1

The Origins of Cognitive Processing Therapy

The prevailing theory of trauma responses in the 1970s consisted of first-order classical conditioning of the fear reaction, along with second-order operant conditioning that generalized the reaction to other triggers (Kilpatrick et al., 1979, 1981). Later, once the posttraumatic stress disorder (PTSD) diagnosis was introduced, there was awareness of the importance of escape and avoidance learning in maintaining the primary symptoms of PTSD. If someone is experiencing strong conditioned emotional reactions, this person is likely to avoid or escape reminders of the trauma that arise in objectively safe situations. Mowrer's (1960) two-factor theory of classical conditioning and operant avoidance became more commonly discussed, along with Foa and Kozak's (1986) emotional processing theory of PTSD, which in turn was based on Lang's (1977) theory that people develop fear networks with stimulus, response, and meaning elements. However, because there were enough clients who responded to their traumatic events with reactions such as "I knew he wasn't going to kill me, but it was such a huge betrayal" or "I feel so much shame and disgust at what they did to me," we had doubts that PTSD was only a fear/anxiety disorder. These exceptions led us to believe that the theory regarding trauma responses needed to be revised. We began to look toward cognitive theories of PTSD.

Theoretical Influences

In the 1960s and 1970s, Aaron T. Beck studied the causes of depression and developed his cognitive theory, which focuses on how people absorb negative and erroneous beliefs from society that leave them feeling ashamed and depressed. He and his colleagues produced a treatment manual for the cognitive therapy of depression (Beck et al., 1979). Although this was one of the first manualized treatments, we wanted something more specific and progressive that would tell therapists how to proceed

session-by-session in the treatment of PTSD. We also wanted to help clients become their own therapists by teaching them new, more balanced ways to cope and think. However, Beck et al.'s cognitive therapy for depression focused on current thoughts, and we believed that, in treating PTSD, we first needed to revisit the traumatic events to see where clients' thinking was impacted by trauma and how, if at all, they had emotionally processed the traumatic events at the time. We started conceptualizing that those who hadn't been able to recover had been "stuck" in their thinking since the time of the traumatic events and began to call such clients' thoughts "Stuck Points."

Additional inspiration came from the work of McCann and colleagues (McCann, Sakheim, & Abrahamson, 1988; McCann & Pearlman, 1990), who developed the constructivist self-development theory of traumatic victimization. This theory was based on Mahoney's (1981) constructivist perspective, in which humans actively create their personal realities, such that new experiences are constrained to fit people's determinations of what "reality" is (Mahoney & Lyddon, 1988). McCann et al. proposed a constructivist theory of trauma in which people construct meaning from events. They theorized that aside from frame of reference (i.e., the need for a stable and coherent framework for understanding experiences), the schemas (i.e., mental structures and needs) that are likely to be affected by trauma are those regarding safety, trust, power/control, esteem, and intimacy. These schemas can be self- or other-directed. Because these constructs appeared so frequently in our discussions with clients, we began to think that we could use the work of McCann and colleagues in a briefer cognitive therapy.

We were also influenced by Hollon and Garber (1988), who proposed that, when someone is exposed to schema-congruent events (e.g., they know from experience that bad events can happen to good people), then, when they experience a traumatic event, they are assimilated into preexisting beliefs with no change to those prior beliefs or interpretations of the event. This is the process that occurs when someone has healthy beliefs and does not develop PTSD. However, for schema-discrepant information (the new event does not match one's prior belief system), one of two things happens. The first possibility is that the person may attempt to alter their memory or interpretation so that it can be assimilated into the person's existing beliefs/schemas without changing the prior beliefs (e.g., "It wasn't a rape, it was a misunderstanding; I must have done something for him to think it was OK"). The other alternative is that the person may change existing beliefs (e.g., "Only strangers rape") to incorporate the new, discrepant information (e.g., "It is possible to be raped by someone you know"). This new learning represents accommodation and is a goal for therapy. Hollon and Garber's proposal, of course, was based on the work of Piaget (1971) but had not been considered in the context of therapy for trauma before.

With this edition of the book, we further refine our understanding of the cognitive processes involved in trauma processing (see Chapter 4) to ultimately facilitate an individualized cognitive conceptualization of a given patient's presentation. More specifically, extending Hollon and Garber's (1988) theory, we clarify that clients with PTSD are *attempting* to assimilate schema-discrepant information. However,

these attempts are unsuccessful and lead to the classic intrusive symptoms of PTSD (unwanted memories, nightmares) because accommodation has not been successful. In this way, the traumatic memory information is left uncategorized or unprocessed and causes symptoms. In this revision, we also help identify a range of cognitions that may interfere with trauma processing and provide additional guidance on assuming a Socratic stance in the provision of cognitive processing therapy (CPT).

In working with trauma survivors, we further realized that sometimes people could alter their beliefs to extremes, even while they were distorting and attempting to assimilate the traumatic events. They overgeneralized their beliefs to whole classes of schemas (e.g., "I always make bad decisions," "No one can be trusted," "I must control everyone around me"). We called this phenomenon "overaccommodation" (Resick & Schnicke, 1992, 1993). Although we were in the early stages of developing CPT, we recognized that it was important to work on failed accommodation of the trauma first and not move to the overaccommodated beliefs until the index (i.e., most distressing) trauma was resolved. For example, once clients stop blaming themselves for the occurrence of the traumatic event, then it is easier to tackle the idea that they can't make good decisions. Accordingly, we placed the work with overaccommodated schemas later in the therapy.

In this edition, we also clarify for therapists that people can assimilate new incoming information into overaccommodated beliefs, seemingly confirming their already negative beliefs. In this way, assimilation is not just about historical appraisals, but rather what happens when people are presented with new information. Due to negative attentional biases, people with PTSD are likely to attend to negative and threat-related information that confirms and is easily assimilated into existing overaccommodated beliefs.

Early Development of CPT

The first study of CPT was an open trial of CPT in groups (Resick & Schnicke, 1992). The first CPT manual was published in 1993 focusing on rape-related PTSD, which included the results of the first 35 participants in group treatment and the first 9 clients in individual treatment (Resick & Schnicke, 1993).

In the process of developing an adaptation of CPT for adults who had been sexually abused as children, Chard (2005) completed the first study of adult survivors of childhood sexual abuse who had been diagnosed with PTSD. She noted that not everyone's beliefs were shattered by trauma (Janoff-Bulman, 1992) because they had negative beliefs that developed as a result of their negative developmental experiences. She observed that if clients had been abused (emotionally, physically, or sexually) as children, or had other prior traumas, they might already have (and perhaps had always had) negative beliefs about themselves and about their roles in the traumatic events (e.g., "I must deserve bad things to happen to me"). Any new trauma would be assimilated without alteration because it was not schema-discrepant, but

schema-congruent. The question then arose: Why would such people have PTSD, if their beliefs were already matching the new events? It is possible that these individuals did not get *new* PTSD; they might have already had it. However, the new events might have strengthened their distorted beliefs about themselves and others and about their roles in traumatic events. In other words, they might be using the new events as "proof" that their prior beliefs were accurate: Their PTSD would worsen, and their beliefs would become more entrenched (Resick, 2001; Resick et al. 2007). On the other hand, even with prior negative schemas about themselves or others, people might still ask, "Why me?" or "Why again?" They might still find new traumatic events to be schema-discrepant because they had done everything they could to change what they perceived to be the cause of prior trauma ("I tried to be perfect"), or they could see how members of other families behaved toward one another and couldn't figure out what they were doing wrong. And in the case of efforts to assimilate, either altering their memory of the event to fit prior positive beliefs or agreeing with prior negative beliefs, if the tactic worked, they wouldn't have PTSD. The repeated intrusive memories of the event occur because somehow the explanations the trauma survivor has come up with have not resolved the issue and they are still in conflict.

Another difference between the theoretical approach that led to CPT and the theories on which other therapies are based lies in the range and type of emotions addressed in CPT. Because PTSD was classified as an anxiety disorder until the publication of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), most of the extant theories on PTSD focused on fear and anxiety. However, as trauma clinicians, we were struck by the amount of "erroneous" guilt, shame, disgust, sadness, and so forth that we found among the clients. In the longitudinal studies we conducted, nearly everyone said that they were afraid during the event—but most people recovered from their fear, and fear did not always seem to be the driving force behind the observed flashbacks, intrusive memories, nightmares, and avoidance. Furthermore, if PTSD were only about fear conditioning, then it wouldn't matter what the trauma was; the rates of PTSD should be equal. The epidemiological studies of PTSD (e.g., Kessler et al., 1995) made it clear that all traumas did not have the same effects: Rape and other interpersonal traumas produced greater rates of PTSD than impersonal traumas such as natural disasters and accidents. Something else was going on besides fear conditioning because the people who had experienced these traumatic events evaluated them in relation to their beliefs and prior experiences.

In addition, self-blame and/or erroneous other-blame, leading to guilt or shame, were almost universal among those with PTSD. By the time Resick wrote an unpublished manual for a generic version of CPT (Resick, 2001), after the events of September 11, 2001, she was differentiating "natural" emotions from "manufactured" emotions. The "natural" emotions are those we humans are hard-wired with and do not need to effortfully think about (e.g., fight–flight leads to fear or anger; losses elicit sadness). The emotions referred to as "manufactured" result from faulty cognitions about the traumatic event. Although natural emotions may take a while to dissipate,

if not avoided, emotions that are generated by thoughts ("It must have been my fault because things like this don't happen to good people") will disappear immediately if the thought is changed with more accurate information. On the other hand, if the traumatized person keeps repeating and believing erroneous statements about the trauma, the negative emotions can last a lifetime.

The first randomized controlled trial (RCT) of CPT compared it with prolonged exposure (PE) and a minimal-attention waitlist among women who had been raped. The large majority of the participants (85%) had experienced other interpersonal traumas, and 41% had experienced childhood sexual abuse (Resick et al., 2002). The findings of this study were also examined to see if the results lasted over time. Resick, Williams, and colleagues (2012) conducted a long-term follow-up, on average, 6 years after treatment with all who could be located (70%) and conducted an intention-to-treat (ITT) analysis using the same measures as baseline. They found that clients who received either CPT or PE continued to have very low symptoms and were not different on PTSD or depression.

The second RCT included women who had experienced any kind of interpersonal violence in adulthood or childhood as their index trauma to begin treatment (Resick et al., 2008). That study dismantled the components of CPT to see if both the cognitive therapy and written accounts were necessary components. It was found that CPT with accounts (later called CPT+A) had no value added to conducting the treatment without the accounts (now CPT), which worked better overall than the written accounts only. The protocol presented in Part II of the book is CPT (without written accounts), although the alternative with written accounts is presented in Chapter 18 in Part III.

Monson et al. (2006) received a grant from the U.S. Department of Veterans Affairs (VA) to conduct the first study of CPT+A with veterans. The majority of participants were male veterans of the Vietnam War. Given that most of them had received treatment for years, and that all had a history of substance abuse, the loss of a PTSD diagnosis in 12 sessions among 40% of these veterans had an immediate impact on the field. Monson et al. also noted that there were more commonalities than differences among trauma survivors, and that the veterans' interpretations of their traumas were very similar to those of the interpersonal violence victims in the earlier studies.

Since these first studies, there have been dozens of RCTs and many program evaluation studies. The next chapter describes the research on CPT and how it has continued to evolve, what populations have been studied, and what factors influence outcomes.

Dissemination of CPT

In 2006, the authors of this manual received funding from the VA Central Office to begin developing materials for disseminating CPT throughout the U.S. VA system. We wrote a treatment manual for active duty military personnel and veterans; developed training materials (e.g., slides with notes, videos, trainers' manual, consultants'

manual); and then trained a first group of national trainers. Because there were so few people in the VA system who had conducted CPT, many of the trainers were from St. Louis (former faculty colleagues, postdoctoral fellows, or graduate students). Up until then, Resick had only conducted 1-day workshops, with no follow-through with case consultation. Monson rightly suggested that we emphasize the teaching of the Socratic method as the most difficult part of the therapy, causing us to think through how to teach this skill to other therapists, who might have been taught to never ask a question or to let thoughts go rather than changing them. We also had to teach the reasoning behind the approach of asking questions that would help clients examine their Stuck Points (erroneous thoughts and beliefs dating from the time of the trauma, as explained earlier), to put them back into the context of what they actually knew at the time, what choices they really had (if they had choices), and why they made the ones they did. We also had to help clients differentiate among intentionality, responsibility, and the unforeseeable. Finally, Chard noted that we needed to include a Stuck Point Log that would serve as a "living" document throughout the therapy. This log would help to keep both a client and a therapist focused on the unhealthy cognitions and not get derailed into more supportive forms of therapy.

The first 2 years of the dissemination project included 22 workshops each year, and then the project was cut back slowly, as more VA therapists completed training that included workshops and case consultation. Along the way, we received thoughtful feedback from the trainers about ways to streamline the handouts and make them more accessible to people with lower education levels or with traumatic brain injuries. We also developed "help sheets" for understanding Stuck Points and for answering challenging questions. As of this writing, tens of thousands of providers have been trained in CPT in the VA. Beyond the VA context, CPT has also been widely disseminated through mental health centers across the United States, as well as in different countries, and through several funded implementation trials testing different strategies for training clinicians in CPT (e.g., LoSavio, Dillon, Murphy, & Goetz, et al., 2019; Monson et al., 2018).

The CPT manuals have been translated into 14 languages thus far, and the therapy appears to work well across cultures (see Chapter 20). Because the cognitive impact of a traumatic event is very individualized, clients across cultures can describe why they think their events happened and what the events mean to them. Even though there may be differences in some concepts, many of them translate well—and even in very strict traditional cultures, it can be pointed out that not all people believe identically and that there is some flexibility in beliefs. People can change their minds.

A Biological Model of PTSD and CPT

The most recent additions to our training and conceptualization involve the connections between the biological underpinnings of PTSD and the reasons why CPT

works. Most of this material reflects research on activation of the amygdala, which triggers strong emotions and sends neurotransmitters throughout the brain to activate the emergency response. Additional factors that were not noticed immediately, but are actually found more frequently in research, are the diminished responsivity and smaller size of the prefrontal cortex (Shin et al., 2006) among those with PTSD.

In a normal fight–flight response, activity in the prefrontal cortex (which is the seat of decision making and control over the amygdala) decreases, along with other immune functions and normal physical processes like digesting food, in order to free all available resources for either running or fighting. The natural emotions accompanying flight and fight are fear and anger. During a life-threatening emergency, it is more important to activate the brain stem and neurotransmitters to aid in the fight–flight response than to think about what to have for dinner or whether to change jobs. However, in a well-modulated emergency response (see Figure 1.1), the prefrontal cortex is activated enough to notice when the danger is over, and to send messages out to the amygdala to stop the fight–flight response and return to normal parasympathetic functioning. In other words, there is a reciprocal relationship between the prefrontal cortex and the amygdala.

In studies of people with PTSD, by contrast, researchers have found that the amygdala shows heightened responsivity while the prefrontal cortex shows greatly decreased activity, and that there is a functional relationship between the two (Shin et al., 2004). Because the amygdala is so highly activated and the activity in the prefrontal cortex is diminished (see Figure 1.2), it takes a person with PTSD much longer to recognize that the perceived danger has ended and to calm down.

In imaging studies, Hariri and colleagues (Hariri et al., 2000, 2003) found that when participants were shown pictures of emotional faces or dangerous objects and

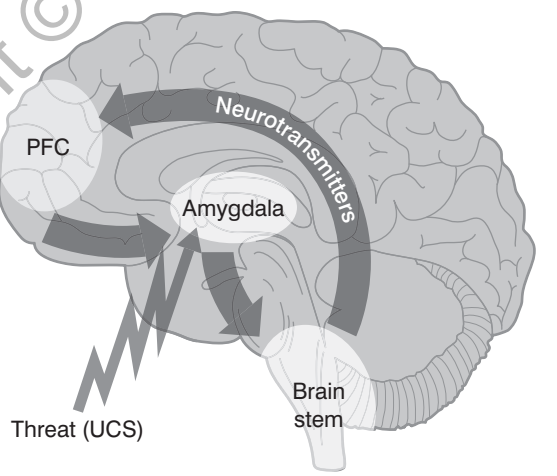

FIGURE 1.1. Well-modulated emergency response. UCS, unconditioned stimulus; PFC, prefrontal cortex.

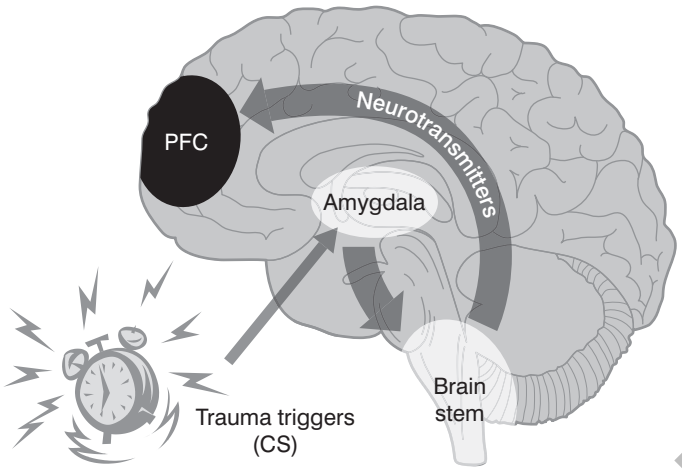

FIGURE 1.2. Emergency response in PTSD. CS, conditioned stimulus. Data from Liberzon and Sripada (2008), Milad et al. (2009), Rauch et al. (2000), and Shin et al. (2001).

were asked either (1) to pick pictures that matched the original pictures, or (2) to label the emotions or objects, in the first case there was no change in the activation of the amygdala. However, when participants were asked to label the objects or to describe whether each picture was of a natural or an artificially created danger, the instruction to use words resulted in the activation of the prefrontal cortex (including Broca's area, which is the speech area), while the amygdala quieted.

It occurred to us that if merely labeling objects or pictures was sufficient to activate the prefrontal cortex and quiet the amygdala, we could accomplish much more with regard to affect regulation through cognitive therapy—specifically, having clients talk *about* and answer questions about the trauma—than through having clients reexperience the images of the traumatic events. In other words, these findings reinforced the idea that cognitive therapy could be a more direct route to change than having clients imagine the traumatic events repeatedly (see Figure 1.3).

Neurobiology also helps us to understand why younger people are more likely to develop PTSD, aside from the fact that physical and sexual abuse, rapes, assaults, car accidents, and combat are all more likely to occur among those who have not reached full adulthood. The prefrontal cortex is not fully developed until humans are well into their 20s, so not only are young people likely to be traumatized, but they also have fewer resources to deal with trauma once it occurs (Johnson et al., 2009). According to Johnson et al. (2009):

> The prefrontal cortex coordinates higher-order cognitive processes and executive functioning. Executive functions are a set of supervisory cognitive skills needed for goal-directed behavior, including planning, response inhibition, working memory, and attention. These skills allow an individual to pause long enough to take stock of a situation, assess his or her options, plan a course of action, and execute it. Poor executive functioning

leads to difficulty with planning, attention, using feedback, and mental inflexibility, all of which could undermine judgment and decision making. (p. 218)

By the time child and adolescent trauma victims receive therapy as adults, they may have settled on cognitions that were constructed at a time when their executive functions were not fully developed. This brain immaturity is probably the reason why so many clients with PTSD have extreme beliefs and have been traumatized repeatedly. CPT may well assist such clients in developing affect regulation, increasing their cognitive flexibility, and changing many assumptions and beliefs that were developed at a period of cognitive immaturity and that were never reexamined because of avoidance symptoms. One of the goals of CPT is to teach these clients greater flexibility in thinking—specifically, to teach them how to think critically about what they have been saying to themselves regarding the reasons why the traumatic events happened and the events' implications about themselves and others.

As noted before, PTSD does not always include the fight–flight response. Some people do not experience fear during the traumatic event, so fear circuitry may not be involved. Ramage et al. (2016) classified active duty military members according to their type of trauma: danger-related traumas (e.g., life threat to self or others) and nondanger traumas (e.g., exposure to images, sounds or smells, traumatic loss, or moral injury). They also included samples of combat veterans without PTSD and civilian controls. As expected, those who had experienced danger-based traumas showed activation in the amygdala, whereas the nondanger group with PTSD looked the same as the two control groups. However, the nondanger PTSD group showed higher responsivity in the precuneus, which is associated with heightened cohesive brain activity at rest, greater introspection, and moral cognition.

CPT offers an approach to treatment that has had profound results for patients.

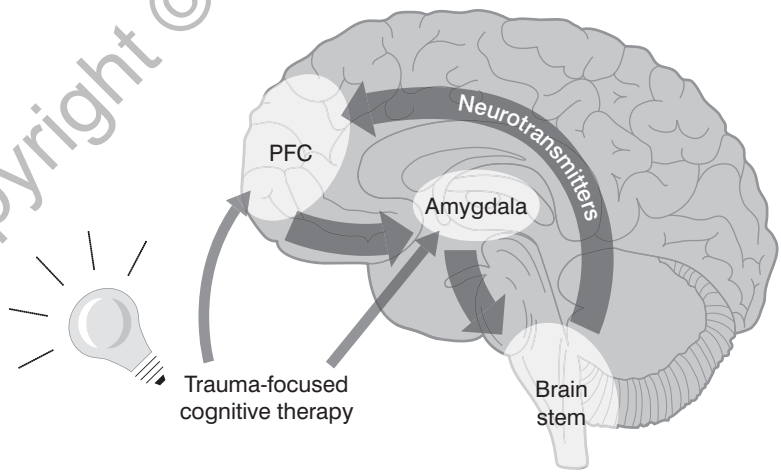

FIGURE 1.3. How cognitive therapy may work: It may force the frontal lobe online, which inhibits the amygdala and prevents extreme emotional responses, even while the trauma circuit is simultaneously and sufficiently activated.

This book provides an explanation of this approach and guides you through implementing the protocol with your patients. Before getting into those specifics, in Chapter 2 we discuss some of the research evidence that supports CPT for treating people struggling with PTSD and other problems stemming from exposure to trauma. Then, before moving into an examination of the protocol itself in Chapter 6, we will discuss assessment, cognitive case conceptualization, and preparing to deliver CPT.

Guilford Publications
370 Seventh Avenue
New York, NY 10001
212-431-9800
800-365-7006
www.guilford.com