Coping Strategies Form Systems that Regulate PTSD Symptoms in Children and Adolescents: Exploring the Regulatory Hypothesis

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(ABSTRACT)

This study investigated the potential regulatory effects of various coping strategies on Posttraumatic Stress Disorder (PTSD). It first divided PTSD symptoms and selected coping strategies into cognitive, social/motivational, and emotional types. The study then conceptualized each of the preceding types of coping strategies as being controlled stress responses and the PTSD symptoms as being semiautomatic stress responses. It lastly proposed that coping strategies be further divided into activating controlled stress response and deactivating controlled stress response. Controlled stress responses are coping strategies that are consciously initiated and implemented. Semiautomatic stress responses are PTSD symptoms that spontaneously emerge without conscious intent. Activating controlled stress responses consisted of the following coping strategies: seeking understanding, avoidant actions, and expressing feelings. Deactivating controlled stress responses encompassed: positive cognitive restructuring, emotion-focused support, and physical release of emotions. Semiautomatic stress responses entailed: reexperiencing, numbing, and arousal symptoms. It was proposed that cognitive, social/motivational, and emotional activating controlled stress responses would increase

corresponding cognitive, social/motivational, and emotional semiautomatic stress responses. In the same vein, it was expected that cognitive, social/motivational, and emotional deactivating controlled stress responses would decrease respective semiautomatic stress responses. To illustrate, it was predicted that with regard to the cognitive regulatory system, its activating cognitive controlled stress response (seeking understanding) would exacerbate the frequency of associated cognitive semiautomatic stress responses (reexperiencing PTSD symptoms) whereas its deactivating cognitive controlled stress response (positive cognitive restructuring) would ameliorate it.

Path analyses were conducted on correlation matrices whose elements represented two coping strategies (e.g., an activating controlled stress response: seeking understanding, and a deactivating controlled stress response: positive cognitive restructuring) and one PTSD symptom cluster of the same nature (e.g., the semiautomatic stress response: reexperiencing). Data were obtained from a sample of sixty-four children and adolescents ages 8-18. The coping strategies were assessed via ratings on items included in the How I Cope Under Pressure (HICUPS) instrument and the PTSD clusters through the use of the Diagnostic Interview for Children and Adolescents (DICA).

Only one hypothesis was partially supported. It was found that the social/motivational activating controlled stress response (avoidant actions) indeed increased social/motivational semiautomatic stress responses (numbing symptoms).

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Coping Strategies Form Systems that Regulate PTSD Symptoms in Children and Adolescents: Exploring the Regulatory Hypothesis

Up to 70% of children and adolescents who lived through a war, 74% who have been victimized, and 63% who have experienced a disaster or accident develop Posttraumatic Stress Disorder (PTSD) (Saigh, Green, & Korol, 1996). These estimates indicate that a certain percentage of children and adolescents who experience a potentially traumatic event do not develop PTSD. Thus, something must account for the observation that some young individuals subsequently manifest PTSD while others do not. It is said that predispositions in the form of vulnerability and protective factors explain the diverse paths the psychological health of children and adolescents take. For instance, a frequently mentioned protective factor is the notion of coping. Coping refers to courses of actions individuals take to diminish their stress. These courses of actions refer to diverse coping strategies aimed at dealing with the cause of the stress. Thus, for instance, children and adolescents may try to reduce the frequency of maladaptive PTSD-related thoughts, social/motivational states, or emotions that produce stress by gaining control over these via compatible coping strategies. Although coping can be a protective factor, it does not always result in positive outcomes. Some coping strategies may in fact increase the frequency of PTSD-related thoughts, social/motivational states, or emotions that create stress. These coping strategies represent vulnerability factors.

Even though the functions carried out by coping strategies that represent protective factors oppose those of coping strategies that stand for vulnerability factors, the two types may coexist and even operate jointly as systems. These systems entail: (a) cognitive, social/motivational, or emotional vulnerability coping strategies that activate

corresponding clusters of PTSD symptoms (i.e., increase their frequency), (b) cognitive, social/motivational, or emotional protective coping strategies that deactivate compatible clusters of PTSD symptoms (i.e., decrease their frequency), and (c) cognitive, social/motivational, or emotional clusters of PTSD symptoms (i.e., PTSD Cluster B: re-experiencing/intrusion; PTSD Cluster C: numbing/avoidance; and PTSD Cluster D: hyperarousal/arousal). It may in fact be the case that activating coping strategies in conjunction with deactivating ones ultimately regulate the frequency of clusters of PTSD symptoms and bring children and adolescents to pre-trauma levels. The preceding statements in a nutshell form the basis of what this document refers to as the regulatory hypothesis.

Having established the theme for the document, it is hopefully clear that it will expand on the topics of (a) PTSD, (b) coping, and (c) the relationship between the two. The introduction to the study treats these topics in four sections. The first section of the paper focuses on PTSD in children and adolescents and divides into two subsections. The first subsection concentrates on the psychometric structure of the disorder. More specifically, it argues on logico-empirical grounds that the current three-fold symptom structure of PTSD representing reexperiencing, numbing, and hyperarousal clusters be further divided into a four-fold one. The second subsection addresses the qualitative structure of PTSD. It recasts clusters of PTSD symptoms as being predominantly cognitive (reexperiencing), social/motivational (numbing), or emotional (hyperarousal) in nature. It additionally differentiates cognitive, social/motivational, and emotional PTSD symptom clusters into those supported by semiautomatic processes versus those governed by controlled processes.

The second section of the document addresses the notion of coping. It divides into three subsections. The first asserts that specific types of coping strategies regulate specific types of PTSD symptoms. The second subsection introduces the counterintuitive idea that coping and PTSD symptoms can be conceptualized as two types of coping strategies. The last subsection claims that coping strategies gradually deautomatize PTSD symptoms.

The third section of the paper introduces the regulatory hypothesis. It divides into two subsections. The first explains why the hypothesis needs to be proposed, developed, and tested. The second subsection then presents a detailed description and explanation of the structural and process aspects of the hypothesis. The last section presents the scientifically formatted hypotheses to be tested in this study.

PTSD in Children and Adolescents

This section makes a case for dividing PTSD into four clusters instead of three. It then conceptualizes clusters of PTSD symptoms in children and adolescents as semiautomatic cognitive, social/motivational, and emotional stress response systems.

The Psychometric Structure of PTSD

The presentation of PTSD in youth is similar to that in adults. However, in children, responding to the event with intense fear, horror, or helplessness (A2) can also be expressed as disorganized or agitated behavior. In addition, the reexperiencing symptom B1 (distressing recollections) can be manifested as repetitive play in which themes or aspects of the trauma are expressed, B2 (distressing dreams) as frightening dreams without recognized content, and B3 (reliving the event) as re-enactment of the trauma.

The symptom structure of PTSD in children and adolescents varies depending on the traumatic event and the PTSD assessment instrument used. However, two clear confirmatory factor solutions emerge from the data. One indicates that four factors "account" for the variance observed in the 17 PTSD DSM-IV-TR symptoms. Thus, instead of the three-cluster structure of PTSD proposed by the task force of the DSM-IV, using the Diagnostic Interview for Children and Adolescents (DICA) on adolescent survivors of the Pol Pot War, Sack, Seeley, & Clarke, 1997, found that their confirmatory factor analysis yielded a four-factor intercorrelated PTSD structure composed of reexperiencing, avoidance, numbing, and hyperarousal symptoms that fit the data better. These findings were later replicated in a second study in which the same instrument yielded a four-factor intercorrelated solution from a sample of victimized homeless adolescents (Stewart, Steiman, Caucce, Cochran, Whitbeck, & Hoyt, 2004).

The Qualitative Structure of PTSD: Nature of Symptoms

It is proposed that three out of the four factors representing the psychometric structure of PTSD be each identified as predominantly cognitive (reexperiencing), social/motivational (numbing), and emotional (hyperarousal) components of the disorder. These are components that logically represent systems or assemblages or combinations of things or parts forming complexes or unitary wholes (American Heritage Dictionary, 2006). In the statistical sense, they represent statistically significant covariances between observed variables. Theoretically, a system originates out of shared underlying maladaptive supports. For instance, the cognitive system representing the reexperiencing PTSD symptom cluster may form out of shared defective cognitive components, such as troublesome working memory modules or poor executive function parts. Thus, it is

reasonable to propose that there exist cognitive, social/motivational, and emotional PTSD systems

The Qualitative Structure of PTSD: Automaticity of Symptoms

Cognitive, social/motivational, and emotional PTSD systems are formed by semiautomatic and controlled stress responses. It is important to keep in mind that only the semiautomatic stress responses constitute the PTSD syndrome with the exception of the active avoidance symptoms (C1 and C2). There is much to be said about controlled and semiautomatic processes, however (c.f., Moors & Houwer, 2006). According to Bargh (1994), the main feature distinguishing automatic from controlled processes is autonomy. Autonomous processes run their course to completion without conscious guidance or monitoring. Furthermore, pure automatic processes are identified as being unconscious, unintentional, effortless/efficient, and uncontrollable. Automatic processes do not interfere with other conscious processes according to one version of automaticity (Posner & Snyder, 1975a, 1975b). The manner by which processes become automatic entails strengthening of computational algorithms or shifts toward one-step memory retrieval (Moors & Houwer, 2006). Both mechanisms come about as a result of repeated practice. Lastly, automatic processes are conditional in the sense that there exist preconditions for their initiation. As such, few processes are indeed purely automatic (Bargh, 1992). Since few processes are purely automatic, most consist of automatic and controlled features. Thus, automatic processes belong to one of three classes: (a) preconscious requiring no conscious input and no intention, (b) postconscious entailing conscious input but no intention, and (c) goal-dependent demanding conscious input and intention to start but no goal to run to completion.

From what has been said up to now, it is clear that traumatic stress responses (PTSD symptoms) are not purely automatic but semiautomatic. For instance, PTSD reexperiencing symptoms are automatic in the sense that they are autonomous (not self-started, -maintained, or -terminated), effortless, and often unintentional (i.e., not pursued as a goal of the organism), but controlled because they are available to consciousness and can be altered or stopped via coping strategies such as cognitive avoidance or distraction. Furthermore, PTSD symptoms interfere with other psychological processes suggesting that they cannot be fully automatic. For this reason, from now on we will refer to traumatic stress responses (PTSD symptoms) as semiautomatic stress responses.

Examples Concerning the Qualitative Structure of PTSD

Reexperiencing Symptoms as Semiautomatic Cognitive Stress Responses. The PTSD syndrome as expressed in children and adolescents consists of a cognitive component associated with reexperiencing symptoms. The symptoms themselves are cognitive semiautomatic stress responses. As an illustration, children and adolescents who experience a traumatic event often feel confused or in a daze during the episode, ruminate, have nightmares, experience and flashbacks of the event. Moreoever, these individuals are more likely to be diagnosed with PTSD even 3 and 6 months after the event (Ehlers, Mayou, & Bryant, 2001).

Numbing Symptoms as Semiautomatic Social/Motivational Stress Responses. Posttraumatic stress disorder is accompanied by a host of social/motivational impairments associated with numbing symptoms experienced by children and adolescents. Indeed, paralleling the social outcomes of traumatized adults, young people who are positive for PTSD exhibit similar semiautomatic social/motivational stress

responses such as less interest in enjoyable things, estrangement from parents and friends, and constricted affect about the event (Pynoos, Calvin, Kathi, William, Steinberg, Eth, Nunez, & Fairbanks, 1987). Moreover, children and adolescents with a diagnosis of PTSD exhibit more internalizing behaviors responsible for social/motivational deficits (e.g., depression) than those without the disorder (McDermott & Cvitnovich, 2000). Indeed, when avoidance (C1-C2) and numbing symptoms (C3-C7) are investigated separately the latter is related to disorders leading to social/motivational difficulties (e.g., alexithymia) while the former is not (Badura, 2003).

Arousal Symptoms as Semiautomatic Emotional Stress Responses. The emotional component of PTSD in young people interestingly also paralleled that of adults. Children and adolescents diagnosed with PTSD manifest semiautomatic emotional stress responses that are expressed as hyperarousal symptoms. These responses represent dysfunctional bioemotional systems resulting from unregulated neurotransmitters, neurohormones, psychophysiology, and neurocognition (Silva, 2004). Further attesting to the contribution of emotional arousal to PTSD in children and adolescents is the observation that reexperiencing and avoidance/numbing symptoms decrease with time while hyperarousal symptoms stay the same (Scheeringa, Zeanah, Myers, & Putnam, 2005).

Coping

Coping Strategies Handle Corresponding Demands

Coping is defined in a variety of ways. Most conceptions view coping as a self-regulatory process (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). This notion is contained in the one of the most cited conceptual definitions of coping. It states that coping refers to constantly changing cognitive and behavioral efforts to

manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person (Lazarus and Folkman, 1984; p. 141). This idea can be expanded to suggest that certain amounts of cognitive, social/motivational, and emotional coping resources are needed to meet specific cognitive, social/motivational, and emotional PTSD symptoms. For instance, certain amounts of working memory are needed to deal with reexperiencing symptoms of PTSD, social and self-regulatory competence to manage numbing symptoms, and emotional regulation strategies to moderate hyperarousal symptoms. It follows that when these resources are not enough to deal with their corresponding demands, a subjective state known as stress ensues. Stress, thus, is the result of appraising cognitive, social/motivational, or emotional demands (semiautomatic stress responses) as taxing or exceeding corresponding cognitive, social/motivational, or emotional resources (controlled stress responses).

Coping Strategies and PTSD Symptoms

Coping is a term used in conjunction with the notion of stress (e.g., one copes with stress). It is clear, then, that of all psychopathological states, PTSD as a stress disorder is most straightforwardly related to the idea of coping. At the core of this disorder is stress caused by a traumatic event as described in the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV Text Revision, 2000) as such:

> **Criterion A.** The person has been exposed to a traumatic event in which both of the following have been present:
>
> (1) The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
>
> (2) The person's response involved intense fear, helplessness, or horror. **Note:** In children, this may be expressed instead by disorganized or agitated behavior.

The link between PTSD symptoms and coping is examined in the children and adolescents coping literature. For instance, Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, (2000) argued that stress responses fall on two dimensions: voluntary and involuntary. Voluntary stress responses are controlled processes that go by the name of coping while involuntary stress responses are processes spontaneously deployed during stressful situations. Consistent with these ideas, their confirmatory factor analytical study conducted on a number of stress responses yielded the following solutions: Two factors representing voluntary engagement and disengagement encompassing controlled regulatory processes (coping) and two other factors standing for involuntary engagement and disengagement composed of stress responses (PTSD symptom). The authors did not refer to the indicator variables as controlled and semiautomatic stress responses, but their features suggested so. For instance, the stress responses representative of involuntary engagement were: rumination, intrusive thoughts, emotional arousal, physiologic arousal, and impulsive actions. One cannot help to notice that these are reexperiencing and hyperarousal symptoms, respectively. Of equal importance were the components making up involuntary disengagement: cognitive interference, involuntary avoidance, inaction, and emotional numbing. These seem to be symptoms included in the numbing/avoidance PTSD cluster. Interestingly, the components constituting the voluntary engagement and disengagement controlled regulatory processes often matched in quality to those making up the involuntary engagement and disengagement stress responses.

In relation to the work on coping, PTSD symptoms seem to be maladaptive ways of coping in the sense that (a) they are outside of the control of the individual, (b) cause

clinically significant distress, and (c) create social/motivational, academic, or occupational impairments. It is also possible that some controlled ways of dealing with stressors might have become semiautomatic with repetition, thereby creating habits that in the context of a traumatic stressor become maladaptive in the sense that they produce clinically significant distress or occupational, social/motivational, or academic difficulties. It is also possible that some of these individuals deal with stressors by engaging semiautomatic processes (such as, emotional/physiological arousal, intrusive thoughts/rumination, and social/motivational numbing) that are outside of their control but are not maladaptive because in the absence of a traumatic experience they neither cause clinically significant distress nor social, academic, or occupational impairment.

Coping Strategies, PTSD Symptoms, and Gradual Deautomatization

Because semiautomatic stress responses (PTSD symptoms) include automatic and controlled aspects, it is clear that they share something in common with controlled stress responses (coping). For instance, the computational algorithms (psychological mechanisms) underlying cognitive semiautomatic stress responses (reexperiencing symptoms) could overlap with those supporting cognitive controlled stress responses (cognitive coping strategies). More importantly, since they share similar characteristics—such as being available to consciousness—but differ in controllability, the one that can be controlled may be used to deautomatize the other. Thus, an example would be cognitive controlled stress responses being used to deautomatize automatic aspects of cognitive semiautomatic stress responses by taking advantage of overlapping controlled features (e.g., conscious availability). This document refers to this process as gradual deautomatization.

PTSD and Coping Strategies as Systems of Regulatory Processes

Up to now this document has introduced some specific ideas upon which its central hypothesis rests. For instance, it continuously emphasized the need to study PTSD symptoms separately and to group them according to their nature. It proposed that coping strategies be treated similarly. Finally, it conceptualized the former notion as one representing semiautomatic stress responses whereas the latter controlled stress responses. This section seeks to establish a relationship between these two types of stress responses.

Individuals are resilient in the aftermath of a traumatic event in the sense that they have cognitive, social/motivational, and emotional competences that allow them to successfully deal with cognitive, social/motivational, and emotional PTSD symptoms. How and why these resources neutralize PTSD symptoms is of central interest to this document. As such, it first focuses on PTSD symptoms as cognitive, social, and emotional semiautomatic stress responses that are maladaptive in the sense of being outside the conscious control of individuals and causing clinically significant distress or social, academic, or occupational impairment. It then treats coping as controlled cognitive, social/motivational, and emotional stress responses that are aimed at reducing or eliminating corresponding maladaptive semiautomatic cognitive, social/motivational, and emotional stress responses (i.e., PTSD symptoms).

The analysis subsequently offers a hypothetical process underlying the link between controlled and semiautomatic stress response processes. At a practical level, this analysis demonstrates how children and adolescents may use controlled stress responses (coping) to indirectly take control of semiautomatic stress responses (PTSD symptoms).

The key contribution this document makes to the corpus of knowledge on coping and PTSD in children and adolescents is to propose that cognitive, social/motivational, and emotional controlled stress responses (coping strategies) create systems whose components regulate target cognitive, social/motivational, and emotional semiautomatic stress responses (PTSD symptoms). It is recalled that systems are assemblages or combinations of things or parts forming complexes or unitary wholes (American Heritage Dictionary, 2006). Regulatory systems contain controlled stress responses of the same type (e.g., cognitive coping strategies) that jointly exacerbate and ameliorate target semiautomatic stress responses (reexperiencing symptoms).

Factors Leading to the Proposal, Development, and Test of the Regulatory Hypothesis

Before introducing the regulatory hypothesis, this document needs to explain why the clinical psychology field needs yet another explanatory mechanism in the area of coping and PTSD. After all, it is well-known that certain types of coping (e.g., passive) exacerbate PTSD symptoms while others (e.g., active) ameliorate them. If the relationship between coping and PTSD could be summarized in this nice linear manner it would only be a matter of concentrating on the main effects of a few coping strategies and forget about mediating or moderating phenomena. However, this is rarely the case in the real world. There are at least three reasons to include more complex mediating and moderating models in the area of coping and PTSD. First, the coping literature indicates that youth engage in not just one or two types of coping strategies, but many. There is no magic coping strategy that is a panacea to all troubles. Second, PTSD consists of many stressing components that cannot be dealt with by only one coping strategy. Third,

sometimes so-called beneficial coping strategies actually exacerbate PTSD symptoms while thought to be maladaptive ones actually ameliorate them.

We previously discussed the study conducted by Compas et al. which implies that individuals rely on groups of diverse coping resources when faced with multifaceted stressors. In the case of PTSD, traumatic events demand that individuals resort to cognitive, social/motivational, and emotional coping resources. Further supporting this contention is the result of a study indicating that about 90% of children and adolescents in one investigation reported frequently relying on 5 coping strategies, out of a total of 10 (Stallard, et al. 2001). For this reason, it is instructive to examine how different coping strategies affect the different symptom clusters making up PTSD.

Semiautomatic stress responses constituting the PTSD syndrome vary in configuration across individuals. Indeed, only one out of 5 reexperiencing symptoms, three out of seven numbing/avoidance symptoms, and two out of five arousal symptoms are needed for a diagnosis of PTSD. This means that different patterns of controlled stress responses (coping strategies) would be needed to deal with corresponding configurations of these semiautomatic PTSD stress responses. In fact, this is perhaps what makes the difference in the time course and permanence of semiautomatic stress responses. That is, to the extent that effective controlled stress responses are enlisted, semiautomatic stress responses may subside or live on forever. For this reason, the relationship between types of controlled stress responses and clusters of PTSD semiautomatic stress responses needs to be studied.

It is believed that certain ways of coping with cognitive, social/motivational, and emotional difficulties are universally adaptive whereas others are globally maladaptive.

However, upon a closer look at the literature on coping and PTSD a number of inconsistencies arise. For instance, the group of controlled stress responses known as avoidance coping at first sight would seem to be maladaptive strategies for dealing with semiautomatic stress responses (PTSD symptoms). However, it has been demonstrated that in certain cases avoidance may not be such a bad thing. For instance, Bonanno, Keltner, Holen, & Horowitz (1995) showed that certain individuals do better when they avoid the cognitive, social/motivational, or emotional processing of traumatic material. On the other hand, other studies indicate that certain avoidance strategies, such as the suppression of unwanted thoughts, exacerbate PTSD symptoms (Asarnow, Glynn, Pynoos, Nahum, Guthrie, Cantwell, & Franklin, 1999; Ehlers, Mayou, & Bryant, 2001). Similar results involve postulated adaptive strategies. To illustrate, distracting oneself from traumatic stimuli, cognitive structuring traumatic knowledge or appraisals, engaging in problem solving, seeking social support, or calming oneself are believed to be adaptive controlled responses; yet, they might be related to higher levels of PTSD just as much as maladaptive ones, such as criticizing oneself, increasing emotionality, blaming others, and withdrawing from social life (Vernberg, La Greca, Silverman, & Prinstein, 1996). In the same vein, other supposedly adaptive strategies used by children and adolescents measured with the Kidcope instrument (Spirito, Stark, & Williams, 1988), such as problem solving and emotion regulation, were positively correlated with frequency of PTSD symptoms assessed via the Post-Traumatic Stress Reaction Index (Frederick, 1987) (Russoniello, Skalko, O'Brien, McGhee, Bingham-Alexander, & Beatley, 2002).

The Hypothesis of Coping Strategies as Regulatory Systems

The regulatory hypothesis examines the claim that groups of controlled stress responses (i.e., coping strategies) are deployed to govern groups of semiautomatic stress responses (i.e., PTSD symptoms). It further investigates the possibility that specific groups of controlled stress responses are effective managers of particular types of semiautomatic stress responses. What is novel about this hypothesis is the contention that there exist markers underlying the transformation of semiautomatic stress responses into controlled ones. These markers take the observable form of increases and decreases in semiautomatic stress responses. Also of novelty is the idea that the activation of controlled stress responses in the face of qualitatively similar semiautomatic stress responses is an indirect method of taking over otherwise involuntary processes that are outside of the reach of conscious control. This process involves the gradual deautomatization of cognitive, social/motivational, and emotional semiautomatic stress responses that have become indomitable and are experienced as uncontrollable clusters of PTSD symptoms. Thus, ironically, losing control over psychological processes that are outside the realm of conscious control is the price paid in exchange for their eventual submission. This submission comes about as cognitive, social/motivational, and emotional semiautomatic stress responses (PTSD symptom) increase as they struggle against given types of controlled stress responses and decrease as they are transformed by different classes of controlled stress responses. For this reason, cognitive, social/motivational, and emotional controlled stress responses that increase symptoms go by the name of activators while those that decrease them receive the name of deactivators.

Structural and Process Aspects of the Regulatory Hypothesis. The regulatory hypothesis offers a structural and a process interpretation. Structurally, it predicts that controlled stress responses (coping strategies) and semiautomatic stress responses (PTSD symptoms) share underlying mechanisms and characteristics. As long as controlled stress responses share underlying mechanisms and characteristics with semiautomatic stress responses (PTSD symptoms), it is possible for the former to gradually gain control over the latter. For instance, PTSD cluster B symptoms (reexperiencing) seem to be predominantly cognitive in nature in the sense that underlying cognitive dysfunctions may best explain their existence (e.g., deficient executive function or working memory). Similarly, certain controlled cognitive stress responses such as seeking understanding or positive cognitive restructuring of the traumatic material require equivalently underlying support systems (executive function, working memory, etc.). Therefore, predominantly cognitive PTSD symptoms may share support mechanisms with cognitive coping strategies. Because of this commonality cognitive controlled stress responses may eventually come to govern the activity of cognitive semiautomatic stress responses.

Process wise, the regulatory hypothesis contends that the preceding state of affairs allows controlled stress responses in the form of coping resources to deautomatize semiautomatic stress responses. The regulatory hypothesis proposes that this transformation is indexed by certain observables, namely, increases and decreases in the emergence of semiautomatic stress responses (PTSD symptoms). Why should this be the case? The hypothesis posits that since controlled stress responses and semiautomatic stress responses share mechanisms of action and characteristics of controlled processes, the activation of the latter becomes one of the preconditions for the activation of the

former. Thus, PTSD symptoms and coping strategies become intimately connected to each other as one facilitates the occurrence of the other. I term this process feature the activation facilitation hypothesis. At a molar level this is a reasonable hypothesis to advance given that controlled stress responses in the form of coping strategies trigger semiautomatic stress responses in the form of PTSD symptoms. For instance, individuals exhibiting partial or full PTSD tend to avoid any cues that may activate corresponding symptoms. In the context of the regulatory hypothesis, controlled stress responses (coping) become another cue that may bring about semiautomatic stress responses (PTSD symptoms).

As the process portion describes how increases in controlled stress responses lead to increases and decreases in semiautomatic stress responses, it offers the following possibilities. Controlled stress responses or other entities can serve as triggering cues that activate semiautomatic stress responses. If controlled stress responses function as cues, then priming of semiautomatic stress responses through spread of activation explains their emergence. Of importance is to point out that whether semiautomatic stress responses initiate due to outside cues or controlled stress responses, the former process requires no conscious or willful initiation, maintenance, and termination. However, individuals must will controlled stress responses into existence regardless of how PTSD symptoms were triggered. Thus, while under certain conditions controlled stress responses may or may not materialize, semiautomatic stress responses must. In the case activation via traumatic cues, through spreading of activation priming semiautomatic stress responses may make corresponding controlled stress responses available; however, individuals still must consciously access, retrieve, and apply them.

Once the dysfunctional mechanisms underlying semiautomatic stress responses (PTSD symptoms) initiate them, it is difficult to consciously guide, monitor, alter, or stop them. It is at this point that controlled stress responses offer their assistance by taking over certain aspects of these maladaptive mechanisms that are outside conscious control. For instance, defective executive function may be responsible for uncontrollable reexperiencing symptoms. Thus, semiautomatic stress responses may not be controlled from within but from without via activating controlled stress responses. This transfer of control paradoxically results in initial increases in semiautomatic stress responses because although given controlled stress responses activate them, these do not modify them. They instead serve to gain control over the initiation, guidance, and monitoring of semiautomatic stress responses. A second type of controlled stress response is needed, namely, deactivating controlled stress responses. These are in charge of altering the content of the semiautomatic stress responses so as to gradually achieve their deactivation.

Notice that all three components of the regulatory hypothesis share underlying supports and thus can theoretically prime each other. However, in order for controlled stress responses to regulate associated semiautomatic stress responses the semiautomatic stress responses must first be activated and then deactivated. This is the reason the regulatory hypothesis proposes that (a) individuals who first rely on activating controlled stress responses to take control of semiautomatic stress responses by increasing them and (b) who subsequently resort to deactivating controlled stress responses to decrease them should be the ones who bring themselves to pre-trauma levels in the natural environment.

Process Models in the Regulatory Hypothesis. The regulatory hypothesis proposes the existence of systems of regulatory coping strategies classified as activators and deactivators. The activators gain control of the initiation, guidance, and monitoring of semiautomatic stress responses (PTSD symptoms) while their counterparts, deactivators, render these inert. There are four models that can be proposed to explain how the preceding events occur. The first is an independence model that states that although activators and deactivators are independent entities, the former increases whereas the latter decreases semiautomatic stress responses. The second model is an ordinary regression model similar to the preceding one where activators and deactivators only correlate with each other but they have main effects on semiautomatic stress responses. This model provides a certain amount of information above and beyond the independence one. Yet, a third model offers an extra piece of information. The moderating model contends that (a) activators and deactivators exert main effects on semiautomatic stress responses, and (b) activators and deactivators jointly influence semiautomatic stress responses. One way to describe the interaction effect in this model is to think of it as predicting that the linear relationship between deactivators and semiautomatic stress responses will change according to the strength of the activator. For instance, at high levels of the activator the linear relationship between deactivators and semiautomatic stress responses would be stronger (a steeper negative slope) than at low levels. In other words, the more a given activating coping strategy is used (e.g., seeking understanding) the more a corresponding deactivating coping strategy (e.g., positive cognitive restructuring) decreases respective PTSD symptoms (e.g., intrusive symptoms). The fourth and last model offers the most information. The mediating specifies the following

components: (a) a direct effect of activators on semiautomatic stress responses, (b) an indirect/mediated effect of activators on semiautomatic stress responses via deactivators, (c) a main effect of deactivators on semiautomatic stress responses, and (d) the spurious effect of activators on the relationship between deactivators and semiautomatic stress responses.

Study Hypotheses

To summarize, the present study tests the core idea that activators (controlled stress responses) indirectly initiate qualitatively matching semiautomatic stress responses due to the fact that they share underlying support mechanisms. This is accompanied by the action of deactivators (controlled stress responses) on qualitatively matching semiautomatic stress responses. Technically speaking, (a) activators and semiautomatic stress responses are positively causally related, (b) deactivators and semiautomatic stress coping responses negatively causally related, and (c) activators and deactivators are positively correlated. It is important to point out that the relationship between activators and deactivators differs in nature from the relationship between these controlled stress responses and semiautomatic stress responses. Activating controlled stress responses prime and make available deactivating controlled stress responses, even though both controlled stress responses must be willed into existence (i.e., processes must be consciously retrieved from long term memory and activated in working memory). This is not true of semiautomatic stress responses. Activating and deactivating controlled stress responses prime, activate, and initiate semiautomatic stress responses, because semiautomatic stress responses cannot be willed into existence.

The preceding ideas fall under two groups of hypotheses. The first set of predictions focuses on the structural aspect of the regulatory hypothesis. They test whether qualitatively matching activating and deactivating controlled stress responses and semiautomatic stress responses form systems. These systems consist of activators that when present increase semiautomatic stress coping responses (i.e., PTSD symptoms) and deactivators that result in corresponding decreases. More specifically, the claims to be tested are the following:

1. Combinations of cognitive controlled stress responses have regulatory effects on cognitive semiautomatic stress responses (cognitive PTSD symptoms). At a more refined level, seeking understanding coping increases reexperiencing symptoms while positive cognitive restructuring coping decreases them.
2. Combinations of social/motivational controlled stress responses have regulatory effects on social/motivational semiautomatic stress responses (motivational/social PTSD symptoms). More specifically, avoidant actions coping increases numbing symptoms whereas emotion-focused support coping decreases them.
3. Combinations of emotional controlled stress responses have regulatory effects on emotional semiautomatic stress responses (emotional PTSD symptoms). That is, expressing feelings coping increases arousal symptoms but physical release of emotions decreases them.

The second group of predictions concerns the process aspect of the regulatory hypothesis. Because the regulatory hypothesis is in its infancy, a number of process models are tested in order to extract the most amount of information for future research. These are:

4.1, 4.2, and 4.3. An independence model in which activating and deactivating controlled stress responses are not related to each other, only to semiautomatic stress responses.

5.1, 5.2, and 5.3. An ordinary regression model in which activating and deactivating controlled stress responses correlate with each other and have main effects on semiautomatic stress responses.

6.1, 6.2, and 6.3. A moderating/interactional model in which activating and deactivating controlled stress responses have main and interactional effects on semiautomatic stress responses.

7.1, 7.2, and 7.3. A mediating model in which activating controlled stress responses have main and indirect effects on semiautomatic stress responses and deactivating ones have main effects on semiautomatic stress responses.

Method

Participants

One hundred and sixty-four children and adolescents were interviewed about four months after exposure to residential fires. There were 71 (43%) African Americans and 93 (57%) European Americans. Seventy-two youth (44%) from the sample were boys and 92 (56%) were girls. The mean age of the sample was 11.18 (SD = 1.31). The sample consisted of children, adolescents, and their families who participated in an NIMH-sponsored study that looked into the effects of residential fires (age 8-18) (Residential Fire Project -- RFP; Jones & Ollendick, 2001). The study sampled from areas in and surrounding five locations: Atlanta, Georgia; Blacksburg and Richmond, Virginia; Charlotte, North Carolina; and Charleston, South Carolina. Recruiting criteria included:

1) the family lost at least 15% of their home or personal belongings, and 2) the family had a child between 8 and 18 years of age. The study sampled families who recently experienced fires in their homes from incident reports sent to the investigators by fire departments, news reports in the newspaper or on television, and information given out to fire victims about the project by Red Cross agencies. Potential participants were informed about the project through letters and telephone calls. Those who replied were screened. Families who met inclusion criteria were asked if they would be interested in participating in the study. Families who agreed were then interviewed. About one third of the families contacted met inclusion criteria and two thirds of them agreed to participate. Approximately ninety percent of these families completed the first interviews.

Procedure

Approximately four months after they experienced a residential fire, children and adolescents were interviewed by advanced graduate students in an APA-approved clinical psychology training program who had been trained in the administration of the measures. These interviews occurred in the participants' homes or in public places such as Red Cross offices, neighborhood churches, libraries, or mental health clinics. The interviewers first obtained informed consent from the parents and the children and adolescents. Then, they interviewed the young people. Finally, the interviewer paid $75 to each family for its participation.

Measures

Coping (How I Cope Under Pressure Scale; HICUPS)

Participants first listened to the following instructions:

"Now I want you to think about what you did to make things better or to make yourself feel better as a result of the fire. Please tell me how much you thought or did EACH of the following things to try and make things better or to make yourself feel better. There are no right or wrong answers. Just indicate how often you did each of these things as a result of the fire".

Children and adolescents then listened to 45 coping strategies one at a time and rated each on four-point scales defined by intervals: 1 = not at all, 2 = a little, 3 = somewhat, and 4 = a lot.

PTSD (Diagnostic Interview for Children and Adolescents – PTSD Module; DICA)

The study used the Posttraumatic Stress Disorder (PTSD) module of the Diagnostic Interview for Children and Adolescents (DICA-R-C and DICA-R-A; Reich, 2000) to assess 15 semiautomatic stress responses constituting part of the disorder. Trained DICA interviewers asked children and adolescents whether they had experienced each PTSD symptom during the past week. The interviewer then judged their answers on a four point continuum: 1 = no, 2 = rarely, 3 = sometimes or somewhat, and 5 = yes. The four point rating scale then served as a basis for deciding whether the children or adolescents interviewed met criteria for specific PTSD symptoms. This was done according to the instruments protocol by dichotomizing the continuous scales in the following manner: 1 = symptom absent and 2, 3, 5 = symptom present. The final total scale for a particular cluster consisted of the sum total of its symptoms being present (reexperiencing range = 0 – 5 symptoms; numbing range = 0 – 5 symptoms; arousal range = 0 – 5 symptoms).

Activating Cognitive Controlled Stress Responses (ACCSR), Deactivating Cognitive Controlled Stress Responses (DCCSR), and Semiautomatic Cognitive Stress Responses (SCSR)

Seeking Understanding (ACCSR). The seeking understanding subscale of the How I Cope Under Pressure Scale (HICUPS; Ayers et al., 1996) assesses "cognitive efforts to find meaning in the stressful situation or to understand it better". It consists of five items: (a) thought about why it happened, (b) asked God to help me understand it, (c) tried to understand it better by thinking about it, (d) thought about what I could learn from the problem, and (e) tried to figure out why things like that happened. The Cronbach's alpha coefficient of internal consistency for this HICUPS subscale was .66. This alpha is close in magnitude to the one yielded in the work conducted by Ayers et al. of .74.

Positive Cognitive Restructuring (DCCSR). This HICUPS subscale assesses whether the child or adolescent "thinks about the situation in a more positive way". It is composed of the following items: (a) tried to notice or think about only the good things in life, (b) told myself it would be over in a short term, (c) reminded myself that things could be worse, and (d) told myself it is not worth getting upset about. The alpha coefficient for this HICUPS subscale was .41; the alpha in the original work was .62.

Reexperiencing PTSD Symptoms (SCSR). This DICA PTSD module subscale taps into the five symptoms making up cluster B, reexperiencing symptoms. The Kuder-Richardson's internal consistency coefficient for this subscale was .73.

Activating Social/Motivational Controlled Stress Responses (ASMCSR), Deactivating Social/Motivational Controlled Stress Responses (DSMCSR), and Semiautomatic Social/Motivational Stress Responses (SSMSR)

Avoidant actions (ASMCSR). This HICUPS subscale measures "behavioral efforts to avoid the stressful situation by staying away from it or leaving it". The Cronbach's alpha coefficient of internal consistency for this HICUPS subscale was .53; the alpha in the original work was .64. There are four items that make up this subscale: (a) tried to stay away from the problem, (b) tried to stay away from things that made me feel upset, (c) avoided the people who make me feel bad, and (d) avoided it by going to my room.

Emotion-Focused Support (DSMCSR). This HICUPS subscale evaluates whether the child or adolescent "involves other people in listening to feelings or providing understanding to help him or her be less upset". It presents the participant with the following statements: (a) talked about how I was feeling with my mother or father, (b) talked about how I was feeling with some adult who is not in my family, (c) talked with my brother or sister about my feelings, and (d) talked with one of my friends about my feelings. The subscale yielded a Cronbach's alpha of .60; the alpha in the original work was exactly the same (.60).

Numbing PTSD Symptoms (SSMSR). This variable was created by aggregating five of the seven cluster C numbing symptoms measured by the DICA-PTSD module. This subscale yielded a Kuder-Richardson's internal consistency coefficient of .74.

Activating Emotional Controlled Stress Responses (AECSR), Deactivating Emotional Controlled Stress Responses (DECSR), and Semiautomatic Emotional Stress Responses (SESR)

Expressing Feelings (AECSR). This HICUPS subscale taps into any "overt expression of feelings either by an action to express feelings, a verbal expression of feelings, or simply an over release of emotion". It yielded a Cronbach's alpha of .57; the alpha in the original work was .59. It consists of the following items: (a) wrote down my feelings, (b) cried to myself, and (c) let out feelings to my pet or stuffed animal.

Physical Release of Emotions (DECSR). This HICUPS subscale looks into whether the child or adolescent engaged in "efforts to physically work off feelings with physical exercise, play, or efforts to physically relax". It consists of the following statements: (a) went bicycle riding, (b) played sports, (c) went skateboarding or roller-skating, and (d) did some exercise. The Cronbach's alpha coefficient of internal consistency for this HICUPS subscale was .65; the alpha in the original work was identical (.65).

Arousal PTSD Symptoms (SESR). The arousal variable was obtained from the five cluster D arousal symptoms assessed by the DICA-PTSD module. The Kuder-Richardson's internal consistency coefficient for this subscale was .74.

Results

Before any main path analyses concerning the hypotheses of interest can be conducted, it is logically necessary to offer empirical evidence for the following:

1. The determined controlled stress responses (coping strategies) represent psychometrically valid variables.

2. The specified semiautomatic stress responses (PTSD symptom clusters) represent psychometrically valid variables.
3. The controlled and semiautomatic stress responses form cognitive, social/motivational, and emotional systems.

Confirmatory Factor Analysis of the How I Cope Under Pressure Scale (Figure 1 and 2)

In order to find out whether the selected controlled stress responses (coping strategies) represented psychometrically valid variables, this study fitted its sample covariance matrix to the established psychometric structure of the HICUPS. To accomplish this, a confirmatory factor analysis was employed. Confirmatory factor analysis is a statistical technique belonging to the family of structural equation models. It relies on mathematical equations to describe hypothetical states of affairs proposed by researchers. The equations are in the family of linear models which job is to estimate population parameters that are able to reproduce the sample covariance matrix (an estimate of the population covariance matrix). Indeed, confirmatory factor analysis (CFA) is equivalent to ordinary regression analysis in that potentially correlated independent variable scores (unobserved/latent or factor scores) and associated weights (beta coefficients) are used to reproduce ("explain or predict") the dependent variable scores (observed/manifest or indicator scores). In CFA, the potentially correlated independent variable (factor) scores reflect potentially correlated factor scores called exogenous variables. Notice that in the same manner that ordinary regression analysis does not specify causal relationships between independent variable scores, CFA does not examine causal relationships between factor scores (nor interaction effects). However the main difference between the two techniques is that both observed independent and

dependent variables in the regression model become dependent or endogenous variables in the CFA model. In this case, factors (factor scores) seek to reproduce ("explain or predict") all observed variable scores. Lastly, associated regression weights (loadings) specify the portion of the standard deviation (or variance) of all observed variables overlapping with ("accounted for") the factors (factor scores) once the effects of other factors (latent variables) and errors are removed. It is easy to see how CFA involves the usual linear parameters. In the case of confirmatory factor analysis, factor score covariances, "factor loadings" (regression coefficients in the factor pattern), and error of measurement covariances (random error, systematic error, and unique variance) are the main guessed parameters. The technique answers the following question: given that one does not have observed (measured) scores or associated weights, how can one estimate the best population scores (and thus factor score covariances) and weights (factor loadings) that reproduce the population covariance represented by the sample covariance? This is all in theory; in practice no factor scores are usually calculated.

Confirmatory factor analysis computes multiple derivatives of all parameters with the objective of finding the set that most likely reproduces the sample covariance matrix. This set of parameters generates a model-implied covariance matrix whose aim is to reproduce the sample covariance matrix. This value is found whenever a minimization function finds a set that maximally decreases the discrepancy between the model-implied and the sample covariance. This function therefore produces a maximum likelihood minimum value. It in effect says, "Given the sample covariance you sampled, I have found the estimates of population parameters that most likely reproduce the following population covariance matrix. Now, this is how well this reproduced population

covariance matrix approximates (fits) the sample covariance matrix that you believe represents the population covariance matrix".

Ayers et al. (1996) tested the psychometric properties of the How I Cope Under Pressure Scale using Cronbach's internal consistency alpha coefficients and confirmatory factor analyses. They did this to ultimately confirm the fit between the data (observed covariance matrix) and three models of coping (problem- focused versus emotion-focused; active versus passive; and problem-focused versus emotion-focused versus distraction versus avoidant versus support-seeking). Ayers et al. created 11 parcels (subscales or groups of items) consisting of 3 to 5 items each. They then conducted 11 separate Cronbach analyses. The results suggested that all subscales exhibited acceptable internal consistencies except for the one that measured the type of coping strategy called expressing feelings (alpha = 34). The researchers subsequently conducted 10 separate confirmatory factor analyses one on each coping type covariance matrix representing relationships between 4 to 5 items. The CFAs demonstrated good fit of the 10 model-implied covariance matrices to the 10 sample covariance matrices. Ayers et al. subsequently used the 10 parcels (subscales or group of items) as observed, endogenous (dependent) variables and hypothesized that five unobserved, exogenous latent variables (factors) would reasonably reproduce their covariance matrix. The model did not fit well, so the researchers reconsidered their theoretical framework by postulating the existence of 4 factors instead. This model fit the data better compared to two other 2 primary factor models.

It is a mystery why Ayers et al. did not conduct one confirmatory factor analysis specifying the 11 parcels as first order factors and the 4 supraordinate classes of coping

as second order factors. One can only speculate that they perceived their sample size to be too small (n = 237) to carry out such confirmatory analysis all at once. However, the to be proposed method is superior to Ayers et al. for two reasons: (a) it accounts for variance not taken into consideration in the Ayers et al.'s example, (b) represents the model better, and most importantly, (c) factor experts, such as Gorsuch (1983; p. 153), do not recommend the computation of multiple confirmatory factor analyses, as this practice entails the same problems stemming from carrying out several t tests (e.g., capitalizing on chance). For these reasons, in addition to their model, this study conducted one confirmatory factor analysis that included all items constituting the 11 parcels (subscales), hypothesized primary factors (subordinate categories of coping), and second order factors (supraordinate classes of coping).

The preceding approach had one weakness, however: only complete data could be used to arrive at a maximum likelihood solution unless a mean-intercept model was proposed. The mean-intercept model, however, is not as reliable as the straightforward maximum likelihood solution carried out with the complete data. This resulted in 97 cases being included in the analyses possibly increasing the instability of the observed correlation matrix. Ayers et al.'s model (Figure. 1) was run first yielding an inadmissible solution due to a reproduced negative error variance associated with the active avoidance subscale. This was then set to 0 meaning that the variable was considered to be measured without error and allowed the analysis to generate an admissible solution. The study's model (Figure 2.), on the other hand, readily produced an admissible solution. This is surprising because complex models represented by a greater number of estimated parameters (90 in our case) and based on small samples tend to (a) be empirically

unidentified or (b) generate reproduced matrices characterized as being conceptually impossible (e.g., negative variances or correlations greater than 1) or nonpositive definite. Nevertheless, both models offered room for improvement in that they did not fit the data well—Ayers et al.'s: $\chi 2(30, n = 97) = 57.26, p < .002$ versus study: $\chi 2(945, n = 97) = 1736.38, p < .000$. Given that these are not hierarchically nested models they could not directly compared. However, model 1 (Ayers et al.'s) produced a model-implied covariance matrix that was much more discrepant (*Comparative Fit Index* = .946) with respect to an independence model than model 2 (*Comparative Fit Index* = .508). Yet, model 2 did a much better job at approximating the values of its covariance matrix to the population covariance matrix (*Root Mean Square Error of Approximation* = .093, *Confidence Interval Lower bound* = .086, *Confidence Interval Upper bound* = .10) compared to model 1 (*Root Mean Square Error of Approximation* = .097, *Confidence Interval Lower bound* = .058, *Confidence Interval Upper bound* = .14).

The preceding findings are not surprising given that this study's model is much more complex and is bound to fit the data less well compared with the much simpler Ayers et al.'s model. Indeed, Ayers et al. reported a $\chi 2(29, n = 247) = 82.77, p < .001$ and an identical *CFI* = .96 in their study. However, given that their model (a) capitalized on chance error), (b) yielded an inadmissible solution on its first run, (c) produced a higher Root Mean Square Error of Approximation, and (d) did not include the expressing feelings subscale, this study decided to trust its findings. This decision is further supported by the observation that the investigation is interested in how well the subscales (parcels or group of items) represent their respective constructs, not on how well they are represented by higher order factors. With respect to 10 subscales, both studies

demonstrated (a) good internal consistencies, (b) parameter estimates that made sense (e.g., observed variables regression weights), (c) statistically significant parameter estimates, and (d) reasonable standard error associated with the parameters.

Confirmatory Factor Analysis of the Diagnostic Interview for Children and Adolescents-PTSD module

To determine whether the specified semiautomatic stress responses (PTSD symptom clusters) represented psychometrically valid variables, the study intended to run a confirmatory factor analysis consisting of seventeen indicators and four factors representing the reexperiencing, numbing, hyperarousal, and active avoidance clusters. Since the collected data took the form of binary categories (symptom absent = 0; symptom present = 1), its variance/covariance matrix required a different parameter estimation method. In this case, weighted least square parameter estimation is preferred over maximum likelihood because matrices derived from binary categories attenuate the magnitude of the variance/covariance elements to be decomposed. Unfortunately, this method necessitates the use of large samples with the minimum number of cases being $p(p + 1)/2$, where p equals the number of observed variables. In our analysis, p = 17 resulting in 17(18)/2 = 153 minimum number of cases to include. Because his type of analysis demands the exclusion of missing cases, it resulted in 112 cases, not enough to meet the 153 threshold. For these reasons, the most reasonable approach at this point was to trust the results of past confirmatory factor analyses studies indicating that a four factor solution better represents the psychometric structure of PTSD.

Confirmatory Factor Analysis Validating the System Hypothesis (Figure 3, 4, and 5)

To determine whether the controlled and semiautomatic stress responses (6 HICUPS and 3 DICA latent variables) formed cognitive, social/motivational, and emotional systems the study took the following approach. It ran a confirmatory factor analysis consisting of three factors (independent latent variables) representing the cognitive, social/motivational, and emotional domain and their corresponding controlled and semiautomatic stress responses (dependent observed variables). This analysis yielded three findings: (a) a poor fit of the model, (b) all controlled stress responses loaded well on their factors, and (c) the cognitive and emotional semiautomatic stress responses did not significantly load on their respective factors (reexperiencing = .06 and arousal = .02) (Figure 3). At first sight this suggests that the semiautomatic stress responses are not part of the same system as the controlled stress responses and the data validated this hunch. A second factor analysis that included a general factor (independent latent variable) and the nine controlled and semiautomatic stress responses once more indicated that reexperiencing and arousal were still not explained by this general factor (Figure 4). That is, their "factor loadings" were not significant. However, when all controlled stress responses were excluded, the general factor (to be called semiautomatic stress response factor) "accounted for" the variance in reexperiencing, numbing, and arousal (Figure 5). All parameters were significant in this case. In retrospect this is expected because even though controlled and semiautomatic stress responses belong to the same domain, they are obviously different given that they represent two distinct types of processes. Adding support to this assertion is the fact that the to be presented path analyses findings suggested some statistically significant relationships between controlled and

semiautomatic stress responses despite the fact that the confirmatory factor analyses suggested that they belonged to different groups.

Path Analyses

Rationale. The technique known as path analysis was chosen because it forces the investigator to clearly express its theoretical framework and accompanying hypotheses. This technique belongs to the family of structural equation models in which observed indicator scores act as exogenous and endogenous variables. Statistically, it is a series of regression analyses where the endogenous (dependent) variables are regressed onto exogenous (independent) or other endogenous variables (dependent). As such, it requires that the researcher meet all of the assumptions expected of regression analysis. Regression analysis, however, only specifies direct (main) or interactional effects of exogenous/independent on endogenous/dependent variables leaving the relationship between the independent/exogenous variables unexplained. Path analysis further decomposes the elements in a variance/covariance matrix representing associations between all exogenous and endogenous variables into direct, indirect, spurious, and unanalyzed effects. In this case, what used to be an exogenous/independent variable in the ordinary regression model can become an endogenous or combination of endogenous and exogenous variable. What is important to keep in mind about path analysis is that it partitions the covariance among variables into finer parts. For instance, suppose variables a, b, and c covary with each other. Take variable a to be the exogenous variable which affects variable b and c (spurious effect between b and c). Furthermore, suppose variable b also affects variable c. This means that variable a covaries with variable c and that possibly variable a also covaries with variable b and c. In this case, the covariance

between variable a and c is can be partitioned into that variance that overlaps between a and c (direct main effect) and the variance that overlaps between a, b, and c (indirect mediating effect).

Direct (main), indirect (mediated), spurious (confounding), and unanalyzed (error) effects are expressed in the form of path regression beta coefficients. As such, a path coefficient serves the same function as a beta coefficient: it expresses a ratio of the covariance between two variables over the variance of one of the variables. Stated differently, a path coefficient expresses the amount of partitioned variance it accounts for in an endogenous/dependent variable when all other relevant variables in the model are held constant. Unanalyzed (correlational) effects are correlation coefficients the researcher does not wish to decompose because he or she does not have a clear idea how to do so. That is, the researcher does not have an informed hypothesis in this case.

The second purpose of a path analysis is model testing. A path analysis is a causal model that in effect makes a hypothesis regarding the correlation matrix. It in effect attempts to reproduce the observed correlation matrix obtained from the collected data with its hypothesized correlation matrix derived from the specifications in the model. This allows for a test of the causal model hypothesis via a computation of the difference between the elements in the observed minus the elements in the hypothesized coefficient matrix. This difference is part of a chi-square analysis conducted to assess the magnitude of the difference between the observed and hypothesized matrices elements. The closer the elements are to each other, the less likely the chi-square will be significant, the better the fit between the hypothetical matrix and the observed one. This test is only a preliminary test as it is most often statistically significant when large samples are used.

One of the main assumptions of path analysis is that the variables are measured without error. This is highly unlikely in social research, however. Thus, researchers instead resort to the use of hybrid structural equation modeling to obtain measurement ("true" plus "error") and structural model parameters in the same analysis. Unfortunately, hybrid structural equation models require the inclusion of only continuous variables and sample sizes larger than the one available in the present study. Nevertheless, an estimate of the measurement error variance accounted for by variables outside of the model can be obtained from the current data by taking the squared root of the squared generalized correlation coefficient minus one. This residual variance represents the operation of variables (random, systematic, or independent) not specified in the current analysis.

To recapitulate, in this study the causal model proposes that some activating coping strategies (seeking understanding, avoidant actions, and expressing feelings) will independently contribute to increases in respective PTSD symptom clusters (reexperiencing, numbing, and hyperarousal). IT further suggests that other deactivating coping strategies (positive cognitive reappraisal, emotion-focused support, and physical release of emotions) will contribute to decreases in corresponding PTSD symptom clusters. Lastly, the model claims that activating coping strategies positively covary with deactivating coping strategies and through these indirectly affect PTSD symptom clusters.

Data Screening and Preparation. To investigate the possible confounding effects of the demographic variables on the interpretation of the path analyses, a multivariate analysis of variance was performed on the data with age (children [8-12] versus adolescents [12-18]), ethnicity (white versus black versus other), and gender as

independent variables and the six coping strategies and the three PTSD clusters as dependent measures. These analyses in essence test whether the demographic variables had moderating effects on dependent variables. If this were the case, separate path analyses would have to be conducted for whatever demographic variable yielded significant differences across coping strategies or PTSD symptom clusters. Fortunately, the multivariate analysis yielded no significant omnibus main or interaction effects of the demographic variables on the coping strategies or the PTSD symptom clusters. This allowed us to collapse the data across gender, age, and ethnicity.

With respect to the matrices used in the actual path analyses, because the number of missing cases across PTSD and coping variables casewise deletion was implemented leaving sixty-seven cases with complete data on all variables. No multivariate moderating analysis could be done on such a small data set, unfortunately. However, the casewise deletion procedure had to be implemented because missing values result in cells of different sizes, unstable correlations, and biased standard errors of estimates. The author decided that parameter estimate instability was a worse risk to take than superfluous moderating effects. Besides, the study's focus was not on the relationship between demographic variables, coping, and PTSD. Still, because it was possible that the complete and shortened data sets were different, the researcher conducted all analyses on both. This method surprisingly led to equivalent solutions characterized by fit tests and parameters of similar magnitudes and levels of statistical significance. Due to the fact that the sample size was still adequate in the "cleaned" data (15 cases per independent variable) and offered advantages over the missing cases matrices, it was reasonable to present the results yielded by "cleaned" data set. All summary statistics describing the

resulting sample are displayed in Table 1. No significant outliers were found in the data and the multivariate shape of the distribution of variables varied from slightly skewed to normal.

Hypotheses Results

Cognitive Controlled Stress Responses Increase and Decrease Cognitive Semiautomatic Stress Responses

Independence Model Hypothesis 4.1. Since the independence model is overidentified, it allows tests of its fit. The model reproduced the sample correlation matrix poorly, $\chi 2(1, n = 67) = 30.12$, $p < ,000$, *CFI* = .078, *RMSEA* = .66, (*Low* = .47, *High*, .89). This suggests that there is still much unexplained variance to be "accounted for". In terms of parameters, the model yielded a main effect of the deactivating cognitive controlled stress response positive cognitive restructuring on the cognitive semiautomatic stress response total number of reexperiencing symptoms (*b* = .57, *S.E.* = .21, C.R. = 2.67, *p* = .00). This effect was nevertheless in the opposite direction. Additionally, a non-significant main effect of the activator seeking understanding on reexperiencing symptoms emerged in this model (*b* = -.24, *S.E.* = .19, *C.R.* = -1.30, p = .19).

Ordinary Regression Model Hypothesis 5.1. Unlike the previous model, the ordinary regression model is just-identified providing no reason to test its fit. As expected, in terms of its parameters, this model replicated the independence model's findings. Whereas the deactivator positive cognitive restructuring reached a statistically significant positive in direction main effect on reexperiencing symptoms (*b* = .57, *S.E.* = .27, *C.R.* = 2.12, *p* = .03) seeking understanding did not (*b* = -.24, *S.E.* = .23, *C.R.* = -1.03, *p* = .30). The model added, however, another piece of information. It suggested a

statistically significant moderate in strength and positive in direction correlation between the activator seeking understanding and the deactivator positive cognitive restructuring (r = .35, *S.E.* = .08, *C.R.* = 4.21, p =.00).

Moderating/Interactional Model Hypothesis 6.1. The study carried out three steps in the case of the moderating/interactional model. First, it computed a moderating vector by multiplying the scores on the positive cognitive restructuring subscale by those on the seeking understanding subscale. Second, it conducted a regression analysis that included the two main effect terms plus the moderating one. This did not yield significant results for the main effects—(a) seeking understanding: b = -.27, *S.E.* = .70, t = -.39, p = .70, and (b) positive cognitive restructuring: b = .54 *S.E.* = .65, t = -.82, p = .42)—nor for the interaction effect, (c) seeking understanding by positive cognitive restructuring: b = .01, *S.E.* = .25, t = 0.49, p = .96. Third, to make sure that these results were not due to possible multicollinearity between the main effect and the interaction vectors, the study included only the interaction effect in a second analysis. The interaction effect again did not reach statistical significance, b = .05, *S.E.* = .05, t = 1.08, p = .28. Moreover, replicating the results of the ordinary regression model, only a positive in direction main effect of positive cognitive restructuring on total number of reexperiencing symptoms emerged, b = .57, *S.E.* = .27, t = 2.09, p = .04, when this was analyzed in conjunction with the seeking understanding term only.

Mediating Model Hypothesis 7.1. (Figure 6). This model generated a positive in direction main effect of the deactivator positive cognitive restructuring on total number of reexperiencing symptoms (b = .57, *S.E.* = .27, *C.R.* = 2.12, p = .03). There was also a positive in direction main effect of the activating cognitive controlled stress response

seeking understanding on the deactivator positive cognitive restructuring (b = .52, *S.E.* = .09, *C.R.* = 6.18, p = .00). No direct effects of seeking understanding on total number of reexperiencing symptoms emerged (b = -.24, *S.E.* = .23, *C.R.* = -1.03, p = .30).

Summary. One consistent result arises from the preceding analyses. The cognitive deactivating controlled stress response positive cognitive restructuring was the only cognitive coping strategy that influenced the cognitive semiautomatic stress response total number of reexperiencing symptoms. Contrary to the hypothesis, however, increases in this controlled stress response accompanied the activation resulted in corresponding increases in the semiautomatic stress response. In the same vein, the cognitive activating controlled stress response seeking understanding indirectly influenced the number of reexperiencing symptoms by affecting the cognitive deactivator positive cognitive restructuring, albeit not as expected. However, the hypothesis that increases in this cognitive coping strategy would accompany increases in total number of reexperiencing PTSD symptoms must be rejected.

Social/Motivational Controlled Stress Responses Increase and Decrease Social/Motivational Semiautomatic Stress Responses

Independence Model Hypothesis 4.2. The independence model reproduced the sample correlation matrix poorly, $\chi 2(1, n = 67) = 6.99$, $p <$,008, *CFI* = .342, *RMSEA* = .30, (*Low* = .12, *High* = .53). With regard to the parameters of interest, there was a positive in direction main effect of the activating social/motivational controlled stress response avoidant actions on the total number of numbing symptoms, (b = .52, *S.E.* = .11, *C.R.* = 2.42, p = .02). However, the direct main effect of the social/motivational

deactivator emotion-focused support on numbing symptoms did not emerge, (b = -.06, *S.E.* = .11, *C.R.* = -.54, p = .59).

Ordinary Regression Model Hypothesis 5.2. As expected, the ordinary regression model replicated the preceding effects (avoidant actions on numbing symptoms: b = .52, *S.E.* = .11, *C.R.* = 2.42, p = .02; emotion-focused support on numbing symptoms: b = -.06, *S.E.* = .11, *C.R.* = -.54, p = .59). Beyond this, the model indicated the existence of a statistically significant weak positive correlation between the social/motivational activator avoidant actions and the deactivator emotion-focused support (r = .20, *S.E.* = .08, *C.R.* = 2.46, p =.01).

Moderating/Interactional Model Hypothesis 6.2. The moderating model did not generate any main or interaction effects (avoidant actions on numbing symptoms: b = .233, *S.E.* = .32, t = .75, p = .46; emotion-focused support on numbing symptoms: b = -.09, *S.E.* = .39, t = -.22, p = .82; avoidant actions by emotion-focused support on numbing symptoms: b = .01, *S.E.* = .14, t = .07, p = .95). The study tested and did not confirm the multicollinearity hypothesis.

Mediating Model Hypothesis 7.2 (Figure 7). The mediating model yielded a positive in direction main effect of the social/motivational activator avoidant actions on both numbing symptoms (b = .25, *S.E.* = .11, *C.R.* = 2.29, p = .02) and the social/motivational deactivator emotion-focused support (b = .29, *S.E.* = .11, *C.R.* = 2.72, p = .01). Unfortunately, the deactivator emotion-focused support did not have a direct main effect on numbing symptoms (b = -.06, *S.E.* = .12, *C.R.* = -.51, p = .61).

Summary. The preceding findings confirm the hypothesis that the social/motivational activating controlled stress response avoidant actions would “account

for" increases in the social/motivational semiautomatic stress response numbing PTSD symptoms. However, the hypothesis that the social/motivational deactivator emotion-focused support would reduce the number of numbing symptoms must be rejected. Lastly, although increases in the social/motivational activator avoidant actions corresponded to increases in the social/motivational deactivator emotion-focused support, the former did not have an indirect mediated effect on numbing symptoms because the latter did not influence these symptoms.

Emotional Controlled Stress Responses Increase and Decrease Emotional Semiautomatic Stress Responses

All Models: Neither the Independence Model (hypothesis 4.3), nor the ordinary regression model (hypothesis 5.3), nor the moderating/interactional model (hypothesis 6.3), nor the mediating model (hypothesis 7.3; Figure 8) generated direct (main), indirect (mediated), or interactional (moderated) effects. For instance, in the mediating model the direct main effect of the emotional activating controlled stress response expressing feelings on the emotional semiautomatic stress response total number of arousal symptoms was characterized by the following parameters: b = .03, *S.E.* = .13, *C.R.* = .20, p = .61. With regard to the emotional deactivating controlled stress response physical release of emotion the parameters were: b = -.04, *S.E.* = .13, *C.R.* = -.28, p = .78. Only the main direct effect of the emotional activating controlled stress response expressing feelings on the emotional deactivating controlled stress response physical release of emotion came close to achieving statistical significance: b = .22, *S.E.* = .12, *C.R.* = 1.81, p = .07).

Summary. All hypotheses proposed concerning the relationship between emotional activating and deactivating controlled stress responses and emotional semiautomatic stress responses must be rejected.

Follow up Statistical Analyses

Given the unfruitful results yielded by the data, it is reasonable to further analyze it to rule out certain possibilities. It is plausible that the results are a consequence of the regulatory hypothesis' main premises. For instance, the hypothesis states that certain controlled stress responses increase corresponding semiautomatic stress responses whereas others decrease them. This implies that the workings of the components constituting the cognitive, social/motivational, and emotional systems proposed by the regulatory hypothesis should be observed most clearly in that group of individuals with the highest number of symptoms. As such, it stands to reason that data collected on individuals diagnosed with PTSD should yield different results in comparison to data obtained from individuals without this diagnosis. This observation motivates the question of different in what way? In the present case, the difference in the degree of linear association between the activating and deactivating controlled stress responses and the semiautomatic stress responses within a given system will be tested in the PTSD and the No PTSD groups with the expectation that the PTSD group will be higher.

The test of the above hypothesis entails comparing a 3 x 3 correlation matrix from one population against another 3 x 3 matrix representing a different population. In our case, we will be testing three such pairs of correlation matrices each pair representing the effects of the components of a system in the PTSD and No PTSD populations (See Table 2.). To do this, the mistake is often made to test pairs of correlation coefficients one at a

time. This procedure capitalizes on chance stemming from experiment-wise error. Instead, a statistical method that tests omnibus hypotheses that include all concerning correlation coefficients is required. Steiger (2004) developed the theory behind such method for testing pattern hypotheses of correlation coefficients derived from independent samples. In our case, the test will analyze differences between each of three elements contained in each pair of 3 x 3 correlation matrices representing the cognitive, social/motivational, and emotional system. To illustrate using the cognitive system, the test will all at the same time test the following hypotheses: (a) correlation coefficient between the activating controlled stress response seeking understanding and the semiautomatic stress response reexperiencing symptoms will be the same in the PTSD and the no PTSD group, (b) correlation coefficient between the deactivating controlled stress response positive cognitive restructuring and the semiautomatic stress response reexperiencing symptoms will be the same in the PTSD and the no PTSD group, and (c) correlation coefficient between the activating controlled stress response seeking understanding and the deactivating controlled stress response positive cognitive restructuring will be the same in the PTSD and the no PTSD group.

Besides the statistical theory, Steiger also created a computer module compatible with Mathematica capable of testing correlation pattern hypotheses in seconds, WBCORR (Within and Between Correlations). This program was used to investigate whether the null results yielded by the path analyses were a consequence of differences in the number of symptoms reported by the PTSD and the No PTSD groups. The procedure was the following: data from a pair of 3 x 3 correlation matrices was input using the computer language of Mathematica, (b) Ordinary Least Square Estimates of the

population parameters calculated, (c) 2-State Generalized Least Square estimates of the difference between population parameters computed, and (d) an omnibus Chi Square significance test indicating whether there were statistical differences between the estimates of population correlation coefficients. Unfortunately, the results were consistent with the path analyses' findings. Neither the cognitive, social/motivational, nor emotional systems yielded significant Chi Square test statistics [$\chi 2(3) = 1.27$, $p > .05$, $\chi 2(3) = 2.95$, $p > .05$, $\chi 2(3) = 5.15$, $p > .05$, respectively]. Although disappointing, these findings add to the satisfaction that comes from being certain about the results, even null results.

Discussion

All in all, this study offered partial support for only one hypothesis. As such, the assertion that cognitive coping regulatory systems exist (hypothesis 1) must be discarded, revised, or further studied. Consistent with this statement, the activating cognitive controlled stress response seeking understanding did not reflect associated increases in the cognitive semiautomatic stress response reexperiencing symptoms. It did, however, have a mediated effect on reexperiencing symptoms. Nevertheless, the mediated effect suggested that the deactivating cognitive controlled stress response positive cognitive restructuring increased, rather than decreased, its target semiautomatic stress response reexperiencing symptoms. This is obviously inconsistent with the prediction that cognitive deactivating coping strategies decrease PTSD symptoms. In concrete terms, the finding suggests that children and adolescents who cope cognitively with traumatic events by making sense of it also make efforts to recast it in positive terms. However, the more the traumatic material is cognitively processed in this manner, the more the number of reexperiencing PTSD symptoms that emerge. This is a counterintuitive finding that

perhaps can be explained by examining the items making up this variable. They were the following: (a) tried to notice or think about only the good things in life, (b) told myself it would be over in a short term, (c) reminded myself that things could be worse, and (d) told myself it is not worth getting upset about. Upon closer examination these statements seem to encapsulate cognitive distraction strategies that do not directly deal with the traumatic material. Seen in this new light, it is not surprising that the effect of positive cognitive restructuring on reexperiencing PTSD symptoms is different from that which the author conceived. Since the variable does not indicate direct cognitive restructuring of traumatic material, it represents a possible model misspecification error on his part.

With regard to the assertion that social/motivational coping regulatory systems exist (hypothesis 2), this too must be discarded, reviewed, or further investigated. In this case, the activating social/motivational controlled stress response avoidant actions exhibited associated increases in the social/motivational semiautomatic stress response numbing symptoms as expected. Additionally, this controlled stress response was associated with increases in the deactivating social/motivational controlled stress response emotion-focused support. Unfortunately, the fact that this deactivating controlled stress response was not associated with changes in numbing symptoms invalidated a portion of the hypothesis. To refresh the memory, avoidant actions constituted the following activities: (a) tried to stay away from the problem, (b) tried to stay away from things that made me feel upset, (c) avoided the people who make me feel bad, and (d) avoided it by going to my room. In the same vein, emotion-focused support encompassed the following actions: (a) talked about how I was feeling with my mother or father, (b) talked about how I was feeling with some adult who is not in my family, (c)

talked with my brother or sister about my feelings, and (d) talked with one of my friends about my feelings. This means that children and adolescents reported avoiding the traumatic material but not their emotions. Furthermore, the more they reported avoiding coming into contact with the traumatic material, the more they focused on talking about their feelings with people. Therefore, it is possible that the participants found facing or talking about the traumatic material more difficult than focusing on something safer like their feelings, for feelings are always there but traumatic events are unusual. Thus, when it comes to children and adolescents it may be best to increase their social participation by helping them deal with their emotions. This seems a reasonable proposal if it were not for the fact that their numbing symptoms did not ameliorate as they talked about their feelings with others. Therefore, it is safe to say that activating children and adolescent's appetitive social/motivational systems so that their numbing symptoms decrease by having them interact with individuals they view as safe or trustworthy is not enough. It is perhaps more effective activating their appetitive social/motivational systems by having them interact with trauma related individuals.

Lastly, no support at all was found for the claim that emotional coping regulatory systems exist (hypothesis 3). Neither the activating (expressing feelings) nor the deactivating (physical release of emotions) emotional controlled stress responses had an effect on its respective semiautomatic stress response (arousal symptoms). This assertion must clearly be discarded. Given that the arousal cluster represents vegetative symptoms typical of emotional states (e.g., difficulty falling or staying asleep, irritability, hypervigilance, and exaggerated startle response) it is perplexing that neither expressing feelings or physically releasing emotional charge was in any manner related to these

vegetative manifestations. Therefore, out of all systems, the emotional one needs the most conceptually rigorous work.

Data-Based Speculations

Looking at the pattern of results the following is observed. A controlled stress response with a positive sign and another with a negative sign emerged even though some of the beta coefficients were small in magnitude and nonsignificant in each of the three coping systems. The fact that the study included three data points (covariance matrices) whose analyses yielded similar coefficient patterns could be a consequence of random chance. However, it could just as well suggest a trend in the right direction. The small nonsignificant coefficients could have been a result of the content of the HICUPS items. That is, it is reasonable to speculate that the six groups of statements standing for the activating and deactivating controlled stress responses may not be the best representatives of cognitive, social/motivational, and emotional coping strategies. Indeed, these six types of coping strategies were not the focus of analysis of Ayers et al., so their names may not correspond to their content. However, the preliminary confirmatory factor analyses conducted at the beginning of the result section offers some evidence that this was not the case. That is, the psychometric validation of the system hypotheses suggest that seeking understanding, positive cognitive restructuring, avoidant actions, emotion-focused support, expressing feelings, and physical release of emotions loaded well on their respective cognitive, social/motivational, and emotional domains. What is reasonable to propose is that the content of the items constituting these six factors did not reflect the author's thinking. That is, although they possessed face validity as indicated by their

variable labels and construct validity as suggested by the CFA results, they nevertheless lacked content validity.

From the preceding observations it is imperative to suggest that researchers spend more time thinking about the nature of activating and deactivating controlled stress responses and semiautomatic stress responses. For instance, what was theorized to be an activating controlled stress response in the case of the cognitive regulatory coping system turned out to be a potential deactivating one. More specifically, the sign of the nonsignificant beta coefficient associated with the effects of the cognitive activator seeking understanding on total reexperiencing symptoms was negative (-.24). A statistically sophisticated researcher could say that this may be due to the nature of the model; that is, to the interaction of this variable with others. However, this phenomenon emerged across all models (independence, regression, moderating, and mediating). Therefore, keeping in mind that one of the beta coefficients was nonsignificant, it seems reasonable to speculate that the study mispecified the cognitive regulatory coping system model. In the same vein, with the exception that it was statistically significant, the converse was true of the purported deactivating response positive cognitive restructuring: its coefficient was positive (.57). The proposed direction, but no the magnitude, of the others activating and deactivating controlled stress responses did follow the theoretical framework's claims.

With regard to the social/motivational regulatory coping system, the effect coefficient summarizing the relationship between the activating social/motivational controlled stress response avoidant actions and total number of numbing symptoms was statistically significant and positive in direction (.25). With the exception that is

nonsignificant, the converse was true of the deactivating social/motivational controlled stress response emotion-focused support: a nonsignificant coefficient negative in direction (-.06).

Lastly, although their coefficients were nonsignificant, the components of the emotional regulatory coping system followed the preceding trends (expressing feelings on total number of arousal symptoms = nonsignificant and positive, .03; physical release of emotions on total number of arousal symptoms = nonsignificant and negative, -.04). The preceding speculations suggest that there may be cognitive, social/motivational, and emotional regulatory coping systems that displayed patterns consistent with the structural and process aspects of the regulatory hypothesis.

One last pattern emerged that was consistent with the process aspect of the regulatory hypothesis. In every case, the activating controlled stress response accompanied increases in the deactivating controlled stress response. The direction of this effect is still a speculation given that it may take field and experimental studies to validate it as the analyses of covariance matrices obviously cannot do so. As the process hypothesis indicates, these two variables may prime each other through spread of activation. Therefore, it is reasonable to suggest that whichever occurs first may trigger the other. However, the theoretical framework clearly states that with regard to the optimal regulation of PTSD symptoms, individuals most likely first resort to the use of activating controlled stress responses to bring out traumatic material and then apply deactivating controlled stress responses to modify and neutralize it.

Theoretical Speculations

Conceptualizing coping as a group of static, non-interacting strategies may lead to overlooking its complexity. Instead, the effect coping has on PTSD symptoms may be best understood by clearly delineating the differential and interactional paths and outcomes that various coping strategies may lead to with regard to the disorder. This is of great importance when dealing with a multifaceted disorder that seems to present a variety of distinct problems, some cognitive, others social/motivational, and still others emotional in nature. Unfortunately, PTSD is most often treated as a unitary construct as opposed to an abstract entity formed from a multifarious pool of symptomatic evidence. Currently Posttraumatic Stress Disorder exists as a conceptual entity, not an operation that must subsequently be translated to operationalizations at the symptom level. It is at this level that the interaction between the disorder and coping strategies would result in the most fruitful findings. The present study attempted to move down a level of analysis to the symptom cluster step in a first attempt at understanding how coping regulates PTSD symptomatology. Finally, the same must be said of coping. Clumping together various coping strategies into groups would yield less information and a more blurred picture of the relationship between coping and PTSD than moving down to a more specific, concrete level of coping analysis.

Because each of the problems PTSD introduces to the lives of children and adolescents is different in nature, various coping strategies must be used to manage them effectively. As an example, it would be counterproductive to deal with the cognitive problems included in the reexperiencing cluster of PTSD by physically releasing emotions. Even if there was a short-term decrease in symptoms, it is most likely that the

intrusive cognitive symptoms will not go away. Instead, it is reasonable to argue that intellectually evaluating one's trauma-related thoughts, appraisals, and perceptions would most likely result in the decrease of maladaptive thoughts as adaptive cognitions increase. Indeed, this is what cognitive processing therapies are supposed to do: challenge and replace unrealistic cognitions with realistic ones by making children and adolescents think about the trauma. Thus, PTSD seems to be a disorder that must be targeted at different levels by resorting to different coping strategies. However, each coping strategy takes the form of an intervention that best decreases a particular group of symptoms. A cognitive group of symptoms require a cognitive coping intervention, a social/motivational class a social/motivational one, and an emotional cluster an emotion-based coping intervention.

In sum, groups of coping strategies may best be regarded as systems that regulate PTSD by increasing and decreasing levels of PTSD symptomatology and eventually bring organisms back to a homeostatic state. These systems may be the standard way of decreasing PTSD. This is in line with the claim that "resilience (not recovery) is the most common response to potential trauma (Bonanno, 2005). Thus, perhaps the great majority of young people naturally engage in self-administered coping interventions that lead to the prevention or reduction of PTSD symptoms. However, sampling these coping interventions and studying them separately via the use of multiple regression analyses would only lead to at best an incomplete picture of how coping strategies regulate PTSD symptoms. This is perhaps responsible for the multitude of inconsistent findings in the literature with regard to how coping affects PTSD symptoms. It would also clear paradoxes such as why certain studies show that PTSD symptoms are increased by so-

called adaptive strategies like active/approach coping and at other times decreased or why supposedly maladaptive techniques such as avoidant coping are sometimes the best solution to PTSD. Hence, path analytical or structural equation models that present a fuller picture of the role of various coping strategies on specific types of PTSD symptoms may eventually pay off.

Practical Implications

Understanding how various coping strategies lead to mental homeostasis in children and adolescents is clearly important for the development of PTSD interventions. Much can be learned from observing processes in natural environments. This is because they can inform the development of interventions by pointing to how PTSD is reduced in "the wild". Understanding how systems of coping strategies operate in real life to increase PTSD resilience would point out to not only beneficial interventions but most importantly detrimental ones. Even of greater importance, this would suggest that interventions that may be thought as detrimental at first might have a healing effect when combined with other interventions. This understanding may furthermore inform clinicians regarding what intervention to use based on what symptoms are present. This is clearly of value given that a great majority of traumatized children and adolescents only present with partial PTSD. Thus, a child who is experiencing only emotional PTSD symptoms may be best treated through the teaching and practice of emotional coping interventions. The clinician may in this case introduce a symptom augmenting emotional coping strategy in conjunction with a diminishing one. Therefore, it is possible that the strategies we have up to now developed to treat PTSD have been naturally and all along

implemented by children and adolescents classified as PTSD-resilient in their natural environments.

Limitations and Future Directions for Research

This study established the factor validity of the How I Cope Under Pressure Scale (HICUPS) Pressure eleven primary and four higher order subscales. It adds to the findings obtained by Ayers et al. concerning the psychometric properties of the HICUPS. Without these advances, the theoretical propositions marshaled in this document could not be given as much credence. All in all, the study offered partial support for only one hypothesis. The findings are not surprising given the exploratory nature of the study and the novelty of the theoretical framework. This endeavor has just begun; thus, it is highly likely that many roadblocks will have to be overcome before we can arrive at a clear picture. Therefore, it is reasonable to propose that further tests will have to be conducted to either modify or completely discard the theoretical framework. Having said this, what can be learned from the results in the meantime?

One of the most pressing limitations of the present research is the lack of compatibility between controlled stress responses and semiautomatic stress responses despite the intentions of the researcher. It is apparent after the fact that the highest degree of compatibility would be reached when the object of the coping strategies is the actual PTSD symptoms. This fact can be illustrated by the following observation. In the present study the object of the coping strategies was the residential fire, not the PTSD symptoms. It is clear that asking participants how they dealt with their experience of the fire is different from asking them how they coped with their PTSD symptoms. This discrepancy

may reasonably attenuate the correlational strength between the selected controlled stress responses and the chosen semiautomatic stress responses.

A second limitation exhibited by the study relates to the use of multiple variables measured at one point in time. The way variables may be deployed in real life may be much different than how they were modeled in the present study. The remedy to this problem would require that researchers conduct in-depth case studies and content analysis of coping strategies and PTSD symptoms to understand the regulatory effects coping exerts on PTSD. At best, however, researchers would benefit from observational, field design studies that can assess how young people cope with PTSD symptoms throughout the course of the day. This type of design would yield a richer picture of how different coping strategies increase, decrease, and stabilize PTSD symptoms. This is an important point, because the time interval most studies rely on to investigate the relationship between coping and PTSD across time is in terms of months as opposed to minutes or hours. For example, predicting whether a coping strategy reduces reexperiencing symptoms after two or three months will obviously give us partial information as to the regulatory nature of coping in relation to PTSD. It is instructive at this point to address an issue that might cause confusion in the future regarding the temporal aspects of the regulatory hypothesis. It is assumed that the nature of regulatory systems change across time. As an illustration, it is not reasonable to propose that cognitive, social/motivational, and emotional controlled stress responses may be deployed differently four weeks after encountering the event compared to three months after it. However, it makes equal sense to assert that the regulatory hypothesis would offer the most amount of information at the beginning of the disorder because it is at this

point that symptoms are at their peak and individuals are struggling to manage them. Later on in the disorder, the regulatory hypothesis premises are at higher risk to be confounded with other variables. For instance, with regard to emotional regulatory systems, controlled stress responses just as much as habituation may be responsible for decreases in autonomic arousal underlying emotional semiautomatic stress responses. Therefore, investigations of the regulatory hypothesis should benefit more from being conducted during the first phases of the PTSD disorder.

Another limitation concerns the mechanisms underlying the chosen statistical technique. It is a statistical truism that a correlation matrix cannot inform the researcher regarding what the best coping-PTSD causal model may be. This is because various hypothesized causal models may equally reproduce the correlation matrix and, thus, different models may fit the same data. Unfortunately, this can only be best remedied by the introduction of experimental designs that clearly provide evidence regarding the cause and effect relationships between coping strategies and PTSD symptoms.

Lastly, it is clear that there is still room for the further decomposition of PTSD cluster symptoms into individual symptoms and coping strategies into individual strategies. This would produce the greatest amount of information with regard to how coping affects and regulates PTSD symptoms. To do so would of course require much larger sample sizes and perhaps would result in the use of statistical techniques that are less adequate in terms of their measurement models. However, multiple indicators of single symptoms may be created and input into structural equation models in order to have a better picture of both the structural and measurement gains in knowledge regarding coping and PTSD.

Conclusion

The present study represents one of the few attempts at disentangling the regulatory mechanisms whereby given types of coping strategies increase and decrease corresponding PTSD symptoms resulting in the return of children and adolescents to pre-trauma status. It is hoped that future research introduces further improvements to the theoretical, methodological, and statistical modeling proposed in this area of study. As a start, it would be instructive to conduct rich qualitative studies based on in-depth observational studies followed by experimental designs. This would hopefully lead to a clearer picture regarding the relationship between coping and PTSD and perhaps would better inform clinical interventions. Paradoxically, much can be learned from those resilient individuals who never come to see us, but who may in time prove invaluable.

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Table 1

Activating and Deactivating Controlled Stress Responses and Semiautomatic Stress Responses Means, Standard Deviations, and Correlations

	Reex	SU	PCR	Numb	AVA	EFS	Arou	EF	PRE
Mean	.88	2.6	2.65	.37	2.56	2.09	.39	1.75	2.09
SD	1.27	.82	.71	.74	.83	.76	.82	.83	.82
Reex	1.00	.04	.22	**.44**	**.27**	.00	**.40**	-.03	.08
SU		1.00	**.61**	.15	**.53**	**.60**	.00	**.36**	**.35**
PCR			1.00	.17	**.66**	**.34**	-.00	.19	**.43**
Numb				1.00	**.27**	.03	**.31**	-.10	.24
AVA					1.00	**.32**	.20	**.34**	**.30**
EFS						1.00	.01	**.35**	**.25**
Arou							1.00	.02	-.03
EF								1.00	.22
PRE									1.00

Note. Significant correlations appear in bold (two-tailed significant test).

Reex = Reexperiencing; SU = Seeking Understanding; PCR = Positive Cognitive Restructuring; Numb = Numbing; AVA = Avoidant actions; EFS = Emotion-Focused Support; Arou = Arousal; EF = Expressing Feelings; PRE = Physical Release of Emotions.

Table 2

Activating and Deactivating Controlled Stress Responses and Semiautomatic Stress Responses Correlation Coefficients in the PTSD and No PTSD Groups

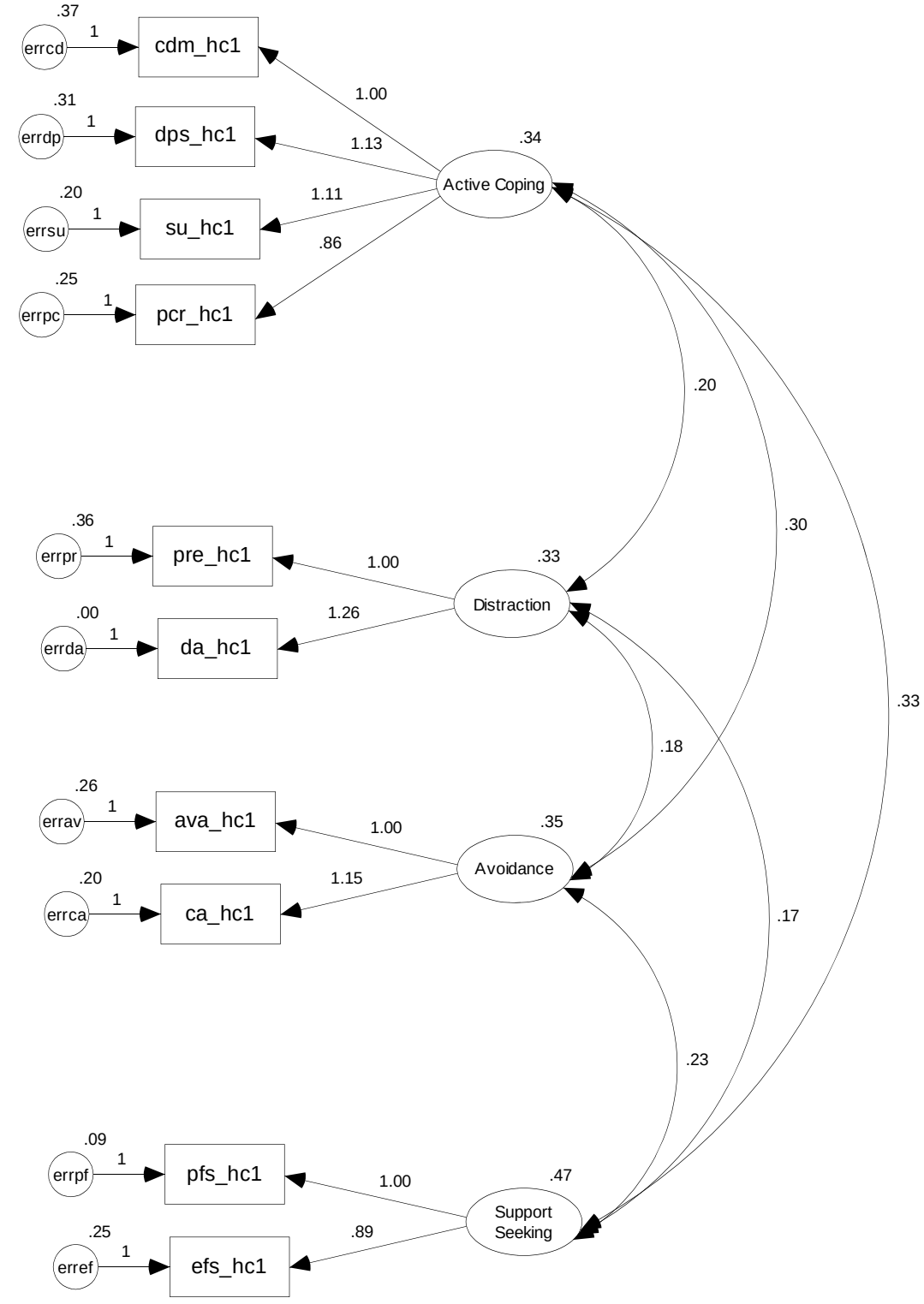
	Reex	SU	PCR	Numb	AVA	EFS	Arou	EF	PRE
Reex	1.00	.50	.21						
SU	.01	1.00	.59						
PCR	.21	**.62**	1.00						
Numb				1.00	.80	.90			
AVA				-.10	1.00	.47			
EFS				**.27**	**.26**	1.00			
Arou							1.00	.02	-.61
EF							.14	1.00	.22
PRE							.05	**.32**	1.00

Note. Significant correlations appear in bold (two-tailed significant test).

PTSD Group = Upper triangle; No PTSD Group = Lower triangle.

Reex = Reexperiencing; SU = Seeking Understanding; PCR = Positive Cognitive Restructuring; Numb = Numbing; AVA = Avoidant actions; EFS = Emotion-Focused Support; Arou = Arousal; EF = Expressing Feelings; PRE = Physical Release of Emotions.

Figure 1. Confirmatory factor analysis validating the psychometric structure of the HICUPS according to Ayers et al.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cdm_hc1
1.00
.31
errdp
1
dps_hc1
1.13
.34
Active Coping
.20
errsu
1
su_hc1
1.11
.86
.25
errpc
1
pcr_hc1
.20
.30
.36
errpr
1
pre_hc1
1.00
.33
Distraction
.00
errda
1
da_hc1
1.26
.33
.18
.26
errav
1
ava_hc1
1.00
.35
Avoidance
.20
errca
1
ca_hc1
1.15
.17
.23
.09
errpf
1
pfs_hc1
1.00
.47
Support
Seeking
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erref
1
efs_hc1
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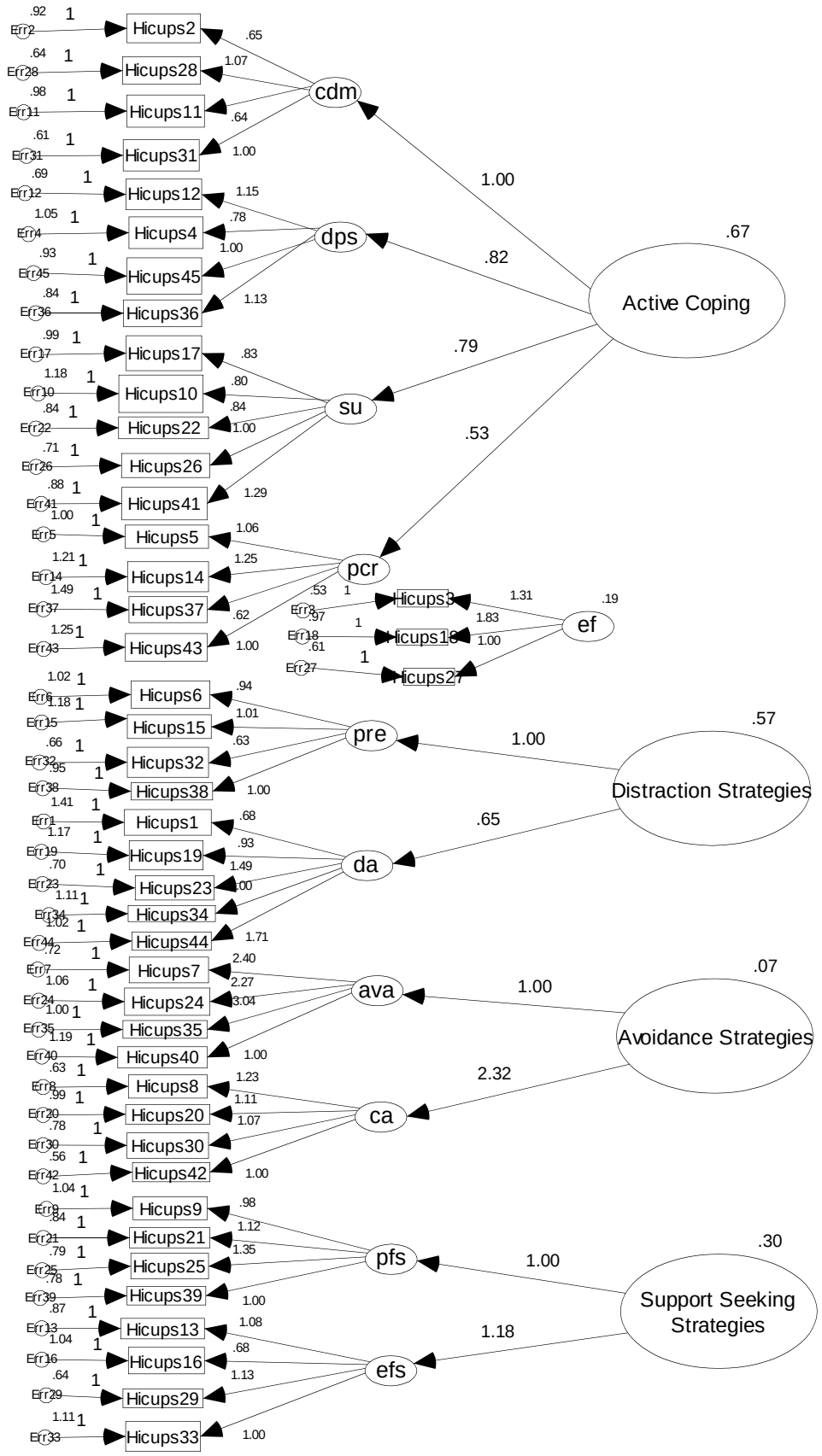
Figure 2. Confirmatory factor analysis validating the psychometric structure of the HICUPS according to the study's model.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Hicups28
Hicups11
Hicups31
Hicups12
Hicups4
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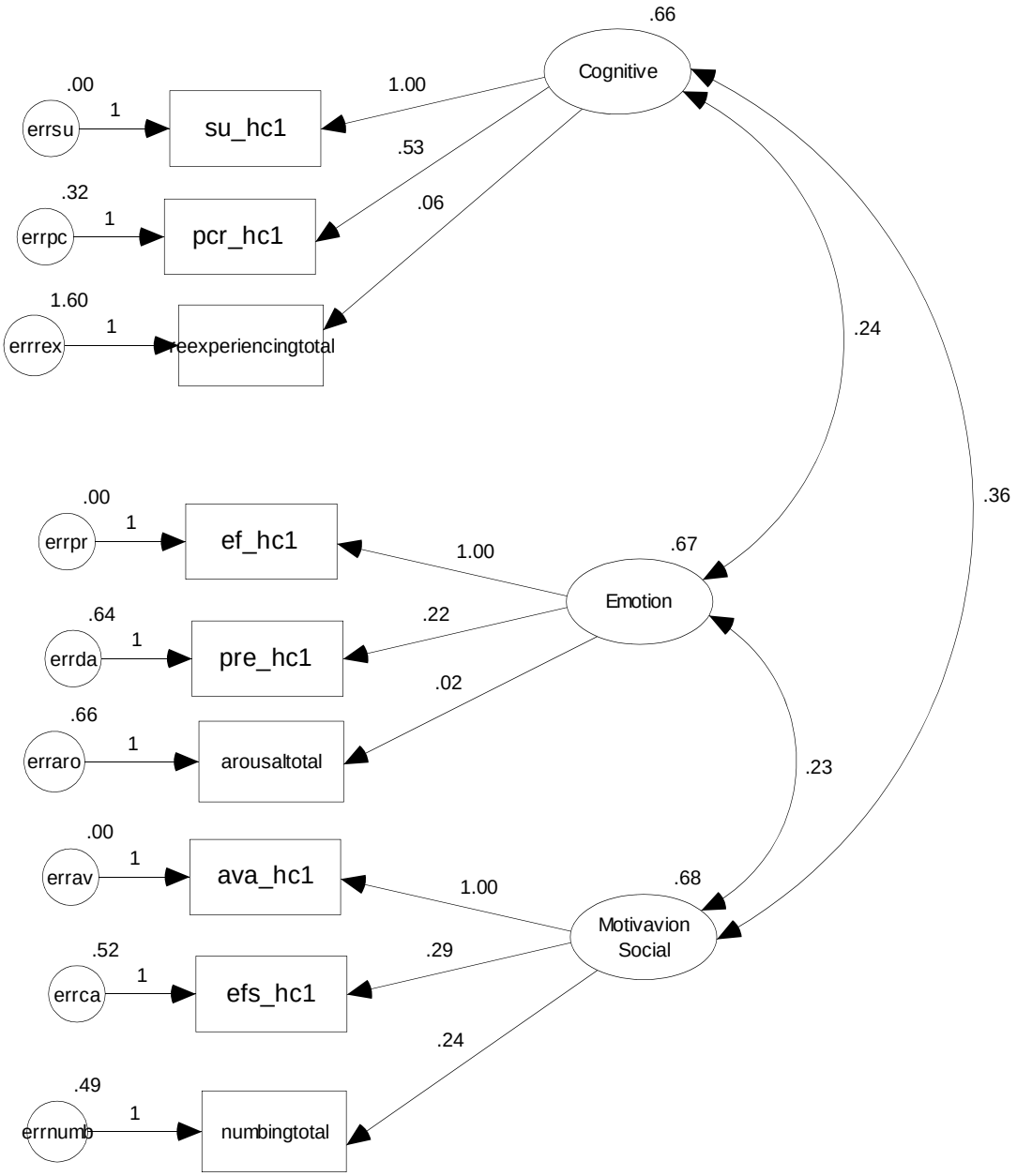
Hicups21
Hicups25
Hicups39
Hicups13
Hicups16
Hicups29
Hicups33
cdm
dps
su
pcr
ef
pre
da
ava
ca
pfs
efs
Active Coping
Distraction Strategies
Avoidance Strategies
Support Seeking Strategies
.67
.57
.07
.30
1.00
.82
.79
.53
.65
2.32
1.18

Figure 3. Confirmatory factor analysis validating the system hypothesis (A).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su_hc1
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.53
.32
errpc
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pcr_hc1
.06
1.60
errrex
1
reexperiencingtotal
.24
.36
.00
errpr
1
ef_hc1
1.00
.67
Emotion
.64
errda
1
pre_hc1
.22
.02
.66
erraro
1
arousaltotal
.23
.00
errav
1
ava_hc1
1.00
.68
Motivavion
Social
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errca
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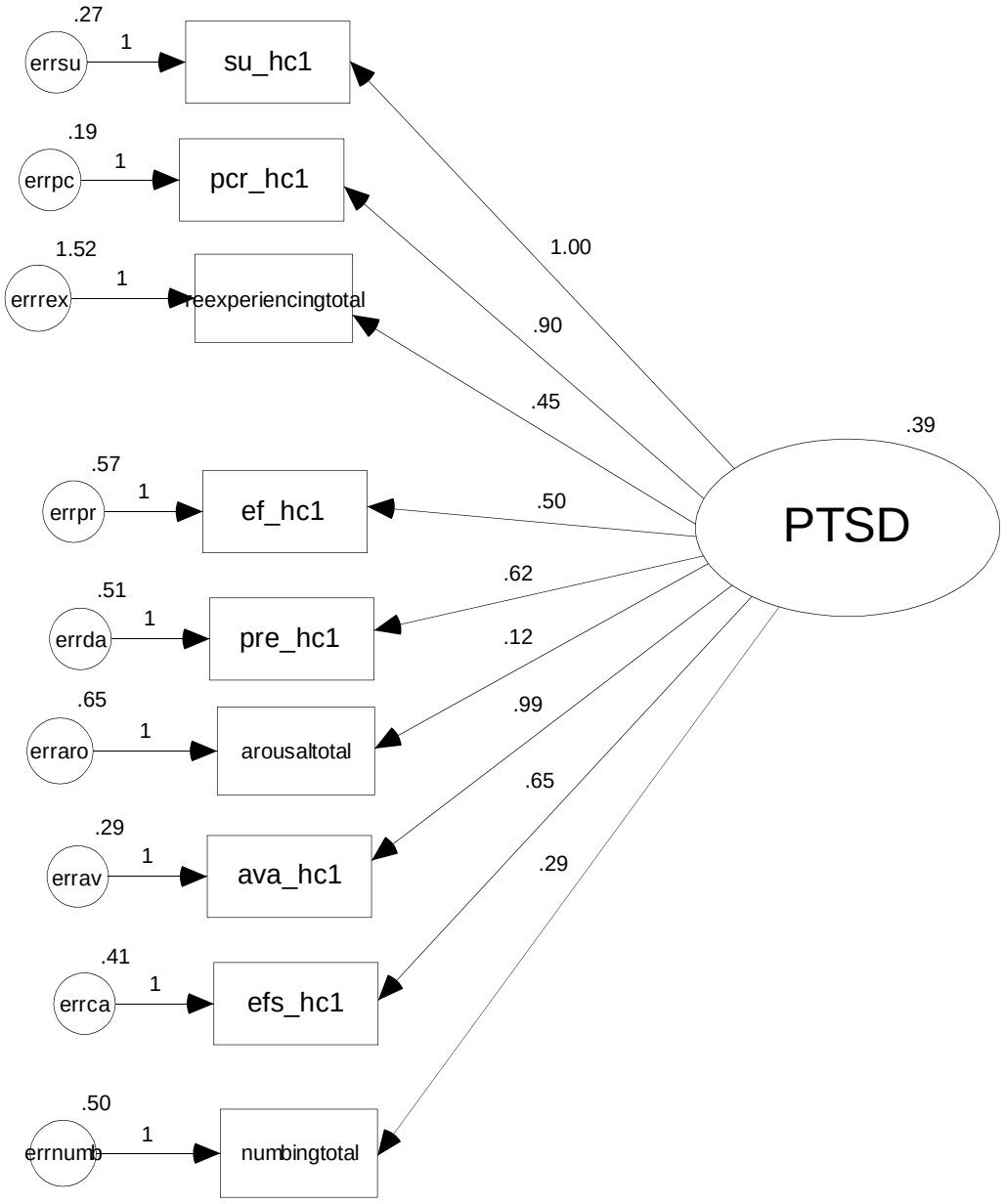
efs_hc1
.29
.24
.49
errnumb
1
numbingtotal

Figure 4. Confirmatory factor analysis validating the system hypothesis (B).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su_hc1
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errpc
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1.00
.90
.45
.39
PTSD
.57
errpr
1
ef_hc1
.50
.62
.51
errda
1
pre_hc1
.12
.65
erraro
1
arousaltotal
.99
.65
.29
errav
1
ava_hc1
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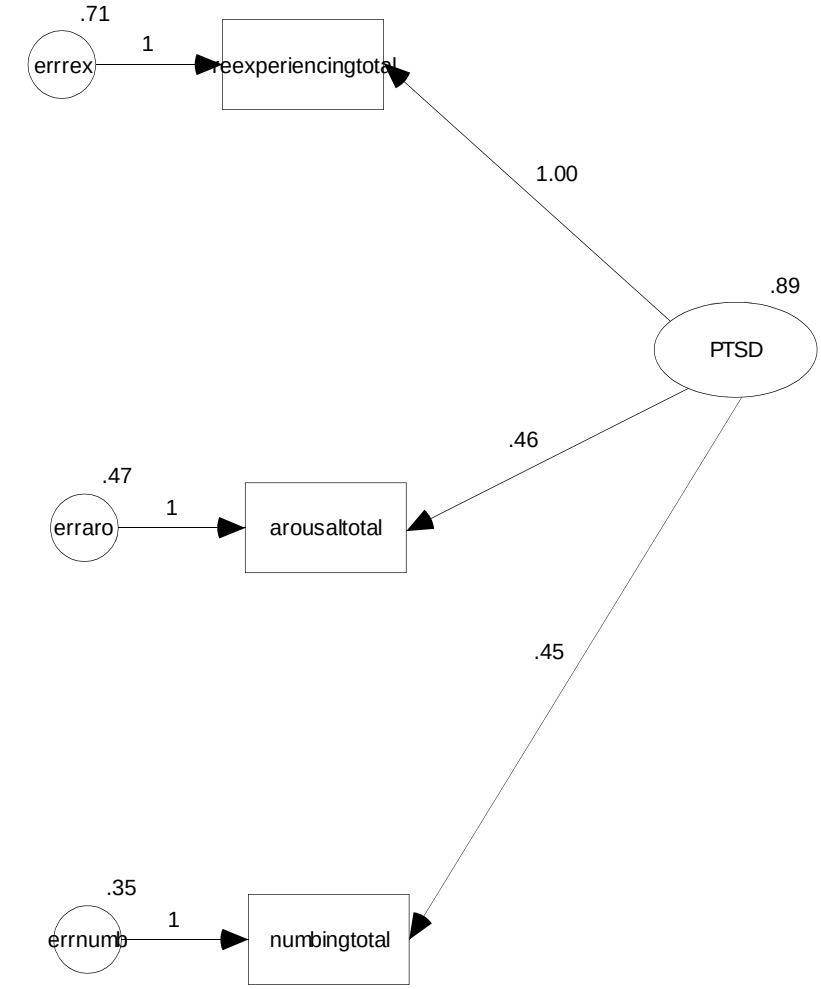
errca
1
efs_hc1
.50
errnumb
1
numbingtotal

Figure 5. Confirmatory factor analysis validating the system hypothesis (C).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reexperiencingtotal
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.89
PTSD
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erraro
arousaltotal
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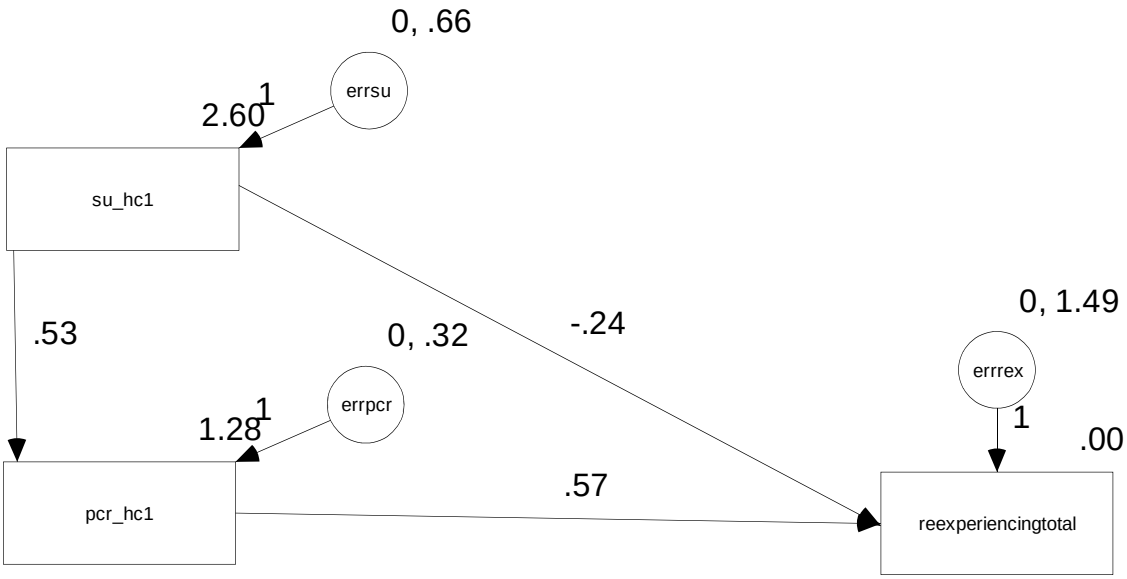
.35
1
errnumb
numbingtotal

Figure 6. Path analytic model of the regulatory hypothesis (cognitive regulatory coping system).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-.24
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errrex
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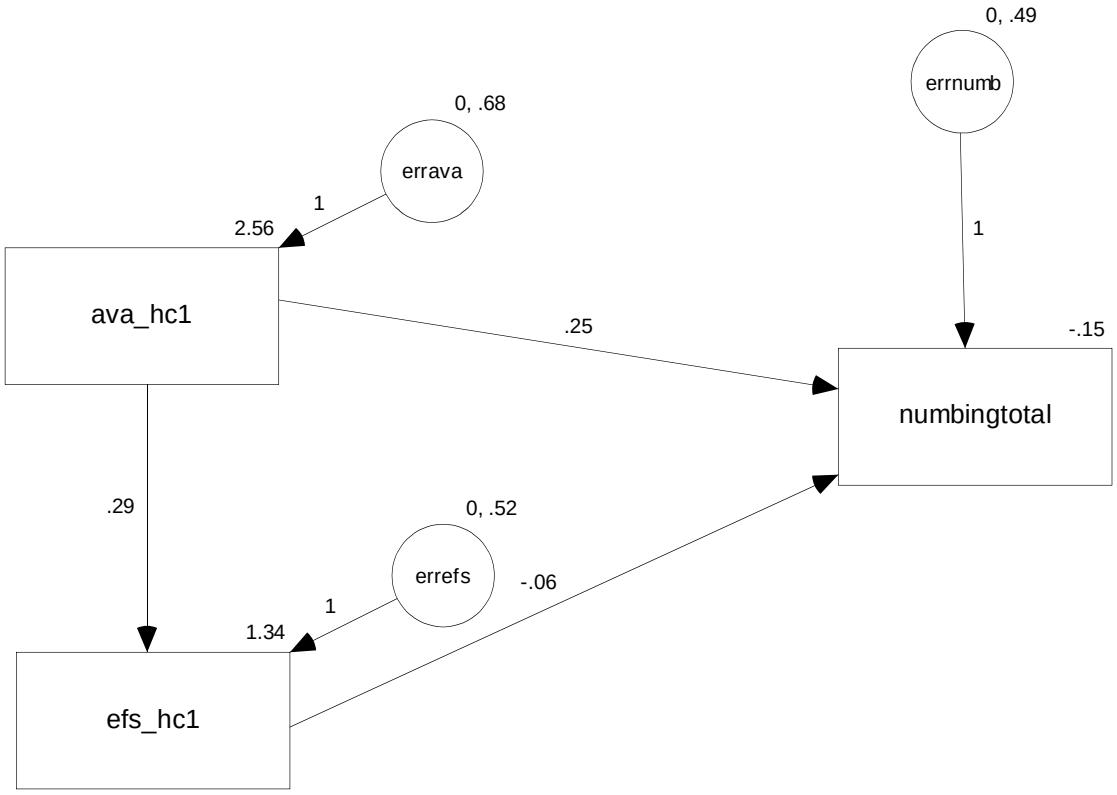
1
.00
pcr_hc1
.57
reexperiencingtotal

Figure 7. Path analytic model of the regulatory hypothesis (social/motivational regulatory coping system).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errava
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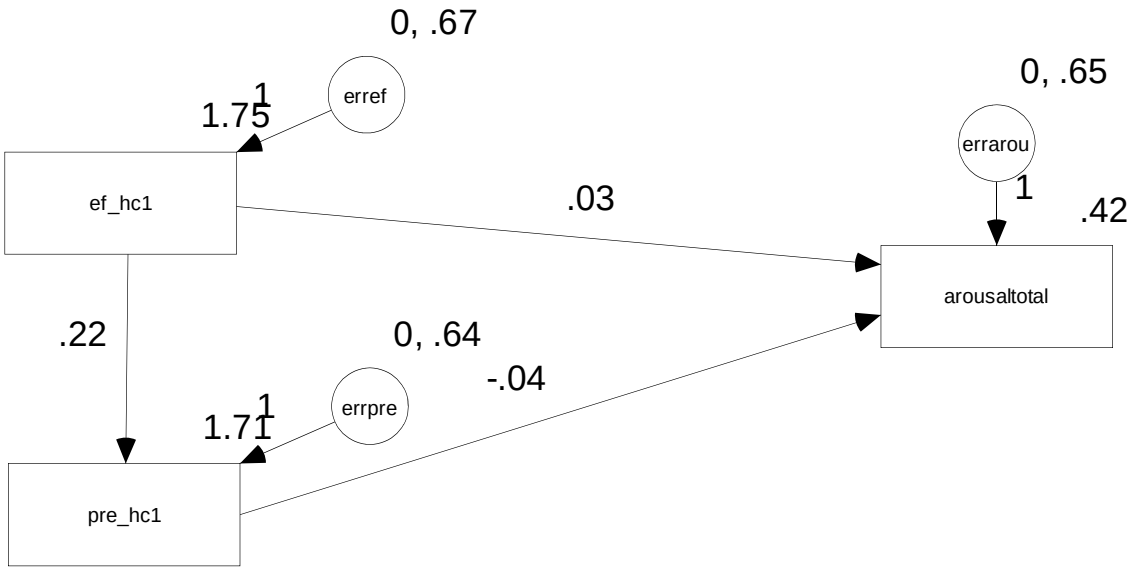
.29
0, .52
errefs
-.06
1
1.34
efs_hc1

Figure 8. Path analytic model of the regulatory hypothesis (emotional regulatory coping system).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