Utilizing Retrospective Accounts of Primary Symptom-Clusters to Predict PTSD over Time in Women Survivors of Domestic or Sexual Assault

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ABSTRACT

The extant theories in PTSD describe significant initial symptom reactions, and these reactions may provide opportunities for clearer early identification and treatment of PTSD. There are empirically identified trajectories of PTSD, which indicates there is a critical starting point to those trajectories. Generally, theories and results suggest that the re-experiencing (Cluster B) and hyperarousal (Cluster E) symptoms are common reactions after traumatic events, while hyperarousal and negative cognitions and mood (Cluster D) clusters are generally identified as the most important and/or predictive. Thus, this dissertation utilized retrospective reports in order to identify initial symptom reactions and then subsequently predict PTSD severity over time. Participants included college women who experienced sexual and relationship violence within the past 2 years. Two primary hypotheses were investigated within the dissertation: 1) Cluster B and E symptoms were expected to be the most prevalent initial reactions reported, and 2) Clusters E and D were expected to significantly predict PTSD severity over time. The results indicated partial support for each hypothesis, such that Cluster B symptoms were among the most prevalent initial reactions and Cluster D was a significant predictor of PTSD severity over time. Specifically, earlier Cluster D ordering interacted with the presence of negative beliefs and loss of positive emotions to predict PTSD severity over time.

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GENERAL AUDIENCE ABSTRACT

There are ideas and theories about how posttraumatic stress disorder (PTSD) starts and gets worse. People develop PTSD in different ways; some develop it very quickly and it is very bad, while others develop it slowly and it may not affect them much at all. The first signs and symptoms may be the best place to look, much like when you first get a cough or a sore throat with a cold or the flu. Generally, research suggests that common reactions are re-living the trauma and having reactions like being on guard all the time. Being on guard all the time also may be one of those important symptoms that will help predict if someone will get PTSD, as well as experiencing things such as thinking harsh things about oneself. This dissertation included reports from people after they had experienced trauma in order to figure out those first symptoms. Then, it used those first symptoms to predict how bad their PTSD was in the weeks and months later. Participants included college women who experienced sexual assault and domestic violence within the past 2 years. The results showed that people often re-live the trauma, but it may not be the most important when predicting whether they will get PTSD or not. Negative thoughts and beliefs about oneself were the most important set of reactions when predicting who will get PTSD and how badly. More importantly, the earlier they had those negative thoughts, the worse their PTSD was in the coming weeks and months.

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1.0 – Utilizing Retrospective Accounts of Primary Symptom-Clusters to Predict PTSD over Time in Women Survivors of Domestic or Sexual Assault

Most individuals experience at least one potentially traumatic event in their lifetime (89.7%; Kilpatrick, Resnick, Milanak, Miller, Keyes, & Friedman, 2013). From these events, approximately 6.8-8.3% of survivors will develop posttraumatic stress disorder (PTSD; Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005; Kilpatrick et al., 2013). Overall, PTSD is a psychological disorder in which individuals experience serious negative impacts to quality of life in many domains, even when compared to other psychopathology (Olatunji, Cisler, & Tolin, 2007). More specifically, PTSD is characterized by 4 primary reactions to a traumatic event, which are categorized into the following clusters: 1) re-experiencing the traumatic event via intrusive thoughts or other means (Cluster B; e.g., images, memories, nightmares, and reactions to traumatic cues), 2) thought or behavioral avoidance of traumatic cues (Cluster C), 3) negative cognitions and mood (Cluster D; e.g., negative beliefs about the self, persistent negative emotions), and 4) hyperarousal (Cluster E; e.g., hypervigilance, anger). From these 4 symptom clusters, the PTSD diagnosis requires at least 1 Cluster B, 1 Cluster C, 2 Cluster D, and 2 Cluster E symptoms (i.e., at least 6 symptoms; American Psychiatric Association, 2013). With 4 clusters, 20 symptoms, and 636,120 ways to be diagnosed (Galatzer-Levy & Bryant, 2013), PTSD is a complex disorder. Given this complexity, it is surprising that there have not been significant investigations into the specific development of PTSD. Currently, extant literature in PTSD is limited to broader risk and protective factors (e.g., Karam et al., 2014; Ozer et al., 2003), broader trajectories of severity (e.g., Bryant et al., 2015), and seminal theories of PTSD (e.g., Brewin, 2013, Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989) that explain PTSD at a broader conceptual level. While there are benefits in identifying broader risk factors, there

exist significant benefits from a specific level of analysis. For example, such an analysis may provide clearer early identification of PTSD onset. Early identification would be improved because if there are trajectories of PTSD symptoms (Bryant et al., 2015), and because PTSD develops via particular mechanisms as stated in the seminal theories (e.g., Brewin, 2013, Ehlers & Clark, 2000; Foa et al., 1989), then it follows that there are likely *important starting points* that can better predict PTSD trajectories over time. Thus, this dissertation first and foremost sought to evaluate the importance of initial symptom/cluster reactions on the longitudinal development of PTSD so that the identification of and prediction of PTSD may be improved.

Currently, the difficulty with identifying and predicting PTSD trajectories may stem from utilizing broad risk and protective factors. For example, Ozer et al. (2003) conducted a seminal meta-analysis of PTSD risk and protective factors, and they found that the effect sizes for the relationships between risk factors and PTSD were only small to moderate. A related area of difficulty stems from findings regarding differential patterns of symptom/cluster expression at the immediate vs. long-term course of the disorder. Specifically, researchers have found that the re-experiencing symptom cluster was generally the most commonly reported and "normal" (McMillen, North, & Smith, 2000, p. 57) PTSD cluster to experience in the immediate aftermath of a traumatic event (Fraguas et al, 2006; McMillen et al., 2000; North, 2004; North et al., 1999; Schell, Marshall, & Jaycox, 2004; Shalev, 1992). However, hyperarousal (Doron-LaMarca et al., 2015; Schell et al., 2004; Shalev, 1992) and emotional numbing (Carper et al., 2015; MacDonald Proctor, Heeren, & Vasterling, 2013) were better predictors of future PTSD compared to the initial re-experiencing cluster. In addition to the extant findings which suggest a distinction between initial PTSD reactions and later predictive power, researchers (Galatzer-Levy & Bryant, 2013; MacDonald et al., 2013; Norris, Tracy, & Galea, 2009) have specifically

noted that PTSD should be investigated in a more specific and nuanced manner with the following recommendations: 1) researchers should explore the heterogeneous nature of PTSD to increase reliability and validity of the field's research (Galatzer-Levy & Bryant, 2013), 2) identify growth patterns that better specify symptom trajectories (MacDonald et al., 2013), and 3) investigate trajectories in particular PTSD clusters (Norris et al., 2009). To date, however, few studies have directly addressed these goals (Doron-LaMarca et al., 2015; Galatzer-Levy, Karstoft, Statnikov, & Shalev, 2014; MacDonald et al., 2013; Schell et al., 2004). Galatzer-Levy et al. (2014) specifically posited that one reason why such investigations have not been carried out was that few researchers have utilized adequate computational models that are able to address PTSD's complexity.

Thus, moving forward this dissertation is based on the above recommendations and addressed 1) the heterogeneous nature of PTSD by utilizing a finer level of analysis (e.g., symptom/cluster level analyses), 2) identifying growth patterns via longitudinal data collection and analysis (i.e., hierarchical linear modeling), and 3) investigating trajectories in particular PTSD clusters. Because this dissertation is one of the first known investigations in this area, its scope was kept to a single component, namely the identification of initial symptom reactions and the extent to which they predict PTSD severity and differential trajectories over time. Additionally, broader risk factors were also examined. Thus, a review of the theoretical and empirical literature on patterns of PTSD symptom-clusters during the acute and long-term phases of the disorder follow.

1.1 – Theoretical Perspectives

There are three seminal theories which attempt to explain PTSD: 1) emotional processing theory (Foa et al., 1989), 2) dual representation theory (Brewin, 2013), and 3) the cognitive

model of PTSD from Ehlers and Clark (2000). These theories will be reviewed first in order to highlight their contributions to the current dissertation.

First, Foa et al. (1989) assert via the emotional processing theory that individuals form an associative fear network after experiencing a traumatic event. The fear network is an individual's idea of and response to a feared stimulus which involves both emotional (e.g., anger, fear, horror, guilt) and behavioral responses (e.g., hypervigilance, startle). Hence, the fear network may involve a PTSD-like response, though Foa et al. (1989) specify that in PTSD the individual needs to have generalized that fear network beyond the traumatic event (e.g., a woman is sexually assaulted by one man and subsequently fears all men). The basis for this fear network perspective originates from Lang's work which highlighted that information about a feared stimulus, responses to that stimulus, as well as meaning attributed to the stimulus and responses all interact and form a network (Lang, 1977; 1979). Similarly, for Foa et al. (1989), they state that it is critical to include both affective and cognitive information, and especially that the information is regarding an interpretation that the stimulus is dangerous. An attribution of dangerousness to previously safe cues is what Foa et al. state is "what distinguishes PTSD from other anxiety disorders" (1989, p. 166). Thus, emotional processing theory appears to highlight symptom D2, "*persistent and exaggerated negative beliefs or expectations about oneself, others, or the world*" (APA, 2013, p. 272), included in the negative cognitions and mood cluster as the critical symptom which indicates a PTSD trajectory. Regarding initial reactions, however, Foa et al. make no specific claims about any particular symptoms or clusters which appear first after a traumatic event. What emotional processing theory does indicate is that a range of reactions activate the fear structure, and Foa et al. specifically reference the *re-experiencing* and *hyperarousal* clusters (i.e., Cluster B, Cluster E, respectively) as those activating reactions (1989,

p.167) which lead to and maintain the attributions of dangerousness, and then subsequent avoidance of feared stimuli. So, while Foa et al. do not outright state a specific path to PTSD, which makes sense given their network perspective, they do appear to infer that *re-experiencing* and *hyperarousal* symptoms may come first, and that *persistent and exaggerated negative beliefs or expectations* is the most important symptom regarding developing PTSD, as well as *avoidance* when maintaining PTSD over time.

The dual representation theory from Brewin (2013) is not based on a fear network structure, such as the emotional processing theory (Foa et al., 1989), which treats traumatic memories as normal memories with a particular response set (i.e., attributions of dangerousness). Instead, Brewin (2013) asserts that traumatic memories are fundamentally distinct from normal memories. He terms these distinct memories as verbally accessible memories (VAM) and situationally accessible memories (SAM). Typical memories as we understand them fall under the VAM category, as they are memories which have been properly encoded with more conscious effort, context, and hence are readily able to be stated or written in a logical order according to dual representation theory. Traumatic memories, by contrast, typically fall into the SAM category because these memories are triggered by situations, or situational cues, and they are triggered involuntarily. Additionally, these SAM memories which contain trauma information also have affective responses which accompany the involuntary memory production. Involuntary memory production with affective responses is specific to the *re-experiencing* cluster, and Brewin originally developed the dual representation theory in order to explain *flashbacks*, though his description of how the traumatic memory is intrusive and contains affective information would also pertain to *intrusive thoughts*, *nightmares,* and *emotional or physical reactions to cues*. Hence, it appears that Brewin's dual representation theory describes

PTSD development primarily with the full set of re-experiencing symptoms. Subsequently, Brewin posited that when the re-experiencing cluster symptoms (e.g., flashbacks, intrusive thoughts, nightmares) were not properly processed into VAM, those symptoms would become emotionally disturbing and then those emotional disturbances would lead to avoidance. *Avoidance* is what Brewin claimed maintains the re-experiencing cluster symptoms via improper processing of the traumatic memory, because the individual cannot utilize their VAM and put the traumatic memory into context and dissociate it from the strong affective responses that go with the SAM. Thus, according to the dual representation theory, *re-experiencing* symptoms are the initial reactions and *avoidance* appears to be the critical factor to developing PTSD.

The final seminal theory is a cognitive model of PTSD by Ehlers and Clark (2000). Similar to the emotional processing theory (Foa et al., 1989), and portions of dual representation theory (Brewin, 2013), Ehlers and Clark state that PTSD develops through improper processing of the traumatic event (e.g., dual representation) that leads to negative attributions, or appraisals, of danger/threat (e.g., emotional processing) and negative personal characterization. The key distinction from emotional processing theory is the specificity of the negative appraisals which Ehlers and Clark describe, including: 1) the traumatic event itself (e.g., "'Nowhere is safe'"; 2000, p. 322, Table 1), 2) self-characterization based on the traumatic event (e.g., "'I attract disaster'"; p. 322, Table 1), 3) initial symptom reactions (e.g., "'I'm going mad'"; p. 322, Table 1), and 4) the subsequent consequences after a traumatic event or symptom reactions, such as physical, social, or loss of resource consequences (e.g., "'I will never be able to lead a normal life again'"; p. 322, Table 1). All of these negative appraisals, according to this cognitive model, negatively influence further PTSD symptoms via negative affective consequences. Putting these critical negative appraisals in symptom terms, they appear to best represent

symptoms from the *negative cognitions and mood cluster:* D2) "*persistent and exaggerated negative beliefs or expectations about oneself, others, or the world*" (APA, 2013, p. 272), and D3) "*persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself or others*" (p. 272). Regarding initial symptom reactions, they explicitly name symptoms which they view as common responses to traumatic events: "Symptoms such as *intrusive [thoughts]* and *flashbacks*, *irritability* and mood swings, *lack of concentration* and *numbing* are common reactions shortly after a traumatic event." (p. 322-23). Thus, it appears that the cognitive model of PTSD (Ehlers & Clark, 2000) asserts *re-experiencing* and *hyperarousal* symptoms as initial reactions, with *negative cognitions and mood* symptoms as the important developing factors for PTSD.

Overall, of the three seminal theories, the emotional processing theory (Foa et al., 1989) and the cognitive model (Ehlers & Clark, 2000) are the most similar regarding initial and important symptom reactions, whereas Brewin's dual representation theory (2013) is partially similar. Specifically, *re-experiencing* and *hyperarousal* are noted as the initial reactions by the emotional processing theory and the cognitive model, where only *re-experiencing* is noted as an initial reaction by the dual representation theory. Regarding important symptom reactions, emotional processing theory and the cognitive model both suggest *negative cognitions and mood* cluster symptoms (specifically pertaining to negative appraisals about the self, world, and others: Symptoms D2 and D3), while dual processing theory suggests *avoidance* is the most important cluster for developing and maintaining PTSD.

Much of the additional theoretical perspectives outside of the three seminal theories further support and align with the initial reactions and critical symptoms/clusters previously identified. For example, social-cognitive theory (Benight & Bandura, 2004) support emotional

processing theory (Foa et al., 1989) and the cognitive model (Ehlers & Clark, 2000) by stating that it is the negative appraisals of one's ability to cope and have support which go on to develop PTSD. Moreover, other cognitive and memory-based models (e.g., Nanney, Constans, Kimbrell, Kramer, & Pyne, 2015; Rubin, Berntsen, & Bohni, 2008) follow and support the cognitive model by Ehlers and Clark (2000) as well as the dual representation theory (Brewin, 2013).

While many of the additional theoretical perspectives support the three seminal theories, there exists a set of theories which primarily highlight the role of the *hyperarousal* cluster as the primary initial reactions to traumatic events as well as the critical developing factor for a PTSD trajectory. This set of theories include the survival mode theory (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988), fear avoidance theory (Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992), and an amygdala-based hyperarousal theory (Weston, 2014). First, survival mode theory (Chemtob et al., 1988) highlights the effects of ongoing hyperarousal, where the hyperarousal cluster symptoms (e.g., hypervigilance and anger) are perceived as useful responses in the acute response to the traumatic event(s) by protecting the individual or helping them cope with it. However, likely involving attributional processes (e.g., Ehlers & Clark, 2000), these hyperarousal responses become maladaptive when the individual's perceptions of threat are more generalized, or the perceived threat does not match the actual threat level of the given situation. Second, fear avoidance theory describes that *anger* (i.e., symptom E1; APA, 2013, p. 272) is preferable to negative emotional states such as fear, shame, guilt, etc., and anger may even be interpreted or perceived as positively valenced. Utilizing anger as a coping mechanism and replacement for other affective components is described as an avoidance tactic which then maintains PTSD symptoms, similar to dual representation theory (Brewin, 2013). Third, and finally, Weston (2014) proposed an amygdala-based theory of PTSD development, in which the

amygdala is stated to process the hyperarousal reactions and other stimuli during the traumatic event. Subsequently, the amygdala is thought to have a significant role in producing further hyperarousal reactions and, via the amygdala's modulation of the hippocampus and interactions with other critical brain regions (e.g., anterior cingulate, orbitofrontal, superior temporal, brainstem, and visual cortex), produces the differences in memory seen in individuals with PTSD and additional PTSD symptoms which maintain the disorder. Each of these theories have differing conceptual components and explanations for the role of *hyperarousal* symptoms; however, they consistently indicate hyperarousal symptoms as the initial reactions and as the developmentally important symptoms which inform PTSD trajectories.

In sum, the theoretical perspectives appear to share commonalities regarding initial reactions but vary in their explanation of developing and maintenance factors of PTSD (see Table 1). Emotional processing theory (Foa et al., 1989) and the cognitive model (Ehlers & Clark, 2000) both indicate *re-experiencing* and *hyperarousal* as the initial symptoms, while Brewin (2013) specifically notes *re-experiencing*. Contrarily, all of the *hyperarousal* theories (Chemtob et al., 1988; Riggs et al., 1992; Weston, 2014) indicate that hyperarousal symptoms are initial reactions to the traumatic event(s). Regarding the important developing and maintenance factors for PTSD, emotional processing theory and the cognitive model indicate *negative cognitions and mood* symptoms are the key factors (i.e., Symptom D2 & Symptom D3), Brewin's dual representation theory (2013) specifies that *avoidance* is the critically important symptom (as does Foa et al.'s emotional processing theory; 1989), and all of the hyperarousal theorists (Chemtob et al., 1988; Riggs et al., 1992; Weston, 2014) assert that *hyperarousal* is the key developmental and maintenance factor for PTSD.

1.2 – Empirical Findings Regarding Symptoms/Clusters

In the theoretical perspectives section, the *hyperarousal cluster* and *re-experiencing cluster* were noted as common initial symptoms following traumatic events (Brewin, 2013; Chemtob et al., 1988; Ehlers & Clark, 2000; Foa et al., 1989; Riggs et al., 1992; Weston, 2014). Building off of these theories, identifying the empirical results regarding initial symptom/cluster reactions as well as the symptoms/clusters which best predict PTSD severity over time provided more specific direction for hypotheses. For example, the dual representation theory (Brewin, 2013) highlights the re-experiencing cluster, as do several others who showed re-experiencing symptoms were common initial reactions after a traumatic event (Fraguas et al, 2006; McMillen, North, & Smith, 2000; North, 2004; North et al., 1999; Schell et al., 2004; Shalev, 1992). However, extant empirical literature largely highlights the impact of the hyperarousal and negative cognitions and mood clusters on PTSD severity over time (Carper et al., 2015; Doron-LaMarca et al., 2015; MacDonald et al., 2013; Rona et al., 2012; Schell, Marshall, & Jaycox, 2004).

While longitudinal studies have emphasized the importance of the hyperarousal and negative cognitions and mood clusters over time (Carper et al., 2015; Doron-LaMarca et al., 2015; MacDonald et al., 2013; Schell et al., 2004), there are several shortcomings which this dissertation seeks to address. These shortcomings include a lack of analyses specifically testing differing trajectories over time, most utilized reports from a significant amount of time after the traumatic exposure and lacked well-known risk factors. For example, Doron-LaMarca et al. (2015) assessed Vietnam Veterans long after their combat experiences (i.e., 2000-2004; at least a 25-year gap), did not incorporate risk factors for the development and maintenance of PTSD, nor did they ground their work in theory. They utilized sophisticated statistical models (i.e.,

structural equation modeling [SEM]), though did not investigate predictions of differing PTSD trajectories. Similarly, MacDonald et al. (2013) also recruited a veteran sample and utilized pre-deployment PTSD symptom-cluster severity to predict post-deployment PTSD symptom-cluster severity. While analyses with pre-deployment factors are commendable for being able to predict PTSD severity before and after trauma exposure, there is a serious confound to these types of studies: any PTSD symptoms that exist pre-deployment cannot be associated with the potentially traumatic event(s) they experience while in combat and thus are instead analyzing accumulated trauma exposure and/or prior PTSD distress predicting further maintenance. In a third SEM-based analysis, Schell et al. (2004) measured PTSD clusters approximately 10 days after traumatic exposure and asked participants to report on their symptoms since the event. This assessment of initial symptom reactions is the best of the cluster-based research, however Schell et al. (2004) did not specifically investigate primary symptom expression. Moreover, their longitudinal timepoints were 3 months and 12 months after the baseline assessment, which provides little in the way of assessing the developmental trajectories in those first months. Finally, Carper et al. (2015) analyzed the PTSD cluster responses in 120 women one month after they experienced a sexual assault. These women reported on their PTSD symptoms in the first month since their attack, and then Carper et al. (2015) utilized these reports to predict PTSD severity over time via linear regression analyses. Their study lacked a-priori hypotheses and were not able to incorporate the primacy of PTSD symptoms and differing trajectories over time. The current dissertation aimed to address these shortcomings with its strong theoretical foundations, the specific prediction of PTSD trajectories over time, and attempted to assess initial symptom reactions.

Within the trauma literature, there are two studies which best supported the current dissertation's aims. They are a part of a larger project which studied individuals longitudinally after they were admitted to an emergency room setting (Galatzer-Levy et al., 2014; Karstoft et al., 2015). Both studies utilized machine learning to predict a chronic, non-remitting PTSD trajectory. Galatzer-Levy et al. (2014) identified a number of symptoms that occur within the first 10 days post-trauma, and instead of identifying clusters they found multiple, specific symptoms which significantly predicted chronic PTSD: nightmares, flashbacks, difficulty concentrating, exaggerated startle, anger/irritability, derealization, and worthlessness. Once again, these symptoms are consistent with the previous PTSD literature which highlights the impact of hyperarousal (i.e., difficulty concentrating, exaggerated startle, anger/irritability), emotional numbing (i.e., derealization, worthlessness), and additionally provides some support to investigate the long-term impact of the re-experiencing cluster (i.e., nightmares, flashbacks). While these studies aligned particularly well with the current dissertation, there are also shortcomings to these investigations which this dissertation addressed. Specifically, these studies utilized exploratory analyses without grounding their work in theoretical perspectives, did not include an adequate range of risk factors, and additionally did not specifically assess which symptom reactions occurred initially.

These collective findings have implications for the current dissertation. First, the *hyperarousal* and *negative cognitions and mood* clusters are likely candidates for predicting PTSD longitudinally. Second, some of these studies also included results that suggested the *re-experiencing* cluster may additionally have predictive value, though this may be via an interaction with hyperarousal and avoidance clusters, which is consistent with existing theoretical perspectives as well (Brewin, 2013; Foa et al., 1989; Weston, 2014).

1.3 – Retrospective Report and Memory in PTSD

Importantly, the decision to investigate initial symptom reactions also critically brings into question *how* to measure them and has significant implications for the background and hypotheses. Specifically, because measuring the actual, observed PTSD symptoms as they occur is either difficult or impossible (given that some symptoms are completely internal and subjective), the decision was made to collect data on retrospective reports of initial symptoms. Retrospective report is often necessary due to an inability to measure at the time of the trauma (e.g., cannot easily predict when a trauma will occur) and additionally because not all PTSD symptoms can be objectively measured (e.g., some symptoms are purely subjective or internal in nature: perceptions of feeling distant or cut-off from other people, intrusive thoughts and memories about the trauma). Because this investigation involved retrospective accounts, it was crucial to further review the literature regarding how memory biases may have affected individual's responses to questions about their initial symptom reactions, as well as whether trauma victims can accurately report on their symptoms.

While a thorough investigation was conducted, the literature in this area is limited; three studies have investigated the correspondence between daily reports of PTSD symptoms and later retrospective reports (Campbell, Krenek, & Simpson, 2017; Nahleen, Nixon, & Takarangi, 2019; Naragon-Gainey et al., 2012). Specifically, Naragon-Gainey et al. (2012) investigated the correspondence between daily reports of PTSD symptoms and retrospective reports in a sample of 132 female sexual assault victims and found through multilevel analyses that correspondence between the daily symptom diaries and retrospective reports was generally high (pseudo R^2 ranged from .55 to .77). The *avoidance* cluster showed the greatest instability across the daily reports relative to the remaining PTSD clusters. They additionally investigated the role of

depression on retrospective reporting and found that depression did not affect retrospective reports. Campbell et al. (2017) utilized a smaller and demographically different sample compared to Naragon-Gainey et al. (i.e., n = 62 and alcohol dependent community members vs. n = 132 female sexual assault victims) and found relatively lower correspondence between daily and retrospective reports (pseudo R^2 ranged from .38 to .60). Notably, aside from the avoidance cluster (pseudo R^2 = .38), the results are generally similar to Naragon-Gainey et al. (2012). Additionally, similar to Naragon-Gainey et al., Campbell et al. (2017) found that *avoidance* exhibited the most instability across the PTSD clusters. Most recently, Nahleen et al. (2019) investigated the memory of sexual assault survivors by comparing their initial symptom reports to those 6 months later and found that generally the symptom reports were largely consistent. However, there were specific effects of PTSD severity on the distorted memory of PTSD symptoms, such that individuals with PTSD were more likely to overreport while individuals who recovered from PTSD symptoms exhibited a bias to underreport initial symptoms.

While there is generally consistent evidence that PTSD symptom reports are largely concordant, there is also consistent and concerning biases which exist and must be taken into context when viewing this dissertation. This limitation is provided initially because there is likely a clear difference between the actual, objective symptoms which occur initially after a traumatic event and the retrospective reports which were conducted as a part of this dissertation which may have differential effects on the prediction of long-term PTSD. However, this does not preclude the usefulness of retrospective accounts as it pertains to practical assessment in the aftermath of traumatic events. Specifically, if the retrospective reports, with memory biases and all, still predict PTSD severity above and beyond well-known risk factors, then that provides use with

avenues to help trauma victims at large consider their own initial PTSD reactions and understand the potential risks of those reactions relative to others as it relates to long-term severity.

1.4 – Putting it All in Context: Broader Empirical Risk Factors

In addition to the specific symptoms and clusters that have been shown to be initial reactions to traumatic events, the broader risk factors which have been well identified were included to add predictive value and to test the importance of initial symptom reports. For example, by including well-known risk factors, one can test the predictive and incremental validity of the initial symptom reactions (i.e., the relative importance or amount of variance explained when controlling for risk factors). The largest basis for the broader risk factors for developing PTSD comes from the seminal meta-analysis by Ozer et al. (2003). They identified the following risk factors: 1) prior trauma, 2) prior psychological adjustment, 3) family history, 4) perceived threat, 5) social support, 6) peritraumatic emotional responses, and 7) peritraumatic dissociation. This is the most widely cited meta-analysis regarding risk and protective factors for PTSD, and yet the effect sizes for any one risk factor range from small to medium (r = .17-.35). Hence, while these risk factors provide relevant information when attempting to identify individuals who will develop PTSD, the trauma field is still searching for more substantive predictors. More recent epidemiological studies have supported these meta-analytic results (Breslau, 2009; Johnson, Maxwell, & Galea, 2009; Karam et al., 2014; Keane, Marshall, & Taft, 2006). These investigations also extend Ozer et al.'s findings and concluded that prior traumatic experience (specifically 4 or more traumatic events; Karam et al., 2014), female gender, the severity of the traumatic experience, and coping efficacy also increase the risks for developing PTSD. Several of these risk factors were included to increase the overall predictive validity of the current dissertation and with the goal of improving the hypothesized models.

1.5 – Hypotheses

The hypotheses for this dissertation fit into 2 categories: 1) frequency of initial reactions, and 2) importance of initial reactions. Regarding initial reactions, it was expected that the *re-experiencing* cluster (i.e., Cluster B) would be the most commonly reported in the immediate aftermath of the traumatic event based on the seminal theories as well as empirical studies which investigated initial reactions after traumatic events (Brewin, 2013; Ehlers & Clark, 2000; Foa et al., 1989; Fraguas et al, 2006; McMillen, North, & Smith, 2000; North, 2004; North et al., 1999; Schell, Marshall, & Jaycox, 2004; Shalev, 1992). Based on the emotional processing theory, the cognitive model, and similar empirical results, the *hyperarousal* cluster (i.e., Cluster E) was also expected to be a frequent initial reaction (Chemtob et al., 1988; Ehlers & Clark, 2000; Foa et al., 1989; Riggs et al., 1992; Weston, 2014). Regarding long-term importance, there were three hypotheses: 1) that the *hyperarousal* cluster would predict PTSD severity over time via the hyperarousal theories and much empirical results (Chemtob et al., 1988; Riggs et al., 1992; Weston, 2014), 2) that *negative cognitions and mood* cluster would predict PTSD severity over time via the emotional processing theory, cognitive model, and some empirical results (Ehlers & Clark, 2000; Foa et al., 1989), and 3) the *re-experiencing* cluster has shown limited evidence for predicting PTSD severity (Galatzer-Levy et al., 2014) and is supported by the dual representation theory (Brewin, 2013), and thus was investigated regarding its predictive value. Each of these hypotheses were tested with the identified risk and protective factors for developing and maintaining PTSD (e.g., social support, self-efficacy, peritraumatic reactions), which were added to the computational models. They were added to improve the overall prediction of long-term PTSD and to more rigorously test the cluster-based hypotheses that previous studies had evidenced.

2.0 – Method

2.1 – Participants

Participants were N = 413 female undergraduate students at a large southeastern U.S. public university. They were recruited electronically on the university's online experiment management system. These women were asked about their traumatic experiences in order to qualify for the full, longitudinal study. When asked about their "worst traumatic event," 294 (71.19%) women reported exposure to a traumatic event as defined by the *DSM 5* (APA, 2013). Of these events, more than half (54.42%, n = 160) happened within the past 2 years. The women were additionally asked about their most recent potentially traumatic event regarding sexual assault or relationship violence, of which 136 women (32.93%) reported within the past 2 years. Women who reported these recent traumatic events qualified for the full longitudinal study. Of the 136 women who qualified based on their responses, 97 completed wave 2 (71.32%), 90 wave 3 (66.18%), and 80 at wave 4 (58.82%).

Due to the strengths of Hierarchical Linear Modeling (HLM), which will be described later in the methods section, the full sample of N = 413 were also utilized in order to assess the consistency of results. One hundred sixty-nine (40.92%) completed wave 2, 151 (36.56%) wave 3, and 131 (31.72%) completed wave 4.

2.2 – Procedure

Students provided informed consent upon accessing the study via the university research portal. Once consent was given, participants filled out initial demographics, information regarding traumatic events, PTSD symptom measures, as well as the remaining questionnaires (i.e., peritraumatic reactions, social support, coping self-efficacy). After completing the

questionnaires, participants were given a page with broader psychological resources including the APA, ABCT, and National Center for PTSD pages as well as the corresponding treatment resources from those organizations. Additionally, upon completing the questionnaires the participants were automatically sent an email thanking them for their participation and this email also contained crisis resources (e.g., emergency hotline numbers, national hotline number, Virginia-based hotline numbers organized by county/area) in case the participants felt they needed more immediate help. After a week passed, the participants received another automated email containing the survey link to the following assessment in the study, which consisted of the PTSD, social support, and trauma coping self-efficacy measures (as their demographics did not need to be continuously collected). With each additional assessment point, participants reviewed the consent form again and signed into the study utilizing their identifying information to confirm that they continued to agree to the study parameters. Once participants completed the 4 assessments over the 4-week timeframe, they received a final email thanking them for their participation. Students were offered extra credit on a 1-point per questionnaire completed basis. In sum, Prior trauma exposure and demographics were measured in week one, and the remaining symptoms were measured once a week.

2.3 – Measures

Demographics. Participants filled out demographic information including age, race/ethnicity, employment status, income, a three-question assessment of alcohol and drug use using 2 items from the Alcohol Use Disorders Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 2001; Saunders, Aasland, Babor, de la Puente, & Grant, 1993) and 1 from the Drug Use Disorders Identification Test (DUDIT; Berman, Bergman, Palmstierna, & Schlyter, 2003)), and mental health treatment history. Brief evaluations of substance use have been

identified to be as valid as the longer assessments (Dawson, Pulay, & Grant, 2010; Smith, Schmidt, Allensworth-Davies, & Saitz, 2010). The AUDIT questions demonstrated good internal consistency as evidenced by Cronbach's α = .80. Only 1 question was taken from the DUDIT and so internal consistency estimates are impossible. Demographics can be found in Table 2.

PTSD Symptoms and Prior Trauma Exposure. The PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013) was utilized to measure PTSD symptom severity. The PCL-5 is a 20 item self-report questionnaire designed to assess for posttraumatic stress disorder where the individual is reporting on the severity of PTSD symptoms in the past month. Recent psychometric analyses reveal that the PCL-5 demonstrates good reliability (Cronbach's α = .91 - .96; test-retest r = .82-84) and confirmatory factor analysis (CFA) supports the cluster-structure as found in the *DSM-5*, with additional models that add more specific factors within the hyperarousal and negative cognitions and mood clusters (Blevins, Weathers, Davis, Witte, & Domino, 2015; Bovin et al., 2015; Wortmann et al., 2016). In this investigation the PCL demonstrated reliability of Cronbach's α = 0.95 in relation to both sexual assault/relationship violence and Cronbach's α = 0.96 in the full sample. The same symptoms listed on the PCL were utilized when asking the women to report on their initial traumatic responses. Specifically, women were asked to recall their symptomology that they experienced within one week following the traumatic event.

The Life Events Checklist for DSM-5 (LEC-5; Weathers, Blake, Schnurr, Kaloupek, Marx, & Keane, 2013) was utilized in order to measure prior trauma exposure. The LEC-5 is a screener for potentially traumatic events where the individual is given a range of potentially traumatic events and asked to indicate the level of exposure to the event in a checklist format. The LEC-5 has not been formally evaluated, though it was utilized in the PCL-5 psychometric

studies listed above (i.e., Blevins et al., 2015; Bovin et al., 2015; Wortmann et al., 2016). In the only known evaluation of the Life Events Checklist (Gray, Litz, Hsu, & Lombardo, 2004), psychometrics showed good convergence with existing trauma history measure and correlated well with PTSD measures.

Peritraumatic Reactions. Peritraumatic reactions were measured via the Peritraumatic Distress Inventory (PDI; Brunet et al., 2001) and the Peritraumatic Dissociative Experiences Questionnaire—Self Report (PDEQ-SR; Marmar, Weiss, & Metzler, 1997). The PDI has shown adequate reliability (Cronbach's α = .75, test-retest = .74) and a two-factor structure (Brunet et al., 2001). Additionally, the PDI has been shown to correlate modestly to moderately with associated constructs (r = .21-.56; Brunet et al., 2001). The PDEQ-SR also has shown adequate reliability (Cronbach's α = .79-.80) as well as consistent one-factor structure (Marmar et al., 1997). In this dissertation, the PDI demonstrated good reliability in relation to sexual assault/relationship violence (Cronbach's α = .82) and slightly better in the full sample (Cronbach's α = .85). The PDEQ-SR showed good internal consistency (Cronbach's α = 0.85) in the sexual assault/relationship violence sample and slightly better (Cronbach's α = 0.88) in the full sample.

Social Support. Social support was measured via the Multidimensional Scale of Perceived Social Support (MSPSS; Zimet, Dahlem, Zimet, & Farley, 1988). The MSPSS psychometrics have been tested many times, with strong reliability coefficients (Cronbach's α = .93-.98; test-retest r = .72-.85). Regarding validity, the MSPSS has shown good divergent and convergent validity as well as a consistent 3-factor structure (Hardan-Khalil & Mayo, 2015). In this sample, the MSPSS demonstrated excellent internal consistency (Cronbach's α = .93).

Coping self-efficacy. Finally, coping self-efficacy was measured via the Trauma Coping Self-Efficacy scale (CSE-T; Benight, Shoji, James, Waldrep, Delahanty, & Cieslak, 2015). The CSE-T demonstrated good reliability (Cronbach's α = .87-.91, test-retest over many time-lags r = .57-.80; Benight et al., 2015). In their thorough psychometric investigation, Benight et al. (2015) also demonstrated good convergent and discriminant validity across a variety of methods, and additionally consistent factor structure and factor invariance. The CSE-T demonstrated excellent internal consistency (Cronbach's α = .92).

2.4 – Data Analysis

R (R Core Team, 2014) was utilized for data analyses. First, simple frequency and descriptive data were utilized to examine the first hypothesis regarding the most common initial symptom reactions. Regarding the long-term importance hypotheses, in order to best analyze the repeated-measures, longitudinal data, hierarchical linear modeling (HLM) was utilized for this dissertation. Given that previous studies' (Carper et al., 2015; Doron-LaMarca et al., 2015; MacDonald et al., 2013; Schell et al., 2004) effect sizes when utilizing clusters or symptoms and future PTSD generally range from medium to large (i.e., d = 0.53 – 1.007; r = .19 - .45), then I anticipated to find similarly moderate to strong effect sizes. The number of subjects needed for adequate power given the expected effect sizes is recommended to be a minimum of 30, and at or above 50 individuals with at least 4 time points would significantly reduce the chance for error in the fixed effect estimates and standard errors (Maas & Hox, 2005; Scherbaum & Ferreter, 2009).

Within HLM, when individuals are measured over time, the analysis is termed a growth model. In an HLM growth model, the first level contains variables measured over time (e.g., time points, symptoms over time) and the second level contains the person-level characteristics (e.g., age, race, initial symptom reactions reported). HLM-based growth models are well suited

for time-series data, as they are able to include all available data (i.e., no need for listwise deletion). Additionally, specifically relevant to growth models, different trajectories over time can be predicted by utilizing the second-level variables to predict differences in the first-level outcomes (because those first-level outcomes are always across time). For example, the long-term importance hypotheses would be evaluated by utilizing second-level variables (i.e., the initial cluster reported) to predict differences in PTSD severity in the intercept (i.e., whether baseline PTSD severity is higher or lower based on the reported initial symptom cluster) or to predict differences in PTSD severity over time (i.e., the slope; whether the trajectory of PTSD severity over time is worse based on the reported initial symptom cluster). Thus, HLM would be able to conduct analyses to replicate and extend previous findings, specifically because it allows for investigation of variation between and within individuals, such that one can explore the relationship of variables across time, including the effect of person-level variables (e.g., initial symptoms/clusters, social support, self-efficacy, peritraumatic reactions). HLM results were computed via packages *nlme* and *lme4*.

3.0 – Results

3.1 – Retrospective Reports of Symptoms

Frequency and ordering data was investigated for women's retrospective reports of their initial symptom reactions one week following trauma exposure. Undergraduate women who experienced recent sexual assault or relationship violence (n = 136) most commonly reported *cognitive avoidance* within the first week after their most recent traumatic event (n = 93, 68.38%), followed by *emotional reactions to cues* (n = 91, 66.91%). The least common symptoms reported were *risky behavior* (n = 26, 19.12%) and *anger* (n = 32, 23.53%). The average total symptom count was 8.50 symptoms (*SD* = 4.73). Regarding the ordering of these symptoms, women reported *negative cognitions and mood* cluster symptoms as being the first symptoms they experienced (n = 46, 33.82%). However, when investigating which symptoms were reported first, *intrusive thoughts* was the earliest symptom reported on average (*M* = 3.56, *SD* = 2.60). Other early symptoms were *emotional reactions to cues* (*M* = 4.70, *SD* = 2.69), and *flashbacks* (*M* = 4.76, *SD* = 2.54). The full list of symptom prevalence and reported ordering can be found in Table 3.

Additionally, the full sample (N = 413) also reported on their initial symptoms one week following trauma exposure in reference to their worst traumatic event. Similar to the women who reported sexual/relationship violence, most reported symptoms were *emotional reactions to cues* (n = 327, 79%), followed by *cognitive avoidance* (n = 293, 71%). The average total symptom count was 8.97 symptoms (SD = 5.01). Regarding ordering, *intrusive thoughts* was again the earliest symptom reported on average (*M* = 4.12, *SD* = 2.81), followed by *emotional reactions to cues* (*M* = 4.58, *SD* = 2.78). These results can also be found in Table 3.

Potential Effects of Time Since Trauma. Time since potential traumatic event was coded into weeks in order to keep same scale as weekly assessments. Regressions were computed to test the associations between symptom counts/presence/ordering and the weeks since the ***most recent traumatic event involving sexual assault/relationship violence.*** Overall symptom count (F[1,133] = 0.21, p = .65) nor cluster counts (F[4,130] = 1.60, p = .18) predicted time since traumatic event. Additionally, while the symptom presence regression did not predict time since traumatic event (F[20,114] = 1.56, p = .07), women who reported *loss of interest* (b = 23.61, t = 2.15, p = .03) and *feeling distant/detached* (b = 15.30, t = 2.08, p = .04) reported a longer amount of time since their trauma, while women who reported *low positive emotions* (b = -26.93, t = -2.68, p = .009) and *anger* (b = -17.48, t = -2.17, p = .03) reported less time since their trauma. Cluster ordering had no effect on time since trauma (F[4,130] = 1.46, p = .22). Similar to symptom presence, while symptom ordering controlling for symptom count did not predict time since trauma (F[21,113] = 1.10, p = .36), later ordering of *feeling distant/detached* was associated with longer time since trauma (b = 2.20, t = 2.15, p = .03).

In contrast, there were significant associations between the amount of time since the ***worst event in the full sample*** and symptom reports. First, women who reported *DSM-5* Criterion A traumatic events as their worst event also indicated that their events happened more recently compared to women who did not indicate a Criterion A event (t[172.99] = 2.41, p = .02). Regarding symptom reports, women who reported higher initial symptom count had more recent events (F[1, 394] = 23.75, p < .0001; b = -9.00, t = -4.87, p < .0001; R^2 = .05). Criterion A status was investigated as an interaction term and had no impact on the recent reports. Similarly, higher cluster counts were associated with more recent events.

3.2 – Hierarchical Linear Modeling (HLM)

Within the HLM results, first women's reports of recent sexual and relationship violence were prioritized, and then the full sample results will be presented later following this primary investigation. Thus, what immediately follows are the results based on sexual and relationship violence and can be found in Tables 4 - 10. Later, the full sample results are labeled differentiating it from the more specialized sample.

HLM Diagnostics. Before computing HLM statistics, one must identify whether HLM is appropriate for the given data. The primary way in which this is done is to calculate the intraclass correlation coefficient (ICC; Garson, 2012). The ICC for the women who experienced sexual assault/relationship violence was ICC = .75. Specifically, this can be interpreted generally in two ways: 1) 75% of the variance in PTSD severity is between persons, and 2) an individual woman's PTSD severity reports are correlated r = .75 with other time points. This ICC is acceptable to then analyze this data with HLM (Garson, 2012).

Unconditional Growth Model. In the unconditional growth model, only time is entered as a predictor. Time was coded where the baseline measurement was set at 0 and the remaining weeks were set at 1, 2, and 3. The intercept, or average PCL-5 score at baseline, was 23.77. Additionally, there was a significant effect of time such that each week the women's symptoms lowered by 2.49 points on average (b = -2.49, t[258] = -4.66, p < .0001). Compared to a Null Model (i.e., a model with only an intercept), time explained Pseudo R^2 = 35.77% of the level-1 random error (i.e., the variance of PTSD symptoms across time; Kreft & de Leeuw 1998; Singer, 1998; Snijders & Bosker, 1999). Model fit was AIC = 3,105.97 and BIC = 3,129.76. Results of the unconditional growth model can be found in Tables 4 - 9 as well as Figure 1.

Cluster/Symptom Models: Cluster/Symptom Presence. The basic number of symptoms endorsed was utilized to test the impact of initially reported PTSD symptom clusters and symptoms on future PTSD severity. Specifically, this was tested with three distinct independent variable sets: 1) the overall symptom count, 2) the symptom count parsed by the PTSD clusters (i.e., number of re-experiencing symptoms [Cluster B], number of avoidance symptoms [Cluster C], number of negative cognitions and mood symptoms [Cluster D], number of hyperarousal symptoms [Cluster E]), and then 3) exploring the effect of individual symptom presence.

Each model began with the unconditional growth model, then subsequently added the cluster/symptom variable(s), then added covariates. Model fit was investigated by comparing the new model fit indices (i.e., AIC, BIC) to the previous model. When AIC and BIC values fell, the variable(s) were retained. If the values increased, the variable was deleted from the model. Non-significant variables were also deleted from the model and fit indices were inspected, following the same rule (if AIC/BIC values fell with deletion, the variable was deleted, if AIC/BIC values rose, the variable was retained within the model). This way, more emphasis was placed on the model fit rather than statistical significance.

Symptom Count Model. When the reported total symptom count within the first week after the traumatic event was added such that it interacted with Time, model fit improved (AIC = 3,042.24; BIC = 3,073.93) compared to the unconditional growth model (decrease of 63.73 & 55.83 points, respectively). Results of this model and the following steps can be found in Table 4. Time was no longer a significant predictor of PTSD severity as it was in the unconditional model (p = .82). Symptom count was a significant predictor of baseline (i.e., intercept) PTSD severity such that for each symptom reported the baseline PTSD severity increased by b = 2.42

points, $t(131) = 9.70$, $p < .0001$. Additionally, symptom count significantly predicted differences in the slope of PTSD severity over time ($b = -0.31$, $t[257] = -2.84$, $p = .005$). Compared to the unconditional growth model, adding symptom count explained 48.03% of the variance in baseline PTSD severity, 13.20% of the variance in the slope of Time on PTSD severity, 4.28% in level-1 error, and 39.14% overall.

Covariates which added to overall model fit included: peritraumatic dissociative experiences (PDEQ), alcohol use (AUDIT), trauma coping self-efficacy over time (TCSE), and weeks since traumatic event. Specifically, model fit improved to AIC = 2912.59 and BIC = 2963.78 (129.65 & 110.15 points, respectively). None of these covariates improved model fit when entered as interaction terms with Time. Peritraumatic dissociative experiences predicted higher baseline PTSD severity ($b = 2.02$, $t[127] = 6.87$, $p < .0001$), as did higher number of alcoholic drinks when drinking ($b = 1.71$, $t[127] = 2.15$, $p = .03$). Weeks since traumatic event was associated with lower PTSD severity at baseline ($b = -.07$, $t[127] = -2.60$, $p = .01$). Additionally, peritraumatic dissociative experiences interacted with trauma coping self-efficacy to predict PTSD severity ($b = -0.04$, $t[252] = -5.03$ $p < .0001$). Time had no direct effect on PTSD severity ($p = .45$). Overall symptom count predicted higher baseline PTSD severity ($b = 1.69$, $t[127] = 7.88$, $p < .0001$) and also predicted differences in the slope of PTSD severity over time ($b = -0.28$, $t[252] = -3.09$, $p = .002$). Figure 2 depicts the impact of symptom count over time. Compared to the unconditional growth model, this model explained 71.78% of the variance in baseline PTSD severity, 55.40% of the variance in the slope of Time on PTSD severity, 7.37% in level-1 error, and 61.18% overall.

Cluster Count Model. In this set of models, the number of symptoms reported in each PTSD symptom cluster was included as a set of independent variables. Results of this model and

the following steps can be found in Table 5. When the set of cluster count variables was added, the best model was one in which only negative cognitions and mood cluster (Clusters D) and hyperarousal cluster (Cluster E) were included. Model fit was AIC = 3038.20 and BIC = 3073.83. Compared to the unconditional growth model, model fit improved by 67.77 points and 55.93 points, respectively. There was no direct effect of Time (p = .90). Reports of higher symptom counts in both Cluster D (b = 3.01, t[130] = 3.94, p = .0001) and Cluster E (b = 4.24, t[130] = 4.64, p < .0001) were associated with higher baseline PTSD severity. Only Cluster D significantly predicted differences in the slope of PTSD severity over time (b = -.78, t[257] = -2.91, p = .004). Compared to the unconditional growth model, this model explained 47.17% of the variance in baseline PTSD severity, 14.47% of the variance in the slope of Time on PTSD severity, 8.53% of level-1 error, and 39.86% overall.

The set of covariates which improved model fit were similar to the overall symptom count model. Model fit improved to AIC = 2911.49 and BIC = 2966.58 (an improvement of 126.71 and 107.25 points, respectively). Time did not have a direct effect (p = .78). Peritraumatic dissociation (b = 2.04, t[126] = 6.88, p < .0001) predicted increased PTSD severity at baseline. Peritraumatic dissociation interacted with trauma coping self-efficacy to predict PTSD severity (b = -.04, t[252] = -5.15, p < .0001). Regarding clusters, more Cluster D (b = 2.18, t[126] = 3.59, p = .0005) and Cluster E (b = 2.74, t[126] = 3.86, p = .0002) symptoms predicted increased PTSD severity at baseline. Regarding interactions with Time, once again Cluster D predicted differences in the slope of Time on PTSD severity (b = -0.71, t[252] = -3.20, p = .002). Figure 3 depicts the impact of Cluster D count over time. Compared to the unconditional growth model, this model explained 70.20% of the variance in baseline PTSD severity, 57.78% of the variance in the slope of Time on PTSD severity, 7.57% in level-1 error, and 60.09% overall.

Symptom Presence Model. First, all symptoms were added with interactions with Time. Interactions and symptoms were iteratively deleted until model fit was optimized based on AIC, BIC, and Pseudo R^2. Model fit was AIC = 3024.01 and BIC = 3075.33 (a decrease of 81.96 and 54.43 points). Results of this model and the following steps can be found in Table 6. In this model PTSD severity significantly decreased over time (b = -1.61, t[257] = -2.75, p = .007). Regarding symptoms, *emotional reactions to cues* (b = 9.02, t[126] = 3.39, p = .0009), *persistent negative beliefs* (b = 7.41, t[126] = 2.90, p = .004), *loss of interest* (b = 11.64, t[126] = 3.29, p = .001), *risky behavior* (b = 8.52, t[126] = 2.36, p = .03), and *exaggerated startle* (b = 6.06, t[126] = 2.40, p = .02) all predicted increased PTSD severity at baseline. Additionally, *loss of interest* predicted differences in the slope of Time on PTSD severity (b = -3.77, t[257] = -2.97, p = .003). Compared to the unconditional growth model, this model explained 44.15% of the variance in baseline PTSD severity, 14.58% of the variance in the slope of Time on PTSD severity, 0.96% in level-1 error, and 36.37% overall.

Similar to previous models, covariates which improved model fit were peritraumatic dissociation, alcohol use, help seeking, trauma coping self-efficacy, and time since trauma. Model fit improved to AIC = 2874.78, BIC = 2949.29. This was an improvement from the previous model of 135.03 and 119.74 points, respectively. PTSD severity decreased over time (b = -1.14, t[252] = .02). Peritraumatic dissociation (b = 2.13, t[121] = 7.44, p < .0001), alcohol use (b = 2.13, t[121] = 2.67, p = .009) predicted increased PTSD severity at baseline, while time since the traumatic event (b = -0.06, t[121] = -2.40, p = .02) predicted decreases. Trauma coping self-efficacy interacted with peritraumatic dissociation (b = -.04, t[252] = -5.60, p < .0001). Regarding symptoms, *emotional reactions to cues* (b = 6.57, t[121] = 3.53, p = .0006), ,loss of interest (b = 8.64, t[121], p = .001), *persistent negative beliefs* (b = 4.67, t[121] = 2.61, p = .01),

startle (*b* = 5.55, *t*[121] = 3.11, *p* = .002), and *risky behavior* (*b* = 6.30, *t*[121] = 2.48, *p* = .002) all predicted increases in PTSD severity at baseline, while *traumatic amnesia* (*b* = -4.77, *t*[) predicted decreases. *Loss of interest* predicted differences in the slope of Time on PTSD severity (*b* = -3.47, *t*[252] = -3.41, *p* = .0008). Figure 4 depicts the impact of *loss of interest* over time. Compared to the unconditional growth model, this model explained 73.14% of the variance in baseline PTSD severity, 58.50% of the variance in the slope of Time on PTSD severity, 8.80% in level-1 error, and 62.67% overall.

Cluster/Symptom Models: Cluster/Symptom Order. First, similar to the symptom count models, each model began with the unconditional growth model, then subsequently added the cluster/symptom order variables, and then covariates. Clusters and symptoms were entered as a set of categorical dummy variables. Regarding the clusters, ordering was simplified to identifying which cluster was reported as being present first, where the reference group was reporting no symptoms. Symptom order was kept as-is, and all symptoms which were unordered were coded as 0, and thus the reference group was also reporting no symptoms. Once again, prioritization was given to model fit which was investigated by comparing the new model fit indices (i.e., AIC, BIC) to the previous model. If any cluster or symptoms were deleted from the model to improve model fit, the reference group was re-specified.

Cluster Order Model. First, overall symptom count was added to control for symptom presence. Results of this model and the following steps can be found in Table 7. When adding just the cluster ordering, Clusters B, C, and E degraded model fit and were thus deleted from the model. Model fit improved to AIC = 3033.81 and BIC = 3085.14, which was a decrease of 68.98 and 45.37 points, respectively. Time had no effect (*p* = .23). Overall symptom count (*b* = 2.49, *t*[129] = 8.03, *p* < .0001) predicted increases in PTSD severity at baseline. Additionally, overall

symptom count predicted differences in the slope of Time (b = -0.55, t[255] = -3.98, p = .0001). Moreover, Cluster D ordering interacted with overall symptom count and Time (b = 0.55, t[255] = 2.34, p = .02). Compared to the unconditional growth model, this model explained 44.76% of the variance in baseline PTSD severity, 16.13% of the variance in the slope of Time on PTSD severity, 0% in level-1 error, and 36.40% overall.

Covariates which improved model fit were peritraumatic dissociation, trauma coping self-efficacy, and time since trauma. Additionally, Cluster B no longer provided better model fit after adding covariates, and so was deleted from the model. Thus, the only cluster which remained in this final model was Cluster D. Model fit was improved to AIC = 2905.32, BIC = 2968.12. This was an improvement from the previous model by 127.1 and 115.62 points, respectively. Time had no direct effect on PTSD severity (p = .10). Peritraumatic dissociation (b = 2.10, t[126] = 7.18, p < .0001) predicted increases in baseline PTSD severity. Trauma coping self-efficacy interacted with peritraumatic dissociation to predict PTSD severity (b = -0.04, t[250] = -5.32, p < .0001). Regarding symptoms, a higher overall symptom count predicted higher PTSD severity at baseline (b = 1.49, t[126] = 5.50, p < .0001). There were two interactions with Time: overall symptom count (b = -0.49, t[250] = -4.26, p < .0001), and a three-way interaction with overall symptom count and Cluster D ordering (b = 0.45, t[250] = 2.34, p = .02). Figure 5 depicts the impact of the interaction among symptom count and Cluster D ordering over time. Compared to the unconditional growth model, this model explained 69.91% of the variance in baseline PTSD severity, 58.37% of the variance in the slope of Time on PTSD severity, 7.08% in level-1 error, and 59.81% overall.

To investigate the specificity of the impact of symptom count on ordering, the cluster count variables were entered and the models re-tested. Results of this model can be found in

Table 8. Ordering of Clusters D and E improved model fit, as did the symptom count of Clusters C, D, and E. Model fit was AIC = 2894.62 and BIC = 2973.00, and when compared to the previous cluster order model was an improvement of AIC = 10.7 and a degeneration of BIC = 4.88 points. There was no effect of Time (p = .27). Peritraumatic dissociation (b = 2.19, t[123] = 7.33, p < .0001) and alcohol use (b = 1.74, t[123] = 2.14, p = .03) both predicted increases in PTSD severity at baseline. Trauma coping self-efficacy interacted with peritraumatic dissociation to predict PTSD severity (b = -.04, t[249] = -5.59, p < .0001). Regarding symptoms, Cluster D count (b = 1.69, t[123] = 2.51, p = .01) and Cluster E count (b = 2.66, t[123] = 3.79, p = .0002) predicted increased PTSD severity at baseline. Additionally, Cluster D ordering also interacted with Time (b = -0.80, t[249] = -3.12, p = .002) and had a three-way interaction with Time and Cluster C count (b = 2.62, t[249] = 2.24, p = .03). Figures 6 depicts the impact of the interaction among Cluster C count and Cluster D ordering over time. Compared to the unconditional growth model, this model explained 69.66% of the variance in baseline PTSD severity, 61.05% of the variance in the slope of Time on PTSD severity, 7.99% in level-1 error, and 59.85% overall.

Effects of symptom presence were then investigated alongside cluster ordering. Results of this model and the following steps can be found in Table 9. The best model resulted in AIC = 2815.78 and BIC = 2943.93, which was an improvement of 89.54 and 24.19 points compared to the cluster order and total symptom count model. There was no effect of time (p = .55). Peritraumatic dissociation (b = 2.26, t[114] = 7.83, p < .0001) predicted increased PTSD severity at baseline. Trauma coping self-efficacy interacted with peritraumatic dissociation to predict PTSD severity (b = -.04, t[245] = -5.83, p < .0001). Regarding symptoms, the presence of low positive emotions (b = 9.71, t[114] = 2.47, p = .01), emotional reactions to cues (b = 4.95, t[114]

= 2.52, p = .01), risky behavior (b = 6.76, t[114] = 2.62, p = .01), predicted increased PTSD severity at baseline; traumatic amnesia (b = -5.40, t[114] = -2.95, p = .004) was associated with decreases in PTSD severity. Cluster D order interacted with low positive emotions (b = -10.57, t[114] = -2.01, p = .05), sleep problems (b = 15.02, t[114] = 3.13, p = .002), and Time (b = 2.10, t[245] = 2.13, p = .03). Time additionally interacted with multiple symptoms, including: concentration problems (b = -3.22, t[245] = -3.13, p = .002), loss of interest (b = -3.31, t[245] = -2.61, p = .01), persistent negative emotions (b = -1.92, t[245] = -2.47, p = .01), and sleep problems (b = 4.56, t[245] = 3.91, p = .0001). Figure 7 depicts the impact of these symptoms over time. Cluster D order and Time also had three-way interactions with low positive emotions (b = 9.44, t[245] = 4.56, p < .0001) and sleep problems (b = -9.21, t[245] = -4.83, p < .0001). These interactions are depicted in Figures 8 - 9.

Symptom Order Model. All symptoms were entered into the model, and then were iteratively deleted to check for improved model fit. Individual symptom orderings did not improve model fit above and beyond overall symptom count and/or covariates; following these steps resulted in a model only containing overall symptom count. Thus, individual symptom orderings did not improve prediction of PTSD severity.

Summary of Model Fit. All of the models were compared to one another in order to identify the model with the most predictive utility. The full model fit results can be found in Table 10. Without covariates, the best initial model was the symptom presence model (AIC = 3009.81, BIC = 3069.03), which explained 48.70% of the variance in baseline PTSD severity, 14.44% of the variance in the slope of Time on PTSD severity, 0.85% in level-1 error, and 39.92% overall. Once adding covariates, the best model was the combination of symptom presence and cluster ordering (AIC = 2815.78, BIC = 2943.93), which explained 74.33% of the

variance in baseline PTSD severity, 90.00% of the variance in the slope of Time on PTSD severity, 6.71% in level-1 error, and 64.65% overall.

3.3 – HLM with Full Sample

In order to consistently test the effect of initial symptom reports and ordering on PTSD severity, the HLM models were re-tested with the full sample of N = 413 women in response to their *worst traumatic event*. As stated earlier, more than half of the full sample endorsed a Criterion A traumatic event (n = 294, 71.19%) and more than half of those individuals reported that event in the past 2 years (n = 160, 54.42%). Similar to the women who reported sexual assault/relationship violence, the ICC for the full sample in response to their worst traumatic event was ICC = .75. Specifically, this can be interpreted generally in two ways: 1) 75% of the variance in PTSD severity is between persons, and 2) an individual woman's PTSD severity reports are correlated r = .75 with other time points.

Full Sample Unconditional Growth Model. In the unconditional growth model, only time is entered as a predictor. Time was coded where the baseline measurement was set at 0 and the remaining weeks were set at 1, 2, and 3. The intercept, or average PCL-5 score at baseline, was 20.25. Additionally, there was a significant effect of time such that each week the women's symptoms lowered by 2.39 points on average (b = -2.39, t[451] = -6.72, p < .0001). Compared to a Null Model (i.e., a model with only an intercept), time explained Pseudo R^2 = 28.19% of the level-1 random error (i.e., the error variance of PTSD symptoms across time). Model fit was AIC = 6,801.54 and BIC = 6,830.02. Results of the unconditional growth model, as well as the following HLM models can be found in Tables 11 – 15. The full sample unconditional growth model is depicted in Figure 10.

Full Sample Cluster/Symptom Models: Symptom Presence. The steps to the full model was entirely similar to the above models, where the impact of initially reported PTSD symptom clusters and symptoms on future PTSD severity was investigated. Again, this was tested with three distinct independent variable sets: 1) the overall symptom count, 2) the symptom count parsed by the PTSD clusters (i.e., number of re-experiencing symptoms [Cluster B], number of avoidance symptoms [Cluster C], number of negative cognitions and mood symptoms [Cluster D], number of hyperarousal symptoms [Cluster E]), and then 3) exploring the effect of individual symptom presence. Model selection was also similar to the above models.

***Full Sample Symptom Count Model*.** Results of this model and the following steps can be found in Table 11. Covariates which added to overall model fit included: peritraumatic dissociative experiences (PDEQ), peritraumatic distress (PDI), drug use (DUDIT), trauma coping self-efficacy over time (TCSE), and weeks since traumatic event. Specifically, model fit improved to AIC = 6,118.01 and BIC = 6,183.70 (improvement of 683.53 & 646.32 points, respectively). Overall symptom count predicted higher baseline PTSD severity (b = 1.01, t[428] = 6.33, $p < .0001$) and also predicted differences in the slope of PTSD severity over time (b = -0.19, t[428] = -2.90, p = .004). Peritraumatic dissociative experiences (b = 1.54, t[428] = 7.52, $p < .0001$), peritraumatic distress (b = 0.29, t[428] = 4.01, $p < .0001$), and frequency of drug use (b = 1.44, t[378] = 2.40, p = .02) predicted higher baseline PTSD severity. Additionally, peritraumatic dissociative experiences interacted with trauma coping self-efficacy to predict PTSD severity (b = -0.03, t[428] = -6.74, $p < .0001$). Time had no direct effect on PTSD severity (p = .67). Compared to the unconditional growth model, this model explained 65.22% of the variance in baseline PTSD severity, 24.06% of the variance in the slope of Time on PTSD

severity, 14.52% in level-1 error, and 54.23% overall. The full sample symptom count model is depicted in Figure 11.

Full Sample Cluster Count Model. When investigating specific cluster counts and similar covariates, model fit improved to AIC = 6,107.90 and BIC = 6,178.26 compared to the unconditional growth model (693.64 & 651.76 points, respectively). Results of this model and the following steps can be found in Table 12. The set of covariates which improved model fit were similar to the overall symptom count model. Regarding clusters, more Cluster D (b = 1.41, t[427] = 4.29, p < .0001) and Cluster E (b = 1.63, t[427] = 3.42, p = .0007) symptoms predicted increased PTSD severity at baseline. PTSD severity decreased over Time (b = -4.24, t[423] = -1.95, p = .05). Regarding interactions with Time, Cluster E predicted differences in the slope of Time on PTSD severity (b = -0.61, t[427] = -3.09, p = .002). The full sample cluster count relationship of Cluster E count over time can be seen in Figure 12. Peritraumatic dissociation (b = 1.45, t[427] = 7.10, p < .0001), peritraumatic distress (b = 0.30, t[427] = 4.29, p < .0001), and drug use (b = 1.43, t[378] = 2.39, p = .02) predicted increased PTSD severity at baseline. Peritraumatic dissociation interacted with trauma coping self-efficacy to predict PTSD severity (b = -0.03, t[427] = -6.35, p < .0001). Compared to the unconditional growth model, this model explained 66.23% of the variance in baseline PTSD severity, 30.02% of the variance in the slope of Time on PTSD severity, 14.26% in level-1 error, and 55.14% overall.

Full Sample Symptom Presence Model. When individual symptom presence was added, model fit compared to the unconditional growth model improved to AIC = 6,102.13, BIC = 6,186.50 (699.41 and 643.52 points, respectively). Results of this model and the following steps can be found in Table 13. Time had no direct effect (p = .16). Regarding symptoms, persistent self-blame (b = 2.54, t[374] = 2.29, p = .02), anger (b = 3.88, t[374] = 2.64, p = .009), risky

behavior (b = 4.41, t[374] = 2.52, p = .01), and sleep problems (b = 4.74, t[374] = 3.81, p = .0002) all predicted increases in PTSD severity at baseline. Anger (b = -1.83, t[428] = -2.65, p = .009) and sleep problems (b = -1.83, t[428] = -6.23, p = .003) predicted differences in the slope of Time on PTSD severity. The impact of these symptoms over time can be seen in Figure 13. Peritraumatic dissociation (b = 1.48, t[424] = 7.18, p < .0001), peritraumatic distress (b = 0.37, t[428] = 5.33, p < .0001), and drug use (b = 1.20, t[374] = 1.98, p = .05) predicted increased PTSD severity at baseline, while trauma coping self-efficacy (b = -0.14, t[428] = -2.06, p = .04) predicted decreases. Trauma coping self-efficacy interacted with peritraumatic dissociation (b = -.03, t[428] = -6.23, p = .003). Compared to the unconditional growth model, this model explained 65.52% of the variance in baseline PTSD severity, 36.53% of the variance in the slope of Time on PTSD severity, 13.83% in level-1 error, and 54.71% overall.

Full Sample Cluster/Symptom Models: Cluster/Symptom Order. The steps to the full model was entirely similar to the above sexual assault/relationship violence models. To review, ordering clusters was simplified to identifying which cluster was reported as being present first, where the reference group was reporting no symptoms. Symptom order was kept as-is, and all symptoms which were unordered were coded as 0, and thus the reference group was also reporting no symptoms. Once again, prioritization was given to model fit which was investigated by comparing the new model fit indices (i.e., AIC, BIC) to the previous model. If any cluster or symptoms were deleted from the model to improve model fit, the reference group was re-specified.

Full Sample Cluster Order Model. Similar to the models predicting PTSD severity after sexual or relationship violence, the best ordering-based model was one which contained cluster ordering and individual symptom presence. Results of this model and the following steps can be

found in Table 14. Model fit was AIC = 4,760.23 and BIC = 4,901.72 (difference of 2,041.31 & 1,928.30 points, respectively). Three individual symptoms predicted increased baseline PTSD severity: traumatic amnesia (b = 5.29, t[266] = 2.94, p = .004), loss of interest (b = 2.98, t[266] = 2.00, p = .05), and risky behavior (b = 3.88, t[266] = 2.03, p = .04). There were also multiple interactions with cluster ordering and symptom presence. Regarding interactions with the hyperarousal cluster ordering (Cluster E ordering), persistent blame (b = 15.40, t[266] = 3.88, p = .0001) and feeling distant/detached (b = -9.20, t[266] = -2.46, p = .01) interacted with Cluster E ordering to predict PTSD severity at baseline. The negative cognitions and mood cluster ordering (Cluster D ordering) interacted with traumatic amnesia (b = -8.39, t[266] = -3.10, p = .002), exaggerated startle (b = 6.98, t[266] = 2.72, p = .007), and sleep problems (b = 5.61, t[266] = 2.20, p = .03). Time had no direct effect on PTSD severity (p = .14), though interacted with concentration problems (b = -1.86, t[349] = -2.74, p = .007) and there was a three-way interaction with Time, Cluster D ordering, and persistent negative beliefs (b = 3.31, t[349] = 2.40, p = .02). These interactions with time are depicted in Figures 14 and 15. Peritraumatic dissociation (b = 1.01, t[266] = 4.08, p = .0001) and peritraumatic distress (b = 0.36, t[266] = 4.33, p < .0001) predicted increased PTSD severity at baseline, while trauma coping self-efficacy (b = -0.22, t[349] = -2.50, p = .01) predicted decreases. Trauma coping self-efficacy also interacted with peritraumatic dissociation (b = -.02, t[349] = -3.37, p = .0008). Compared to the unconditional growth model, this model explained 63.14% of the variance in baseline PTSD severity, 31.12% of the variance in the slope of Time on PTSD severity, 18.48% in level-1 error, and 53.78% overall.

Full Sample Symptom Order Model. All symptoms were entered into the model and interactions with Time and overall symptom count were investigated. Once again, the specific

symptom orderings did not improve model fit above and beyond the overall symptom count model. Thus, individual symptom orderings did not improve prediction of PTSD severity.

Full Sample Summary of Model Fit. All of the full sample models were compared to one another in order to identify the model with the most predictive utility. The full model fit results can be found in Table 15. The best model was the combination of symptom presence and cluster ordering (AIC = 4,760.23, BIC = 4,901.72), which explained 63.14% of the variance in baseline PTSD severity, 31.12% of the variance in the slope of Time on PTSD severity, 18.48% in level-1 error, and 53.78% overall.

4.0 – Discussion

This investigation was designed to analyze the impact of reported PTSD symptoms within the immediate aftermath of a traumatic event on the eventual development and maintenance of PTSD via longitudinal regression models in a sample of women exposed to sexual or relationship violence. Moreover, this potential impact of reported symptoms was also investigated in the full sample of college-age women in reference to their worst traumatic event.

4.1 – Summary of Results

Regarding the results' alignment with the hypotheses: 1) the re-experiencing cluster symptoms were indeed the most frequent reactions recalled as initial symptoms, 2) the hyperarousal cluster (Cluster E) largely was not predictive of a severe trajectory; negative cognitions and mood cluster (Cluster D) was, and 3) the re-experiencing cluster (Cluster B) was not a significant predictor of PTSD severity trajectories over time. Overall, increased symptom count (whether via overall count, particular clusters, or symptoms) generally predicted increased PTSD severity at the baseline measurement. This is consistent with existing research which has shown that acute traumatic stress reactions predict PTSD (Bryant, 2003; Hansen, Hyland, Armour, 2016). Regarding the specific impact of cluster count predicting later PTSD severity, increased Cluster D count (i.e., negative cognitions and mood) was a consistent predictor of higher baseline PTSD severity and interacted with time such that women who reported more Cluster D symptoms then decreased more over time when controlling for all other variables. Additional symptoms which impacted baseline PTSD severity were emotional reactions to cues, startle, risky behavior, and traumatic amnesia. The number of symptoms also had an impact when investigating the order effects. Consistently, there was a three-way interaction with time, symptom count, and ordering. More specifically, in the cluster ordering models, Cluster D

interacted with symptom count and time. PTSD severity increased over time when women reported Cluster D symptoms as being their first symptoms and also reported a higher number of initial symptoms. Loss of interest and persistent negative beliefs appeared to be driving these Cluster D effects, where the interactions with time appeared to be due to loss of interest. This is in contrast to many of the other interactions with time which showed that symptoms generally decreased over time (as consistent with resiliency research in PTSD; Bonanno et al., 2012; Bryant, 2003; Bryant et al., 2015). These cluster-based findings were supported in the full sample as well, wherein both negative cognitions and mood and the hyperarousal clusters (i.e., Clusters D & E) were influential when predicting PTSD severity related to the worst traumatic event ever experienced. Thus, it appears that reporting Cluster D symptoms early in the aftermath of a trauma may have long-term implications.

Individual symptom presence and cluster ordering provided the best model according to fit statistics as well as variance explained. Three clusters were represented in this model: re-experiencing, negative cognitions and mood, and hyperarousal (i.e., Clusters B, D, & E). Specifically regarding re-experiencing symptoms, the combination of initial presence of physical reactions to cues and earlier Cluster D ordering were related to increased PTSD severity. Additionally, emotional reactions to cues were also associated with more PTSD severity. Regarding negative cognitions and mood symptoms, traumatic amnesia was associated with decreases in PTSD severity whereas low of positive emotions was associated with increased PTSD severity. If women reported low of positive emotions and Cluster D symptoms as their first symptom, their PTSD symptoms remained worse over time. Cluster D ordering also interacted with sleep problems, such that if women reported Cluster D symptoms as their first symptom and did not report initial sleep problems, they appeared to maintain a sub-clinical

PTSD presentation over time. Similarly, the full sample best model was also the combination symptom presence and cluster order model which also highlighted the impact of Cluster D across time. In contrast to the models predicting PTSD in sexual/relationship violence-related events, only Cluster D and Cluster E were represented in this model. Additionally, Cluster D ordering interacted with persistent negative beliefs instead of low positive emotions, though the overall pattern of Cluster D ordering and symptom presence over time remained the same.

While there were clear statistical interactions between Cluster D and time, it is also critical to note that other cluster/symptom presence may have had long-term effects on PTSD severity. Specifically, in the cluster count model, the rate of PTSD severity over time was positive, meaning that variables which predicted higher baseline severity would then continue over time. Thus, Cluster E predicted more PTSD severity at baseline, which then means that the increased PTSD severity then persisted over time. Similarly, sleep problems in the best overall model interacted with time such that individuals who reported sleep problems had more persistent PTSD severity. Researchers have consistently shown that increased hyperarousal has similar effects and is associated with increased PTSD severity over time and are consistent with the results found here (Doron-LaMarca et al., 2015; MacDonald et al., 2013).

4.2 – Implications

The immediate implications are that negative cognitions and mood symptoms (i.e., Cluster D symptoms) may have more impact on the eventual development and maintenance of PTSD than previously thought. Consistent with prior research (Bonanno et al., 2012; Bryant, 2003; Bryant et al., 2015), PTSD severity generally decreased over time, with the notable exception of women who recalled and reported more symptoms early on and additionally reported that their first symptoms (within one week) were within the negative cognitions and

mood cluster. Moreover, these results additionally indicate that *low positive emotions* may be driving this relationship in women who have experienced sexual assault or relationship violence. *Low positive emotions* as a symptom is fully detailed as "persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings)" (APA, 2013). Previously, *low positive emotions* was worded as "restricted range of affect (e.g., unable to have loving feelings)" (APA, 2011; Friedman, 2013). Both definitions include the dulling/numbing of emotions, of which the previous PTSD cluster was named (APA, 2011). Importantly, emotional numbing has significant theoretical ties to the development and maintenance of PTSD through connections to avoidance, negative beliefs, and emotional distress (Brewin, 2013; Ehlers & Clark, 2000; Foa et al., 1989, Riggs et al., 1992).

Persistent negative beliefs, also per seminal PTSD theories (e.g., Ehlers & Clark, 2000; Foa et al., 1989), was critical in predicting PTSD severity over time in the full sample. Persistent negative beliefs are the mechanism by which Ehlers and Clark (2000) theorized that PTSD develops and also the primary treatment target in cognitive processing therapy (CPT; Resick, Monson, & Chard, 2016). The cognitive model of PTSD (Ehlers & Clark, 2000) may then be the best way to conceptualize the development of PTSD compared to the dual representation theory (Brewin, 2013) and the emotional processing theory (Foa et al., 1989) which do not put much focus on negative cognitions but rather the re-experiencing and hyperarousal symptoms.

While hyperarousal symptoms had some support for predicting PTSD severity in this investigation, as per the effect of *sleep problems* on long-term PTSD severity, sleep problems interacted with Cluster D ordering to produce the steepest recovery trajectory (see Figure 9). This, and the above symptom-specific interactions likely need to be interpreted with significant caution, as they may be sample-specific effects or due to random statistical artifacts. Moreover,

these findings indicate that commonly reported symptoms (e.g., Cluster B re-experiencing symptoms) may not be as impactful as suggested by Brewin (2013) in his dual representation theory or by Foa et al. (1989) in conceptualizing PTSD as more of a fear-based network. These symptoms do clearly make an impact, as noted in the many symptoms and cluster-based findings which showed that Cluster B and Cluster E symptoms were associated with increased baseline PTSD severity, but largely were not associated with or predicted severity over time. Instead, there appears to be a core cognitive and affective component captured best by the Cluster D symptoms which are then associated with long-term PTSD severity.

What was additionally striking is the avoidance cluster (Cluster C) had seemingly no impact on PTSD severity in either associations with baseline severity or long-term reactions. Each theory which explains PTSD development contains a significant component on the importance of avoidance in the development and maintenance of PTSD (Brewin, 2013; Ehlers & Clark, 2000; Foa et al., 1989, Riggs et al., 1992). There may be multiple explanations for this finding, both of which align with the theoretical perspectives rather than diverging from them. The first is that avoidance as symptoms are necessary for the development of PTSD, but that the symptoms are not good predictors of the *severity* of PTSD over time. In this explanation, avoidance of cognitions or of external stimuli is diagnostic in nature, but its presence and/or order in the development of PTSD may not yield much predictive information. The second explanation is that avoidance may be inherently contained within the other PTSD symptoms such as *low positive emotions*, which may be an indicator of significant emotional avoidance. In this explanation, symptoms that indicate a serious difficulty coping with trauma are also avoidance tactics. Integrating the above points, perhaps as a field we should revisit and pay significant attention to Cluster D and its importance on the development and maintenance of PTSD while

also considering that additional symptoms (e.g., low positive emotions) may be better indicators of the theoretically important avoidance construct.

Importantly, these implications also logically have direct clinical implications regarding early identification of PTSD and potential targets for early intervention. Trauma victims who report early experiences with negative cognition and mood cluster (Cluster D) symptoms may be at higher risk for PTSD longitudinally and these symptoms may be better targets for treatment compared to the fear- or intrusive-based symptoms which are targets in many interventions for PTSD. Early interventions for acute stress disorder have shown promise with targeting negative cognitions through CBT (Bryant, Harvey, Dang, Sackville, & Basten, 1998; Bryant, Moulds, & Nixon, 2003; Ehlers et al., 2003). Additionally, borrowing from the depression field, many of the negative cognitions and mood symptoms share diagnostic similarity with depression and could thus be treated as such. For example, if trauma victim presents with *low positive emotions* (anhedonia), then evidence-based practices may recommend behavioral activation and/or interpersonal psychotherapy (IPT) for depression (Markowitz et al., 2015; Wagner, Zatzick, Ghesquiere, & Jurkovich, 2007). Such intervention tactics would align greatly with existing empirical data which suggest that social support (and logically the behavioral activation which goes with social support) is the best protective factor against PTSD (see Ozer et al., 2003).

4.3 – Limitations

This dissertation is not without limitations. First and foremost, these analyses were based on retrospective reports of symptoms, which has potential impact on the accuracy of reports. Importantly, however, is that accuracy of these reports does not logically mean that the retrospective reports themselves are not meaningful or do not have significant associations with PTSD severity. The time since the traumatic event did not have any significant impact on the

reporting of symptoms in the women who experienced sexual assault, and also did not impact PTSD severity reports in the full sample as evidenced by the HLM results described above. Instead, this limitation warrants caution when applied to interpreting these retrospective reports as an indicator of actual sequences of symptoms. For example, there are consistent findings that highlight individuals with PTSD have memory deficits in working memory, verbal memory, non-trauma related autobiographical memory (e.g., episodic memory), and that traumatic narratives show more disorganization compared to traumatic narratives in non-PTSD individuals (Brewin, 2013; Isaac et al., 2006). Additionally, we do not know how the biases which exist in remembering PTSD symptomology (Nahleen et al., 2019) interact to predict long-term PTSD severity as the literature is significantly limited in this area. Furthermore, interpreting the symptom-specific results should be done so with a message of strong caution that such a specific level of report may be largely affected by these memory biases and without replication may be sample-specific effects. Similarly, another limitation is that these findings are from a sample of college women and thus may not generalize to males or other traumatic event-specific populations (e.g., natural disasters, mass violence, combat). For example, data exist which show that differing trauma types may result in differing severity and symptomology (Graham, Legarreta, North, DiMuzio, McGlade, & Yurgelun-Todd, 2016; Smith, Summers, Dillon, & Cougle, 2016). Thus, the cluster effects and symptom-specific effects found within this dissertation may be likely to change depending on the trauma type investigated, and future researchers are encouraged to replicate and/or test these same findings across trauma type. Further, temporally a significant portion of the sample reported that their worst traumatic events occurred approximately 2 years ago or older, which then means prediction of PTSD severity at baseline should be considered as predicting PTSD severity at however much time has elapsed

since their traumatic event. While the time since traumatic event was controlled for, there may still be an impact from the variability of differing times since trauma exposure, as in the difference between a victim who is in their 4th month of coping compared to one in their 11th or 12th. While difficult, future research may attempt to directly compare trauma victims in the same temporal stages in order to reduce this variability.

4.4 – Conclusions

In the first attempt at identifying the impact of specific cluster and symptom ordering and presence on PTSD severity, it was found that the negative cognitions and mood cluster was the best predictor of long-term severity. Additional support was found for the hyperarousal cluster, as well as *emotional reactions to cues* from the re-experiencing cluster. The cluster-based results were consistent across trauma types and added value above and beyond well-known risk and protective factors of PTSD. These findings may have implications for reviewing and updating theoretical perspectives to adjust the focus to the negative cognitions and mood cluster which appears to best align with Ehlers and Clark's cognitive model (2000). Moreover, there may be treatment implications from focusing on the negative cognitions and mood cluster, namely less time spent targeting re-experiencing symptoms and more on the negative beliefs and loss of positive emotions, which may additionally assist in the early identification and intervention after a traumatic event.

References

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders (5th edition.).* Arlington, VA: American Psychiatric Publishing, Inc.

Babor, T.F., Higgins-Biddle, J.C., Saunders, J.B., & Monteiro, M.G. (2001). AUDIT. The Alcohol Use Disorders Identification Test. Guidelines for Use in Primary Care, 2nd ed. Available at: http://whqlibdoc.who.int/hq/2001/WHO_MSD_MSB_01.6a.pdf

Benight, C. C., & Bandura, A. (2004). Social cognitive theory of posttraumatic recovery: The role of perceived self-efficacy. *Behaviour research and therapy*, *42*(10), 1129-1148.

Benight, C. C., Shoji, K., James, L. E., Waldrep, E. E., Delahanty, D. L., & Cieslak, R. (2015). Trauma Coping Self-Efficacy: A context-specific self-efficacy measure for traumatic stress. *Psychological trauma: theory, research, practice, and policy*, *7*(6), 591.

Berman, A., Bergman, H., Palmstierna, T., & Schlyter, F. (2003). The Drug Use Disorders Identification Test (DUDIT) Manual. Stockholm, Sweden: Karolinska Institutet.

Blevins, C. A., Weathers, F. W., Davis, M. T., Witte, T. K., & Domino, J. L. (2015). The posttraumatic stress disorder checklist for DSM-5 (PCL-5): Development and initial psychometric evaluation. *Journal of Traumatic Stress*, *28*(6), 489-498.

Bonanno, G. A., Mancini, A. D., Horton, J. L., Powell, T. M., LeardMann, C. A., Boyko, E. J., ... & Millennium Cohort Study Team. (2012). Trajectories of trauma symptoms and resilience in deployed US military service members: prospective cohort study. *The British Journal of Psychiatry*, *200*(4), 317-323.

Bovin, M. J., Marx, B. P., Weathers, F. W., Gallagher, M. W., Rodriguez, P., Schnurr, P. P., & Keane, T. M. (2016). Psychometric properties of the PTSD Checklist for Diagnostic and Statistical Manual of Mental Disorders–Fifth Edition (PCL-5) in veterans. *Psychological Assessment*, *28*(11), 1379.

Breslau, N. (2009). The epidemiology of trauma, PTSD, and other posttrauma disorders. *Trauma, Violence, & Abuse*, *10*(3), 198-210.

Brewin, C. R. (2013). Episodic memory, perceptual memory, and their interaction: Foundations for a theory of posttraumatic stress disorder. *Psychological Bulletin*, *140*(1), 69–97.

Brunet, A., Weiss, D. S., Metzler, T. J., Best, S. R., Neylan, T. C., Rogers, C., ... & Marmar, C. R. (2001). The Peritraumatic Distress Inventory: a proposed measure of PTSD criterion A2. *American Journal of Psychiatry*, *158*(9), 1480-1485.

Bryant, R. A., Harvey, A. G., Dang, S. T., Sackville, T., & Basten, C. (1998). Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. *Journal of consulting and clinical psychology*, *66*(5), 862.

Bryant, R. A., Moulds, M. L., & Nixon, R. V. (2003). Cognitive behaviour therapy of acute stress disorder: a four-year follow-up. *Behaviour research and therapy*, *41*(4), 489-494

Bryant, R. A., Nickerson, A., Creamer, M., O'Donnell, M., Forbes, D., Galatzer-Levy, I., ... & Silove, D. (2015). Trajectory of post-traumatic stress following traumatic injury: 6-year follow-up. *The British Journal of Psychiatry*, *206*(5), 417–423.

Campbell, S. B., Krenek, M., & Simpson, T. L. (2017). The Role of Patient Characteristics in the Concordance of Daily and Retrospective Reports of PTSD. *Behavior Therapy*.

Carper, T. L., Mills, M. A., Steenkamp, M. M., Nickerson, A., Salters-Pedneault, K., & Litz, B. T. (2015). Early PTSD symptom sub-clusters predicting chronic posttraumatic stress following sexual assault. *Psychological Trauma: Theory, Research, Practice, and Policy, 7*(5), 442–447.

Chemtob, C. M., Roitblat, H. L., Hamada, R. S., Carlson, J. G., & Twentyman, C. T. (1988). A cognitive action theory of posttraumatic stress disorder. *Journal of Anxiety Disorders, 2*, 253 – 275.

Crespo, M., & Fernández-Lansac, V. (2016). Memory and narrative of traumatic events: A literature review. *Psychological trauma: theory, research, practice, and policy, 8*(2), 149.

Cuccinelli, K. T. (2014). Domestic and Sexual Violence in Virginia. *Commonwealth of Virginia Office of the Attorney General.*

Dawson, D. A., Pulay, A. J., & Grant, B. F. (2010). A comparison of two single-item screeners for hazardous drinking and alcohol use disorder. Alcoholism: Clinical and Experimental Research, 34(2), 364-374.

Doron-LaMarca, S., Niles, B. L., King, D. W., King, L. A., Pless Kaiser, A., & Lyons, M. J. (2015). Temporal Associations Among Chronic PTSD Symptoms in U.S. Combat Veterans: Chronic PTSD Over Time. *Journal of Traumatic Stress, 28*(5), 410–417.

Dunmore, E., Clark, D. M., & Ehlers, A. (2001). A prospective investigation of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour research and therapy*, *39*(9), 1063-1084.

Ehlers, A. (2010). Understanding and treating unwanted trauma memories in posttraumatic stress disorder. *Zeitschrift Für Psychologie/Journal of Psychology*.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*(4), 319–345.

Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., Fennell, M., Herbert, C., & Mayou, R. (2003). A randomized controlled trial of cognitive therapy, a self-help booklet, and repeated assessments as early interventions for posttraumatic stress disorder. *Archives of general psychiatry*, *60*(10), 1024-1032.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior therapy*, *20*(2), 155-176.

Fraguas, D., Terán, S., Conejo-Galindo, J., Medina, O., Cortón, E. S., Ferrando, L., … Arango, C. (2006). Posttraumatic stress disorder in victims of the March 11 attacks in Madrid admitted to a hospital emergency room: 6-month follow-up. *European Psychiatry*, *21*(3), 143–151.

Garson, G. D. (2012). *Hierarchical linear modeling: Guide and applications*. Sage.

Galatzer-Levy, I. R., & Bryant, R. A. (2013). 636,120 ways to have posttraumatic stress disorder. *Perspectives on Psychological Science*, *8*(6), 651–662.

Galatzer-Levy, I. R., Karstoft, K.-I., Statnikov, A., & Shalev, A. Y. (2014). Quantitative forecasting of PTSD from early trauma responses: A Machine Learning application. *Journal of Psychiatric Research*, *59*, 68–76.

Graham, J., Legarreta, M., North, L., DiMuzio, J., McGlade, E., & Yurgelun-Todd, D. (2016). A preliminary study of DSM–5 PTSD symptom patterns in veterans by trauma type. *Military psychology*, *28*(2), 115-122.

Gray, M. J., Litz, B. T., Hsu, J. L., & Lombardo, T. W. (2004). Psychometric properties of the life events checklist. *Assessment*, *11*(4), 330-341.

Isaac, C. L., Cushway, D., & Jones, G. V. (2006). Is posttraumatic stress disorder associated with specific deficits in episodic memory?. *Clinical psychology review*, *26*(8), 939-955.

Johnson, J., Maxwell, A., & Galea, S. (2009). The epidemiology of posttraumatic stress disorder. *Psychiatric Annals*, *39*(6).

Karam, E. G., Friedman, M. J., Hill, E. D., Kessler, R. C., McLaughlin, K. A., Petukhova, M., ... & Girolamo, G. (2014). Cumulative traumas and risk thresholds: 12-month PTSD in the World Mental Health (WMH) surveys. *Depression and Anxiety*, *31*(2), 130-142.

Karstoft, K.-I., Galatzer-Levy, I. R., Statnikov, A., Li, Z., Shalev, A. Y., & Four members of the Jerusalem Trauma Outreach and Prevention Study (J-TOPS) group. (2015). Bridging a translational gap: using machine learning to improve the prediction of PTSD. *BMC Psychiatry*, *15*(1), 30.

Kaysen, D. L., Lindgren, K. P., Lee, C. M., Lewis, M. A., Fossos, N., & Atkins, D. C. (2010). Alcohol-involved assault and the course of PTSD in female crime victims. Journal of traumatic stress, 23(4), 523-527.

Keane, T. M., Marshall, A. D., & Taft, C. T. (2006). Posttraumatic stress disorder: etiology, epidemiology, and treatment outcome. *Annu. Rev. Clin. Psychol.*, *2*, 161-197.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of general psychiatry*, *62*(6), 593-602.

Kilpatrick, D. G., Resnick, H. S., Milanak, M. E., Miller, M. W., Keyes, K. M., & Friedman, M. J. (2013). National estimates of exposure to traumatic events and PTSD prevalence using DSM-IV and DSM-5 criteria. *Journal of traumatic stress*, *26*(5), 537-547.

Kreft, I., De Leeuw, J. (1998). *Introducing Multilevel Modeling*. London: Sage Publications.

Lane, R. D., Ryan, L., Nadel, L., & Greenberg, L. (2015). Memory reconsolidation, emotional arousal, and the process of change in psychotherapy: New insights from brain science. *Behavioral and Brain Sciences*, *38*.

Lang, P. J. (1977). Imagery in therapy: An information processing analysis of fear. *Behavior Therapy*, *8*(5), 862-886.

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology*, *16*(6), 495-512.

Lensvelt-Mulders, G., van Der Hart, O., van Ochten, J. M., van Son, M. J., Steele, K., & Breeman, L. (2008). Relations among peritraumatic dissociation and posttraumatic stress: A meta-analysis. *Clinical Psychology Review*, *28*(7), 1138-1151.

Maas, C. J., & Hox, J. J. (2005). Sufficient sample sizes for multilevel modeling. *Methodology*, *1*(3), 86-92.

MacDonald, H. Z., Proctor, S. P., Heeren, T., & Vasterling, J. J. (2013). Associations of postdeployment PTSD symptoms with predeployment symptoms in Iraq-deployed Army soldiers. *Psychological Trauma: Theory, Research, Practice, and Policy*, *5*(5), 470–476.

Markowitz, J. C., Petkova, E., Neria, Y., Van Meter, P. E., Zhao, Y., Hembree, E., ... & Marshall, R. D. (2015). Is exposure necessary? A randomized clinical trial of interpersonal psychotherapy for PTSD. *American Journal of Psychiatry*, *172*(5), 430-440.

Marmar, C. R., Weiss, D. S., & Metzler, T. J. (1997). The peritraumatic dissociative experiences questionnaire. *Assessing psychological trauma and PTSD*, *2*, 144-168.

McMillen, J. C., North, C. S., & Smith, E. M. (2000). What parts of PTSD are normal: intrusion, avoidance, or arousal? Data from the Northridge, California, earthquake. *Journal of Traumatic Stress*, *13*(1), 57–75.

McNeish, D. M., & Stapleton, L. M. (2016). The effect of small sample size on two-level model estimates: A review and illustration. *Educational Psychology Review*, *28*(2), 295-314.

Nahleen, S., Nixon, R. D., & Takarangi, M. K. (2019). Current PTSD symptomatology distorts memory for past symptoms. *Psychiatry research*, *274*, 330-334.

Nanney, J. T., Constans, J. I., Kimbrell, T. A., Kramer, T. L., & Pyne, J. M. (2015). Differentiating between appraisal process and product in cognitive theories of posttraumatic stress. *Psychological Trauma: Theory, Research, Practice, and Policy*, *7*(4), 372.

Naragon-Gainey, K., Simpson, T. L., Moore, S. A., Varra, A. A., & Kaysen, D. L. (2012). The correspondence of daily and retrospective PTSD reports among female victims of sexual assault. *Psychological assessment*, *24*(4), 1041.

Norris, F. H., Tracy, M., & Galea, S. (2009). Looking for resilience: Understanding the longitudinal trajectories of responses to stress. *Social Science & Medicine*, *68*(12), 2190–2198.

North, C. S. (2004). Psychiatric Effects of Disasters and Terrorism: Empirical Basis from Study of the Oklahoma City Bombing.

North, C. S., Nixon, S. J., Shariat, S., Mallonee, S., McMillen, J. C., Spitznagel, E. L., & Smith, E. M. (1999). Psychiatric disorders among survivors of the Oklahoma City bombing. *JAMA*, 282(8), 755-762.

O'Kearney, R., & Perrott, K. (2006). Trauma narratives in posttraumatic stress disorder: A review. *Journal of traumatic stress*, *19*(1), 81-93.

Olatunji, B. O., Cisler, J. M., & Tolin, D. F. (2007). Quality of life in the anxiety disorders: a meta-analytic review. *Clinical psychology review*, *27*(5), 572-581.

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychological bulletin*, *129*(1), 52.

Resick, P. A., Monson, C. M., & Chard, K. M. (2016). *Cognitive processing therapy for PTSD: A comprehensive manual*. Guilford Publications.

Riggs, D. S., Dancu, C. V., Gershuny, B. S., Greenberg, D., & Foa, E. B. (1992). Anger and post-traumatic stress disorder in female crime victims. *Journal of Traumatic Stress*, 5(4), 613–625.

R Core Team (2014). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. http://www.R-project.org

Rubin, D. C. (2011). The coherence of memories for trauma: Evidence from posttraumatic stress disorder. *Consciousness and Cognition*, *20*(3), 857-865.

Rubin, D. C., Berntsen, D., & Bohni, M. K. (2008). A memory-based model of posttraumatic stress disorder: evaluating basic assumptions underlying the PTSD diagnosis. *Psychological review*, *115*(4), 985.

Rubin, D. C., Dennis, M. F., & Beckham, J. C. (2011). Autobiographical memory for stressful events: The role of autobiographical memory in posttraumatic stress disorder. *Consciousness and cognition*, *20*(3), 840-856.

Saunders, J. B., Aasland, O. G., Babor, T. F., de la Puente, J. R., and Grant, M. (1993). Development of the Alcohol Use Disorders Screening Test (AUDIT). WHO collaborative

project on early detection of persons with harmful alcohol consumption. II. *Addiction* 88:791-804.

Schell, T. L., Marshall, G. N., & Jaycox, L. H. (2004). All Symptoms Are Not Created Equal: The Prominent Role of Hyperarousal in the Natural Course of Posttraumatic Psychological Distress. *Journal of Abnormal Psychology*, *113*(2), 189–197.

Scherbaum, C. A., & Ferreter, J. M. (2009). Estimating statistical power and required sample sizes for organizational research using multilevel modeling. *Organizational Research Methods*, *12*(2), 347-367.

Shalev, A.Y. (1992). Posttraumatic stress disorder among injured survivors of a terrorist attack. *The Journal of Nervous and Mental Disease, 180,* 505-509.

Shalev, A. Y., Freedman, S., Peri, T., Brandes, D., Sahar, T., Orr, S. P., & Pitman, R. K. (1998). Prospective study of posttraumatic stress disorder and depression following trauma. *American Journal of Psychiatry*.

Singer, J.(1998). Using SAS PROC MIXED to fit multilevel models, hierarchical models, and individual growth models. *Journal of Education and Behavioral Statistics, 24 (4),* 323-355.

Smith, P. C., Schmidt, S. M., Allensworth-Davies, D., & Saitz, R. (2010). A single-question screening test for drug use in primary care. *Archives of internal medicine*, *170*(13), 1155-1160.

Smith, H. L., Summers, B. J., Dillon, K. H., & Cougle, J. R. (2016). Is worst-event trauma type related to PTSD symptom presentation and associated features?. *Journal of anxiety disorders*, *38*, 55-61.

Snijders, T., & Bosker, R. (1999). *Multilevel Analysis: an introduction to basic and advanced multilevel modeling.* London: Sage Publications.

Sullivan, C., Jones, R. T., Hauenstein, N., & White, B. (2017). Development of the Trauma-Related Anger Scale. *Assessment*.

Ullman, S. E., & Peter-Hagene, L. C. (2016). Longitudinal relationships of social reactions, PTSD, and revictimization in sexual assault survivors. *Journal of interpersonal violence*, *31*(6), 1074-1094.

Virginia Sexual and Domestic Violence Action Alliance (2017). 2017 Action Alliance Directory of Sexual & Domestic Violence Member Agencies. List available at www.vsdvalliance.org

Wagner, A. W., Zatzick, D. F., Ghesquiere, A., & Jurkovich, G. J. (2007). Behavioral activation as an early intervention for posttraumatic stress disorder and depression among physically injured trauma survivors. *Cognitive and Behavioral Practice*, *14*(4), 341-349.

Weathers, F.W., Blake, D.D., Schnurr, P.P., Kaloupek, D.G., Marx, B.P., & Keane, T.M. (2013). *The Life Events Checklist for DSM-5 (LEC-5).* Instrument available from the National Center for PTSD at www.ptsd.va.gov

Weathers, F.W., Litz, B.T., Keane, T.M., Palmieri, P.A., Marx, B.P., & Schnurr, P.P. (2013). *The PTSD Checklist for DSM-5 (PCL-5).* Scale available from the National Center for PTSD at www.ptsd.va.gov.

Weston, C. S. E. (2014). Posttraumatic stress disorder: a theoretical model of the hyperarousal subtype. *Frontiers in psychiatry*, 5.

Wortmann, J. H., Jordan, A. H., Weathers, F. W., Resick, P. A., Dondanville, K. A., Hall-Clark, B., ... & Mintz, J. (2016). Psychometric analysis of the PTSD Checklist-5 (PCL-5) among treatment-seeking military service members. *Psychological assessment*, *28*(11), 1392.

Zimet, G. D., Dahlem, N. W., Zimet, S. G., & Farley, G. K. (1988). The multidimensional scale of perceived social support. *Journal of personality assessment*, *52*(1), 30-41.

Table 1. Theories and the corresponding initial reactions and important clusters/symptoms

Theory	**Initial Cluster/Symptoms**	**Long-term Importance**
Emotional processing theory (Foa et al., 1989)	Re-experiencing and hyperarousal (inferred)	Negative cognitions and mood (symptom D2)
Dual Representation (Brewin, 2013)	Re-experiencing	Avoidance
Cognitive Model (Ehlers & Clark, 2000)	Re-experiencing and hyperarousal	Negative cognitions and mood (symptoms D2, D3)
Hyperarousal theories (Chemtob et al., 1988; Riggs et al., 1992; Weston, 2014)	Hyperarousal	Hyperarousal

Table 2. Sample Demographics

	Recent Sexual/Relationship Violence Sample (n = 136)		Full Sample (N=413)	
	n / M	*% / SD*	*n / M*	*% / SD*
Age	19.26	1.18	19.31	1.25
Race/Ethnicity				
Caucasian	109	80.14	316	76.51
African-American	8	5.88	24	5.81
Hispanic	1	0.74	7	1.69
Asian	11	8.09	36	8.72
Pacific Islander	0	0.00	2	0.48
Multiracial	5	3.68	18	4.36
Other	2	1.47	5	1.21
Did not provide race	0	0.00	5	1.21
Employed				
Yes	42	30.88	111	26.87
No	94	69.12	301	72.88
Criterion A Event				
Yes	136	100.00	294	71.19
No	0	0.00	119	28.81
Sexual/Relationship Violence				
Yes	136	100.00	177	42.86
No	0	0.00	236	57.14
Help Seeking				
Yes	38	27.94	80	19.37
No	98	72.06	333	80.63
Time Since Traumatic Event (Weeks)	44.92	32.92	183.64	188.01
PTSD Severity (PCL5)	24.85	18.62	20.49	17.38
Peritraumatic Dissociation (PDEQ-SR)	11.59	7.59	9.98	7.37
Peritraumatic Emotions (PDI)	23.16	10.52	21.02	10.91
Social Support (MSPSS)	43.93	12.78	46.77	11.82
Trauma Coping Self-Efficacy (CSE-T)	33.86	11.18	37.14	11.77
Alcohol Frequency (from AUDIT)	1.76	1.04	1.68	1.08
Alcohol Use (from AUDIT)	0.75	1.10	0.66	1.14
Drug Use (from DUDIT)	0.61	1.11	0.38	0.87

Table 3. Reported Symptom Counts and Orderings in the First Week after Trauma Exposure

	Recent Sexual/Relationship Violence Sample (n = 136)		Full Sample (N=413)	
	Frequency / Presence	*% First / Average Order*	Frequency / Presence	*% First / Average Order*
Overall Symptom Count	8.58 (SD = 4.93)	-	8.97 (SD = 5.01)	-
Cluster B	2.35 (SD = 1.5)	43.16% (n = 36)	2.73 (SD = 1.49)	27.36% (n = 113)
Intrusive thoughts	*86 (63.24%)*	*3.56 (SD = 2.60)*	*279 (67.55%)*	*4.20 (SD = 2.89)*
Nightmares	*43 (31.62%)*	*5.86 (SD = 3.55)*	*185 (44.79%)*	*6.09 (SD = 3.31)*
Flashbacks	*37 (27.20%)*	*4.76 (SD = 2.54)*	*153 (37.05%)*	*5.78 (SD = 3.43)*
Emotional Reactions to Cues	*91 (66.91%)*	*4.70 (SD = 2.69)*	*327 (79.18%)*	*4.70 (SD = 2.84)*
Physical Reactions to Cues	*63 (46.32%)*	*5.22 (SD = 2.56)*	*183 (44.31%)*	*5.68 (SD = 3.58)*
Cluster C	1.33 (SD = 0.81)	11.76% (n = 16)	1.29 (SD = 0.81)	7.99% (n = 33)
Cognitive avoidance	*93 (68.38%)*	*5.30 (SD = 3.43)*	*293 (70.94%)*	*6.13 (SD = 3.44)*
Behavioral avoidance	*88 (64.71%)*	*5.72 (SD = 3.20)*	*240 (58.11%)*	*6.85 (SD = 3.34)*
Cluster D	2.95 (SD = 2.03)	33.82% (n = 46)	2.85 (SD = 2.11)	26.88% (n = 111)
Traumatic amnesia	*49 (36.03%)*	*5.41 (SD = 3.97)*	*112 (27.12%)*	*5.95 (SD = 4.25)*
Persistent negative beliefs	*66 (48.53%)*	*5.27 (SD = 3.87)*	*170 (41.16%)*	*5.66 (SD = 3.99)*
Persistent self-blame	*86 (63.24%)*	*4.83 (SD = 3.50)*	*194 (46.97%)*	*5.20 (SD = 3.81)*
Persistent negative emotions	*81 (59.56%)*	*5.30 (SD = 3.66)*	*260 (62.95%)*	*5.53 (SD = 3.84)*
Loss of interest	*33 (24.26%)*	*9.21 (SD = 3.80)*	*124 (30.02%)*	*9.70 (SD = 3.69)*
Feeling distant/detached	*52 (38.24%)*	*7.65 (SD = 3.90)*	*160 (38.74%)*	*8.11 (SD = 3.93)*
Low positive emotions	*34 (25.00%)*	*9.75 (SD = 4.69)*	*155 (37.53%)*	*8.39 (SD = 4.34)*
Cluster E	1.95 (SD = 1.63)	5.15% (n = 7)	2.11 (SD = 1.61)	10.41% (n = 43)
Anger/Irritable behavior	*32 (23.53%)*	*9.78 (SD = 3.95)*	*106 (25.67%)*	*9.09 (SD = 3.96)*
Risky behavior	*26 (19.12%)*	*9.92 (SD = 5.09)*	*49 (11.86%)*	*11.14 (SD = 4.64)*
Hypervigilance	*55 (40.44%)*	*7.20 (SD = 4.30)*	*166 (40.19%)*	*7.80 (SD = 4.54)*
Exaggerated Startle	*54 (39.71%)*	*7.09 (SD = 4.11)*	*144 (34.87%)*	*7.83 (SD = 4.61)*
Concentration Problems	*48 (35.29%)*	*8.67 (SD = 4.22)*	*197 (47.70%)*	*8.21 (SD = 4.51)*
Sleep Problems	*50 (36.76%)*	*7.21 (SD = 4.25)*	*209 (50.61%)*	*7.70 (SD = 4.76)*

Table 4. Overall Symptom Count Model Predicting PTSD Severity

	Step 1: Unconditional Growth Model			Step 2: Symptoms			Step 3: Adding Covariates		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	23.77	14.98	< .0001	2.63	1.05	.29	4.90	1.05	.30
Symptom Count				2.42	9.70	< .0001	1.69	7.87	< .0001
PDEQ-SR							2.02	6.87	< .0001
Alcohol Use							1.71	2.15	.03
CSE-T							-0.10	-0.89	.37
Weeks since trauma							-0.07	-2.60	.01
PDEQ-SR*CSE-T							-0.03	-5.03	< .0001
Time	-2.49	-4.66	< .0001	0.25	0.23	.82	0.55	0.61	.54
Symptom Count				-0.31	–2.84	.005	-0.28	-3.09	.002
Random Effects	Variance		*SD*	Variance		*SD*	Variance		*SD*
Intercept		290.94	17.06		151.20	12.30		82.11	9.06
Time		15.30	3.91		13.28	3.64		6.99	2.64
Level-1 error		55.33	7.44		55.57	7.45		51.25	7.16
	AIC = 3105.97; BIC = 3129.76 R^2 = .07; R^2 error = .35			AIC = 3042.24; BIC = 3073.93 R^2 = 0.39; R^2 Intercept = .48; R^2 Time = .13; R^2 error = -.004			AIC = 2912.59; BIC = 2963.78 R^2 = 0.61; R^2 Intercept = 0.72; R^2 Time = .54; R^2 error = .07		

Table 5. Cluster Count Model Predicting PTSD Severity

	Step 1: Unconditional Growth Model			Step 2: Symptoms			Step 3: Adding Covariates		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	23.77	14.98	< .0001	6.41	2.89	.0042	7.15	1.53	.13
Cluster D Count				3.01	3.94	.0001	2.18	3.59	.0005
Cluster E Count				4.24	4.64	< .0001	2.74	3.86	.0002
Alcohol Use							1.57	1.93	.06
PDEQ-SR							2.04	6.88	< .0001
CSE-T							-0.09	-.82	.41
Weeks since trauma							-0.05	-1.97	.05
PDEQ-SR*CSE-T							-0.04	-5.15	< .0001
Time	-2.49	-4.66	< .0001	-0.12	-0.13	.90	0.22	0.28	.78
Cluster D Count				-0.78	-2.91	.004	-0.71	-3.20	.002
Random Effects	Variance		*SD*	Variance		*SD*	Variance		*SD*
Intercept		290.94	17.06		153.70	12.40		86.70	9.31
Time		15.30	3.91		13.13	3.62		6.47	2.54
Level-1 error		55.33	7.44		55.43	7.44		51.14	7.15
	AIC = 3105.97; BIC = 3129.76 R^2 = .07; R^2 error = .35			AIC = 3083.20; BIC = 3073.83 R^2 = .40; R^2 Intercept = .47; R^2 Time = .14; R^2 error = .09			AIC = 2911.49; BIC = 2966.58 R^2 = .60; R^2 Intercept = .70; R^2 Time = .58; R^2 error = .08		

Table 6. Symptom Presence Model Predicting PTSD Severity

	Step 1: Unconditional Growth Model			Step 2: Symptoms			Step 3: Adding Covariates		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	23.77	14.98	< .0001	8.27	3.31	.001	6.63	1.46	.14
Emotional Cues				9.02	3.39	.0009	6.57	3.53	.0006
Traumatic Amnesia				-3.37	-1.29	.20	-4.74	-2.57	.01
Negative Beliefs				7.41	2.90	.004	4.67	2.61	.01
Loss of Interest				11.64	3.29	.001	8.64	3.29	.001
Risky Behavior				8.52	2.36	.02	6.30	2.48	.01
Exaggerated Startle				6.06	2.40	.02	5.55	3.11	.002
Alcohol Use							2.13	2.67	.009
PDEQ-SR							2.13	7.44	<. 0001
CSE-T							-0.07	-0.71	.48
Weeks since trauma							-0.06	-2.40	.02
Help Seeking							3.61	1.85	.07
PDEQ-SR*CSE-T							-0.04	-5.60	< .0001
Time	-2.49	-4.66	< .0001	-1.61	-2.75	.007	-1.14	-2.31	.02
Loss of Interest				-3.77	-2.97	.003	-3.47	-3.41	.0008
Random Effects	Variance		*SD*	Variance		*SD*	Variance		*SD*
Intercept		290.94	17.06		162.18	12.74		78.15	8.84
Time		15.30	3.91		13.07	3.62		6.35	2.52
Level-1 error		55.33	7.44		54.80	7.40		50.46	7.10
	AIC = 3105.97; BIC = 3129.76 R^2 = .07; R^2 error = .35			AIC = 3009.89; BIC = 3069.03 R^2 = .36; R^2 Intercept = .44; R^2 Time = .15; R^2 error = .01			AIC = 2874.78; BIC = 2949.29 R^2 = .63; R^2 Intercept = .73; R^2 Time = .58; R^2 error = .09		

Table 7. Cluster Order and Overall Symptom Count Model Predicting PTSD Severity

	Step 1: Unconditional Growth Model			Step 2: Symptoms			Step 3: Adding Covariates		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	23.77	14.98	< .0001	2.21	0.77	.44	6.06	1.30	.19
Cluster D Order				1.39	0.23	.82	-0.49	-0.10	.92
Symptom Count				2.49	7.97	< .0001	1.49	5.50	< .0001
D Order*Count				-0.19	-0.33	.74	0.30	0.66	.51
PDEQ-SR							2.10	7.18	< .0001
CSE-T							-0.09	-0.87	.38
Weeks since trauma							-0.05	-2.17	.03
PDEQ-SR*CSE-T							-0.04	-5.32	< .0001
Time	-2.49	-4.66	< .0001	1.49	1.21	.23	1.67	1.63	.10
Cluster D Order				-3.39	-1.38	.17	-2.99	-1.49	.14
Symptom Count				-0.55	-3.98	.0001	-0.49	-4.26	< .0001
D Order*Count				0.55	2.34	.02	0.45	2.34	.02
Random Effects	Variance		*SD*	Variance		*SD*	Variance		*SD*
Intercept		290.94	17.06		155.21	12.46		87.53	9.36
Time		15.30	3.91		12.59	3.55		6.37	2.52
Level-1 error		55.33	7.44		55.42	7.44		51.41	7.17
	AIC = 3105.97; BIC = 3129.76 R^2 = .07; R^2 error = .35			AIC = 3036.98; BIC = 3084.39 R^2 = .36; R^2 Intercept = .45; R^2 Time = .16; R^2 error = .00			AIC = 2905.32; BIC = 2968.19 R^2 = .60; R^2 Intercept = .70; R^2 Time = .58; R^2 error = .07		

Table 8. Cluster Order and Cluster Count Model Predicting PTSD Severity

	Step 1: Unconditional Growth Model			Step 2: Symptoms			Step 3: Adding Covariates		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	23.77	14.98	< .0001	5.59	1.94	.05	2.40	0.49	.63
Cluster D Order				-0.90	-0.15	.88	3.69	0.76	.45
Cluster C Count				1.19	0.57	.57	2.31	1.37	.17
Cluster D Count				2.93	3.48	.0007	1.69	2.51	.01
Cluster E Count				4.19	4.55	< .0001	2.66	3.79	.0002
D Order*C Count				-0.27	-0.08	.94	-1.34	-0.47	.64
Alcohol Use							1.74	2.14	.03
PDEQ-SR							2.19	7.33	< .0001
CSE-T							-0.03	-0.30	.77
Weeks since trauma							-0.05	-1.82	.07
PDEQ-SR*CSE-T							-0.04	-5.59	< .0001
Time	-2.49	-4.66	< .0001	1.21	1.01	.32	1.10	1.11	.27
Cluster D Order				-2.52	-1.02	.31	-2.12	-1.06	.29
Cluster C Count				-1.52	-1.79	.08	-1.06	-1.53	.13
Cluster D Count				-0.85	-2.70	.007	-0.80	-3.12	.002
D Order*C Count				3.24	2.25	.03	2.63	2.24	.03
Random Effects	Variance		*SD*	Variance		*SD*	Variance		*SD*
Intercept		290.94	17.06		158.59	12.59		88.29	9.39
Time		15.30	3.91		12.33	3.51		5.96	2.44
Level-1 error		55.33	7.44		55.16	7.43		50.91	7.14
	AIC = 3105.97; BIC = 3129.76 R^2 = .07; R^2 error = .35			AIC = 3023.23; BIC = 3082.37 R^2 = .37; R^2 Intercept = .45; R^2 Time = .19; R^2 error = .00			AIC = 2894.62; BIC = 2973.00 R^2 = .60; R^2 Intercept = .70; R^2 Time = .61; R^2 error = .07		

Table 9. Cluster Order and Symptom Presence Model Predicting PTSD Severity

	Step 1: Unconditional Growth Model			Step 2: Symptoms			Step 3: Adding Covariates		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	23.77	14.98	< .0001	8.00	2.95	.004	9.41	2.13	.03
Cluster D Order				-3.31	-0.83	.41	-2.18	-0.73	.47
Emotional Cues				4.84	1.70	.09	4.95	2.52	.01
Physical Cues				-0.29	-0.09	.93	-0.34	-0.15	.88
Traumatic Amnesia				-3.33	-1.27	.21	-5.40	-2.95	.004
Loss of Interest				3.62	0.78	.43	3.99	1.14	.26
Negative Emotions				6.03	2.14	.03	3.09	1.40	.17
Low Positive Emotions				10.24	2.03	.04	9.71	2.47	.01
Risky Behavior				6.13	1.70	.09	6.76	2.62	.01
Concentration Problems				8.30	2.57	.01	4.60	1.81	.07
Sleep Problems				0.58	0.16	.87	-4.84	-1.68	.1
D Order*Phys Cues				10.94	1.96	.05	7.75	1.98	.05
D Order*Low Positive				-15.00	-2.17	.03	-10.57	-2.01	.05
D Order*Sleep Problems				9.37	1.56	.12	15.02	3.13	.002
PDEQ-SR							2.26	7.83	< .0001
CSE-T							-0.12	-1.20	.23
Weeks since trauma							-0.02	-0.94	.35
PDEQ-SR*CSE-T							-0.04	-5.83	< .0001
Time	-2.49	-4.66	< .0001	-1.10	-1.24	.22	-0.41	-0.61	.55
Cluster D Order				2.68	2.06	.04	2.10	2.13	.03
Negative Emotions				-1.93	-1.85	.06	-1.92	-2.46	.01
Loss of Interest				-2.97	-1.76	.08	-3.31	-2.61	.01
Low Positive Emotions				-3.23	-1.68	.09	-2.03	-1.42	.16
Concentration Problems				-2.71	-1.98	.05	-3.23	-3.14	.002
Sleep Problems				4.90	3.11	.002	4.56	3.91	.0001
D Order*Low Positive				8.83	3.11	.002	9.44	4.56	< .0001
D Order*Sleep Problems				-9.81	-3.76	.0002	-9.21	-4.83	< .0001
Random Effects	Variance		*SD*	Variance		*SD*	Variance		*SD*
Intercept		290.94	17.06		151.65	12.31		74.67	8.64
Time		15.30	3.91		9.22	3.04		1.52	1.24
Level-1 error		55.33	7.44		55.03	7.42		51.62	7.18
	AIC = 3105.97; BIC = 3129.76 R^2 = .07; R^2 error = .35			AIC = 2960.12; BIC = 3073.37 R^2 = .40; R^2 Intercept = .48; R^2 Time = .40; R^2 error = .01			AIC = 2815.78; BIC = 2943.93 R^2 = .65; R^2 Intercept = .74; R^2 Time = .90; R^2 error = .07		

Table 10. Sexual/Relationship Violence HLM Final Model Fit Comparison

Model Name	**AIC**	**BIC**	**Intercept R^2**	**Time R^2**	**Overall R^2**	**Residual R^2**
Overall Symptom Count	2912.59	2963.78	.72	.54	.61	.07
Cluster Count	2911.49	2966.58	.70	.58	.60	.08
Symptom Presence	2874.78	2949.29	.73	.58	.63	.09
Cluster Order Symptom Count	2905.32	2968.19	.70	.58	.60	.07
Cluster Order Cluster Count	2894.62	2973.00	.70	.61	.60	.07
Cluster Order Symptom Presence	2815.78	2943.93	.74	.90	.65	.07

Table 11. Full Sample Overall Symptom Count Model Predicting PTSD Severity

	Unconditional Growth Model			Final Model		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	20.25	23.90	< .0001	4.91	1.48	.14
Symptom Count				1.01	6,33	< .0001
PDEQ-SR				1.54	7.52	< .0001
PDI				0.28	4.01	.0001
Drug Use				1.44	2.40	.02
CSE-T				-0/11	-1.55	.12
Weeks since trauma				-0.004	-1.42	.16
PDEQ-SR*CSE-T				-0.03	-6.74	< .0001
Time	-2.39	-6.72	< .0001	-0.31	-0.42	.67
Symptom Count				-0.19	-2.90	.004
Random Effects	Variance		*SD*	Variance		*SD*
Intercept	236.66		15.38	82.29		9.07
Time	9.06		3.01	6.88		2.62
Level-1 error	58.62		7.66	50.11		7.08
	AIC = 6801.54; BIC = 6830.01 R^2 = .03; R^2 error = .28			AIC = 6118.01; BIC = 6183.70 R^2 = 0.54; R^2 Intercept = 0.65; R^2 Time = .24; R^2 error = .15		

Table 12. Full Sample Cluster Count Model Predicting PTSD Severity

	Unconditional Growth Model			Final Model		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	20.25	23.90	< .0001	7.23	2.26	.02
Cluster D Count				1.41	2.39	.02
Cluster E Count				1.63	3.42	.0007
PDEQ-SR				1.45	7.10	< .0001
PDI				0.30	4.29	< .0001
Drug Use				1.43	2.39	.02
CSE-T				-0.13	-1.87	.06
Weeks since trauma				-0.004	-1.64	.10
PDEQ-SR*CSE-T				-0.03	-6.35	< .0001
Time	-2.39	-6.72	< .0001	-0.78	-1.41	.16
Cluster E Count				-0.61	-3.09	.002
Random Effects	Variance		*SD*	Variance		*SD*
Intercept	236.66		15.38	79.92		8.94
Time	9.06		3.01	6.34		2.52
Level-1 error	58.62		7.66	50.26		7.09
	AIC = 6801.54; BIC = 6830.01 R^2 = .03; R^2 error = .28			AIC = 6107.90; BIC = 6178.26 R^2 = 0.55; R^2 Intercept = 0.66; R^2 Time = .30; R^2 error = .14		

Table 13. Full Sample Symptom Presence Model Predicting PTSD Severity

	Unconditional Growth Model			Final Model		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	20.25	23.90	< .0001	8.50	2.64	.009
Self-blame				2.53	2.29	.02
Anger				3.88	2.64	.009
Risky Behavior				4.41	2.52	.01
Sleep Problems				4.74	3.81	.0002
PDEQ-SR				1.48	7.18	< .0001
PDI				0.37	5.33	< .0001
Drug Use				1.20	1.98	.05
CSE-T				-0.14	-2.06	.04
Weeks since trauma				-0.005	-1.63	.10
PDEQ-SR*CSE-T				-0.03	-6.23	< .0001
Time	-2.39	-6.72	< .0001	-0.65	-1.41	.16
Anger				-1.83	-2.65	.008
Sleep Problems				-1.83	-3.03	.003
Random Effects	Variance		*SD*	Variance		*SD*
Intercept	236.66		15.38	81.59		9.03
Time	9.06		3.01	5.74		2.40
Level-1 error	58.62		7.66	50.51		7.11
	AIC = 6801.54; BIC = 6830.01 R^2 = .03; R^2 error = .28			AIC = 6102.13; BIC = 6186.50 R^2 = 0.55; R^2 Intercept = 0.66; R^2 Time = .37; R^2 error = .14		

Table 14. Full Sample Cluster Order Symptom Presence Model Predicting PTSD Severity

	Unconditional Growth Model			Final Model		
Fixed Effects	*b*	*t*	*p- value*	*b*	*t*	*p- value*
Intercept	20.25	23.90	< .0001	13.04	3.06	.002
Cluster D Order				-1.27	-0.47	.64
Cluster E Order				2.85	1.11	.27
Traumatic Amnesia				5.29	2.94	.004
Negative Beliefs				1.61	0.85	.39
Self-blame				0.49	0.34	.74
Distant/Detached				1.74	1.12	.26
Loss of Interest				2.98	2.00	.05
Risky Behavior				3.88	2.03	.04
Exaggerated Startle				-1.35	-0.81	.42
Concentration Problems				1.33	0.86	.39
Sleep Problems				0.61	0.35	.73
D Order*Memory				-8.39	-3.10	.002
D Order*Beliefs				-1.17	-0.40	.69
D Order*Startle				6.98	2.72	.007
D Order*Sleep				5.61	2.20	.03
E Order*Self-blame				15.40	3.88	.0001
E Order*Distant				-9.20	-2.46	.01
PDEQ-SR				1.01	4.08	.0001
PDI				0.36	4.33	< .0001
CSE-T				-0.22	-2.50	.01
Weeks since trauma				-0.006	-1.61	.11
PDEQ-SR*CSE-T				-0.02	-3.37	.0008
Time	-2.39	-6.72	< .0001	-1.05	-1.49	.14
Cluster D Order				-0.96	-0.94	.35
Negative Beliefs				-0.81	-0.93	.35
Concentration Problems				-1.86	-2.74	.007
D Order*Beliefs				3.31	2.40	.02
Random Effects	Variance		*SD*	Variance		*SD*
Intercept	236.66		15.38	87.23		9.34
Time	9.06		3.01	6.24		2.50
Level-1 error	58.62		7.66	47.20		6.87
	AIC = 6801.54; BIC = 6830.01 R^2 = .03; R^2 error = .28			AIC = 4760.23; BIC = 4901.72 R^2 = 0.54; R^2 Intercept = 0.63; R^2 Time = .31; R^2 error = .19		

Table 15 Full Sample HLM Final Model Fit Comparison

Model Name	**AIC**	**BIC**	**Intercept R^2**	**Time R^2**	**Overall R^2**	**Residual R^2**
Overall Symptom Count	6118.01	6183.70	.65	.24	.54	.15
Cluster Count	6107.90	6178.26	.66	.30	.55	.14
Symptom Presence	6102.13	6186.50	.66	.37	.55	.14
Cluster Order Symptom Presence	4760.23	4901.72	.63	.31	.54	.19

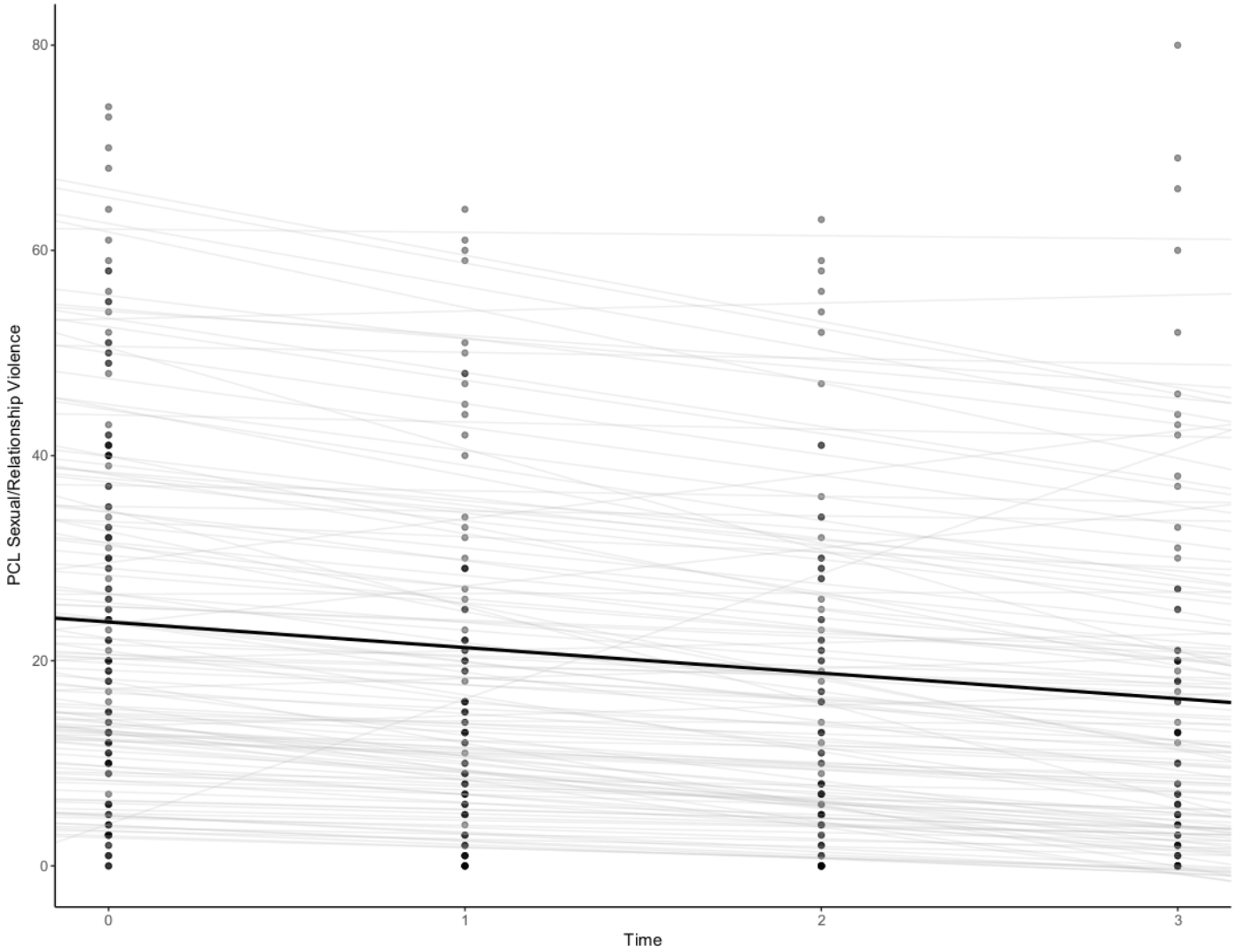

Figure 1. Unconditional Growth Model of PTSD Severity in Sexual/Relationship Violence Survivors.

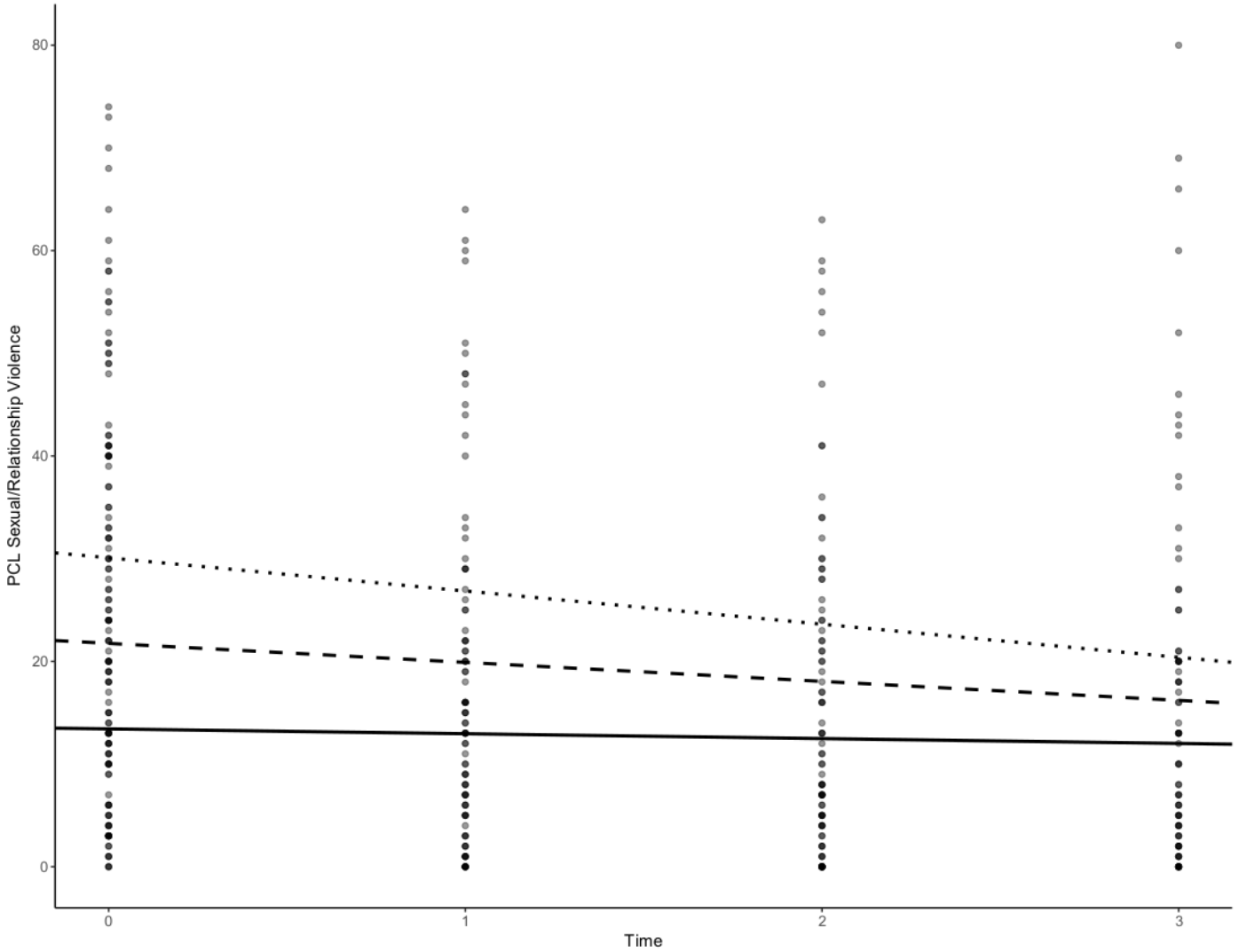

Figure 2. Symptom Count Effects on PTSD Severity in Sexual/Relationship Violence Survivors. Dotted line represents 1SD above the mean, dashed line represents mean, and solid line represents 1SD below the mean.

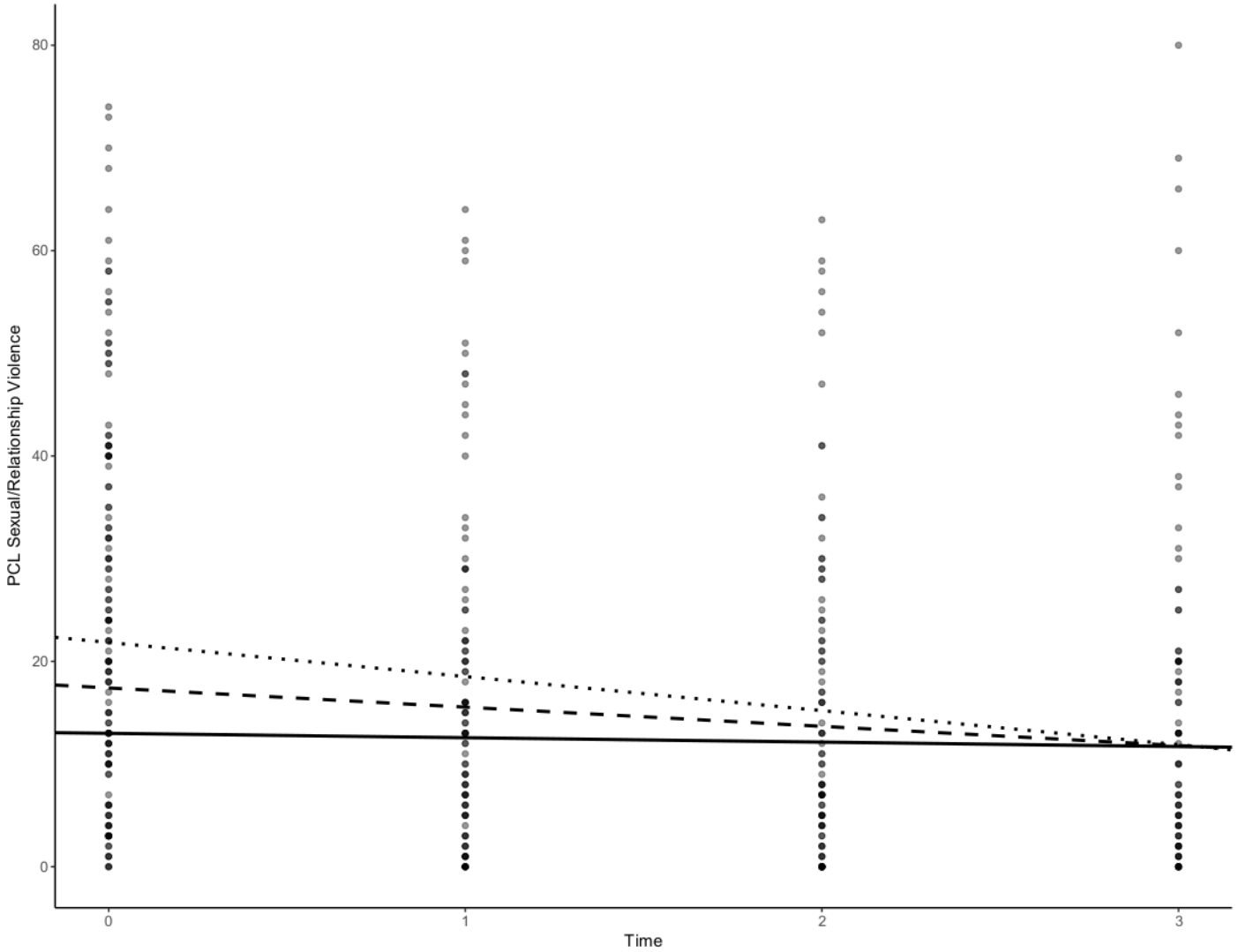

Figure 3. Cluster D Count Effects on PTSD Severity in Sexual/Relationship Violence Survivors. Dotted line represents 1SD above the mean, dashed line represents mean, and solid line represents 1SD below the mean.

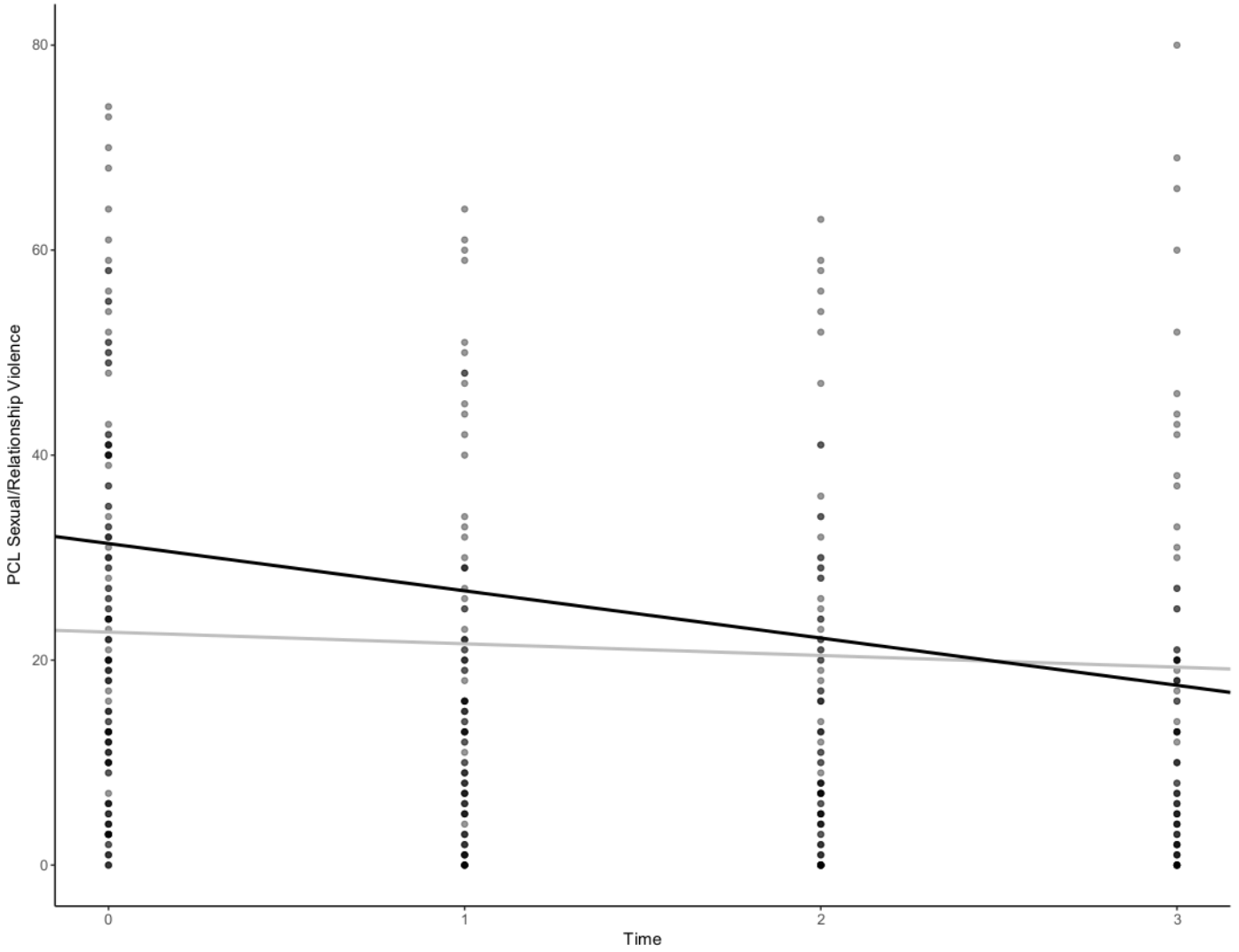

Figure 4. Loss of Interest Effects on PTSD Severity in Sexual/Relationship Violence Survivors. Solid black line represents reporting Loss of Interest in first week after trauma, gray line represents not reporting Loss of Interest.

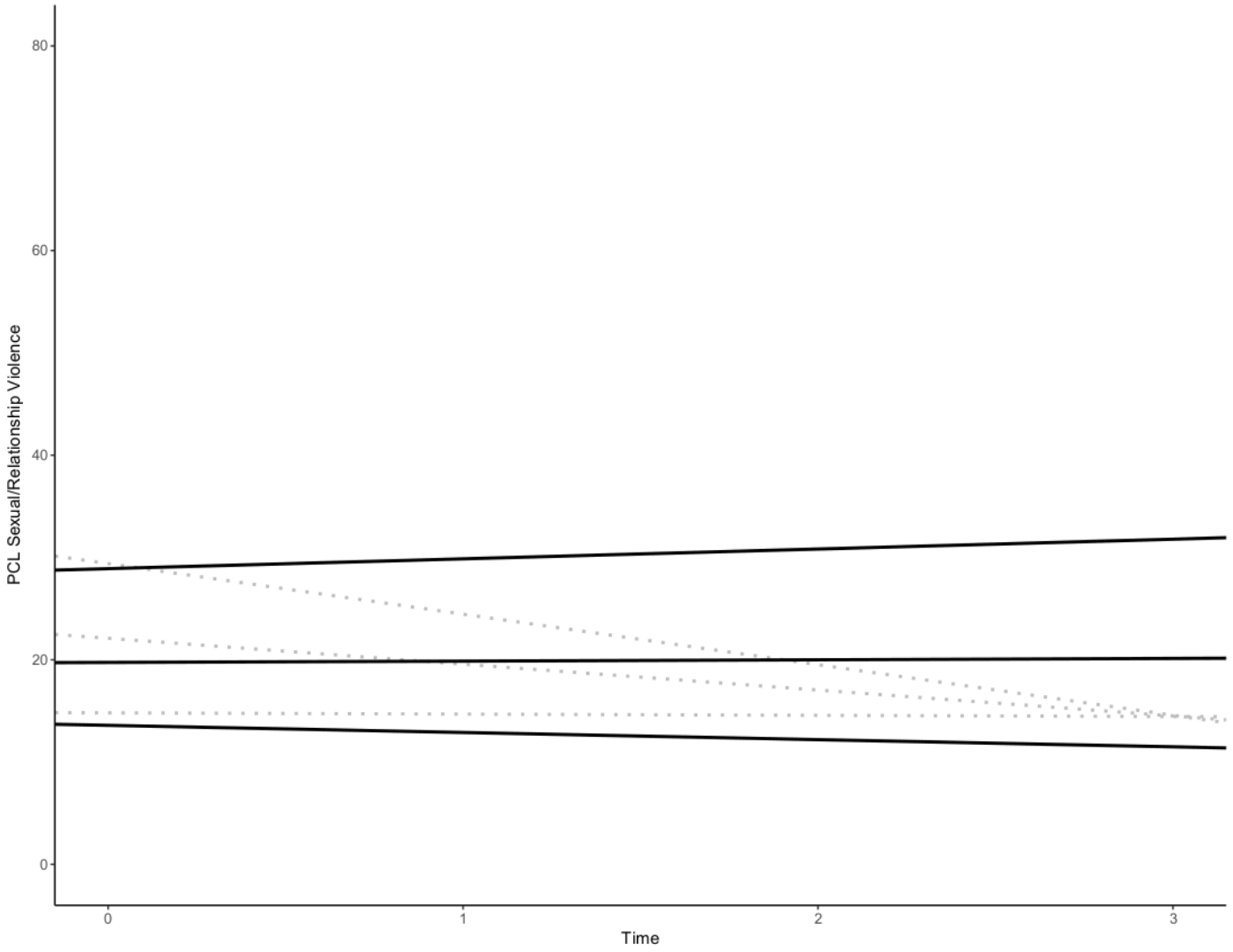

Figure 5. Interaction of Symptom Count and Cluster D ordering on PTSD Severity in Sexual/Relationship Violence Survivors. Solid line represents differing levels of Symptom Count and earlier Cluster D symptoms. Dotted gray lines represent individuals who did not report Cluster D symptoms as their initial symptom reaction.

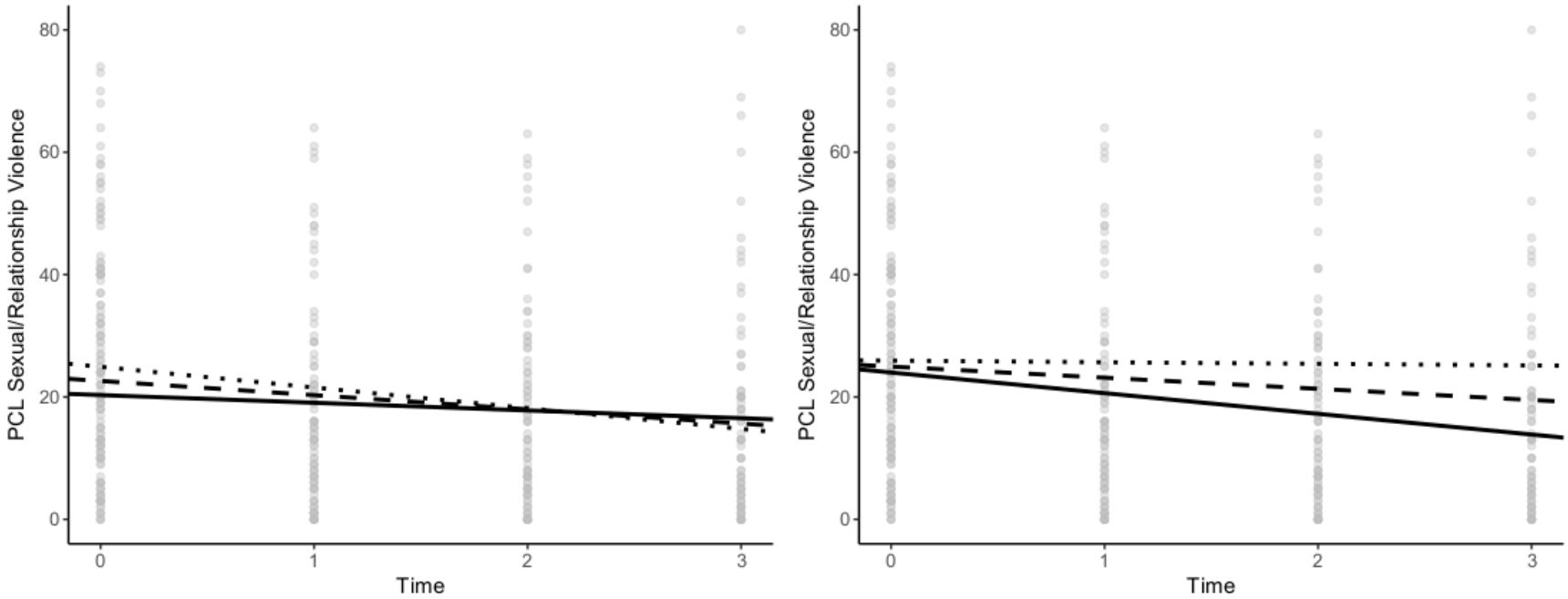

Figure 6. Interaction of Cluster C Count and Cluster D ordering on PTSD Severity in Sexual/Relationship Violence Survivors. The left graph represents individuals who did not report Cluster D symptoms as their first symptom. The right represents individuals who did report Cluster D symptoms first. Dotted line represents 1SD above the mean, dashed line represents mean, and solid line represents 1SD below the mean.

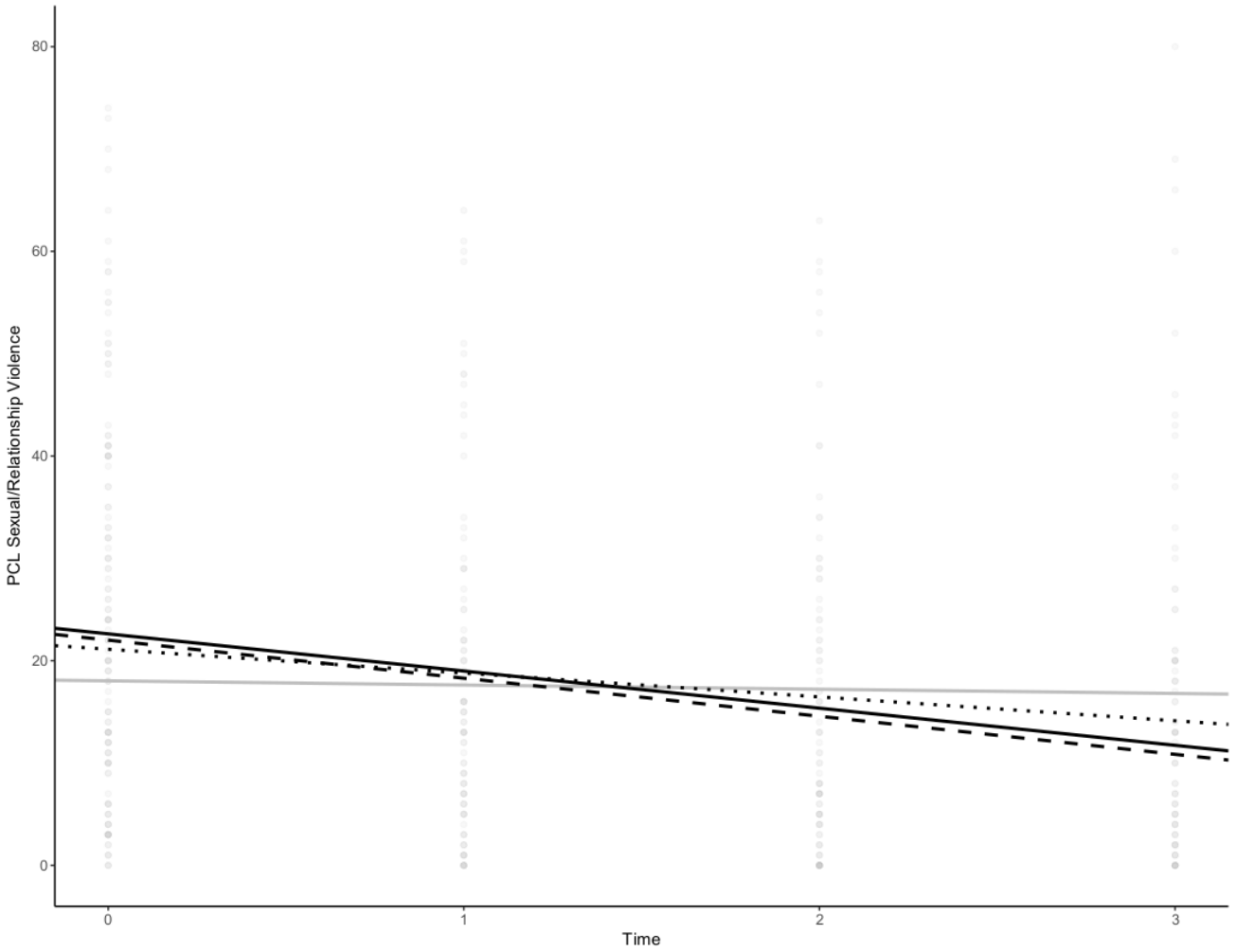

Figure 7. Impact of Concentration Problems, Loss of Interest, and Persistent Negative Emotions on PTSD Severity in Sexual/Relationship Violence Survivors. Solid gray line represents the intercept and slope when these symptoms are not present. Solid black line represents Concentration Problems, dashed black Loss of Interest, and dotted black Persistent Negative Emotions.

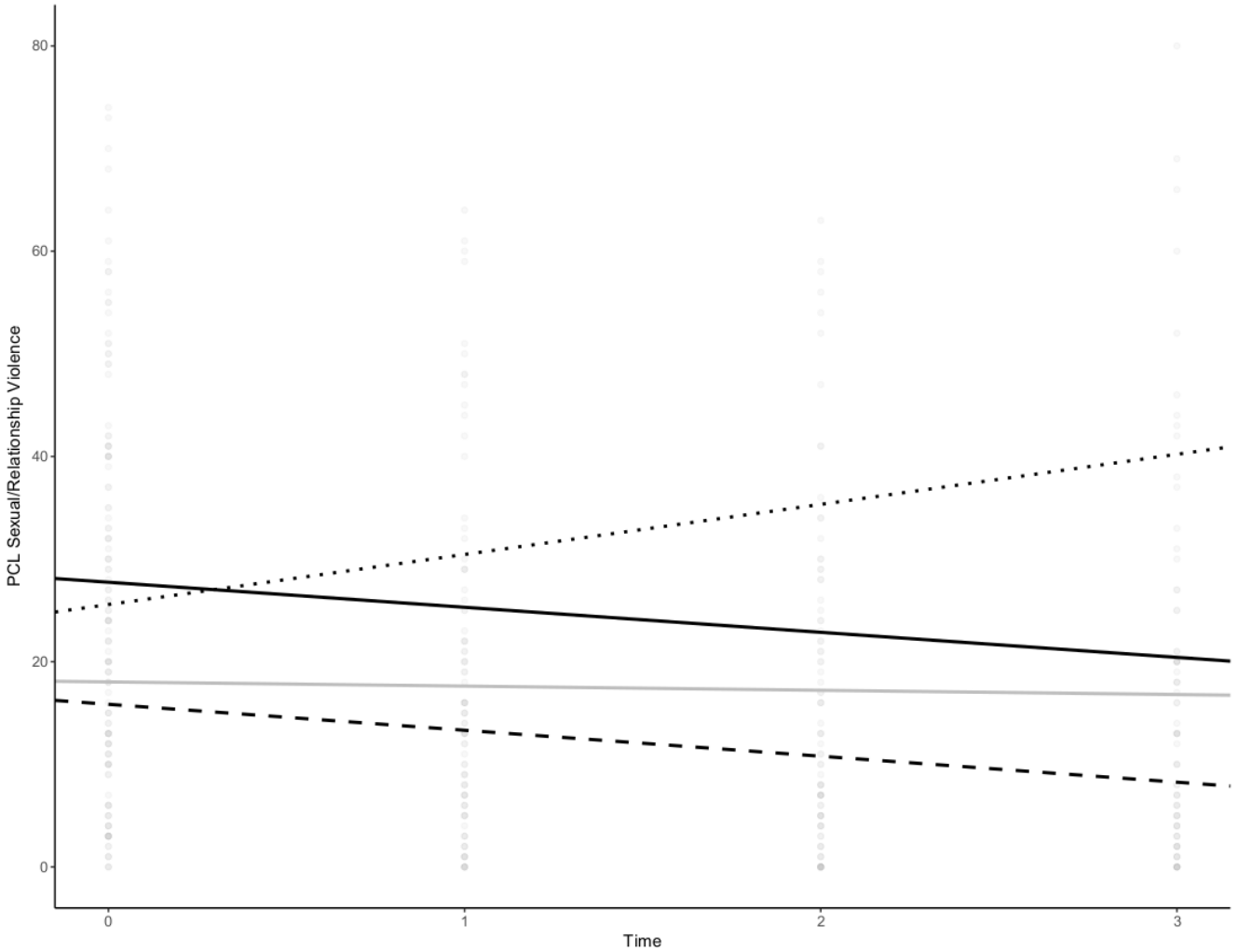

Figure 8. Interaction of Cluster D Order and Low Positive Emotions on PTSD Severity in Sexual/Relationship Violence Survivors. Solid gray line represents the intercept and slope when Low Positive Emotions are not present and Cluster D symptoms are not reported as the initial symptoms. Solid black line represents when Low Positive Emotions only are present, dashed black Cluster D Order only, and dotted black is when both are present.

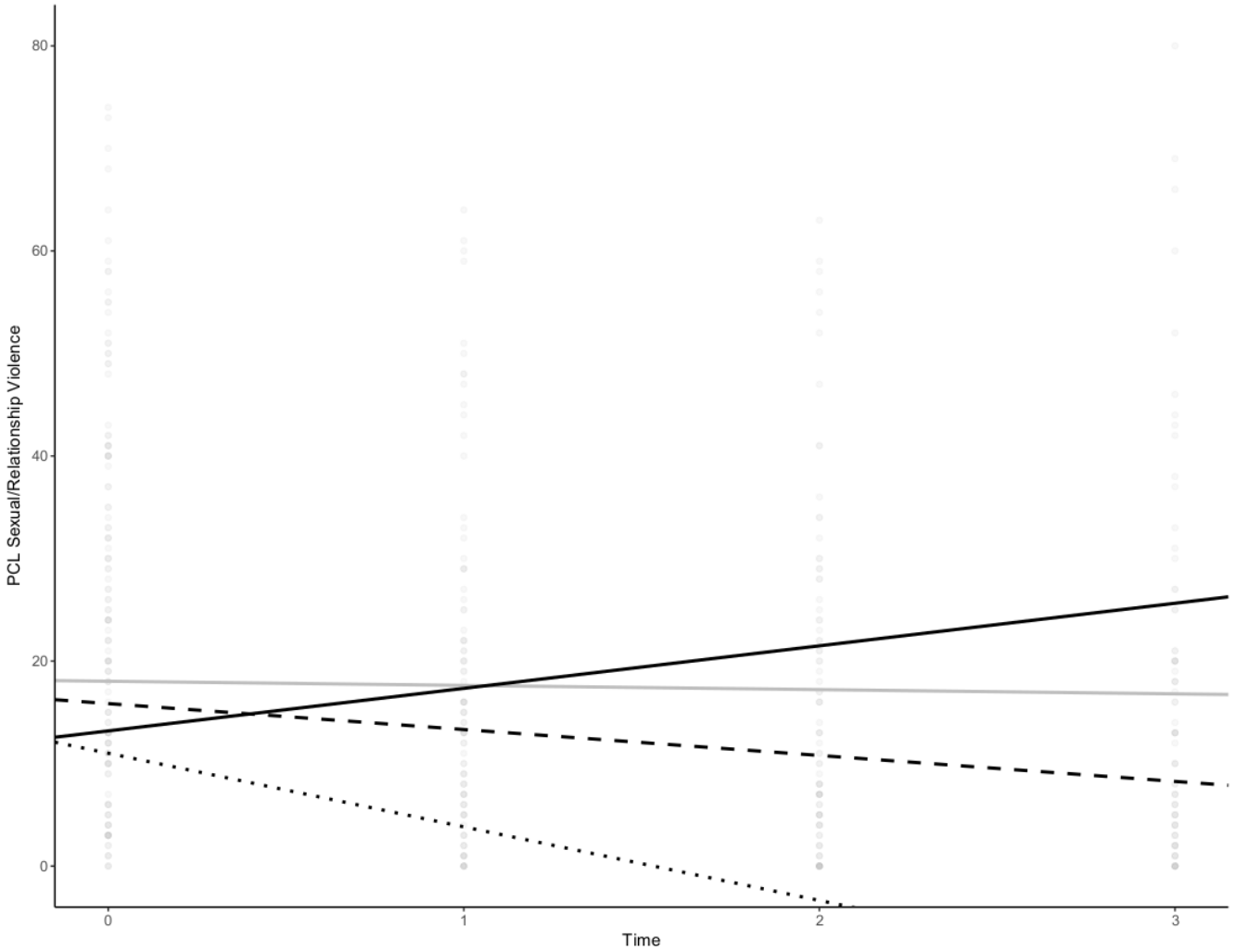

Figure 9. Interaction of Cluster D Order and Sleep Problems on PTSD Severity in Sexual/Relationship Violence Survivors. Solid gray line represents the intercept and slope when Sleep Problems are not present and Cluster D symptoms are not reported as the initial symptoms. Solid black line represents when Sleep Problems only are present, dashed black Cluster D Order only, and dotted black is when both are present.

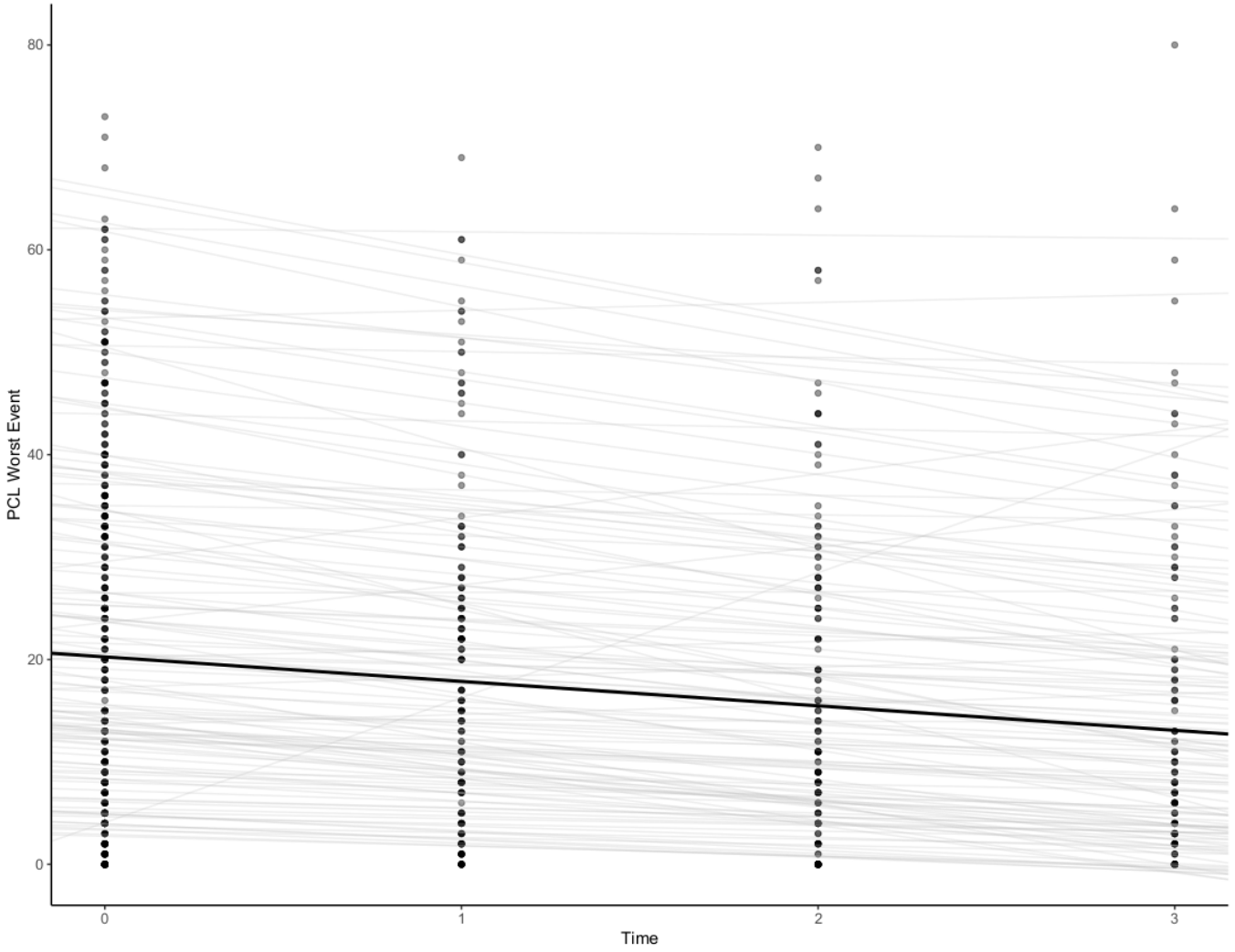

Figure 10. Unconditional Growth Model of PTSD Severity in the Full Sample.

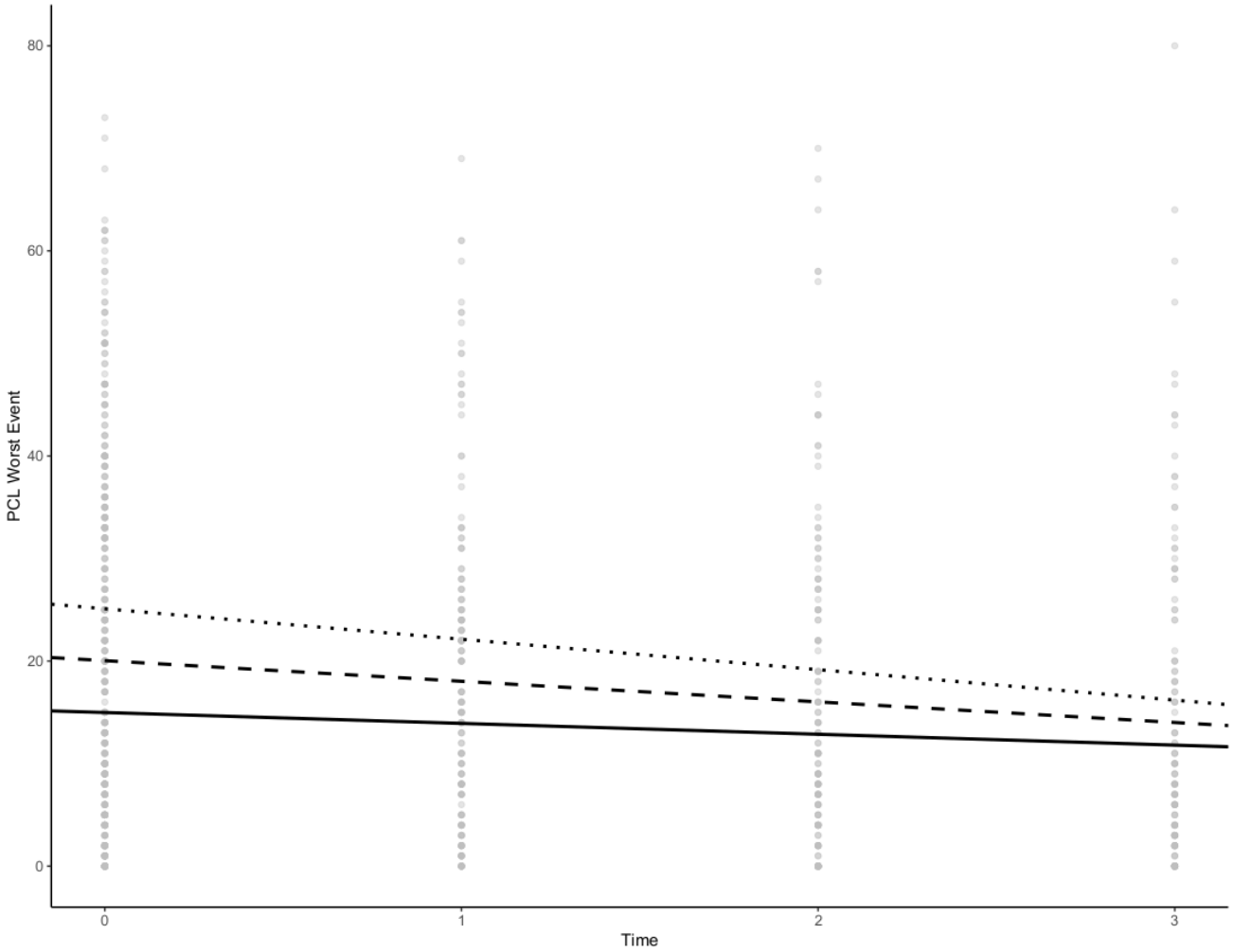

Figure 11. Symptom Count Effects on PTSD Severity in the Full Sample. Dotted line represents 1SD above the mean, dashed line represents mean, and solid line represents 1SD below the mean.

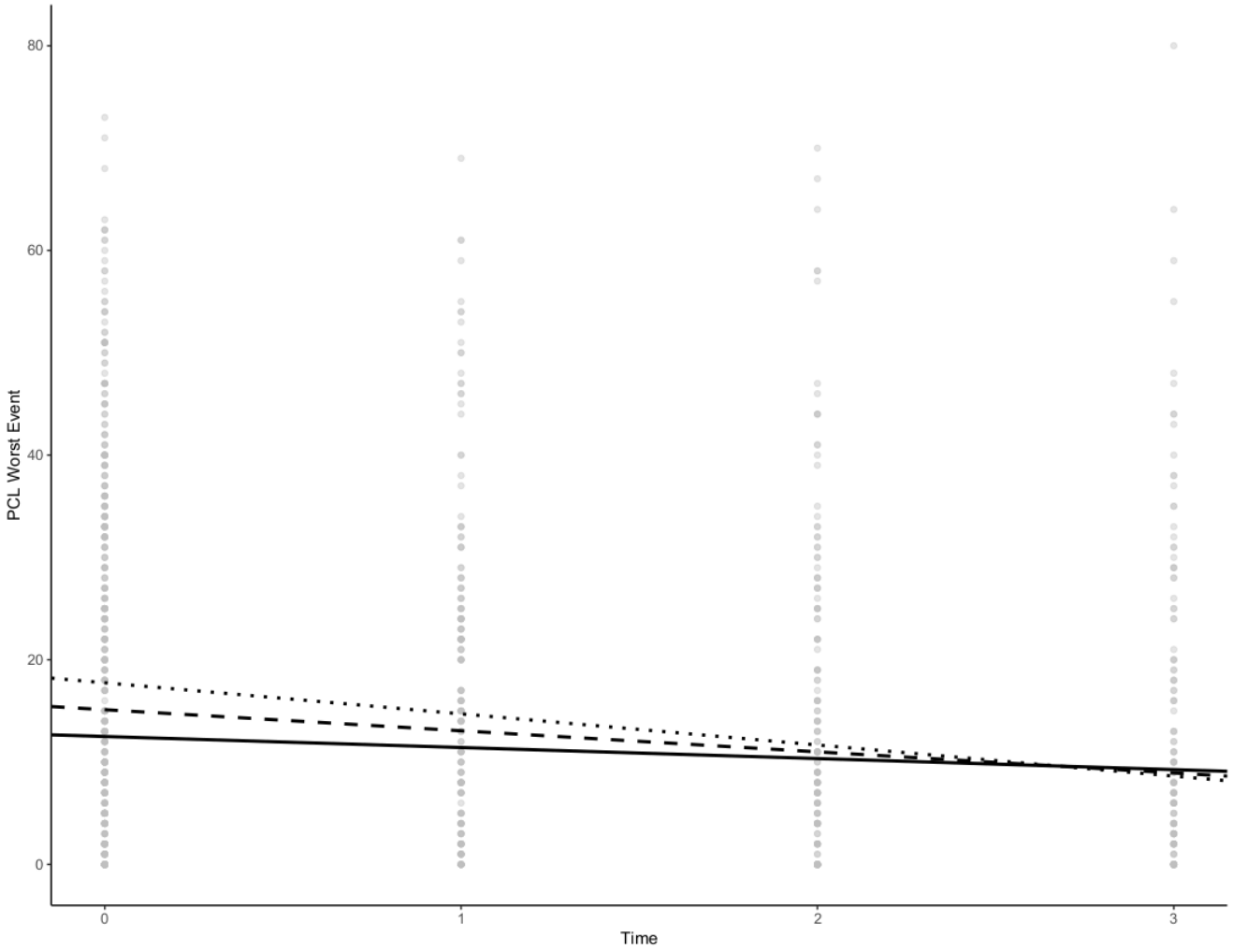

Figure 12. Cluster E Count Effects on PTSD Severity in the Full Sample. Dotted line represents 1SD above the mean, dashed line represents mean, and solid line represents 1SD below the mean.

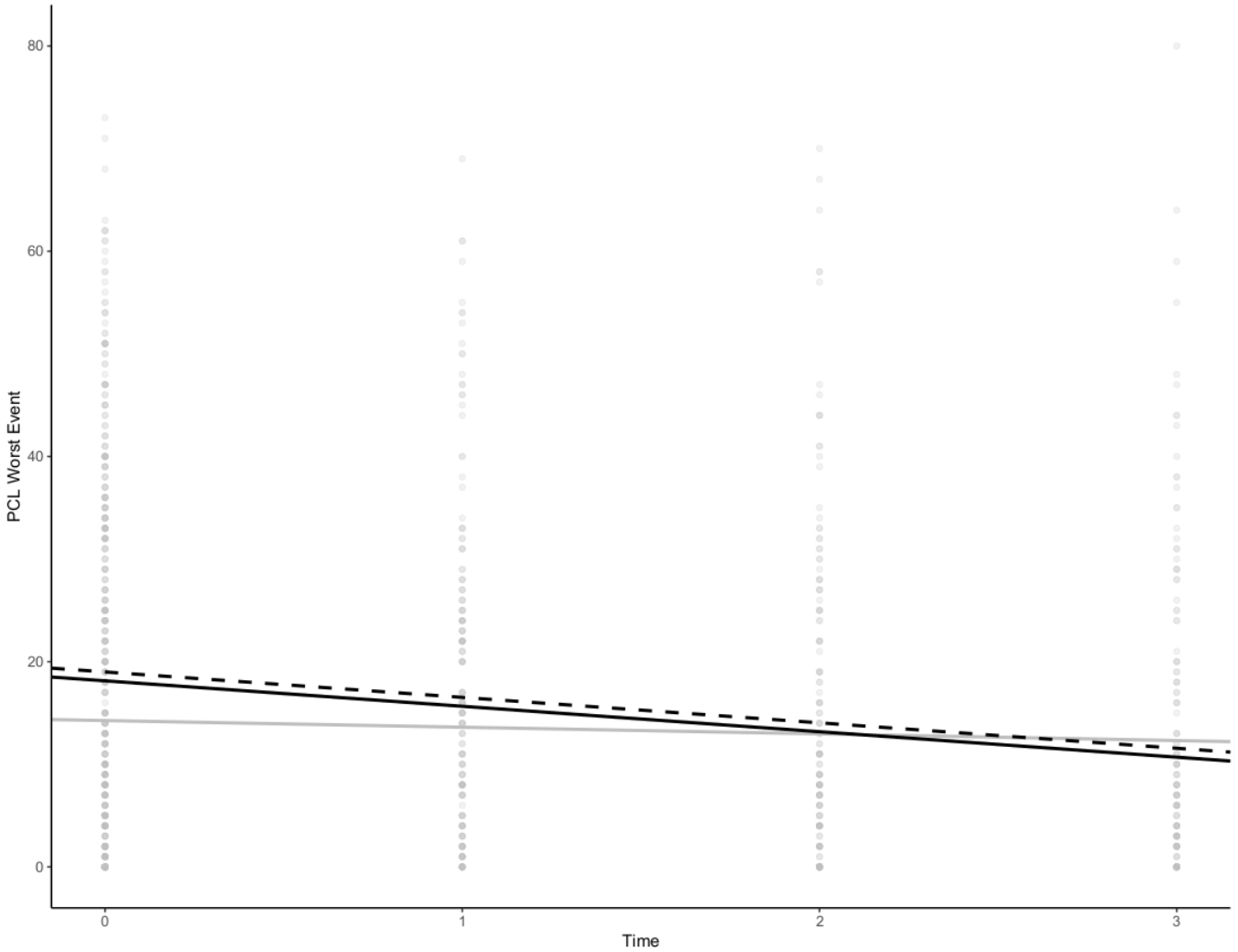

Figure 13. Anger and Sleep Problems Impact on PTSD Severity in the Full Sample. Solid black line represents reporting Loss of Interest in first week after trauma, gray line represents not reporting Anger or Sleep Problems.

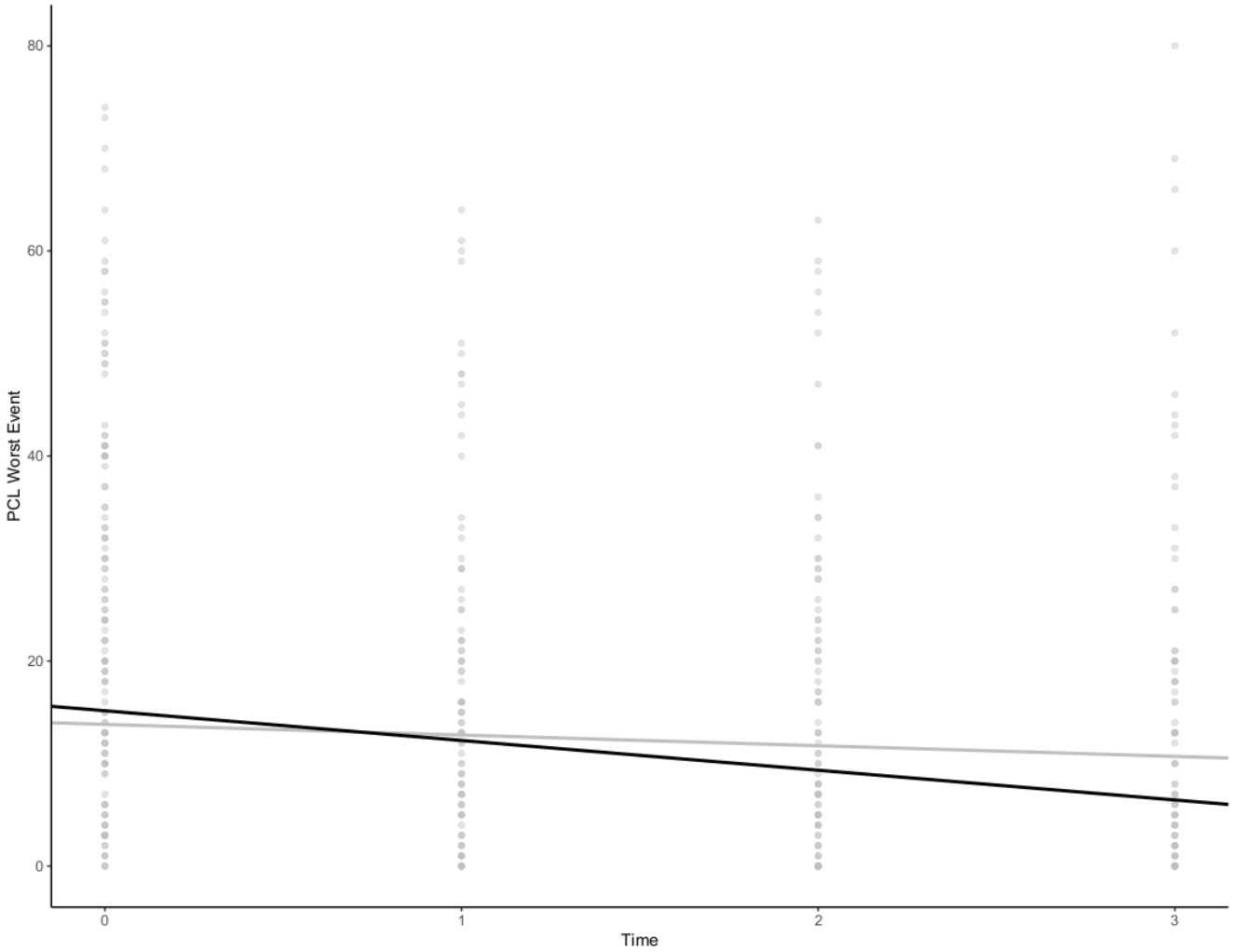

Figure 14. Concentration Problems Impact on PTSD Severity in the Full Sample. Solid black line represents reporting Concentration Problems in first week after trauma, gray line represents not reporting Concentration Problems.

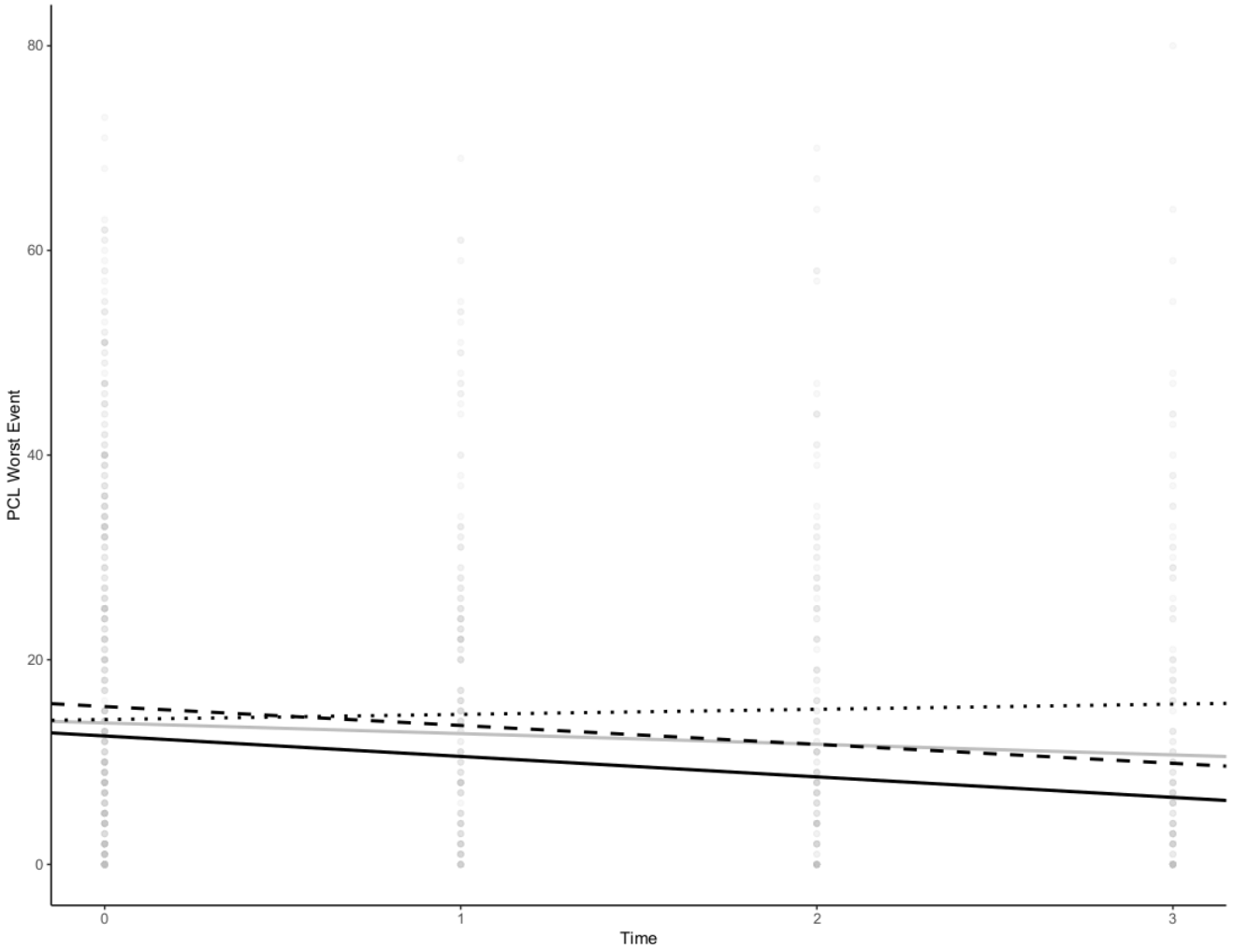

Figure 15. Interaction of Cluster D Order and Negative Beliefs on PTSD Severity in the Full Sample. Solid gray line represents the intercept and slope when Negative Beliefs are not present and Cluster D symptoms are not reported as the initial symptoms. Solid black line represents when Cluster D Order only is present, dashed black Negative Beliefs only, and dotted black is when both are present.