with a mean of 3, and M_{ij} were based on the values in Table 1. At t = 6, new parameter values for a_i and K_i, were incorporated. Of note, this time point was selected for introducing new parameter values to ensure there was enough time for any symptom decline to occur. The means of a_i were changed to 0.50 with a *SD* of 0.01, with the means of K_i now set to 2.50 with a

correlation of 0.10.[11] The resulting data for all 100,000 subjects is presented in Figure 12. The simulation indicated that stress-induced paranoia/dissociation grew at a substantially lower rate compared to all other BPD symptoms. Around age 16, the growth of most symptoms declined except for chronic feelings of emptiness. These differences in trajectories are largely due to variability that was introduced in parameters **a**, with the decline in symptom severity mostly attributable to changes in the **K** parameters. Although some decline was observed, the model clearly underperforms when tasked with modeling negative linear growth in symptoms.

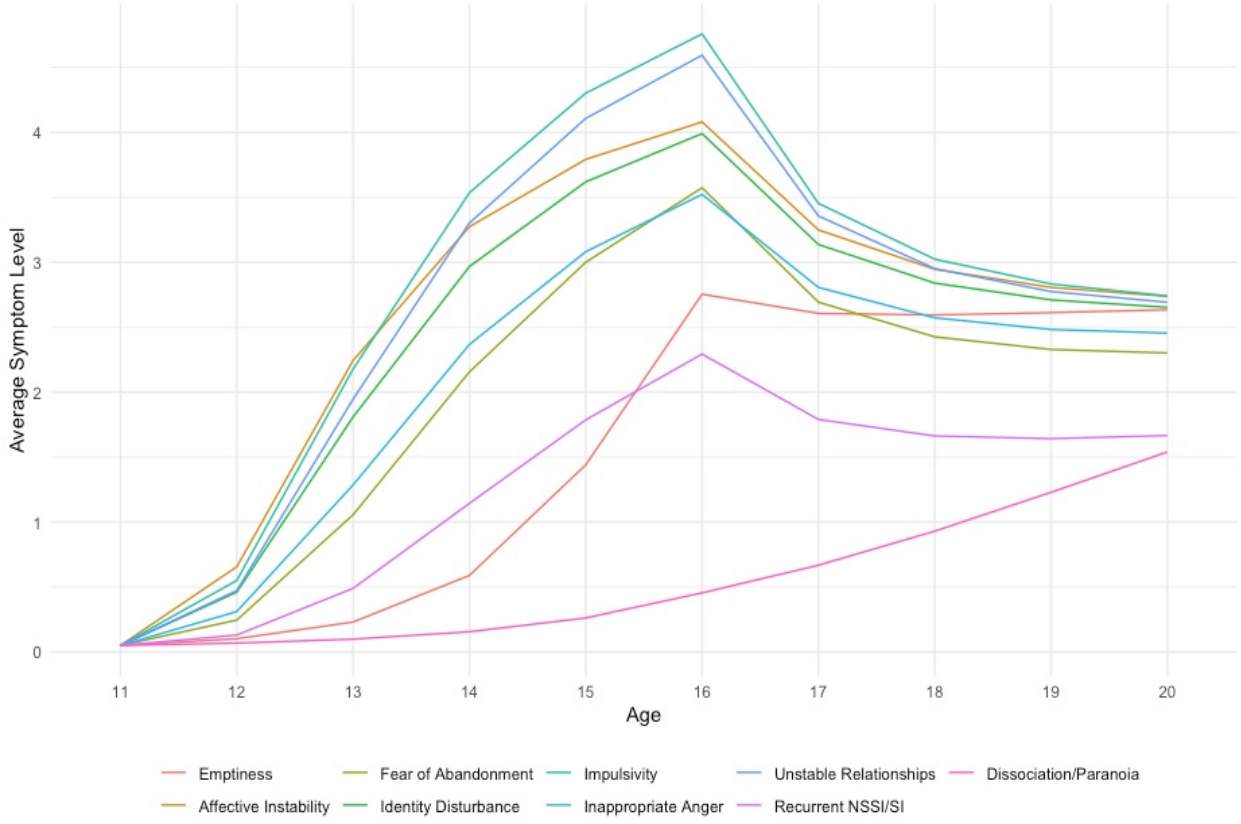

Figure 12. Variability in Symptom Onset and Average Trajectories (N=100,000).

[11] Of note, these values were selected arbitrarily, and other values could have been. Parameters **K**, however, required some additional attention in selecting appropriate values, as means lower than 2.5 led to significant instability and lack of model convergence.

As a supplemental analysis, a related simulation was conducted using the same parameter values as the above simulation, with the exception that the M_{ij} weights at $t = 6$ were specified to be considerably smaller than the original M_{ij} weights. Surprisingly, reducing M_{ij} at $t = 6$ led to negligible changes, and the resultant trajectories were identical to the symptom trajectories found in Figure 12. This indicates that reducing the interaction weights once symptoms have already experienced some degree of growth does not significantly impact the overall symptom trajectories. In other words, changes in the behavior of the model during later periods of development appear to be primarily driven by K_i rather than M_{ij}.

Simulation 6: Symptom Reduction in Response to Behavioral Intervention

In the final simulation, the ability of the mutualism model to account for change in symptoms after receiving treatment was investigated. With respect to treatment, we focus specifically on DBT, as DBT is widely regarded as the gold standard treatment for BPD. The primary goal of DBT is to reduce the core symptoms of BPD, such as impulsivity and emotion dysregulation, while simultaneously improving distress intolerance and interpersonal skills (Linehan, 1999). From a mutualism perspective, reducing the connectivity between BPD symptoms should theoretically lead to decreases in the overall symptom severity. In Simulation 6, we investigated this possibility by examining whether the formalized mutualism model could adequately account for treatment effects. To test this scenario, data was generated for only a single individual. For the purpose of this example, M_{ij} were deliberately selected in such a way that affective instability had the strongest associations with all other symptoms, followed by impulsivity and unstable interpersonal relationships. a_i were sampled from an uncorrelated normal distribution with a mean of 2 and *SD* of 1, and K_i were drawn from a correlated distribution with a mean of 3 ($r = 0.25$). Results for the single subject are presented in Figure 13.

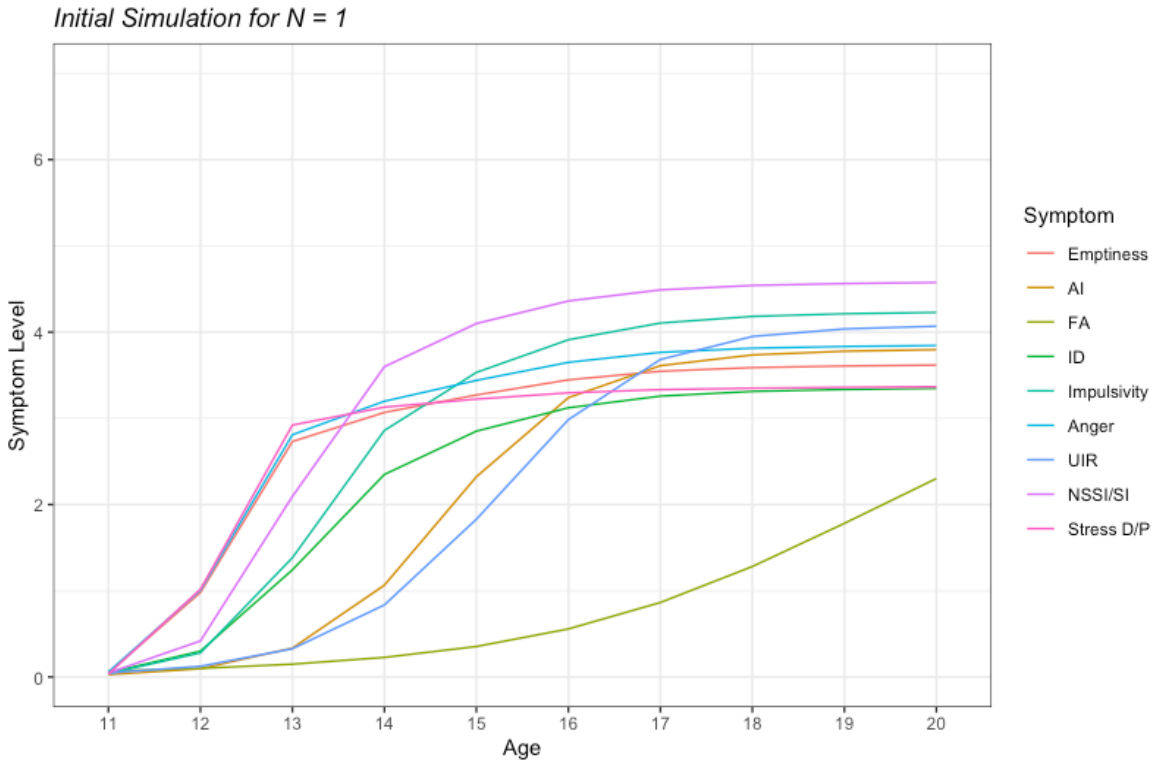

Figure 13. Initial Growth Trajectories for One Subject.

Note. AI = affective instability; FA = fear of abandonment; ID = identity disturbance; UIR = unstable interpersonal relationships; NSSI/SI = non-suicidal self-injury/suicidal ideation; Stress D/P = stress-induced dissociation and paranoia.

After simulating the initial data, model parameters were manipulated to serve as a proxy for DBT treatment. Although DBT can have positive impacts on multiple symptoms, we focused on how the model changes when weights of central symptoms are reduced after some development has taken place. Consequently, the simulation was initially updated to include new M_{ij} weights at $t = 4$. The new M_{ij} values were identical to the original values, except the M_{ij} weights between affective instability to all other symptoms were reduced. The results are presented in the upper-left panel of Figure 14. Compared to the initial trajectories (see Figure 13), changing the M_{ij} weights related to affective instability at $t = 4$ resulted in minor decreases in the overall growth trajectories. Similar to what was documented in Simulation 5, this suggested that solely reducing the strength of the M_{ij} weights does not appear to produce significant decreases in the overall growth trajectories.

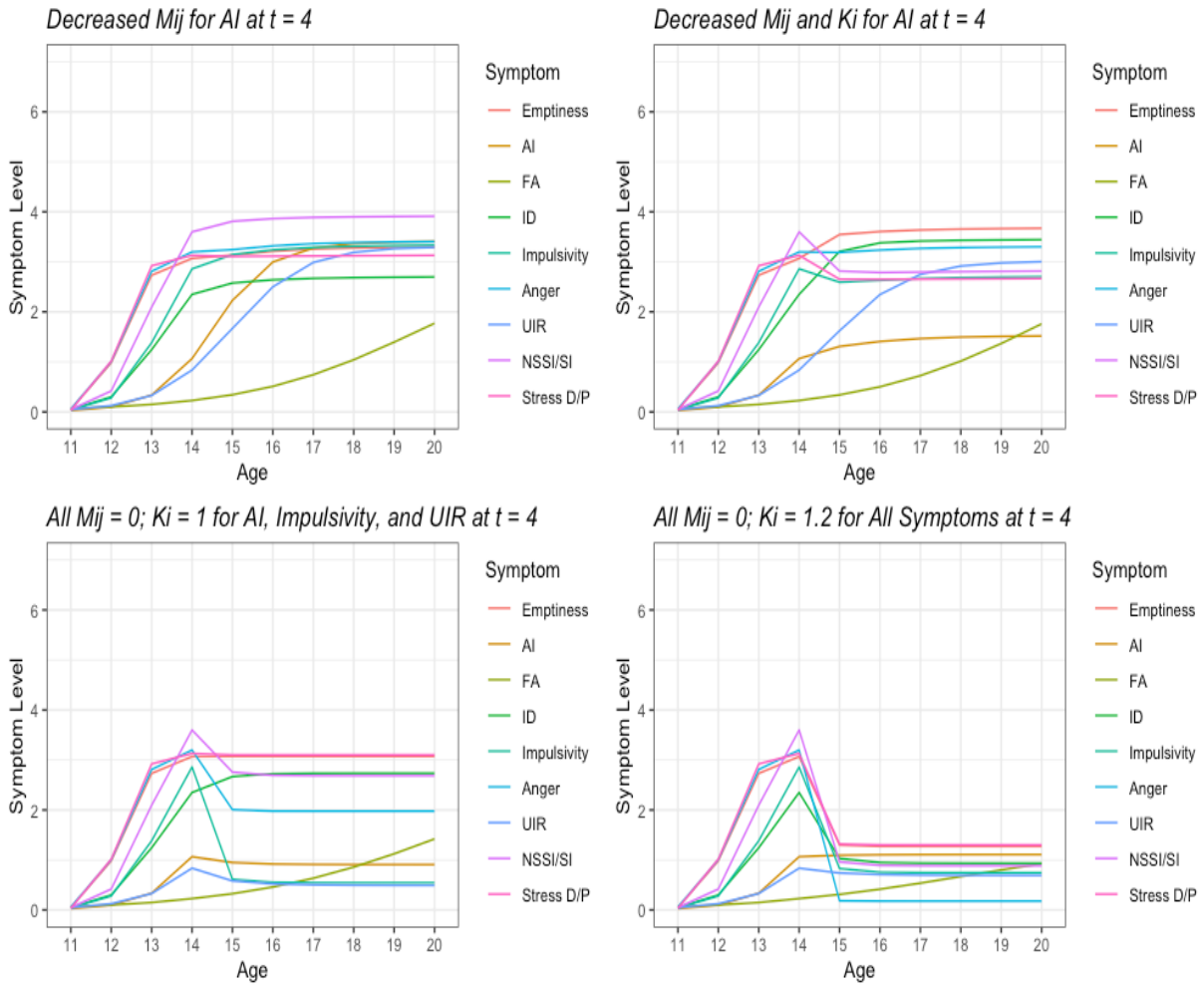

Figure 14. Changes in Symptom Trajectories Across Four Scenarios.

Note. AI = affective instability; FA = fear of abandonment; ID = identity disturbance; UIR = unstable interpersonal relationships; NSSI/SI = non-suicidal self-injury/suicidal ideation; Stress D/P = stress-induced dissociation and paranoia.

To further document the behavior of the model in response to treatment, another iteration of this simulation was conducted. The same parameter values were used except the mean of K_i for affective instability at $t = 4$ was set to 1 instead of 3. This implies that the association between affective instability and other symptoms—in addition to its total genetic and environmental vulnerability—was reduced at the fourth time point. Results are displayed in the upper right-hand corner of Figure 14 and indicated decreased growth for affective instability and NSSI/SI. The severity of the remaining symptoms decreased only slightly. This suggested that to

achieve more realistic treatment effects, further changes to the model are needed. As a result, two other scenarios were examined below to determine if the model could reasonably account for treatment effects with additional changes.

In the first supplemental scenario, M_{ij} for symptoms with moderate to high influence (i.e., affective instability, impulsivity, and unstable interpersonal relationships), were set to 0 at $t = 4$. The mean of K_i associated with these symptoms was similarly reduced at $t = 4$ and set to 1. The results are displayed in the lower-left quadrant of Figure 14 and indicated that all symptoms decreased in severity over time. Affective instability, intense anger, impulsivity, unstable interpersonal relationships, fear of abandonment, and NSSI/SI experienced the most change, while chronic feelings of emptiness and stress-related dissociation/paranoia exhibited minimal change overall.

To examine whether trajectories differed when parameters **M** and **K** were changed for all symptoms, a final simulation was implemented in which M_{ij} and K_i at $t = 4$ were changed to 0 and 1, respectively, for all symptoms. However, this model failed to converge, and the means of K_i were slightly increased to 1.20, leading to proper convergence. The results of this simulation are displayed in the bottom-right hand corner of Figure 14. All symptoms exhibited very clear declines in their trajectories between the fourth and fifth time point. Taken together, Simulation 6 suggested that targeting environmental and genetic factors may be more fruitful in decreasing the overall severity of BPD relative to targeting specific symptom associations.

Reviewing Explanatory Adequacy of the Formal Model

The simulations conducted to this point were designed to assess the explanatory adequacy of the formalized model. Evaluating the adequacy of the formalized theory corresponds to the fourth step of the TCM and assess whether the model in question explains the empirical

phenomena of interest. In the present study, the mathematical model was implemented via computer simulations and was found to successfully produce a positive manifold amongst BPD symptoms. By manipulating various assumptions and parameter values, the ability of the model to account for several key features of BPD was also explored. In the following section, we extend our evaluation of the formalized mutualism model by examining its overall worth and utility based on the TCM recommendations (Borsboom et al., 2021).

CHAPTER FOUR: EVALUATING THE UTILITY OF THE FORMAL THEORY

In the fifth and final stage of the TCM, the overall worth or utility of the formal theory is assessed. The goal of this step is not merely to show that the theory can explain the target system; rather, it is to assess the quality of the theory and determine whether further refinements are necessary. Although several metrics may be suitable for evaluating the overall utility of a formal theory, the TCM stresses the value of assessing explanatory breadth and precision (Borsboom et al., 2021; Robinaugh et al., 2021).

Explanatory Breadth

Evaluating the explanatory breadth of a theory often involves considering its ability to explain additional phenomena that are anticipated to emerge from the target system (Borsboom et al., 2021). For example, researchers may wish to examine the extent that a theory can explain an array of diverse phenomena, and whether these explanations are transferable to different contexts. As discussed in Chapter Three, the formal mutualism model of BPD provided a tenable explanation for the positive manifold of BPD symptoms by successfully reproducing strong positive correlations amongst its symptoms. Moreover, by manipulating different parameters and assumptions, we tested whether the formal model could adequately explain or reproduce several associated elements of the disorder. The model's capacity to explain these features is reviewed below.

Differences in the propensity to developing BPD

The second simulation investigated whether the model could account for between-person differences in the propensity to developing BPD. This was tested by examining the behavior of

the model after manipulating certain growth parameters. The results of this simulation indicated that individuals with higher K_i values had substantially more growth across symptoms, suggesting that greater exposure to genetic or environmental risk factors led to more severe BPD symptoms. This finding aligns with our original hypothesis and is congruent with other theories of BPD that highlight the significance of these vulnerabilities in predicting future increases in symptoms. (Chapman et al., 2017). Notably, the initial starting values (x0) and M_{ij} weights need not be different to achieve these results, reiterating the importance of the growth parameters.

Although the results of this simulation were encouraging, it is worth mentioning that the formalized model was unable to account for the possibility that some individuals may have zero growth. In simpler terms, setting the growth parameters to zero for certain individuals led to convergence issues due to the logistic nature of the mathematical model (van der Maas et al., 2006). While the model could robustly account for some symptoms having very little to no growth, the model failed to converge when all symptoms were specified to have zero change.

Variability in symptom relationships and greater importance of select symptoms

The third simulation tested whether the model could account for differences in the relationships between BPD symptoms, and explored the possibility that some symptoms may be more influential in the development of other symptoms. These changes were implemented by allowing M_{ij} to differ, with some symptoms quantified as having a greater effect on the development of other symptoms. For example, affective instability was modeled as the most influential symptom overall.

The results of Simulation 3 displayed a similar growth pattern as Simulations 1 and 2, with some minor differences. Upon closer inspection, it was revealed that solely changing the weights in matrix **M** did not produce as significant of changes between the symptom

relationships as expected. Rather, it appeared that additional modifications were required in order to achieve greater variability between symptoms, as well as greater overall variability between the individual symptom trajectories. In particular, the simulations suggested that allowing a_i to vary across symptoms was necessary to increase overall variability, as these parameters not only influence the initial growth of each symptom, but indirectly influence the strength of the mutualistic interactions. Thus, this simulation demonstrated how more realistic between-and within-person variability can be achieved by permitting the growth parameter values to vary between symptoms.

Variability in the onset of BPD symptoms

The goal of Simulation 4 was to test whether the model could account for differences in onset of various BPD symptoms. Considering that some research suggests that certain BPD symptoms may emerge earlier in development than others, it was important to examine if the model could flexibility account for this possibility.

The results of Simulation 4 indicated that modeling symptoms to have varying onsets can be achieved by setting the mean of a_i for a given symptom to be relatively low initially and introducing a larger value at a later point in the simulation. Notably, no other changes were necessary to achieve these results. This indicated that even with higher K_i or M_{ij} parameters, the progression of a symptom can be delayed solely by manipulating a_i.

Symptom declines in adulthood

The extant literature suggests that a large proportion of individuals who experience BPD traits in adolescence will no longer exhibit these traits as adults (Sharp & Wall, 2018). The ability of the model to reproduce this type of symptom trajectory was examined in Simulation 5. Results suggested that modifying K_i and M_{ij} at $t = 6$ led to only modest symptom declines,

suggesting that the model poorly accounted for linear decay in growth. Although the model seems insufficient at handling decreases in these parameters, it does appear to perform well in instances where growth parameters may increase. This is crucial, as the risk factors captured in parameters **K** are unlikely to be invariant across development. For example, a 17-year-old who encounters additional life stressors is unlikely to have the same carrying capacity as they did when they were 12 years old. Therefore, the ability of the model to flexibly account for potential increases in an individual's carrying capacity represents a strength of the model.

Reductions in BPD symptoms with appropriate interventions

In developing a theoretical model of BPD, it is necessary to consider how and if the model can account for intervention effects. Across several BPD theories, treatment is often acknowledged, though specific predictions about how treatment influences the development of BPD symptoms is seldom discussed. From a mutualism perspective, reducing the connections between symptoms, particularly central symptoms, should facilitate a reduction in symptom severity. This possibility was probed in Simulation 6 by simulating data for one individual. Strong symptom associations were specified for affective instability to other symptoms, followed by moderately strong associations for impulsivity and unstable interpersonal relationships. The simulation was updated at $t = 4$ to serve as a proxy for receiving treatment that targeted affective instability. This was accounted for in the simulation by decreasing the weights from affective instability to all other BPD symptoms. Nonetheless, solely changing the M_{ij} weights for affective instability yielded very minimal changes. Indeed, even when additional changes were introduced into the model by adjusting K_i for affective instability, symptom reductions were still mostly limited to affective instability and NSSI/SI, with only minor changes observed for the remaining symptoms. To achieve a more realistic response to treatment, it was revealed that changes to M_{ij}

and K_i were necessary not only for affective instability, but for impulsivity and unstable interpersonal relationships as well (which were initially specified to have moderate influence).

From a clinical perspective, these findings implied that effective interventions must address the interdependence among BPD symptoms in addition to the genetic and environmental factors believed to contribute to the persistence of these symptoms. In other words, solely targeting the relationships between symptoms does not appear to meaningfully decrease symptom severity over time. Rather, results indicated that targeting both symptom relationships and possible causal factors, such as various genetic or environmental vulnerabilities, is necessary to achieve significant reductions in symptoms. This is consistent with recent findings that highlight the importance of considering both common cause factors and symptom dynamics to better understand how symptoms respond to intervention (O'Driscoll et al., 2022).

Some evidence-based treatments include strategies that target the relationships between symptoms and the vulnerabilities associated with their expression. For example, the emotion regulation skills embedded in DBT may help reduce the association between affective instability and NSSI by providing individuals with adaptive coping strategies that can be used instead of NSSI to regulate negative emotions and/or their response to stressful life events (Linehan, 1993). Although this scenario is simplistic and does not consider other factors that can impact treatment outcomes (e.g., therapeutic alliance, adherence to treatment), it illustrates how DBT can simultaneously affect both the connectivity between symptoms and possible putative vulnerabilities.

Explanatory Precision

Compared to explanatory breadth, the task of evaluating the explanatory precision of a theory is a lengthier process. A common approach for evaluating explanatory precision is to

compare theory-implied data models to empirical data models, which provide insight into the formal model's ability to replicate BPD symptoms in real-world settings (Haslbeck et al., 2021). Empirical data models include any representation of real-world data, such as descriptive statistics, correlation coefficients, factor models, or other statistical summaries of the data (Robinaugh et al., 2021). Models can be directly compared to identify similarities or differences between the theory-implied data and observed data.

Consequently, the explanatory precision of the formal theory was assessed by conducting a final simulation to generate data that was then compared to real-world observations of BPD. The last simulation was intended to generate data that could mirror longitudinal, epidemiological data on BPD symptoms from adolescence to young adulthood and incorporated the following features: a) variability in M_{ij} weights between symptoms and across subjects, b) asymmetrical associations amongst BPD symptoms, with affective instability specified as the most central symptom, c) variability in a_i to recognize differences in growth rates between individuals and symptoms, and d) different parameters for a subset of individuals to ensure sufficient variability in the propensity to developing BPD symptoms is reflected. With respect to the latter, we simulated data akin to Simulation 2, such that the first 50,000 subjects and the last 50,000 subjects were assigned different parameter values to promote greater variability in the dataset. Furthermore, measurement error was incorporated into the simulated data with a *SD* of 0.10, meaning most errors will fall within 0.10 units above or below the actual data point.

These decisions were intended to generate data that closely aligned with community-based observations of BPD. The explanatory precision of the formal model was evaluated by comparing empirical data models derived from the simulated data to models estimated based on three real-world longitudinal datasets. Despite the extensive empirical literature on BPD, only a

small number of studies were identified that reported the associations between individual BPD symptoms over time. As a result, comparisons between theory-implied data and real-world data were considered preliminary in nature.

Real-World Data Sources for Evaluating the Formalized Models

The following longitudinal datasets were used to evaluate the explanatory precision of the formalized mutualism model of BPD.

Pittsburgh girls study data

The Pittsburgh Girls Study (PGS; $N = 2{,}450$) is based on a large urban sample of girls recruited from the Pittsburgh area when girls were between five to eight years old. Data collection was based on an accelerated longitudinal design and girls and their caregivers were interviewed once a year in their homes. Families were deliberately oversampled from low-income neighborhoods and at least 25% of families living at or below the poverty line were contacted. Overall, approximately 2,875 families were deemed eligible to participate in the study and 85% agreed to participate at Wave 1 (Hipwell et al., 2002).

BPD was assessed in all study participants starting at age 14 using the International Personality Disorder Examination – Screener (IPDE-S; Lenzenweger et al., 1997).The IPDE-S is a self-report measure with nine *true* or *false* items that correspond to BPD diagnostic criteria. Scores greater than four are considered clinically significant (Smith et al., 2005).

Fourth R control trial data

The second longitudinal dataset was obtained from an ongoing cluster randomized controlled trial of "*Fourth R,*" a school-based prevention program focused on reducing dating violence (Crooks et al., 2008). Adolescents were recruited from 24 public middle schools across a large metropolitan area in Texas and were randomly assigned to the intervention or control

group. Data used for this study were based on participants assigned to the control group (N = 3,028). Individuals who obtained parental consent to participate were annually assessed from age 12 to age 15 (grades 7 through 10). Unfortunately, demographic information was not made available. However, prior studies using variations of this dataset have reported that most participants identified as either Hispanic, Black, or Asian, and the proportion of male and female participants was fairly equal (e.g., Lu et al., 2021; Temple et al., 2021).

BPD traits were assessed via the Borderline Personality Features Scale for Children-11 (BPFSC-11), which is a shortened, 11-item version of the original BPFSC (Crick et al., 2005; Sharp et al., 2014). The BPFSC-11 is a self-report measure designed to assess borderline personality features in adolescents and children aged 9 and older. Items are rated utilizing a 5-point Likert scale ranging from *not true at all* to *always true.* While the BPFSC-11 captures BPD features, it is important to note that the questions do not directly map onto *DSM-5* criteria for BPD. This is particularly evident for symptoms related to recurrent NSSI/SI and stress-induced paranoia/dissociation, which are not captured in this 11-item measure. Therefore, comparisons between this dataset and the simulated data were limited to only seven BPD symptoms.

Adolescent risk behaviors data

The third dataset was based on a sample of youth who were recruited to participate in a larger prospective study on factors that promote high-risk HIV behaviors in children and adolescents. English-speaking children in the greater Washington D.C. area that were either in the 5^{th} or 6^{th} grade were eligible to participate. Children who received parental consent to participate in the study were then re-assessed annually for up to nine years. At the first assessment wave, 277 adolescents (44% female) between ages 9 and 13 (Mean = 11, *SD* = 0.81) gave informed consent/assent to participate (Collado et al., 2014). For the purpose of the current

study, data was reorganized such that BPD trajectories were examined only for same-aged individuals over time. This resulted in 198 useable cases at age 11, 254 cases at age 12, 228 cases at age 13, and 133 cases at age 14. Remaining ages had an insufficient number of cases and were not analyzed.

Borderline personality traits were assessed using the Borderline Personality Subscale of the Coolidge Personality and Neuropsychological Inventory for Children (CPNI; Coolidge et al., 2002). The borderline subscale of the CPNI consists of nine items that correspond to the diagnostic criteria of BPD. Items are rated on a 4-point Likert scale ranging from *strongly false* to *strongly* true. This questionnaire is intended to be completed by a primary caregiver on behalf of the child or adolescent (Coolidge et al., 2000).

Data inspection based on the final simulation

Parameter values and R code used in the final stimulation are provided in Appendix A and B, respectively. R code for the simulations can also be found using the following link: https://osf.io/npb2w/. Prior to comparing the simulated data to the real-world data, trajectories of four random subjects were inspected and are presented in Appendix A, Figure S1. A random number generator was used to determine which subject data should be plotted. The random number generator resulted in the following four numbers: 23440, 81604, 84681, 44681, which conveniently resulted in data for two subjects in each parameter group. Differences in trajectories between subjects from the different groups were evident, with subject 23440 and subject 44681 having substantially greater growth and variability in their trajectories.

As a supplemental inspection, we also examined whether the simulated data produced prevalence rates of BPD that mirrored real-world estimates. Since the simulation does not clearly identify subjects whose symptoms cross the threshold for clinical significance, we characterized

symptoms as meeting the clinical threshold if their value was ≥2 *SDs* from the mean. Using *DSM-5* diagnostic guidelines, subjects were then characterized as meeting criteria for BPD if at least 5 symptoms were clinically elevated at a given time point.

The prevalence rates at each age are presented in Table 2 and resulted in a gradual increase in the proportion of individuals meeting criteria for BPD at each age. Age 16 had the highest prevalence of BPD, with 1.23% of the sample meeting diagnostic criteria. These estimates appeared to decrease slightly with age, which is consistent with research that documents BPD symptoms to peak in adolescence and slowly decline into adulthood (Sharp & Wall, 2018). Most importantly, these estimates were comparable to recent surveys of BPD that suggest the prevalence rate to be around 1.6% in the general population (Chapman et al., 2017).

Table 2. Proportion of Subjects Meeting Criteria for BPD.

Age	% of Subjects Meeting Diagnostic Criteria for BPD
11	0.000
12	0.014
13	0.16
14	0.79
15	1.21
16	1.23
17	1.14
18	1.06
19	1.00
20	0.95

Tests for facilitating data comparisons

In comparing the theory-implied data to real-world data, a series of empirical data models were estimated. First, Pearson correlations were computed for all datasets at each time point using the corFIML() function in the *psych* package (Revelle & Revelle, 2015). Correlation matrices for the simulated and real-world data are presented in the supplemental materials (see Appendix C, Tables S1-S25).

Second, to examine the extent to which the theory-implied data structure aligned with the empirical data structures, cross-sectional, single-factor models were estimated across datasets. For both the theory-implied and real-world data, models were estimated using the *lavaan* package (Rosseel, 2012) with robust maximum likelihood estimation (MLR) and full-information maximum likelihood (FIML) to account for any non-normality or missing data, respectively (Enders & Bandalos, 2001; Satorra & Bentler, 1994).[12] Models were identified by fixing the factor variance to 1 and factor mean to 0. Goodness of fit was assessed using robust variants of the following fit indices: the comparative fix index (CFI), the Tucker–Lewis Index (TLI), and the root mean square error of approximation (RMSEA). Models with robust CFI/TLI values greater than or equal to 0.95 and robust RMSEA values below 0.06 indicated good fit to the data (Hu & Bentler, 1999).

Assuming adequate goodness of fit was observed, the baseline models were expanded to a multi-group structure, such that one group was comprised of individuals in the simulated dataset, and the other group consisted of subjects in one of the real-world datasets. The purpose of this analysis was to assess for configural invariance, which was tested by estimating the same factor structure in both groups and inspecting whether the model achieved acceptable fit statistics (Van de Schoot et al., 2012). If configural invariance is supported, this indicates that the same factor structure is tenable across the theory-implied and real-world data under consideration.

In the event that configural invariance was supported, metric invariance was subsequently tested to determine if the strength of the relationship between the individual items and BPD factor were equivalent across datasets. Metric invariance was tested by constraining factor

[12] Although PGS data were binary, some evidence suggests that MLR performs similarly to other estimation approaches and thus this method was used to facilitate consistency across estimation methods (e.g., Kilic et al., 2020).

loadings to be equal across datasets and comparing this model to the configural model (Van de Schoot et al., 2012). If metric invariance was supported, strong invariance was also tested by constraining item intercepts (in addition to factor loadings) across datasets. The strong invariance model is then compared to the metric invariance model to determine if the added constraints result in significantly poorer fit.

Due to the sensitivity of the chi-square difference test (Cheung & Rensvold, 2002), measurement invariance was considered to hold at a given level if changes in CFI and RMSEA were ≤ 0.01 and ≤ 0.015, respectively (Chen, 2007). Invariance of factor loadings implies that the process used to generate the simulated data reasonably mirrors the underlying factor structure of real-world assessments of BPD. Moreover, evidence of strong invariance suggests that the baseline levels (intercepts) of BPD items are similar. Taken together, the support for measurement invariance suggests that the formalized mutualism model is capable of producing data with structural relationships among BPD symptoms that are comparable to real-world data patterns. If supported, this also provides greater confidence in the formal model's ability to accurately represent the underlying target system of interest.

CHAPTER FIVE: MODEL COMPARISONS RESULTS

Single-Factor Models

The results of the confirmatory single-factor models suggested that most models had adequate fit to the data. While there were some instances of poorer fit, these models were still deemed sufficient enough to proceed with the multi-group analyses. However, one problematic case was noted. The single-factor model at age 11 that was estimated using simulated data did not converge. This was likely due to the fact that, consistent with mutualism, the simulated BPD symptoms were largely uncorrelated with one another at age 11. The only dataset with observations at age 11 was similarly noted to have suboptimal fit to the data (R-CFI = .88; R-TLI = .84; R-RMSEA = .084), and thus a multi-group model was not estimated at age 11.

Multi-Group Models

The results of the multi-group models can be found in Table 3. Levels of invariance that were statistically supported are italicized in the table. Findings from each of the specific comparisons (i.e., multi-group model comparing the simulated data to each of the empirical data sources) are described in greater detail below. Of note, Sensitivity checks associated with the multi-group model comparisons are presented in Table 4.

Table 3. Multi-Group Models at Each Age.

Age	Data	Invariance Level	SB- χ^2 (*YB*)	*df*	R-CFI	R-TLI	R-RMSEA	$\Delta\chi^2$ (Δdf)
12	**Sim Data vs. Fourth R**	*Configural*	959.02 (1.49)	28	.941	.911	.031	--
		Metric	3805.96 (1.48)	34	.763	.707	.057	2948.4 (6)***
12	**Sim Data vs. High-Risk**	Configural	2268.50 (1.31)	54	.833	.777	.033	--
13	**Sim Data vs. Fourth R**	*Configural*	191.00 (1.92)	28	.995	.993	.015	--
		Metric	1969.26 (1.88)	34	.946	.933	.046	1960.9 (6)***
13	**Sim Data vs. High-Risk**	*Configural*	69.62 (1.71)	54	1.00	.999	.003	--
		Metric	159.76 (1.67)	62	.998	.997	.007	103.27 (8)***
		Strong	790.33 (1.79)	70	.981	.981	.019	419.89 (8)***
14	**Sim Data vs. Fourth R**	*Configural*	224.89 (1.89)	28	.997	.996	.016	--
		Metric	1954.84 (1.80)	34	.974	.968	.045	2257.60 (6)***
14	**Sim Data vs. High-Risk**	*Configural*	157.62 (1.56)	54	.999	.999	.008	--
		Metric	202.85 (1.56)	62	.999	.998	.008	44.83 (8)***
		Strong	512.91 (1.54)	70	.995	.995	.014	342.54 (8)***
14	**Sim Data vs. PGS**	*Configural*	246.56 (1.73)	54	.998	.997	.011	--
		Metric	496.46 (1.68)	62	.995	.994	.015	310.34 (8)***
		Strong	1800.13 (1.63)	70	.981	.981	.028	1711 (8)***
15	**Sim Data vs. Fourth R**	*Configural*	240.08 (1.83)	28	.998	.997	.016	--
		Metric	1845.23 (1.76)	34	.984	.980	.043	1962.7 (6)***
15	**Sim Data vs. PGS**	*Configural*	289.88 (1.71)	54	.998	.998	.012	--
		Metric	552.60 (1.66)	62	.996	.996	.016	311.67 (8)***
		Strong	1949.38 (1.60)	70	.987	.987	.029	1954.89 (8)***
16	**Sim Data vs. PGS**	*Configural*	363.69 (1.70)	54	.998	.998	.014	--
		Metric	687.99 (1.66)	62	.996	.996	.018	383.93 (8)***
		Strong	2114.66 (1.59)	70	.989	.989	.030	2205.40 (8)***
17	**Sim Data vs. PGS**	*Configural*	361.17 (1.72)	54	.998	.998	.014	--
		Metric	621.68 (1.68)	62	.997	.997	.017	293.05 (8)***
		Strong	1952.65 (1.60)	70	.991	.991	.029	2226.37 (8)***
18	**Sim Data vs. PGS**	*Configural*	421.15 (1.73)	54	.998	.998	.015	--
		Metric	721.54 (1.72)	62	.997	.997	.019	310.22 (8)***
		Strong	1864.28 (1.62)	70	.992	.992	.029	2111.38 (8)***
19	**Sim Data vs. PGS**	*Configural*	439.98 (1.73)	54	.998	.998	.016	--
		Metric	759.78 (1.72)	62	.997	.997	.019	338.55 (8)***
		Strong	1769.23 (1.61)	70	.993	.993	.028	1925.45 (8)***
20	**Sim Data vs. PGS**	*Configural*	419.93 (1.72)	54	.999	.998	.015	--
		Metric	667.18 (1.70)	62	.998	.997	.018	265.49 (8)***
		Strong	1564.66 (1.60)	70	.994	.994	.026	1622.82 (8)***

*Note. SB-χ^2 = Satorra-Bentler corrected chi-square; YB = Yuan-Bentler correction; df = degrees of freedom; R-CFI = robust comparative fit index; R-TLI = Robust Tucker-Lewis index; R-RMSEA = robust root-mean-square error of approximation; Sim Data = simulated data; PGS = Pittsburgh Girls Study; Δχ2 = change in chi-square based on non-robust chi-square statistic. *p < .05. **p < .01. ***p < .001.*

Fourth R data compared to simulated data

In the *Fourth R* dataset, BPD symptoms were measured annually from age 12 to age 15. Given that this dataset did not have indicators related to NSSI/SI and stress-related dissociation and paranoia, these items were omitted from the simulated dataset for comparison purposes. Overall, the configural models had acceptable fit across age. However, constraining factor loadings resulted in significant decrements to model fit based on changes in R-CFI and R-RMSEA at all ages, and strong invariance was not explored. Collectively, this particular dataset and the simulated dataset exhibited similarities in factor structure but the relationships between observed variables and the latent variable were not comparable.

Adolescent risk behaviors data compared to simulated data

In the Adolescent Risk Behaviors dataset, BPD symptoms were annually rated by a primary caregiver from age 11 to age 14. Model comparisons focused only on ages 12-14 due to the poor fit and convergence issues noted at age 11. Overall, the multi-group model revealed poor fit to the data at age 12, and thus no further tests of invariance were pursued. At ages 13 and 14, model fit was considered acceptable for the configural models, and tests of metric invariance were pursued. At both ages, constraining factor loadings to be equal across groups did not significantly reduce the overall fit of the model. Strong invariance was subsequently tested and supported at age 14 but not age 13. As a whole, these findings indicated that BPD had consistent measurement properties across datasets at age 13 and 14; however, only age 14 had comparable item intercepts.

PGS data compared to simulated data

In the PGS data, BPD symptoms were measured on a yearly basis across ages 14-20. Configural models demonstrated good fit to the data across age, enabling further tests of

measurement invariance. Equivalence of factor loadings was supported at all ages, suggesting that the relationship between the indicators and latent construct were consistent across datasets. Evidence of strong invariance was also found at all ages except for age 14. This suggested that the representation of BPD between the theory-implied data and PGS data were not only conceptually similar but demonstrated notable overlap in their overall statistical patterns.

Sensitivity checks

To ensure sample sizes differences between the simulated data and the real-world data sources were not biasing results, a smaller subset of individuals from the simulated dataset were randomly selected and used for the purposes of re-running the configural, multi-group models at age 14. This age was chosen as it was the only age that was shared across all datasets. Thus, we randomly selected 2,500 individuals from the simulated data to be compared to the *PGS and Fourth R* datasets, which resulted in a more balanced number of subjects across groups. For the Adolescent Risk Behaviors dataset, only 250 individuals were randomly selected from the simulated data. This allowed the multi-group models to be balanced in terms of group sample sizes and served as a general sensitivity check. Results are presented in Table 4 and suggested a slight reduction in model fit, though still exceedingly good fit overall. Therefore, differences in sample size do not appear to be biasing the results.

Table 4. Sensitivity Check for Multi-Group Models.

Age	Data Source	SB- χ^2 (YB)	*df*	R-CFI	R-TLI	R-RMSEA
14	Adolescent Risk Behaviors and Simulated	72.96 (1.44)	54	.951	.935	.051
14	Fourth R and Simulated	179.59 (1.90)	42	.977	.966	.062
14	PGS and Simulated	174.13 (1.72)	54	.965	.953	.041

*Note. SB-χ^2 = Satorra-Bentler corrected chi-square; YB = Yuan-Bentler correction; df = degrees of freedom; R-CFI = robust comparative fit index; R-TLI = Robust Tucker-Lewis index; R-RMSEA = robust root-mean-square error of approximation; $\Delta\chi 2$ = change in chi-square based on non-robust chi-square statistic. *p < .05. **p < .01. ***p < .001.*

CHAPTER SIX: DISCUSSION

The present study represents the first attempt at formalizing a developmental theory of BPD. The formalized theory was developed by adapting assumptions of the dynamic mutualism model of intelligence, which acknowledges the importance of direct symptoms relationships, as well as varying genetic and environmental predispositions, in explaining the emergence and progression of these symptoms. As demonstrated by computer simulations, the formalized model successfully reproduced a positive manifold amongst BPD symptoms around age 13, with more pronounced correlations beginning at age 16. Further, the model adequately explained or produced several robust phenomena associated with BPD, including variability in symptom presentation, between-person differences in the propensity to developing symptoms of BPD, variability in the onset of symptoms, potential for dissimilar relationships among symptoms, and maturation effects. The ability of the formal model to account for intervention effects was also explored; however, the model performed poorly in this scenario and was unable to properly account for negative linear growth. Lastly, the prevalence rate of BPD was calculated based on the final simulation. During adolescence and adulthood, the prevalence rate of BPD was similar to current estimates of BPD in the general population (Ellison et al., 2018; Tomko et al., 2014). This provided some confidence with respect to the model's explanatory breadth and ability to replicate real-world data patterns.

The present study also evaluated the explanatory precision of the formal model by assessing how well the theory-implied data aligned with real-world observations of BPD. Model comparisons indicated that there was some evidence to suggest that the formal model emulated

real-world factor structures of BPD across multiple datasets and at several ages. Although these comparisons were preliminary in nature, it is notable that these similarities were observed, especially considering the variability in methodologies (e.g., self-report vs parent report vs clinician reported).

Findings were most robust between the simulated data and PGS data, in which support for configural and metric invariance was found at all ages; strong invariance was supported at all ages except age 14. This indicated that the measurement model exhibited a consistent relationship between the observed variables and the underlying construct across both datasets, and that the scale of measurement operated similarly in both contexts. In contrast, the equivalence of item intercepts was not supported in the majority of other data comparisons. This indicated that individuals in one dataset may have scored systematically higher or lower on certain items. Considering BPD was assessed using different scales of measurement, it is possible that these differences may have contributed to this level of non-invariance. Alternatively, it is conceivable that further refinements to the model are warranted in order to produce item intercepts that align closer to real-world assessments.

Taken together, our results suggested that BPD symptoms behaved similarly in both simulated and real-world contexts. Such a finding is critical, as it highlights the capacity of the formal model to mimic real-world phenomena and suggests at least some degree of explanatory precision (Robinaugh et al., 2021). Some evidence in support of the model's explanatory breadth was also found, as it adequately explained several key features associated with BPD. Although more rigorous tests are needed, these findings provide some confidence in the model's depiction of the target system and its overall utility.

Limitations

Despite these encouraging findings, these results must be considered in light of several limitations. First, the etiology and development of BPD is complicated, and it is unlikely that any model will adequately capture its many nuances and complexities. The formalized mutualism model of BPD is no exception, as its representation of BPD is inherently incomplete and does not fully address the numerous complexities underlying its development. For instance, several variables that presumably influence the development of BPD, such as co-occurring psychopathology, were not explicitly defined in the formal model. The model was instead hypothesized to capture a range of genetic/environmental influences which are subsumed within the **K** parameters (van der Maas et al., 2006). This approach acknowledges an additive effect of these risk factors but does not distinguish the relative contribution that each factor has on the development of a particular symptom. Thus, any hypothesized genetic or environmental factors are purely speculative based on the literature and are do not have unique parameter values associated with each risk factor. Instead, genetic and environmental vulnerabilities are represented as an amalgamation of factors, which lacks specificity and ignores potential interactions between these variables (van der Maas et al., 2017).

While the lack of specificity regarding genetic and environmental factors is a criticism of both our formal model and the mutualism model of intelligence, identifying and mathematically operationalizing variables that are most important in the development of BPD would greatly enhance the model's utility and explanatory precision. Extensions of the mutualism model of intelligence have proposed an option for modeling gene-environment interactions via “multiplier effects” (van der Maas et al., 2017); however, this still requires knowledge of which variables

interact, the strength of their interactions, and when these interactions occur, which is largely unknown and likely differs between individuals.

Second, while the simulated data provides information on the strength of the associations between symptoms, it does not provide clear opportunities to examine differences between individuals who may meet diagnostic criteria for BPD, those who have subthreshold symptoms, and those who have little to no symptoms. The present study attempted to circumvent this concern by calculating the proportion of subjects who had at least 5 symptoms that were two or more standard deviations above the sample mean. This resulted in the prevalence of BPD to be 1.2% at age 16, 1.1% at age 18, and just under 1% at age 20 in the theory-implied dataset. These estimates are fairly comparable to recent data that indicate the prevalence of BPD to be around 1.6% in the general population (Chapman et al., 2017). However, it may still be helpful if the simulation was able to cleanly identify individuals who meet or do not meet the clinical threshold for a given symptom.

Third, the simulation resulted in negligible correlations between BPD symptoms at age 11. Although this was expected given the assumptions of the model, the available observed data suggests that some correlations are already developed between symptoms at this age. As such, it may be necessary to modify the age range of the model to account for some modest degree of symptom growth to be present by age 11.

Fourth, the formalized model does not appear well-equipped for handling non-linear change. This was particularly evident when examining the model's ability to account for changes in symptoms during/after treatment, as well as the model's ability to capture null growth and decreasing symptom trajectories into adulthood. Consequently, it may be more reasonable to use this model when investigating the BPD trajectories of those at risk for developing the disorder,

rather than using it to understand treatment outcomes or the trajectories of a broader population. While it is challenging to ascertain one's risk of developing a given disorder, individuals with well-documented risk factors for BPD (e.g., childhood trauma) may be a more suitable target population for this model. This logic is consistent with other dominant theories of BPD that have centered their theoretical frameworks on individuals suspected of having an early vulnerability to BPD (Crowell et al., 2009). Alternatively, it is possible that additional constraints or adaptations could be made to allow the model to better handle these non-linear fluctuations.

Fifth, as previously stated, the model does not provide a comprehensive depiction of BPD, nor does it account for all of the defining features associated with BPD. For example, while the model indirectly accounts for the influence of co-occurring psychopathology on the development of BPD, it does not explain or predict how subsequent psychopathology may develop. Relatedly, the influence of co-occurring symptoms is not accounted for in matrix **M**, which may be relevant to consider to fully understand the dynamics of these symptoms. Of note, this remains a limitation for most theories of BPD, and the specific mechanisms that foster co-occurring psychopathology are hardly described. Thus, although the formalized mutualism model of BPD has some advantages and strengths over verbal theories, it is not immune to experiencing many of the same limitations as existing theories. For example, the developmental extension of Linehan's biosocial theory acknowledges that BPD tends to have elevated co-occurrence with other disorders, though attributes these elevations to the communal liabilities that are shared between BPD and other internalizing and externalizing disorders (Crowell et al., 2009). While this may be true, this explanation lacks precision and offers poor predictive utility, again highlighting some challenges with verbally formulated theories.

In evaluating the strengths and limitations of the formal model, it is also important to consider how the decisions made during its development may affect the interpretation of results. For example, the chosen time step in the model was selected to reflect an annual assessment period over the course of ten years, serving as a proxy for ages 11-20. This decision was made given available evidence that suggests BPD traits to emerge around age 11 and increase throughout adolescence (Guilé et al., 2018). This approach was also necessary to facilitate comparisons between real-world data, which similarly assessed BPD traits at yearly intervals. However, it is possible that the time scale used does not accurately capture the dynamics between symptoms throughout this developmental period. As longitudinal assessments of BPD at younger ages continue to increase, it is likely that adjustments to the time scale will be needed.

Furthermore, in accordance with the original mutualism model of intelligence, many of the parameter values employed in the present study were selected somewhat arbitrarily, and other values may have led to minor differences in results. For example, the weights selected for matrix **M** were based on empirical evidence, when possible, though some decisions were ultimately made without empirical guidance. Nonetheless, determining appropriate parameter values remains a challenge for most clinical psychology simulation studies, especially when there is a lack of empirical data to inform such decisions.

Finally, the current study did not account for the possibility that symptoms may develop differently as a function of gender, race, or other demographic factors (e.g., socioeconomic status). Some BPD research has found some support for gender differences, though findings in the literature are far from equivocal (e.g., Hoertel et al., 2014; Silberschmidt et al., 2015; Zlotnick et al., 2002). In a similar vein, this study did not consider the potential influence of time-varying factors, which fluctuate throughout development and may impact the symptom

expression of BPD (Conway et al., 2018). These factors encompass a variety of influences, such as stress or traumatic life events, substance use, physical or mental health comorbidities, and other dynamic risk or protective factors that are unique to the individual. As such, we consider this an opportunity for prospective research that was beyond the scope of this study.

Future directions

The present study provides an initial foundation for developing formalized models of BPD and related conditions. Future studies will be imperative for advancing the current understanding of BPD, its development, and how to best serve adolescents and adults who experience these symptoms. Relatedly, future research examining scenarios that were not explored in the current study will be essential for better understanding limitations of this model and identifying area of improvement. For example, the present study did not examine the possibility that some symptoms of BPD undergo increasingly specialized growth processes, resulting in some mutualistic interactions becoming stronger and some becoming weaker. As illustrated by van der Maas et al. (2006), this idea can be simulated by holding the mean of M_{ij} constant across age while allowing the *SD* of M_{ij} to increase over time. This results in some interactions strengthening with age while others decrease in strength or even become "competitive" with one another. This suggests that change in one or more symptoms may be constrained if both symptoms are competing for the same resources (van der Maas et al., 2017).

As discussed previously, the data comparisons in the present study were limited to a small range of data sources with varying methodologies and sample characteristics. Given that other simulation studies in the field of psychology have yet to compare their simulated data to real-world data (Robinaugh et al., 2019; Wang et al., 2023), this was still considered a strength.

Nevertheless, studies that utilize more diverse data sources to assess the efficacy of the formal model are expected to provide valuable insight for future improvement.

Finally, future research that explores scenarios beyond the scope of the present study paper—such as the impact of co-occurring psychopathology, shorter assessment intervals for BPD, and adaptations of the model to accommodate non-linear growth—will be essential for a more comprehensive evaluation of the strengths and limitations of the formal mutualism model of BPD.

Conclusion

In summary, the present study represents an initial attempt at formalizing a developmental model of BPD. This model was developed based on several assumptions of the dynamic mutualism model of intelligence and provides a preliminary glimpse into the potential data outcomes if the theoretical assumptions were true. Moreover, although the mutualism model of BPD has some overlap with other existing theories, our theoretical model is one of the first to explicitly incorporate the interactive effects that BPD symptoms have on each other. The attention to broader causal elements together with local interactions that are specific to the individual aligns well with recent scholarly discussions that emphasize the significance of both common cause elements and local symptom interactions in understanding and treating psychopathology (O'Driscoll et al., 2022). Moving forward, it will be crucial to continue exploring the dynamic interplay among BPD symptoms and identify how these interactions are shaped by both time-varying and static factors. Doing so may not only advance our understanding of BPD, but also facilitate improvements in the assessment and treatment of this disorder.

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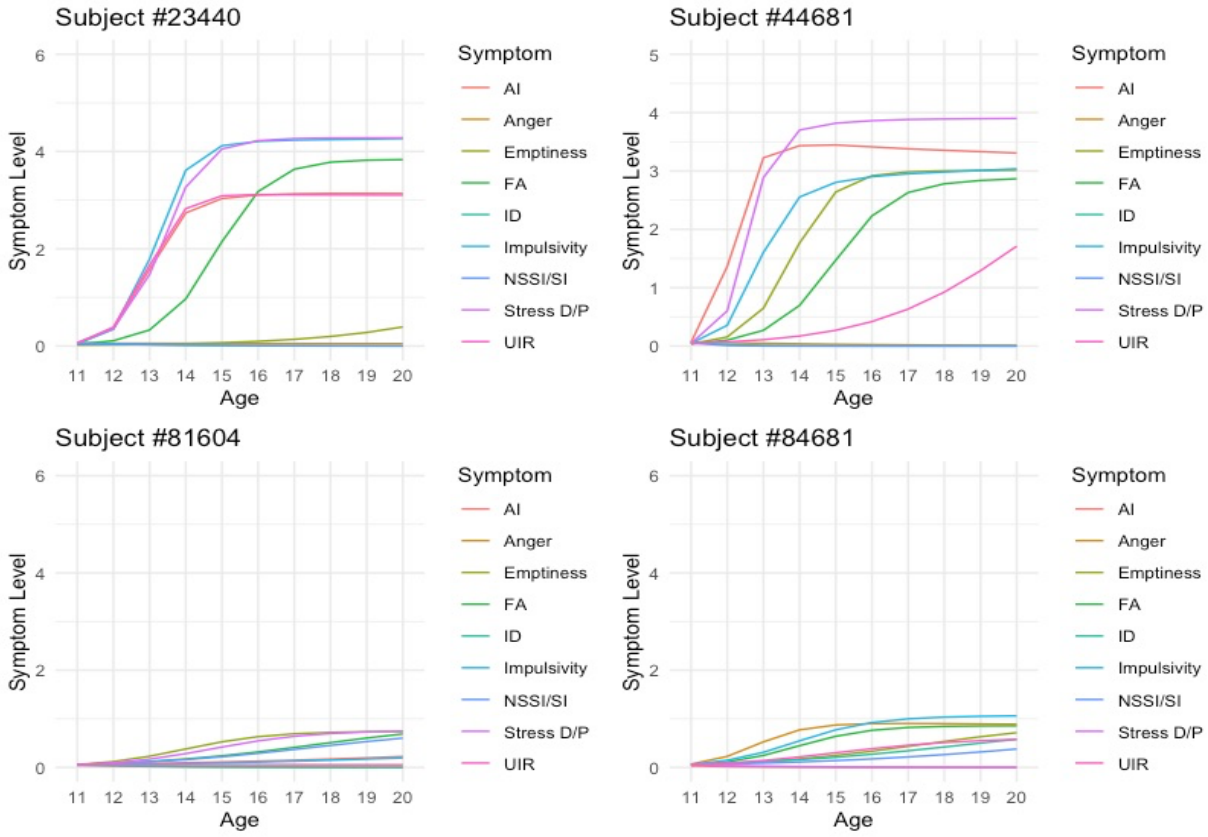
APPENDIX A: FINAL SIMULATION VALUES

Parameter Values and Assumptions Used in the Final Simulation

Parameter	Group 1	Group 2
M_{ij}	Randomly sampled from a multivariate normal distribution based on the M_{ij} weights in Table 1; SD range: 0.02 – 0.06	Randomly sampled from a multivariate normal distribution where all M_{ij} = 0.001; SD range: 0.01 – 0.04
x_0	Means = 0.05; SD = 0.01	Means = 0.05; SD = 0.01
a_i	Means ranged from 0.2 to 1.55; SD = 1	Means = 0.5; SD = 0.50
K_i	Means = 3, correlated distribution; r = 0.25	Means = 0.75, uncorrelated distribution; SD = 0.10

Note. Group 1 represented individuals likely to be at greater risk for developing BPD, or subthreshold levels of BPD. Group 2 represented a normative or healthy sample of individuals.

Developmental Trajectories of Four Random Subjects from the Final Simulation



APPENDIX B: R CODE FOR THE FINAL SIMULATION

```
# Code was adapted based on van der Maas et al. (2006)

# Packages:
library(deSolve)
library(MASS)
library(mvtnorm)

# Mutualism model function:
lotka_volterra <- function(t, y, parms) {
  with(as.list(parms), {
    dy <- a*y*(1-y/K) + a*(y * M %*% y)/K
    list(dy)
  }) }
# Initial M matrix:
M <- matrix(c(0.05, 0.15,   0.05,   0.10,   0.05,   0.10,   0.05,   0.01,   0.01,
              0.02, 0.05,   0.02,   0.10,   0.02,   0.10,   0.02,   0.01,   0.01,
              0.05, 0.15,   0.05,   0.10,   0.05,   0.10,   0.05,   0.01,   0.01,
              0.02, 0.15,   0.02,   0.05,   0.02,   0.10,   0.02,   0.01,   0.01,
              0.05, 0.15,   0.05,   0.10,   0.05,   0.10,   0.05,   0.01,   0.01,
              0.02, 0.15,   0.02,   0.10,   0.02,   0.05,   0.02,   0.01,   0.01,
              0.05, 0.15,   0.05,   0.10,   0.05,   0.10,   0.05,   0.01,   0.01,
              0.05, 0.15,   0.05,   0.10,   0.05,   0.10,   0.05,   0.05,   0.01,
              0.05, 0.15,   0.05,   0.10,   0.05,   0.10,   0.05,   0.01,  0.05),
            nrow = 9, ncol = 9, byrow = TRUE)

# Alt M Matrix for "healthy" subjects;
MHealth<- matrix(0.001,   nrow = 9, ncol = 9, byrow = TRUE)

# Function to generate new Mij for each subject. SD specified later;
generate_random_matrix <- function(original_matrix, std_dev_range, n_subjects) { ra
ndom_matrices <- list()
    for (i in 1:n_subjects) {
      std_dev <- runif(1, min = std_dev_range[1], max = std_dev_range[2])
random_matrix <-original_matrix + matrix(rnorm(length(original_matrix), sd = std_
dev), nrow = nrow(original_matrix))
      diag(random_matrix) <- .05  # Fix the diagonal to 0.05
      random_matrices[[i]] <- random_matrix
}
    return(random_matrices) }

# Generate Mij weights:
 # Define SD Ranges;
std_dev <- c(0.02, 0.06) # elevated group
std_dev_h <- c(0.01, 0.04) # "healthy" group
# Generate a list of data using above function then combine both lists
```

```r
MC<- generate_random_matrix(M, std_dev, 50000)
MH<- generate_random_matrix(MHealth, std_dev_h, 50000)
M_list<- c(MC, MH)
```

Define Other Key Parameters:

```r
#-- 1) Define number of subjects
n_subjects = 100000

#-- 2) Define number of time points
t <- seq(1, 10, by = 1)

#-- 3) Generate random starting values for all subjects
nrows <- n_subjects; ncols <- 9
y0 <- matrix(rnorm(nrows * ncols, mean=0.05, sd=0.01),
                                                          nrow = nrows
, ncol = ncols)
```

Implement Simulation:

```r
sim_results <- list()

# Run the simulation for each subject
for (i in 1:n_subjects) {

  if (i <= 50000) { # first half
    meanA<- c(1, 1.55, 0.4, 0.2, 1.2, 1, 1.2, 0.3, 0.7)
    a <- rnorm(9, mean = meanA, sd = 1)
    mean_k<- rep(3, 9)
    cov_mat_k <- matrix(0.25, nrow = 9, ncol = 9) + 0.1 * diag(9)
    # Sample a K vector from the correlated distribution
    K <- mvrnorm(n = 1, mu = mean_k, Sigma = cov_mat_k)

  } else {
    # Second half of subjects with different parameter values
    a <- rnorm(9, mean = 0.5, sd = 0.5)
    K <- rnorm(9, mean = 0.75, sd = 0.1)
  }
  parms <- list(K = K, a=a, M=M_list[[i]])
  out <- ode(y = y0[i, ], times = t, func = lotka_volterra, parms = parms)

  # Store the simulation results for the current subject
  sim_results[[i]] <- data.frame(time = out[,1], y = out[, -1])
}

# Combine the simulation results for all subjects into a single data frame
sim_data <- as.data.frame(do.call(rbind, sim_results))

# Create unique ID for each subject
sim_data$subject <- rep(1:n_subjects, each = nrow(out))
```

APPENDIX C: CORRELATION TABLES

Table S1. Correlations at Age 11 (T1) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	-.01	1.00							
FA	.00	.00	1.00						
ID	.00	.00	.00	1.00					
IMP	.00	.00	.00	.00	1.00				
ANG	.00	.00	.00	.01	.00	1.00			
UIR	.00	.00	.00	.00	-.01	.00	1.00		
NSSI	.01	.00	.00	.00	.00	.00	.00	1.00	
D/P	.00	.00	.00	.00	.00	.00	.00	.00	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S2. Correlations at Age 11 (T1) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.18	1.00							
FA	.06	.08	1.00						
ID	.02	.04	.01	1.00					
IMP	.14	.20	.07	.03	1.00				
ANG	.13	.18	.05	.03	.14	1.00			
UIR	.15	.20	.07	.03	.18	.14	1.00		
NSSI	.04	.06	.02	.01	.04	.04	.04	1.00	
D/P	.09	.13	.04	.02	.10	.09	.10	.02	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S3. Correlations at Age 13 (T3) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.29	1.00							
FA	.12	.17	1.00						
ID	.09	.14	.05	1.00					
IMP	.23	.32	.14	.10	1.00				
ANG	.21	.30	.12	.10	.23	1.00			
UIR	.24	.32	.14	.11	.26	.23	1.00		
NSSI	.10	.15	.06	.05	.11	.11	.11	1.00	
D/P	.16	.22	.09	.08	.18	.16	.17	.07	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S4. Correlations at Age 14 (T4) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.42	1.00							
FA	.21	.27	1.00						
ID	.16	.24	.11	1.00					
IMP	.35	.46	.23	.18	1.00				
ANG	.32	.44	.20	.19	.35	1.00			
UIR	.36	.46	.23	.19	.39	.36	1.00		
NSSI	.18	.24	.11	.10	.20	.18	.19	1.00	
D/P	.25	.34	.16	.14	.27	.26	.28	.13	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S5. Correlations at Age 15 (T5) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.51	1.00							
FOA	.28	.34	1.00						
ID	.23	.31	.16	1.00					
IMP	.44	.55	.31	.26	1.00				
ANG	.41	.54	.27	.26	.44	1.00			
UIR	.45	.56	.31	.26	.49	.45	1.00		
NSSI	.25	.31	.16	.15	.26	.25	.26	1.00	
D/P	.32	.42	.22	.20	.35	.34	.36	.19	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S6. Correlations at Age 16 (T6) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.56	1.00							
FA	.33	.40	1.00						
ID	.28	.37	.20	1.00					
IMP	.50	.61	.36	.31	1.00				
ANG	.47	.59	.33	.31	.51	1.00			
UIR	.51	.61	.36	.31	.55	.51	1.00		
NSSI	.29	.36	.21	.19	.31	.30	.31	1.00	
D/P	.38	.47	.27	.24	.41	.40	.42	.23	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S7. Correlations at Age 17 (T7) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.56	1.00							
FA	.33	.40	1.00						
ID	.28	.37	.20	1.00					
IMP	.50	.61	.36	.31	1.00				
ANG	.47	.59	.33	.31	.51	1.00			
UIR	.51	.61	.36	.31	.55	.51	1.00		
NSSI	.29	.36	.21	.19	.31	.30	.31	1.00	
D/P	.38	.47	.27	.24	.41	.40	.42	.23	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S8. Correlations at Age 18 (T8) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.62	1.00							
FA	.40	.46	1.00						
ID	.35	.44	.26	1.00					
IMP	.57	.68	.43	.38	1.00				
ANG	.53	.66	.39	.38	.58	1.00			
UIR	.58	.68	.43	.38	.62	.58	1.00		
NSSI	.35	.42	.26	.24	.38	.36	.37	1.00	
D/P	.45	.54	.34	.30	.48	.46	.49	.29	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S9. Correlations at Age 19 (T9) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.64	1.00							
FA	.42	.48	1.00						
ID	.37	.46	.28	1.00					
IMP	.59	.69	.45	.41	1.00				
ANG	.55	.67	.41	.40	.60	1.00			
UIR	.60	.70	.45	.40	.64	.60	1.00		
NSSI	.37	.44	.28	.26	.40	.38	.39	1.00	
D/P	.47	.56	.36	.32	.50	.48	.51	.31	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S10. Correlations at Age 20 (T10) for Simulated Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.66	1.00							
FA	.43	.49	1.00						
ID	.39	.47	.29	1.00					
IMP	.61	.71	.47	.42	1.00				
ANG	.57	.69	.43	.42	.61	1.00			
UIR	.61	.71	.47	.42	.65	.62	1.00		
NSSI	.39	.45	.29	.27	.42	.39	.41	1.00	
D/P	.49	.57	.37	.34	.52	.50	.52	.32	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S11. Correlations at Age 14 for PGS Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.28	1.00							
FA	.25	.23	1.00						
ID	.36	.32	.22	1.00					
IMP	.18	.22	.19	.19	1.00				
ANG	.23	.44	.16	.12	.24	1.00			
UIR	.25	.24	.25	.14	.16	.17	1.00		
NSSI	.16	.10	.11	.10	.05	.11	.10	1.00	
D/P	.34	.32	.29	.34	.19	.25	.21	.13	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S12. Correlations at Age 15 for PGS Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.28	1.00							
FA	.29	.20	1.00						
ID	.30	.31	.25	1.00					
IMP	.13	.18	.14	.16	1.00				
ANG	.25	.46	.21	.25	.21	1.00			
UIR	.27	.20	.25	.23	.20	.21	1.00		
NSSI	.16	.12	.12	.08	.04	.10	.08	1.00	
D/P	.28	.32	.27	.28	.19	.32	.23	.08	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S13. Correlations at Age 16 for PGS Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.29	1.00							
FA	.32	.21	1.00						
ID	.34	.35	.30	1.00					
IMP	.13	.21	.14	.18	1.00				
ANG	.26	.49	.20	.25	.24	1.00			
UIR	.25	.24	.27	.22	.18	.24	1.00		
NSSI	.18	.13	.13	.14	.06	.07	.11	1.00	
D/P	.31	.36	.27	.31	.18	.30	.21	.07	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S14. Correlations at Age 17 for PGS Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.31	1.00							
FA	.29	.18	1.00						
ID	.35	.30	.25	1.00					
IMP	.18	.19	.14	.17	1.00				
ANG	.27	.47	.20	.26	.24	1.00			
UIR	.29	.24	.24	.24	.19	.23	1.00		
NSSI	.18	.14	.09	.10	.07	.12	.11	1.00	
D/P	.33	.33	.23	.29	.22	.28	.26	.08	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S15. Correlations at Age 18 for PGS Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.30	1.00							
FA	.27	.18	1.00						
ID	.36	.33	.28	1.00					
IMP	.16	.23	.14	.20	1.00				
ANG	.27	.46	.23	.25	.23	1.00			
UIR	.30	.23	.23	.22	.19	.23	1.00		
NSSI	.20	.11	.12	.11	.07	.08	.09	1.00	
D/P	.33	.33	.29	.32	.17	.34	.24	.12	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S16. Correlations at Age 19 for PGS Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.34	1.00							
FA	.25	.20	1.00						
ID	.39	.36	.27	1.00					
IMP	.17	.22	.19	.20	1.00				
ANG	.27	.43	.18	.24	.22	1.00			
UIR	.25	.23	.17	.22	.23	.18	1.00		
NSSI	.19	.12	.15	.15	.08	.11	.10	1.00	
D/P	.36	.32	.33	.35	.19	.27	.18	.14	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S17. Correlations at Age 20 for PGS Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.36	1.00							
FA	.29	.21	1.00						
ID	.38	.33	.25	1.00					
IMP	.23	.27	.23	.21	1.00				
ANG	.30	.45	.24	.26	.27	1.00			
UIR	.32	.29	.20	.23	.25	.20	1.00		
NSSI	.18	.09	.12	.12	.12	.09	.07	1.00	
D/P	.35	.35	.23	.34	.24	.37	.29	.10	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S18. Correlations at Age 12 for Fourth R Data.

	E	AI	FA	ID	IMP	ANG	UIR
E	1.00						
AI	.49	1.00					
FA	.51	.57	1.00				
ID	.58	.68	.64	1.00			
IMP	.40	.59	.52	.58	1.00		
ANG	.35	.42	.43	.46	.50	1.00	
UIR	.59	.63	.60	.64	.52	.46	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships.

Table S19. Correlations at Age 13 for Fourth R Data.

	E	AI	FA	ID	IMP	ANG	UIR
E	1.00						
AI	.53	1.00					
FA	.56	.61	1.00				
ID	.64	.72	.69	1.00			
IMP	.42	.65	.54	.59	1.00		
ANG	.36	.49	.45	.50	.54	1.00	
UIR	.61	.68	.66	.71	.57	.49	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships.

Table S20. Correlations at Age 14 for Fourth R Data.

	E	AI	FA	ID	IMP	ANG	UIR
E	1.00						
AI	.59	1.00					
FA	.58	.59	1.00				
ID	.70	.71	.68	1.00			
IMP	.45	.60	.52	.58	1.00		
ANG	.33	.39	.36	.42	.48	1.00	
UIR	.62	.68	.60	.70	.53	.38	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships.

Table S21. Correlations at Age 15 for Fourth R Data.

	E	AI	FA	ID	IMP	ANG	UIR
E	1.00						
AI	.61	1.00					
FA	.58	.62	1.00				
ID	.69	.71	.69	1.00			
IMP	.51	.59	.50	.58	1.00		
ANG	.33	.39	.36	.40	.45	1.00	
UIR	.66	.68	.60	.70	.54	.38	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships.

Table S22. Correlations at Age 11 for Risk Behaviors Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.29	1.00							
FA	.39	.16	1.00						
ID	.31	.29	.33	1.00					
IMP	.27	.31	.20	.25	1.00				
ANG	.36	.43	.22	.25	.36	1.00			
UIR	.25	.29	.28	.45	.26	.28	1.00		
NSSI	.12	.21	.14	.06	.34	.24	.18	1.00	
D/P	.28	.34	.21	.23	.35	.39	.25	.44	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S23. Correlations at Age 12 for Risk Behaviors Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.22	1.00							
FA	.41	.20	1.00						
ID	.34	.28	.33	1.00					
IMP	.27	.26	.38	.30	1.00				
ANG	.42	.52	.31	.34	.40	1.00			
UIR	.30	.35	.25	.40	.20	.35	1.00		
NSSI	.16	.17	.15	.21	.29	.19	.14	1.00	
D/P	.30	.21	.39	.35	.28	.31	.25	.29	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S24. Correlations at Age 13 for Risk Behaviors Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.36	1.00							
FA	.30	.12	1.00						
ID	.43	.37	.21	1.00					
IMP	.25	.31	.20	.28	1.00				
ANG	.38	.57	.25	.45	.40	1.00			
UIR	.19	.26	.12	.29	.08	.36	1.00		
NSSI	.12	.16	.08	.26	.17	.29	-.02	1.00	
D/P	.30	.31	.19	.41	.22	.33	.11	.20	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.

Table S25. Correlations at Age 14 for Risk Behaviors Data.

	E	AI	FA	ID	IMP	ANG	UIR	NSSI	D/P
E	1.00								
AI	.26	1.00							
FA	.46	.09	1.00						
ID	.27	.46	.17	1.00					
IMP	.14	.32	.33	.38	1.00				
ANG	.29	.57	.19	.53	.28	1.00			
UIR	.23	.25	.17	.38	.04	.36	1.00		
NSSI	.08	.21	.07	.10	.18	.16	.03	1.00	
D/P	.16	.16	.08	.24	.15	.14	.19	.15	1.00

Note. E = chronic feelings of emptiness; AI = affective instability; FA = fear of abandonment; ID = identity disturbance; IMP = impulsivity in two or more areas; ANG = intense/inappropriate anger; UIR = unstable interpersonal relationships; NSSI = recurrent non-suicidal self-injury of suicidal ideation; D/P = stress-induced dissociation/paranoia.