Processes of change in cognitive therapy for posttraumatic stress disorder



Milan Wiedemann

Wolfson College

University of Oxford

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To All The Stars

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Wolfson College, Oxford
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Abstract

Although trauma-focused cognitive therapy for PTSD is effective and recommended in international treatment guidelines as a first-line intervention for PTSD, the psychological processes through which this treatment drives clinical improvement have rarely been investigated. The aim of this thesis is to improve our understanding of how cognitive therapy for PTSD works by testing predictions made by Ehlers and Clark's (2000) cognitive model of PTSD in two large datasets of patients receiving cognitive therapy in routine care ($N_1 = 330$; $N_2 = 343$). To increase the transparency and reproducibility of my work, I developed research software tools that help implement the methods used in this thesis in a transparent and reproducible way.

I used bivariate latent change score models to test whether changes in the PTSD symptoms are preceded by changes in theory-derived cognitive processes and coping strategies during treatment. The results show that changes in PTSD symptoms were preceded by changes in negative appraisals, flashback quality of unwanted memories, and unhelpful responses to intrusions, but not vice versa. The relationship between changes in trauma memory disorganisation and changes in PTSD symptoms was bidirectional.

To investigate sudden symptom improvements during cognitive therapy for PTSD I developed the R package *suddengains*. First, I examined how cognitive factors change before, during, and after sudden gains in PTSD symptom severity. The results indicate that sudden gains were accompanied, and to a smaller degree preceded, by improvements in negative appraisals and flashback quality of unwanted memories. A second study extended these results by exploring improvement in individual PTSD, depression, and anxiety symptoms during sudden gains in total PTSD symptoms. The results suggest that patients experience different patterns of sudden symptom improvements, while showing similar overall treatment outcomes.

The studies presented in this thesis contribute further evidence supporting predictions about maintenance factors and clinical recovery during treatment by Ehlers and Clark's (2000) cognitive model of PTSD. This thesis also contributed to clinical research methodology by developing research software that facilitates reproducible analyses as well as a collaborative evaluation and further development of methods evaluating processes of change in psychological therapies. Overall, this thesis supports interventions targeting the identification and modification of cognitive and behavioural processes in PTSD.

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List of Abbreviations

AIC . . . . . Akaike Information Criterion

BIC . . . . . Bayesian Information Criterion

BAI . . . . . Beck Anxiety Inventory

BDI . . . . . Beck Depression Inventory

CT . . . . . Cognitive Therapy

CT-PTSD . . Cognitive Therapy for Posttraumatic Stress Disorder

CFI . . . . . Comparatie Fit Index

DSM . . . . . Diagnostic and Statistical Manual of Mental Disorders

FE . . . . . Fixed Effect

FIML . . . . . Full Information Maximum Likelihood

GAD-7 . . . . General Anxiety Questionnaire 7-item scale

NHS . . . . . National Health Service

ICD . . . . . International Classification of Diseases

LCSM . . . . . Latent Change Score Model

LL . . . . . Log-likelihood

ML . . . . . Maximum-likelihood

PDS . . . . . Posttraumatic Diagnostic Scale

PHQ-9 . . . . Patient Health Questionnaire 9-item scale

PSS-I . . . . . TSD Symptom Scale Interview

PTCI . . . . . Posttraumatic Cognitions Inventory

PTSD . . . . . Posttraumatic Stress Disorder

RIQ . . . . . Responses to Intrusions Quetsionnaire

RMSEA . . . . Root Mean Square Error Approximation

SBQ . . . . . Safety Behaviours Questionnaire

SEM . . . . . Structural Equation Modelling

SMD . . . . . Standardised Mean Difference

SG . . . . . Sudden Gain

TLI . . . . . Tucker-Lewis Index

TMQ . . . . . Trauma Memory Questionnaire

UMQ . . . . . Unwanted Memories Questionnaire

WHO . . . . . World Health Organisation

1 Introduction

1.1 Posttraumatic stress disorder (PTSD)

Posttraumatic stress disorder (PTSD) is a disorder that some people develop in response to a traumatic event. The *Adult Psychiatric Morbidity Survey 2014* of Mental Health and Wellbeing in England included a representative sample of 7500 adults and found that around one third (31.4%) of individuals reported having experienced at least one traumatic event, and 4.4% met the DSM-IV diagnostic criteria for PTSD (McManus et al., 2016). PTSD can be extremely disabling when left untreated and has been associated with a lower quality of life compared to nonclinical controls (Olatunji et al., 2007), as well as considerable economic cost and burden to the health services (Layard et al., 2007).

PTSD is characterised by repeated unwanted re-experiencing of the event, hyperarousal, emotional numbing, and avoidance of reminders of the trauma (American Psychiatric Association, 2000, 2013; World Health Organization, 1993, 2018). Depending on the diagnostic classification system, somewhat different sets of criteria need to be fulfilled for a PTSD diagnosis. According to the DSM-IV classification (American Psychiatric Association, 2000) the criteria listed in Table 1.1 need to be met. This includes a definition of exposure to a traumatic event (Criterion A); a number of different symptoms across a range of symptom clusters

Table 1.1: Diagnostic criteria of PTSD according to DSM-IV

Criterion	Symptom
Stressor	
A1	Experienced, witnessed or confronted with traumatic event
A2	Response involved intense fear, helplessness, or horror
Reexperiencing	
B1	Recurrent, intrusive, distressing recollections of the event
B2	Recurrent, distressing dreams of the event
B3	Acting or feeling as if the traumatic event were recurring
B4	Intense psychological distress at exposure
B5	Physiological reactivity on exposure
Avoidance	
C1	Efforts to avoid thoughts, feelings, or conversations
C2	Efforts to avoid activities, places, or people
C3	Inability to recall an important aspect
C4	Markedly diminished interest or participation in activities
C5	Feeling of detachment or estrangement from others
C6	Restricted range of affect
C7	Sense of foreshortened future
Arousal	
D1	Difficulty falling or staying asleep
D2	Irritability or outbursts of anger
D3	Difficulty concentrating
D4	Hypervigilance
D5	Exaggerated startle response
Duration	
E	Duration of at least one month
Functioning	
F	Significant distress or impairment of functioning

Note. The descriptions of the criteria and symptoms were shorted for this table.

(Criteria B, C, and D); symptoms must be experienced for longer than one month (Criterion E); and associated with a significant increase in distress or reduction in being able to carry out everyday activities (Criterion F).

Some of the diagnostic criteria for PTSD were updated in the latest DSM-5 classification (American Psychiatric Association, 2013). Criterion A has been debated in the literature, with some researchers suggesting that it should be removed from the diagnosis (e.g., Brewin et al., 2009), and it was updated in the recent version of the DSM-5 (for a review see Stein et al., 2016) to no longer include the

subjective response to trauma (A2 in DSM-IV). The latent structure of PTSD symptoms has also been debated, leading to changes in the number of symptoms and clusters from DSM-IV to DSM-5 (Rasmussen et al., 2018). Accumulating evidence about the role of trauma-related negative appraisals in the development and maintenance led to the addition of a new symptom cluster '*Negative alterations in cognition and mood*', which includes some of the DSM-IV symptoms of the avoidance/numbing cluster (for a detailed review see Friedman, 2013).

A systematic literature review of 112 psychometric studies presented supporting evidence for the DSM-IV and DSM-5 classifications but also highlighted competing models with varying underlying factor structures, suggesting that further research is needed and that the inclusion of a greater number of clusters may improve the description of PTSD (Armour et al., 2016). Further details and implications of changes in the PTSD diagnostic criteria between DSM-IV and DSM-5 are discussed elsewhere (e.g., Hoge et al., 2014) and are not the focus of this thesis. This thesis used datasets of patients diagnosed on the basis of DSM-IV.

The recent version of the International Classification of Diseases (ICD-11; World Health Organization, 2018) has taken a different approach and has narrowed the definition of PTSD by focusing on the three core symptom clusters of *reexperiencing*, *avoidance* and *hypervigilance* when defining PTSD. The remaining symptoms in DSM-5 overlap with those of the new disorder 'Complex PTSD', which is defined as PTSD symptoms plus symptoms of affect deregulation, negative self-concept and problems in maintaining relationships. Regardless of the set of criteria chosen for the different diagnostic systems, all agree on the same core symptoms of PTSD (i.e., reexperiencing, avoidance, and hypervigilance/hyperarousal) and take into account that individuals can experience different symptom profiles.

Depression, anxiety, and substance use disorders are often comorbid with PTSD. In the US *National Comorbidity Study*, Kessler (1995) found that a large proportion of respondents ($N = 5877$) who developed PTSD after a traumatic event also developed at least one other disorder (79% of women; 88% of men) at a later point during their life. The most common comorbid conditions were depression, anxiety,

and substance use disorders (Kessler, 1995; Kessler et al., 2005). On the other hand, a history of anxiety disorders or depression is a risk factor for developing PTSD after trauma exposure (e.g., Ozer et al., 2003).

1.2 Ehlers and Clark's cognitive model of PTSD

Ehlers and Clark (2000) proposed a cognitive model to explain the development and maintenance of PTSD, see Figure 1.1 for a conceptual illustration. Ehlers and Clark's (2000) model proposes that two cognitive processes lead to a perceived sense of internal or external current threat in PTSD: (1) negative appraisals of the traumatic event or its aftermath (e.g., '*I am inadequate*', '*I have to be on guard all the time*') and (2) the disjointed nature of trauma memories, which leads to reexperiencing symptoms. Ehlers and Clark further propose that individuals with PTSD engage in a range of unhelpful cognitive and behavioural coping strategies that maintain the problem.

Experimental and prospective studies of trauma survivors have established strong evidence that the key factors hypothesised in Ehlers and Clark's (2000) cognitive model play an important role in the development and maintenance of PTSD. Results from prospective studies of trauma survivors (e.g., Beierl et al., 2019; Dunmore et al., 2001) and experimental studies of individuals with PTSD (e.g., Sachschal et al., 2018) highlighted the importance of trauma-related negative appraisals in the development and maintenance of PTSD. The central role of trauma memory disorganisation and flashback characteristics is supported by prospective longitudinal studies of trauma survivors (e.g., Ehring et al., 2008; Halligan et al., 2003; Michael et al., 2005) as well as experimental analogue studies of student volunteers (e.g., Halligan et al., 2002). The importance of unhelpful cognitive coping strategies (e.g., thought suppression, rumination, and intentional numbing) and safety behaviours in the development of PTSD has been shown in prospective longitudinal studies of trauma survivors (e.g., Beierl et al., 2019; Ehring et al., 2008;

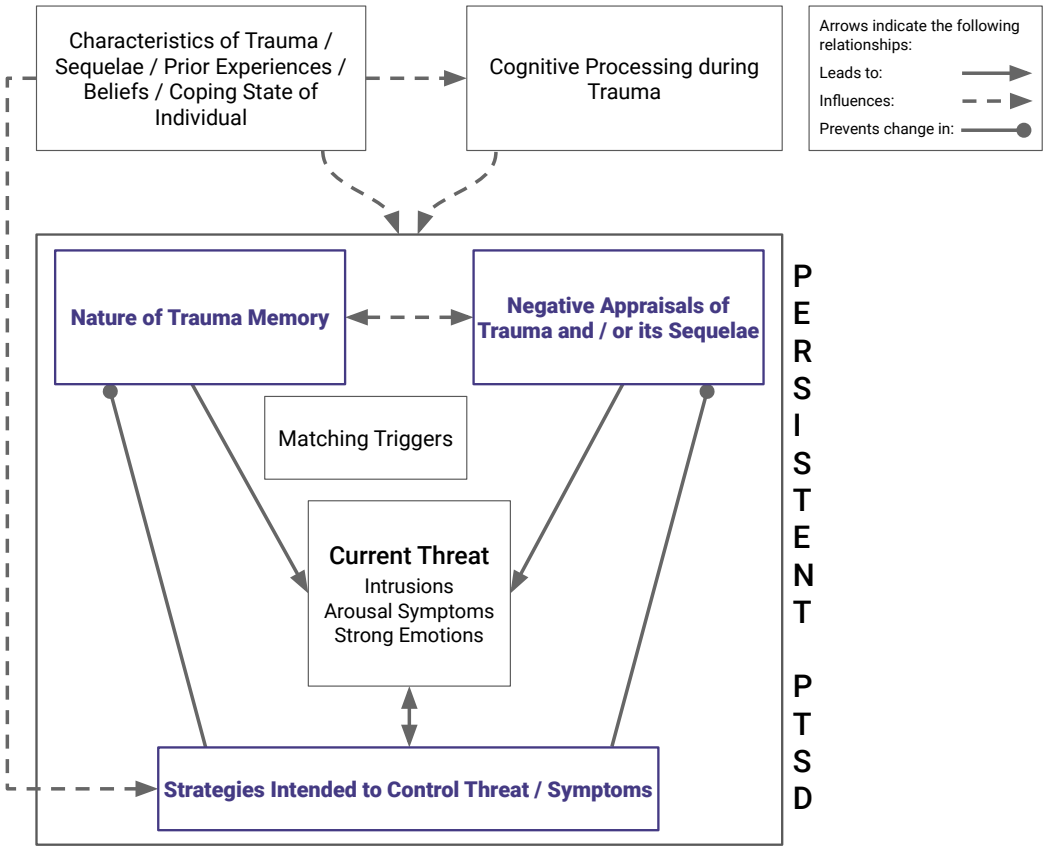


Figure 1.1: Conceptual illustration of Ehlers and Clark's (2000) cognitive model of PTSD. Cognitive and behavioural maintaining factors are highlighted in purple. (Reprinted with permission from the authors).

Kleim et al., 2012). The empirical evidence of Ehlers and Clark's (2000) cognitive model will be discussed in more detail in later chapters of this thesis.

Other cognitive-behavioural models of PTSD also emphasise key processes like trauma-related appraisals, trauma memory characteristics, and unhelpful coping strategies in the development and maintenance of PTSD (e.g., Brewin, 2014; Foa & Kozak, 1986; Foa & Riggs, 1993; Resick & Schnicke, 1992). However, there are also some differences. These models primarily differ with respect to the hypothesised effect of PTSD on trauma memories and the hypothesised processes that prevent change in the maintaining factors (see Brewin & Holmes, 2003; Dalgleish, 2004; Ehlers et al., 2012). Alternative treatment protocols have been developed based on some of these models, for example 'Prolonged Exposure Therapy' (Foa & Riggs,

1993) and 'Cognitive Processing Therapy' (Resick & Schnicke, 1992).

1.3 Cognitive therapy for PTSD (CT-PTSD)

A number of psychological therapies have been developed for posttraumatic stress disorder, with meta-analyses of randomised controlled trials providing strong evidence for their short and long-term efficacy (e.g., Cusack et al., 2016; International Society for Traumatic Stress Studies, 2020; Kline et al., 2018; Mavranezouli et al., 2020; National Institute for Health and Care Excellence, 2018). International treatment guidelines recommend trauma-focused cognitive behavioural therapies as first-line interventions for this condition (American Psychological Association, 2017; International Society for Traumatic Stress Studies, 2020; National Institute for Health and Care Excellence, 2018).

One of the treatments that is strongly recommended is trauma-focused cognitive therapy for PTSD (CT-PTSD), which is based on Ehlers and Clark's (2000) cognitive model of PTSD. CT-PTSD has been shown to be efficacious in randomised controlled trials (e.g., Ehlers et al., 2005; Ehlers et al., 2003; Ehlers et al., 2014) when compared against a self-help condition, a wait-list condition, or emotion-focused supportive therapy. Furthermore, CT-PTSD has also shown to be effective in routine clinical care (e.g., Ehlers et al., 2013).

The treatment aims to reduce the patient's sense of current threat by changing problematic meanings of the trauma and its consequences, elaborating and updating the memories of the trauma with information that gives them a less threatening meaning at present, discriminating triggers of intrusive memories, and changing behaviours and cognitive processes that maintain PTSD, such as rumination and safety behaviours. A conceptual illustration of the goals in CT-PTSD based on Ehlers et al. (2005) is shown in Figure 1.2.

The key treatment procedures in CT-PTSD include (see Ehlers & Wild, 2015): (a) Based on Ehlers and Clark's (2000) model of PTSD the therapist and patient develop an *individualised case formulation* together. Further treatment procedures

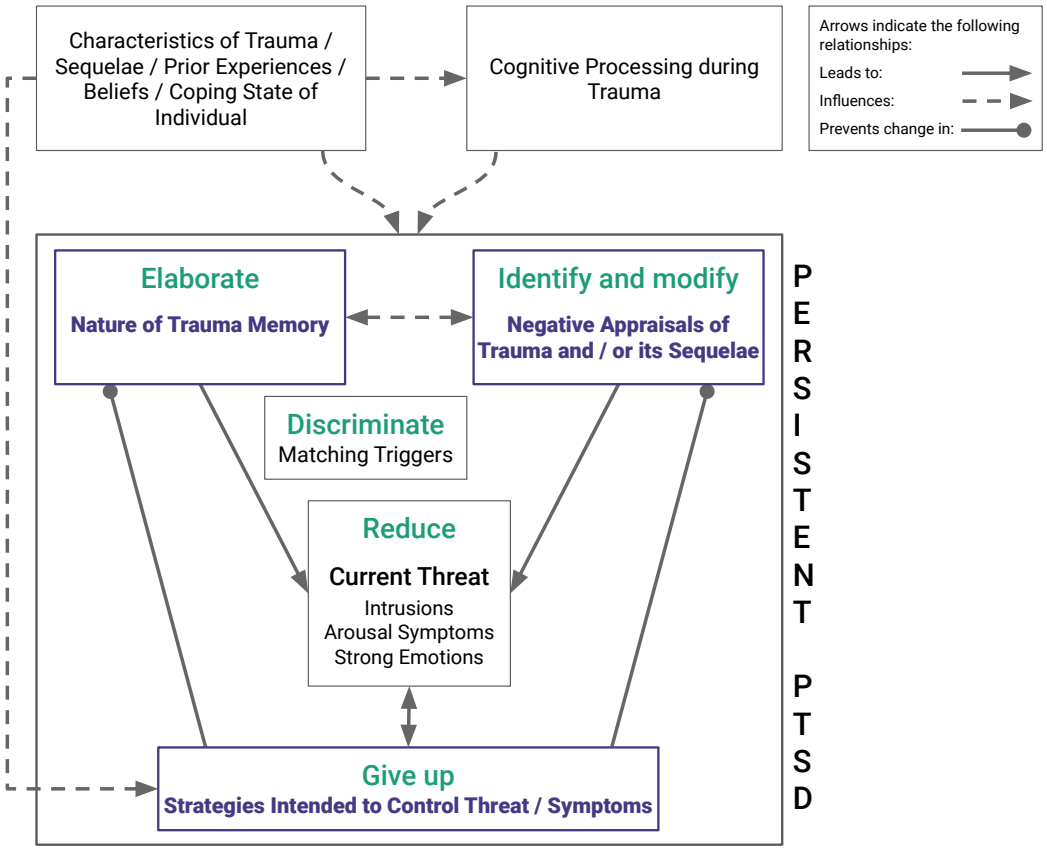


Figure 1.2: Conceptual illustration of treatment goals in CT-PTSD based on Ehlers et al. (2005) and Ehlers and Wild (2015). Cognitive and behavioural maintaining factors are highlighted in purple, treatment goals are highlighted in green. (Reprinted with permission from the authors).

are tailored to the individual formulation; (b) *Reclaiming/rebuilding your life assignments* are designed to reclaim or rebuild activities and social contacts to address the patients' perceived change after trauma; (c) *Changing problematic appraisals* of the traumas and their sequelae involve guided discovery and behavioural experiments throughout treatment. This work is closely linked to the updating trauma memories procedures; (d) The *updating trauma memories* procedure involves the following three steps: (1) accessing memories of the worst moments during the traumatic events and their threatening meanings, (2) identifying information that updates these meanings to be less threatening, and (3) linking the new meanings to the worst moments in the trauma memory; (e) *Discrimination training with triggers of reexperiencing* involves systematically spotting idiosyncratic triggers and learning

to discriminate between 'Then' (cue in the traumatic event) and 'Now' (cues in a new safe context); (f) A *site visit* completes the memory updating and trigger discrimination work; (g) *Dropping unhelpful behaviours and cognitive processes* includes behavioural experiments and discussing their advantages and disadvantages; (h) A *therapy blue print* outlines the main learning points from treatment and includes a plan for any set backs. The work on negative trauma-related appraisals is closely linked to memory work and tailored to the individual case-formulation.

1.4 Investigating processes of change in psychological therapies for PTSD

It is important to empirically test the processes through which therapy is thought to work (Kazdin, 2007, 2009). Building on the supportive evidence from experimental and prospective studies, Ehlers and Clark's (2000) cognitive model of PTSD also needs to be tested in populations receiving treatment for PTSD. Ehlers and Clark's (2000) model assumes the same processes that are involved in maintaining PTSD are also responsible for improvement during treatment. This hypothesis needs to be tested in clinical samples receiving CT-PTSD using appropriate statistical methods.

Research into the processes through which individuals improve during therapy may facilitate refinements of existing therapies and advance patient care. This is particularly important because a subgroup of individuals who receive an evidence-based psychological treatment for PTSD do not benefit as much as others. Systematic reviews and meta-analyses highlight the problem that some participants show considerable residual symptoms at the end of treatment, or drop out before finishing treatment (for reviews see Bisson et al., 2013; Schottenbauer et al., 2008). Lewis et al. (2020) reported that the pooled dropout rate across 115 randomised controlled trials was 16% (95% CI [14% to 18%]), while some reviews and meta-analyses suggest that up to half of the patients who finish treatment still show substantial residual PTSD symptoms (Bradley et al., 2005; Schottenbauer et al., 2008).

A closer examination of the processes that predict clinical improvement during treatment may have important clinical implications and guide adaptations to current treatment protocols. There is some initial evidence that trauma-focused therapies for PTSD work for the reasons suggested by the underlying theoretical models, in particular that they change problematic beliefs (e.g., Brown et al., 2018; Kleim et al., 2013). However, recent reviews conclude that further research is needed to investigate cognitive and behavioural factors that are involved in clinical improvement (Brown et al., 2018; Cooper, Clifton, et al., 2017; McNally & Woud, 2019).

To examine the processes through which treatments work, it is important to assess potential therapy processes and outcome variables at repeated time points during treatment. This allows to test the longitudinal associations between two (or more) constructs using appropriate statistical models that match predictions made by the underlying theoretical models. Recent methodological developments (e.g., latent change score modelling) allow to examine whether *changes* in a therapy processes are preceding *changes* in symptom severity (Goldsmith et al., 2018; Grimm et al., 2012) – a prediction that is commonly made by theoretical models underlying psychological therapies.

However, only looking at the processes of change throughout the entire treatment might miss important aspects of therapeutic change that happen suddenly and during shorter time intervals. Sudden gains (Tang & DeRubeis, 1999) have been investigated in a range of psychological disorders and therapeutic approaches to learn about key moments in therapy when large and clinically meaningful improvements occur suddenly from one session to the next (Shalom & Aderka, 2020).

The following two chapters review the literature on longitudinal processes of clinical improvement (see Chapter 1.5) and sudden symptom improvements (see Chapter 1.6) during psychological therapies for PTSD.

1.5 Review of longitudinal processes of clinical improvement

To examine processes of change during psychological therapies for PTSD some studies have analysed associations between changes in theory-derived therapy processes and changes in PTSD symptoms during treatment. Several theories of PTSD highlight the central role of negative trauma-related appraisals in the development and maintenance of PTSD (e.g., Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1992). Consequently, most evidence-based psychological therapies for PTSD address negative appraisals either directly or indirectly during treatment (Schnyder et al., 2015). A meta-analysis of 16 randomised controlled trials including 994 participants highlighted that psychological therapies for PTSD are efficacious in reducing negative trauma-related appraisals (Diehle et al., 2014). In an overview of the recent literature discussing the role of negative appraisals in PTSD, McNally and Woud (2019) argue that associations between hypothesised processes and symptom improvements might suggest a causal link from cognition to symptoms, but it is also important to establish the temporal precedence and consider other important cognitive aspects of PTSD, for example characteristics of the trauma memory.

Brown et al. (2018) conducted a systematic literature review to examine psychological treatment studies for adolescents and adults that investigated associations between PTSD symptoms and negative appraisals. The authors included 65 PTSD studies in their review and found that 15 addressed the directionality of changes between symptoms and negative appraisals in some way. Out of these studies, 11 found evidence for change in appraisals preceding PTSD symptoms change, while four studies found evidence for a reverse or bidirectional relationship. Although these results provide evidence for the theoretical prediction that cognitive change precedes symptom change during psychological treatments of PTSD, it is important to note the differences in clinical samples, treatment approaches, methodological approaches, and investigated time intervals used to address this research question. Most studies were secondary analyses of randomised controlled trials comparing a

variety of treatment approaches with considerable differences in sample sizes (M = 118.53, SD = 64.89, Range = 29 to 268), while only few studies were done in routine clinical care (e.g., Kleim et al., 2013; Kumpula et al., 2017). More studies are needed to evaluate whether change processes identified in randomised controlled trials act in a similar way in routine clinical practice.

More importantly, to our knowledge, only seven studies are based on multiple assessments of negative appraisals and PTSD symptoms during the course of treatment in adults (Cooper, Zoellner, et al., 2017; Kleim et al., 2013; Kumpula et al., 2017; McLean, Su, et al., 2015; McLean et al., 2019; Trachik et al., 2018; Zalta et al., 2014), see Table 1.2 for a summary. Out of these studies, one found no evidence that cognitive changes in trauma-related guilt predicted PTSD symptoms at the following assessment point in active-duty military personnel and veterans (Trachik et al., 2018), and one study found a reciprocal relationship between negative appraisals and PTSD symptoms when investigating 4-week intervals during treatment (McLean, Su, et al., 2015). The other five studies provided evidence that session-by-session changes in negative appraisals preceded changes in PTSD symptoms. Kleim et al. (2013) specifically addressed the temporal associations between session-by-session changes in negative appraisals and PTSD symptoms in a sample of 268 participants receiving CT-PTSD in routine clinical care. The authors applied a bivariate latent growth modelling approach to first estimate the trajectories for PTSD symptoms and negative appraisals separately and then tested the temporal associations between changes in each construct. The results of this study suggest that changes in negative cognitions predicted subsequent changes in PTSD symptoms, whereas no evidence was found for the reverse relationship. Taken together, there is strong evidence that changes negative appraisals are driving changes in PTSD symptoms in a range of clinical samples and different cognitive behavioural treatment approaches.

Although most studies investigated the longitudinal associations between negative appraisals and PTSD symptoms during treatment implementing advanced statistical techniques (e.g., lagged mixed-effects models or bivariate latent growth

models), other statistical models can test more directly whether *changes* in negative appraisals (or other therapy processes) predict subsequent *changes* in PTSD symptoms. It is therefore important to test this relationship using statistical models that match more directly the predictions made by theoretical models of clinical improvement during cognitive therapy, while also considering other key cognitive and behavioural maintenance factors of PTSD. To be able to evaluate such models and review whether methods are appropriate and implemented correctly, it would be beneficial if the code for these analyses were openly available. However, to the best of our knowledge, no study that investigated the longitudinal associations of negative appraisals and PTSD symptoms has shared their analytic code publicly.

With few exceptions (e.g., McLean et al., 2019, who investigated emotion regulation in prolonged exposure therapy), other theory-derived process measures have rarely been assessed on multiple occasions during PTSD treatment. Most studies could therefore only analyse pre-treatment severity or changes from the beginning to the end of treatment in therapy process measures. Only by investigating the longitudinal associations of maintenance factors and PTSD symptoms during treatment can the change processes linked to clinical improvement be adequately evaluated.

Several psychological theories also highlight the importance of trauma memory characteristics and unhelpful coping strategies such as avoidance in the development and maintenance of PTSD (e.g., Brewin et al., 2010; Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1992). The disorganised nature of trauma memories plays a key role in Ehlers and Clark's cognitive model of PTSD. There has been a debate about the exact definition and assessment of the relevant aspects of memory disorganisation (see Brewin, 2016; Ehlers et al., 2012). Prospective studies have shown that individuals who report higher levels of disorganised or disjointed memories shortly after a traumatic experience develop more PTSD symptoms in the long term (e.g., Beierl et al., 2019; Halligan et al., 2003). A few studies, with small sample sizes, have provided mixed results on the changes in memory disorganisation and fragmentation during psychological treatment in adults (Bedard-Gilligan et al.,

2017; Foa et al., 1995; Kindt et al., 2007; Mundorf & Paivio, 2011; van Minnen et al., 2002) as well as children and adolescents (Kangaslampi & Peltonen, 2019; Meiser-Stedman et al., 2017). Some studies find that changes in narrative incoherence during treatment are associated with changes in PTSD symptoms (e.g., Mundorf & Paivio, 2011). Other studies do not find evidence for such association (e.g., Bedard-Gilligan et al., 2017). Different definitions and methods of assessing the extent of trauma memory disorganisation (e.g., independent coding of trauma narratives versus self-report measures) complicate the comparability between studies and most samples included only one type of trauma (cf. Bedard-Gilligan et al., 2017). To the best of our knowledge, only one study has investigated whether changes in PTSD symptoms during treatment are associated with subsequent changes in memory characteristics. In a sample of 29 children and adolescents Meiser-Stedman et al. (2017) found no evidence for this effect. Therefore, further studies with larger sample sizes are clearly needed.

Intrusive memories of the traumatic event are a core clinical feature of PTSD that can be very distressing for people with this disorder. Michael et al. (2005) highlighted that while intrusive trauma memories in people with and without PTSD have many common characteristics (e.g., sensory impressions), some characteristics of intrusive memories (e.g., nowness, distress, lack of context, and easy triggering) distinguish between those with and without PTSD. In a cross-sectional and prospective study, the authors found that these characteristics were an important predictor of PTSD symptoms. Some other studies explored session to session changes in these characteristics and showed that they improve during treatment. For example, Hackmann et al. (2004) and Speckens et al. (2006) found that the experienced nowness, distress, and vividness of intrusive memories decreased during CT-PTSD. However, these studies did not statistically test how changes in characteristics of intrusive memories relate to changes in PTSD symptoms, and samples were relatively small.

According to Ehlers and Clark (2000) individuals who engage in unhelpful cognitive and behavioural coping strategies with the aim of reducing perceived

threat or symptoms after trauma are at greater risk of persisting PTSD symptoms than those who do not. Prospective and experimental studies support the importance of cognitive coping strategies like *thought suppression*, *rumination*, and *emotional numbing* in preventing change in PTSD symptoms (e.g., Beierl et al., 2019; Ehring et al., 2008; Michael et al., 2007; Murray et al., 2002). Behavioural coping strategies like *safety seeking behaviours* have also shown to be important predictors of PTSD symptom severity in prospective studies (e.g., Beierl et al., 2019; Dunmore et al., 2001; Ehring et al., 2008), however less research has examined the role of cognitive coping strategies and safety seeking behaviours in the context of psychological treatments for PTSD. A treatment study of 95 veterans with PTSD receiving exposure therapy showed that reductions in safety behaviours were associated with lower depression and PTSD symptoms at post-treatment (Goodson & Haeffel, 2018). Brady et al. (2015) analysed video tapes of 58 patients receiving cognitive therapy for PTSD and found that higher levels of rumination and worrying during early sessions were associated with worse treatment outcomes. A better understanding of how changes in common coping strategies are related to changes in PTSD symptoms is needed to evaluate their role in clinical improvement. To the best of our knowledge, no study has yet investigated the temporal associations between changes in coping strategies and PTSD symptoms during therapy.

Table 1.2: Studies investigating lagged associations between negative appraisals and PTSD symptoms during treatment in adults

Study	Treatment	n	Assessment points*	Measures	Method	Findings
Cooper et al. (2017)	PE or Sertraline	134	10	PTCI/PSS-SR	Lagged mixed-effect model	Appraisals → PTSD
Kleim et al. (2013)	CT	268	10	PTCI/PDS	Bivariate latent growth curve	Appraisals → PTSD
Kumpula et al. (2017)	PE	46	4	PTCI/PDS	Lagged mixed-effect model	Appraisals → PTSD
Zalta et al. (2014)	PE	64	11	PTCI/PDS	Lagged mixed-effect model	Appraisals → PTSD
McLean et al. (2015)	PE or SC†	159	7	PTCI/PSS-I	Lagged mixed-effect model	Appraisals ↔ PTSD
Trachik et al. (2018)	EXP	42	2	TRGI/PCL-M	Multiple lagged regressions	Appraisals — PTSD
McLean et al. (2019)	PE or PCT	216	6	PTCI/PSS-I	Causal steps approach‡	Appraisals → PTSD

Note. * Assessment points during therapy that were included in the analysis investigating the lagged association. PE = Prolonged Exposure Therapy; CT = Cognitive Therapy; SC = Supportive Counceling; EXP = Exposure Therapy; PCT = Present-centered therapy. † PE or SC were delivered in combination with either naltrexone or a placebo. PTCI = Posttraumatic Cognitions Inventory; PSS-SR = Posttraumatic Stress Disorder Symptom Scale - Self Report; PSS-I = Posttraumatic Stress Disorder Symptom Scale - Interview; PDS = Posttraumatic Diagnostic Scale; PCL-M = PTSD Checklist - Military Version; TRGI = Trauma-related Guilt Inventory. ‡ Bootstrapped confidence intervals. → Unidirectional lagged relationship; ↔ Bidirectional lagged relationship. — No evidence for a lagged relationship.

1.6 Review of sudden symptom improvements

Sudden gains are large and stable symptom improvements experienced by a patient from one therapy session to the next. Tang and DeRubeis (1999) developed three criteria to identify sudden gains: (1) the gain should be large in absolute magnitude, (2) large relative to the previous symptom score, and (3) large relative to symptom fluctuation. The authors applied these criteria to a sample of 61 patients who received cognitive–behavioural therapy for depression and found that the 24 patients (39%) who experienced a sudden gain reported better outcomes at the end of treatment and at follow-up compared with all other patients who did not experience a sudden gain. Further, coding of video recordings of the sessions by independent raters showed that patients with sudden gains showed a greater shift in cognitions during the session immediately before the sudden gain in comparison to a control session within the same patients. Tang and DeRubeis (1999) found that patients reported an increase in therapeutic alliance immediately after the sudden gain and hypothesised that sudden gains lead to a better therapeutic alliance, which enables further improvements during therapy.

Several studies have replicated and expanded upon Tang and DeRubeis's (1999) findings and methods in different psychological treatments and disorders, primarily analysing data from randomised controlled trials, for example, posttraumatic stress disorder (PTSD, e.g., Kelly et al., 2009), generalised anxiety disorder (e.g., Deschenes & Dugas, 2013), social anxiety disorder (e.g., Hofmann et al., 2006), panic disorder (e.g., Clerkin et al., 2008), and obsessive–compulsive disorder (e.g., Aderka, Anholt, et al., 2011). The positive relationship between sudden gains and better outcomes at the end of therapy was replicated in treatments for depression, anxiety, and PTSD (for reviews see Aderka et al., 2012; Shalom & Aderka, 2020). A recent meta-analysis by Shalom and Aderka (2020) highlighted the clinical importance of sudden gains on treatment outcomes in 50 studies (Hedge's $g = 0.68$ at the end of treatment and Hedge's $g = 0.68$ at follow-up[1]).

[1]The length of follow-up assessments varied from 1.5 to 12 months ($M = 6.09$, $SD = 2.98$) between studies.

Similarly, six studies found that sudden gains in PTSD symptoms are linked to better treatment outcomes at the end of psychological therapy for PTSD (Aderka, Appelbaum-Namdar, et al., 2011; Doane et al., 2010; Jun et al., 2013; Kelly et al., 2009; König et al., 2014; Krüger et al., 2014), and only one study found no such association (Haugen et al., 2015), see Table 1.3. Out of the six studies reporting an association between sudden gains and posttreatment outcomes, two studies also reported an association between sudden gains and better outcomes at follow-up (Aderka, Appelbaum-Namdar, et al., 2011; Kelly et al., 2009), two reported no effect on follow-up measures (König et al., 2014; Krüger et al., 2014), and two did not investigate this question (Doane et al., 2010; Jun et al., 2013). To our knowledge, only two studies with relatively small samples ($n = 63$ and 26) investigated sudden gains in treatments for PTSD in routine clinical care (Aderka, Appelbaum-Namdar, et al., 2011; Doane et al., 2010). Further research in larger routine clinical care samples is needed to evaluate how common sudden gains are in routine clinical settings and how they are related to outcome.

Table 1.3: Studies investigating sudden gains in participants with PTSD

Study	Treatment	n	Sessions	SG measure	% with SG	Findings
Kelly et al. (2009)	CPT	24	12	PDS	46%	SGs associated with better PTSD and depression outcomes
	CPT-C	24	12	PDS	42%	-
	WA	26	6	PDS	31%	-
Doane et al. (2010)	PE	23	10	PSS-R	52%	SGs associated with better PTSD outcomes
Aderka et al. (2011)†	PE	63	12-15	CPSS	49%	SGs associated with better PTSD and depression outcomes
Jun et al. (2013)	PE	116	10	PSS-SR	42%	SGs associated with better PTSD outcomes
	Sertraline	84	10	PSS-SR	31%	-
Keller et al. (2014)	PE	116	10	BDI‡	21%	SGs associated with better depression outcomes
	Sertraline	84	10	BDI‡	14%	-
König et al. (2014)	DET	47	24	IES-R	23%	SGs associated with better PTSD outcomes
	CPT	55	24	IES-R	23%	-
Krüger et al. (2014)	DBT	34	24	PDS	25%	SGs associated with better PTSD outcomes
	TAU-WL	38	24	PDS	8%	-
Gibby et al. (2015)†	TF-CBT	74	20	CDI‡	35%	SGs not associated with better PTSD outcomes
	TF-CBT	74	20	IES	65%	SGs not associated with better depression outcomes
Haugen et al. (2015)	Diverse	36	19%	OQ-42.5‡	19	SGs not associated with better treatment outcomes

Note. SG = Sudden gain. SG measure = Measure used to identify sudden gains; % with SG = Percentage of patients experiencing at least one sudden gain; PE = Prolonged Exposure; TF-CBT = Trauma-focused Cognitive Behavioural Therapy; Diverse = Combination of psychodynamic and CBT; CPT = Cognitive Processing Therapy; CPT-C = Cognitive Processing Therapy (cognitive components only); WA = Written Account; DET = Dialogical Exposure Therapy; CPT = Cognitive Processing Therapy; DBT = Dialectical Behaviour Therapy; TAU-WL = Treatment as usual; CPSS = Child PTSD Symptom Scale; PSS-SR = Posttraumatic Symptom Scale-self-report; CDI = Children's Depression Inventory; IES = Impact of Events Scale; OQ-45.2 = Outcome Questionnaire-45.2; BDI = Beck Depression Inventory; PDS = Posttraumatic Diagnostic Scale; IES-R = Impact of Events Scale Revised. † Study with children or adolescent samples. ‡ Measure used to identify sudden gains does not specifically assess PTSD symptoms.

Most sudden gains studies based on the criteria suggested by Tang and DeRubeis (1999) provide information about which criteria were used to identify sudden gains and whether they were adapted. However, to the best of our knowledge, no study provides sufficient details about how the criteria were applied to the data to permit a full computational replication or made the analytic code that was used to identify sudden gains openly available. From the published literature it is not possible to know whether sudden gains were identified using programs like R, SPSS, Excel, or other methods. A freely available and open source solution to identifying sudden gains in longitudinal data could help to increase the transparency and comparability of sudden gains studies.

1.6.1 Cognitive processes associated with sudden gains[2]

An important question is what processes of change contribute to sudden gains. It has not as yet been examined how changes in cognitive factors thought to contribute to the maintenance of PTSD (e.g., negative appraisals or trauma memory characteristics; Brewin, 2014; Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1992) are associated with sudden gains in PTSD symptoms. These theories would predict that changes in these cognitive factors not only accompany sudden gains in symptoms but also predict them because changes in cognitions are thought to drive symptom change.

Change processes associated with sudden gains have primarily been investigated in studies in treatments for depression and social anxiety. Support for the hypothesis that cognitive change precedes sudden gains was found in some depression studies (e.g., Tang & DeRubeis, 1999; Tang et al., 2005), whereas other studies of patients with depression or social anxiety did not find such an association (e.g., Andrusyna et al., 2006; Bohn et al., 2013; Hofmann et al., 2006; Vincent & Norton, 2019). Reasons for the reported discrepancies in change processes associated with sudden

[2]The work presented in this chapter is based on published work: Wiedemann, M., Stott, R., Nickless, A., Beierl, E. T., Wild, J., Warnock-Parkes, E., Grey, N., Clark, D. M., & Ehlers, A. (2020). Cognitive processes associated with sudden gains in cognitive therapy for posttraumatic stress disorder in routine care. *Journal of Consulting and Clinical Psychology.* https://doi.org/10.1037/ccp0000488

gains in depression may partly be due to differences in the clinical samples and treatments. Replications of sudden gains studies in comparable clinical samples are scarce (cf. Wucherpfennig, Rubel, Hollon, et al., 2017). Further, the methods used to address the question of which processes are associated with sudden gains differ in the time points at which the process variables were measured (e.g., baseline differences, between-session changes immediately prior to the sudden gain, or within-session changes in the pregain session) and the methods to select a comparison group (within-patient comparisons, between-patient comparisons, or both). Although these methods aim to answer a similar research question, the differences are likely to influence the results and complicate the comparison between studies (Vincent & Norton, 2019; Wucherpfennig, Rubel, Hofmann, et al., 2017).

In addition to testing processes and predictors preceding sudden gains, recent studies have also investigated processes following sudden gains. Wucherpfennig, Rubel, Hofmann, et al. (2017) replicated Tang and DeRubeis's (1999) findings that sudden gains in depression lead to an improvement in the therapeutic alliance and further found that patients reported an increase in coping skills following sudden gains. Further research investigating how other clinically relevant factors change following sudden gains may help understand the processes of change.

To our knowledge, no study has yet investigated the cognitive changes associated with sudden gains in PTSD treatments. Cognitive change processes that may be related to sudden gains can be derived from Ehlers and Clark's (2000) cognitive model of PTSD. This model suggests that excessively negative appraisals of the trauma and/or its sequelae and certain characteristics of the trauma memory (disjointed recall of moments without context information, leading to a 'here and now' quality of the memories) play a major role in the maintenance of PTSD. Other cognitive-behavioural models of PTSD have also highlighted appraisal and memory processes (e.g., Brewin, 2014; Foa & Riggs, 1993; Resick & Schnicke, 1992). As reviewed above (see Chapter 1.5), prospective studies (e.g., Beierl et al., 2019; Ehring et al., 2008) and psychological treatment studies (e.g., Hackmann et al., 2004; Kleim et al., 2013; McLean, Yeh, et al., 2015) provide evidence in

support of predictions about maintenance factors of PTSD by cognitive-behavioural models. Investigating these maintaining factors in greater detail may help to better understand the processes involved in the occurrence of sudden gains.

Results regarding baseline predictors of sudden gains are inconsistent (for a review see Shalom & Aderka, 2020). Some studies found that higher quality of life and the absence of comorbidity were predictive of sudden gains in depression (Lemmens et al., 2016), or younger age in PTSD (Jun et al., 2013), whereas others found no baseline predictors (e.g., Aderka, Anholt, et al., 2011; Aderka, Appelbaum-Namdar, et al., 2011; Hunnicutt-Ferguson et al., 2012).

1.6.2 Individual symptom changes

Depression and anxiety disorders are often comorbid conditions in patients with PTSD that need to be addressed in treatment (Kessler, 1995; Kessler et al., 2005). Trauma-focused cognitive-behavioural therapies for PTSD are effective in reducing depression and anxiety symptoms as well as the core phenomena of PTSD, such as intrusive memories and avoidance of trauma reminders (e.g., Bisson et al., 2007). However, a more nuanced understanding of the relationships between improvements in PTSD symptoms and comorbid symptoms is needed. Some studies investigated sudden symptom improvements that are experienced by a subgroup of patients with PTSD to learn more about key moments in therapy (e.g., Jun et al., 2013; Keller et al., 2014). An example would be someone who lived with PTSD for many years, experiencing nightmares, flashbacks, avoidance, feelings of guilt and high arousal for much of the time and drastically improves during therapy from one treatment session to the next.

Recent studies aimed to identify predictors of sudden gains as well as associated change processes that facilitate and maintain sudden symptom improvements. Current studies use a binary grouping (*sudden gain* versus *no sudden gain*) to investigate the processes involved in sudden symptom changes. However, it is unclear whether sudden gains are a homogeneous phenomenon across participants and whether the same psychological processes are involved across all gains. Although there is

growing evidence for the importance of therapeutic alliance in the consolidation of symptom improvements made during sudden gains in depression symptoms (e.g., Wucherpfennig, Rubel, Hofmann, et al., 2017; Zilcha-Mano, Errázuriz, et al., 2019), findings regarding processes preceding sudden gains are less consistent.

Detailed examinations of exactly how individual symptom patterns change during sudden gains are rare, especially with respect to simultaneous changes in comorbid symptoms. If PTSD patients with sudden gains differ in the symptom patterns that bring about the sudden gain, it is possible that this may be related to different treatment-specific factors (e.g., updating trauma memories) or non-specific factors (e.g., the therapist's support). For example, a sudden symptom improvement that is mainly characterised by reductions in hyperarousal could be driven by dropping maintaining safety behaviours in a behavioural experiment – or a non-specific treatment process like feeling safe in the therapeutic relationship.

Sudden improvements in symptom severity

Three criteria were suggested by Tang and DeRubeis (1999) to identify sudden gains based on session to session changes in sum scores on the Beck Depression Inventory (BDI; Beck & Steer, 1993a), a 21-item measure to assess the severity of depressed mood. Subsequent studies generally followed the original criteria with some using adaptations due to specific research questions or methodological concerns (for a review see Shalom & Aderka, 2020). All adaptations however follow the original procedure to consider symptom severity as a sum measure of individual symptoms for identifying sudden gains and categorise participants primarily into two groups (*sudden gain* versus *no sudden gain*). The majority of sudden gains studies investigated sudden improvements (or deteriorations) in measures used to assess the severity of a particular psychological disorder that was addressed in treatment (e.g., PTSD, depression, or social anxiety). The methodological approach uses a summary statistic of a multiple-item measure (e.g., sum score) to detect meaningful changes in the latent construct that these items are measuring, however information about individual items or subgroups of items is lost. This may be

problematic if there are concerns that not all items are equally good indicators of the latent construct that is used to identify sudden gains. Given the current evidence it is unclear whether taking more information into account than just the sum score of one measure might be more suitable for some research questions when identifying and analysing sudden symptom improvements.

Considering individual PTSD symptoms

PTSD is very heterogeneous compared to other disorders (e.g., social phobia) with many different symptom combinations leading to the same diagnosis. Galatzer-Levy and Bryant (2013) highlight that due to the criteria of PTSD in the *Diagnostic and Statistical Manual of Mental Disorders*, the number of possible symptom combinations is considerably larger for PTSD in comparison to social phobia or depression. Although there are differences in how PTSD is classified (e.g., American Psychiatric Association, 2000, 2013; World Health Organization, 2018), all approaches agree on the core symptoms of PTSD (i.e., reexperiencing, avoidance, and hypervigilance/hyperarousal) and take into account that individuals can experience different symptom profiles. It is therefore also possible that participants experience sudden gains in PTSD symptoms in different ways.

Network studies of DSM-IV and DSM-5 PTSD symptoms also highlighted the heterogeneity of these PTSD diagnoses, especially with regards to which symptoms are most central (Birkeland et al., 2020). A review of 20 cross-sectional network studies of PTSD symptoms found that symptoms within each DSM-IV and DSM-5 cluster generally showed the strongest connections (Birkeland et al., 2020). One common finding however was that '*trauma amnesia*' was amongst the least connected symptoms in most network studies (Birkeland et al., 2020) – this observation is also consistent with theoretical criticism of this construct (e.g., Berntsen & Rubin, 2014; Rubin et al., 2008). In light of these findings, a better picture about which symptoms change during sudden gains is needed. Only one study investigated the associations between sudden gains and treatment outcome in different PTSD symptoms clusters. A study of 72 women receiving cognitive processing therapy found that sudden

gains were associated with better treatment outcome in avoidance and hyperarousal but not in intrusion symptoms (Kelley et al., 2009). To our knowledge no study has yet investigated individual symptom changes during sudden gains in PTSD symptoms, or in any other disorder.

Considering comorbid symptoms

O'Donnell et al. (2004) addressed the question of whether PTSD and depression are separate disorders following trauma by exploring the prevalence of PTSD, depression, and comorbid PTSD and depression, as well as investigating predictors of symptom severity and diagnosis. The authors analysed a sample of 363 injury survivors after the traumatic event and two follow-up assessments (3 and 12 months) and found that a similar proportion of participants developed PTSD (4%), PTSD with comorbid depression (6%), or depression (5%) at 3 months. Investigating predictors of these diagnostic categories and transitions between the follow-up assessments, the results of this study suggest that PTSD with and without comorbid depression describe a similar construct while depression describes a separate construct. Kleim et al. (2012) investigated predictors of the development of depression and PTSD 6 months in $n = 222$ assault survivors. The authors found that disorder-specific cognitive factors were best predictors of symptom severity, for example hopelessness and self-devaluative thoughts for depression and cognitive responses to intrusive memories and persistent dissociation for PTSD. The authors also found that comorbidity of PTSD and depression was high with around half (47%) of the participants with PTSD also meeting criteria for major depression and around two thirds (68%) of those with depression also meeting criteria for PTSD. The results of these studies suggest that while there are shared factors that predict the symptom severity of PTSD and depression, disorder-specific factors also exist. Some studies investigated the network structure of PTSD, depression, and anxiety symptoms. Price et al. (2019) analysed a sample of 1184 participants who met PTSD Criterion A of the DSM-5 to explore commonalities of PTSD, depression, and anxiety symptoms. The authors identified four groups of different PTSD symptoms (intrusions and

avoidance, hyperarousal, dysphoria, and negative affect) and one that consisted of all depression and anxiety symptoms. The anxiety symptom '*Inability to relax*' and the PTSD symptom '*Restricted or diminished positive emotion*' were the main connecting symptoms between these communities. Given the overlap between clinical constructs it is therefore of interest to explore how depression and anxiety symptoms change when investigating sudden gains in PTSD symptoms.

PTSD treatment studies provide some evidence that improvement in PTSD symptoms is accompanied by improvements in depression and anxiety symptoms (e.g., Ehlers et al., 2005; Ehlers et al., 2013; Liverant et al., 2012; Zoellner et al., 2019). Some PTSD sudden gain studies have also looked at the effect of sudden gains on treatment outcomes in comorbid symptoms (e.g., depression and anxiety) and generally found a positive association (Aderka, Appelbaum-Namdar, et al., 2011; Doane et al., 2010; Kelly et al., 2009). Two studies found that some participants also experienced sudden gains in depression symptoms during treatment for PTSD (8%, Kelly et al., 2009; 18%, Keller et al., 2014). While sudden gains in PTSD symptoms are often accompanied by meaningful improvements in depression or anxiety severity across patients, it is possible that this result is driven by a minority of cases. To our knowledge, no study has yet investigated comprehensively how a range of comorbid symptoms change when an individual experiences a sudden gain in PTSD symptoms. Studying individual symptom changes may reveal subcategories of sudden gains according to the relative degree of improvement in different PTSD or comorbid symptoms.

1.7 Aims of this thesis

This thesis presents a set of analyses of two large consecutive samples of patients ($N_1 = 330$; $N_2 = 343$) who received CT-PTSD in routine clinical care. The overall aim of the thesis is to test the predictions derived from Ehlers and Clark's (2000) cognitive model of PTSD that symptom changes during therapy are driven by change in negative appraisals, memory characteristics, and unhelpful cognitive

and behavioural coping strategies. To increase the transparency of my work and facilitate replications as well as further methodological improvements, I aim to develop software tools that implement the research methods used in this thesis.

Chapter 2 describes the development of two R packages. Chapter 2.2 presents the development of the R package *lcsm*. This package assists with the implementation of latent change score modelling, a method that allows to specifically test whether changes in one construct (e.g., a therapy processes variable like negative appraisals) are preceding changes in another construct (e.g. PTSD symptom severity). Chapter 2.3 reviews the methods used in previous studies to investigate sudden symptom improvements and presents the development of the R package *suddengains*, which fully automates the identification of sudden gains in longitudinal data.

Chapter 3 investigates whether changes in theory-derived therapy process measures precede changes in PTSD symptom during therapy. This study tests the key cognitive and behavioural processes through which CT-PTSD aims to drive clinical improvement according to the underlying cognitive model by Ehlers and Clark (2000).

Chapter 4 examines sudden and stable symptom improvements during CT-PTSD. Chapter 4.1 analyses associations between changes in negative appraisals and memory disorganisation around the time of sudden gains in PTSD symptoms. Chapter 4.2 investigates patterns of individual PTSD, depression, and anxiety symptom changes during sudden gains in PTSD symptoms.

Chapter 5 discusses the results from this thesis within the wider literature on Ehlers and Clark's (2000) cognitive model of PTSD and considers implications for current psychological treatments. Benefits of open research practices and developing research software in light of current challenges in psychological treatment research are also discussed. The chapter concludes with potential areas for future clinical and methodological research.

2

Development of R packages for implementing clinical research methodology

2.1 Methodological challenges in psychological treatment research

As well as the clinical research gaps reviewed in Chapters 1.5 and 1.6, there is scope to improve the implementation and reporting of statistical methods within psychotherapy research. In psychotherapy research questions such as '*How does this therapy work?*' are becoming more common and prominent. A range of primarily quantitative research methods are now being used to investigate how existing psychological therapies work. Most of these methods consist of multiple data analytic steps, involving data manipulations (e.g., restructuring data from '*wide*' format to '*long*' format) and sequential statistical analyses (e.g., longitudinal structural equation modelling) and therefore rely on analytic code.

Coding errors can happen at any point during the analysis and may remain unnoticed. In some cases the consequences can be significant. For example, Goldacre et al. (2019) described a randomised controlled trial where coding errors led to the experimental and control groups being reversed, ultimately requiring a retraction of

the paper (see Aboumatar & Wise, 2019). Coding errors are common and most researchers are not adequately trained to detect them (e.g., Wilson et al., 2014). It has been suggested that sharing the analytic code and research software tools that were used may assist in detecting coding errors and thereby improve the quality of the research (e.g., Goldacre et al., 2019; Naudet et al., 2018; Stodden et al., 2016).

Using and sharing analytic code can have benefits for the researcher (e.g., having a complete documentation of the analysis), as well as advantages for the wider research community (e.g., evaluating, replicating, or adapting a statistical method). Open analytical scripts do not only allow other researchers to evaluate the correctness of the code, but also allow them to replicate or build on the existing code if it is shared under an appropriate license (for a review see Fortunato & Galassi, 2020). Although there are some technical challenges when sharing analytic code, it has been argued that many of the common concerns (e.g., time investment) around sharing analytic code do not apply (e.g., Goldacre et al., 2019) and free solutions are available to help researchers share their analytic code online (e.g., Munafò et al., 2017; Perkel, 2019). In line with open research practices, sharing well-documented code along with the data is considered best practice (Peng, 2011), however data-sharing may not always be feasible or permitted and can be a complex issue. In that case, it has been argued that sharing the analytic code alone can be helpful too as it allows other researchers to evaluate or replicate the reported methods in more detail (Barnes, 2010; Minocher et al., 2020). In addition to improving transparency and reproducibility, sharing the analytic code may also assist other researchers in learning new methodological skills. Although the importance of sharing analytic code has been discussed before and is becoming the norm in some disciplines (e.g., Stodden et al., 2013), it is currently less common in psychotherapy research.

For example, to the best of our knowledge, none of the studies reviewed in this thesis, which investigated longitudinal processes of change or sudden gains during treatment for PTSD, publicly shared their analytic code. Furthermore, no sudden gains study based on the criteria suggested by Tang and DeRubeis (1999) has made their computational methods for applying the sudden gains criteria to

their data openly available. This is despite the fact that most studies analysing processes of change in psychological treatments for PTSD were performed using a statistical programming language that relies on analytic scripts (e.g., R or Python) or a program with the option to export scripts that represents the analysis (e.g., jamovi, JASP, SPSS). This means that it is currently hard to determine whether the described methods have been implemented as intended, and this subsequently complicates replications as well as comparisons across different studies.

Most studies reviewed in this thesis employ similar methodological approaches such as manipulating data to represent a time lag or identifying sudden gains. These analytic steps could be specified in publicly available code so that it is easier to perform the same analyses across different datasets (for reviews see Ince et al., 2012; Lowndes et al., 2017). Although a wide range of statistical analyses are implemented in open source software packages, more specialised analyses such as identifying sudden gains are not yet available.

The statistical programming language R (R Core Team, 2020) is popular among researchers in clinical psychology due to its comprehensive functionality, which can be extended through add-on R 'packages' to meet the requirements of specific research questions. Due to advances in software tools that simplify the development of such packages (e.g., Wickham, 2015; Wickham & Bryan, 2020; Wickham, Hester, et al., 2020) it is becoming easier for researchers without a formal training in programming to develop add-on R packages. These packages can be publicly shared with other researchers, for example through the Comprehensive R Archive Network (CRAN) or alternative online repositories.

To address some of the methodological gaps reviewed in Chapters 1.5 and 1.6, this Chapter describes the development of two R packages that facilitated the analyses of the studies presented in Chapters 3 and 4. The development of free and open source research software that assists with the implementation of longitudinal analyses and the identification of sudden gains may help researchers to be more efficient and improve the transparency as well as consistency of reporting of psychotherapy research studies.

2.2 *lcsm*: An R package for latent change score modelling

Different statistical methods have been used to investigate changes in psychological constructs over time (for reviews see Preacher, 2015; Usami et al., 2019). Longitudinal Structural Equation Modelling (SEM) has been particularly popular to examine longitudinal processes because of its flexibility to build statistical models that match a particular psychological theory. Many psychological theories make predictions about changes over time and testing these predictions using appropriate statistical models is important to evaluate the evidence of these theories.

Latent change score models (LCSMs) are used across disciplines in the behavioural sciences to study how constructs change over time (e.g., Hawley et al., 2017; Kievit et al., 2018; King et al., 2009; King et al., 2018). This framework can be extended to specifically examine how changes in one construct are associated with changes in another construct (Grimm et al., 2012), which can have advantages compared to other statistical techniques (e.g., autoregressive cross-lagged models). This approach may be particularly suitable to study a core question in psychotherapy research: 'Are changes in a therapy process (e.g., negative appraisals) related to subsequent changes in a treatment outcome measure (e.g., PTSD symptoms)?'. This chapter introduces the R package *lcsm*, a tool that aims to help users understand, analyse, and visualise different LCSMs.

2.2.1 Methodological overview

Latent change score modelling builds on concepts from classical test theory, which assumes that the observed score (X) of an individual (i) at a particular time (t) can be expressed as the individual's 'true score' (lx) and the individual's 'unique score' (u) at that time, see Equation (2.1).

$$X_{ti} = lx_{ti} + u_{ti} \tag{2.1}$$

The specification of the latent structures in LCSMs is so that the model can estimate a latent factor that captures the change in latent 'true scores' between two time points. By combining two longitudinal structural equation modelling methods, namely 'latent growth curve models' and 'autoregressive cross-lag models', LCSMs can provide a detailed examination of within-person changes in one (i.e., univariate) or more constructs (e.g., bivariate) over time (Grimm et al., 2017; McArdle & Hamagami, 2001). A common modelling approach is to first understand the individual trajectories of each construct in a univariate LCSM, before combining both models to examine their relationships in a bivariate LCSM. The notation of the parameters in this chapter mainly follow tutorials by Grimm et al. (2012) and Grimm et al. (2017).

Univariate LCSM

A univariate LCSM aims to describe the changes of individuals (i) in one construct (X) over time (t). The LCSM framework offers different options to describe this change: a constant change parameter ($\alpha_x \times s_{xi}$), a proportional change parameter ($\beta_x \times x_{[t-1]_i}$), and an autoregressive effect of the change scores ($\phi_x \times \Delta x_{[t-1]_i}$). Equation (2.2) shows how the change in one construct (X) at a specific time point (t) is constructed when using these three parameters. The constant change parameter alone is similar to linear change because it has the same effect on all change scores, see the paths from `g2` to the change scores `dx2` to `dx5` in Figure 2.1. Proportional change describes whether the 'change score' at time (t) is determined by the 'true score' of the same construct at the previous time point ($t-1$), see the paths labelled `beta_x` in Figure 2.1. Autoregressions of the change scores describe whether previous changes are associated with subsequent changes of the same construct, see the paths labelled `phi_x` in Figure 2.1. Note that in this example all parameters are constrained to be equal over time.

$$\Delta x_{[t]_i} = \alpha_x \times s_{xi} + \beta_x \times x_{[t-1]_i} + \phi_x \times \Delta x_{[t-1]_i} \tag{2.2}$$

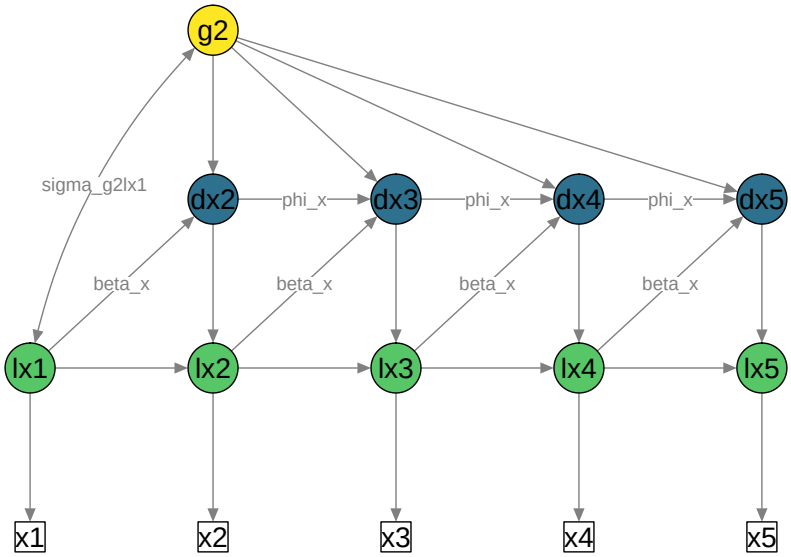


Figure 2.1: Simplified path diagram of univariate LCSM. White squares = Observed variables (x1 to x5); Green circles = Latent true scores (prefix 'l'); Blue circles = Latent change scores (prefix 'd'); Yellow circle = Constant latent change factor. Single-headed arrows = Regressions; Double-headed arrows = Covariance. `beta_x` = Proportional change factor; `phi_x` = Autoregression of change scores; `sigma_g2lx1` = Covariance of change factor (`g2`) with initial true score (`lx1`). Unique scores (ux_t) and unique variances (σ^2_{ux}) are not shown in this figure for simplicity. Note that this figure was created using the `plot_lcsm()` function of the *lcsm* package.

Bivariate LCSM

The univariate LCSM can be extended to a bivariate LCSM to examine associations between two constructs over time. Depending on the research question there are different 'coupling' options to model the associations between two constructs (for a review see Grimm et al., 2017). The following overview focuses on the extension that allows to examine how changes in one construct are associated with changes in another construct (Grimm et al., 2012). This particular bivariate LCSM can address research questions like: 'Do *changes* in negative appraisals precede *changes* in PTSD symptoms during therapy?'.

A simplified path diagram of a bivariate LCSM with five repeated measurements and these parameters is shown in Figure 2.2. Equation (2.3) shows how changes of a bivariate LCSM with lagged coupling parameters are constructed. The first line of each equation represents the parameters that describe the within construct changes, while the second line of each equation represents parameters that describe

the between construct coupling parameters. In this case, change in construct X now consist an additional element, that is change in the other construct (Y) at the previous time point ($\xi_{\text{lag}_{xy}} \times \Delta y_{[t-1]_i}$), see Equation (2.3a). The model that describes changes in construct Y, see Equation (2.3b), includes a constant change factor (α_y) and an autoregressive parameter of the latent change scores (ϕ_y). The parameter $\xi_{\text{lag}_{yx}} \times \Delta x_{[t-1]_i}$ estimates whether changes in construct Y at one time point (t) are determined by changes in construct X at the previous time point (*t-1*).

$$\begin{aligned} \Delta x_{[t]_i} = \alpha_x \times s_{xi} + \beta_x \times x_{[t-1]_i} + \phi_x \times \Delta x_{[t-1]_i} + \\ \xi_{\text{lag}_{xy}} \times \Delta y_{[t-1]_i} \end{aligned} \tag{2.3a}$$

$$\begin{aligned} \Delta y_{[t]_i} = \alpha_y \times s_{yi} + \phi_y \times \Delta y_{[t-1]_i} + \\ \xi_{\text{lag}_{yx}} \times \Delta x_{[t-1]_i} \end{aligned} \tag{2.3b}$$

It is also important to consider whether to examine concurrent or lagged relationships between two constructs. In some cases it may be desirable to examine concurrent relationships, for example when the underlying theory predicts that both constructs change simultaneously. For a more detailed discussion on this topic see Wang et al. (2009) and Goldsmith et al. (2018). Coefficients are usually constrained to be equal over time, but this can be changed by allowing variation between specific time points. For example, if the associations between two constructs could vary across different parts of the investigated time points (e.g., different treatment phases).

2.2.2 Why is a package needed?

Multiple script-based software packages support structural equation modelling and can be used for analysing LCSMs, for example *lavaan* (Rosseel, 2012), *OpenMx* (Neale et al., 2016), or *Mplus* (Muthén & Muthén, 2017). Other software packages like *JASP* (JASP Team, 2019) or Ωnyx (von Oertzen et al., 2015) also offer a graphical user interface for building and analysing structural equation models. The R package *RAMpath* (Zhang et al., 2015) offers a framework for analysing

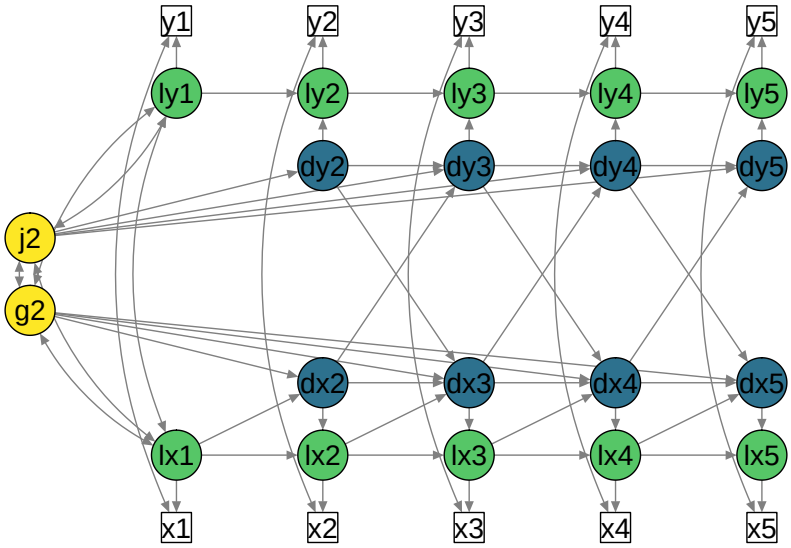


Figure 2.2: Simplified path diagram of bivariate LCSM with lagged change to change coupling parameters. White squares = Observed variables; Green circles = Latent true scores (prefix 'l'); Blue circles = Latent change scores (prefix 'd'); Yellow circles = Constant latent change factors. Single-headed arrows = Regressions; Double-headed arrows = Covariance. Unique scores (ux_t, uy_t) and unique variances (σ^2_{ux}, σ^2_{uy}) are not shown in this figure for simplicity. Note that this figure was created using the `plot_lcsm()` function of the *lcsm* package.

longitudinal structural equation models and it can also estimate basic univariate and bivariate LCSMs. Although there exist many tutorials on how to implement latent change score models in different software packages (e.g., Ghisletta & McArdle, 2012; Grimm et al., 2017; Kievit et al., 2018; Klopack & Wickrama, 2020), to the best of our knowledge, there is currently no tool that automatically generates syntax for LCSMs with different model specifications.

Specifying LCSMs in current software packages can be complex and cumbersome, especially with larger numbers of repeated measures and when testing sequential models with increasing complexity. Syntax for complex models can be hard to program, is lengthy, and thus prone to errors. For example, *lavaan* syntax for a bivariate LCSM with 10 repeated measures can consist of between 200 to 300 lines of code. Klopack and Wickrama (2020) also highlighted this drawback in a recent tutorial on latent change score modelling in *Mplus*, *'Models can be cumbersome to program in available software packages.'* (p. 100). A tool that generates syntax for different model specifications may not only help to streamline the analytic steps

Table 2.1: Main functions of the *lcsm* R package and their dependencies

Function	Description	Dependency‡
Specify lavaan syntax		
`specify_uni_lcsm()`	Specify *lavaan* syntax for univariate LCSM	*base*
`specify_bi_lcsm()`	Specify *lavaan* syntax for bivariate LCSM	*base*
Fit models		
`fit_uni_lcsm()`	Fit univariate LCSM	*lavaan*
`fit_bi_lcsm()`	Fit bivariate LCSM	*lavaan*
Extract results		
`extract_fit()`	Extract fit statistics from *lavaan* objects	*broom*
`extract_param()`	Extract parameter estimates from *lavaan* objects	*broom*
Simulate data		
`sim_uni_lcsm()`	Simulate data from univariate LCSM parameters	*lavaan*
`sim_bi_lcsm()`	Simulate data from bivariate LCSM parameters	*lavaan*
Additional functions		
`plot_trajectories()`	Plot individual trajectories of cases	*ggplot2*
`plot_lcsm()`	Plot simplified LCSM path diagram	*semPlot*

Note. More details about each function can be found in the package documentation or using the `help()` function in R. ‡ This column lists additional R packages that are required by the functions of the *lcsm* package.

involved in latent change score modelling, but also help researchers to reduce errors in code and facilitate a transparent way of reporting analyses.

2.2.3 Overview of the package

The *lcsm* package combines the strengths of existing R packages for SEM by providing a framework that makes these packages work together efficiently. The current version of the package (Version 0.1.3) provides a set of functions that help with visualising longitudinal data, generating *lavaan* syntax for different univariate and bivariate LCSMs, fit univariate and bivariate LCSMs, and extract estimated parameters as well as fit statistics. Further functions allow to plot simplified path diagrams and to simulate data to explore the effects of different parameters. An overview of the main functions and a short description is presented in Table 2.1. The interactive application *shinychange* (https://milanwiedemann.shinyapps.io/shinychange) supplements this package and illustrates how the *lavaan* syntax and path diagrams change depending on different model specifications.

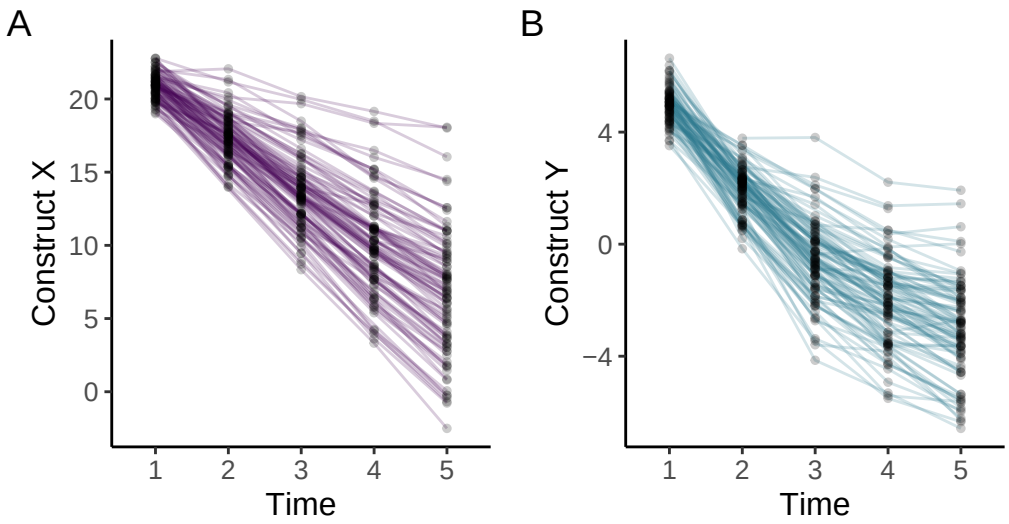


Figure 2.3: Longitudinal plots of five repeated measurements.

Visualise longitudinal data

Visualising individual trajectories of repeated measurements may be helpful to understand the data and inform modelling decisions. A more detailed overview of the rational for visualising longitudinal data is described in Ghisletta and McArdle (2012) and Grimm et al. (2017). The function `plot_trajectories()` offers an easy way to visualise longitudinal data. Figure 2.3 was created using this function to visualise individual trajectories of 5 repeated measurement for two constructs (X and Y) from an example dataset. This function is built using the R package *ggplot2* (Wickham, 2016) and additional *ggplot2* functions can be added to extend the plot.

Generate *lavaan* syntax for different LCSMs

The functions `specify_uni_lcsm()` and `specify_bi_lcsm()` can be used to generate *lavaan* syntax for different univariate and bivariate model specifications and varying time points. The LCSM parameters that should be included in the *lavaan* syntax can be specified as a `list()` using the `'model'` argument of these functions. For example, to include a constant and proportional change factor, the following argument needs to be added `'model = list(alpha_constant = TRUE, beta = TRUE)'`. For bivariate LCSMs 'coupling' parameters can be specified using the `'coupling'` argument, for example `'coupling = list(xi_lag_xy =`

Table 2.2: Available specifications for univariate LCSMs and bivariate coupling options

Option	Description
Univariate model options	
`alpha_constant`	Constant change factor
`alpha_piecewise`	Piecewise constant change factor
`alpha_piecewise_num`	Change point of piecewise constant change factor
`alpha_linear`	Linear change factor
`beta`	Proportional change factor
`phi`	Autoregression of change scores
Coupling options	
`coupling_piecewise`	Piecewise coupling parameters
`coupling_piecewise_num`	Change point of piecewise coupling parameters
`delta_con_xy`	Change score x (t) determined by true score y (t)
`delta_con_yx`	Change score y (t) determined by true score x (t)
`delta_lag_xy`	Change score x (t) determined by true score y (t-1)
`delta_lag_yx`	Change score y (t) determined by true score x (t-1)
`xi_con_xy`	Change score x (t) determined by change score y (t)
`xi_con_yx`	Change score y (t) determined by change score x (t)
`xi_lag_xy`	Change score x (t) determined by change score y (t-1)
`xi_lag_yx`	Change score y (t) determined by change score x (t-1)

Note. Covar = Covariance. More details about each model option as well as further customisations can be found in the package documentation using `help(specify_uni_lcsm)` or `help(specify_bi_lcsm)`. Bivariate model options allow for concurrent (`con`) and lagged (`lag`) coupling between two constructs.

`TRUE)'` to add a parameter that estimates whether changes in construct X at time point (t) are determined by changes in construct 'Y' at the previous time point ($t-1$). The *lcsm* package offers a concurrent (`con`) and lagged (`lag`) version of each coupling parameter.

The *lcsm* package adds comments to the *lavaan* syntax to provide more information about each section and to facilitate further manual adaptations (e.g., freeing up parameters that are fixed over time). Table 2.2 shows the full list of available univariate and bivariate model specifications that are implemented in the current version of the *lcsm* package. The interactive online application *shinychange* allows users to explore how the number of repeated measures and different parameters affect the *lavaan* syntax, see Figure A.1.

Fit models

The functions `fit_uni_lcsm()` and `fit_bi_lcsm()` can be used to estimate univariate and bivariate LCSMs using the *lavaan* package (Rosseel, 2012). By default Full Information Maximum Likelihood (FIML) is used to estimate a model. This method makes the assumption that data is Missing Completely at Random (MCAR) or Missing at Random (MAR) and includes individuals with incomplete data (Baraldi & Enders, 2010).

As shown in the univariate example below, the user specifies the dataset in wide[1] format using the `'data'` argument. A list of variables representing the repeated measures need to be specified in the `'var'` argument. The parameters for the univariate LCSM can be specified using the `'model'` argument in the same way described in the section above. The code example below shows how to fit a univariate LCSM. A simplified path diagram of specified model is shown in Figure 2.1.

```
# Fit univariate latent change score model
fit_uni_lcsm(data = data_bi_lcsm,
             var = c("x1", "x2", "x3", "x4", "x5"),
             model = list(alpha_constant = TRUE,
                          beta = TRUE,
                          phi = TRUE))
```

As shown in the example below, for bivariate LCSMs it is possible to specify two sets of repeated measures using the arguments `'var_x'` and `'var_y'` as well as two different univariate models (`'model_x'` and `'model_y'`). Furthermore, the argument `'coupling'` can be used to specify a list of between construct coupling parameters. Additional parameters can be added to the LCSM simply by adding it to the list of parameters and setting it to `TRUE`. For example, to also estimate a proportional change parameter in `'model_y'`, the element `'beta = FALSE'` needs to be changed to `'beta = TRUE'`. A simplified path diagram of the model specified in this example is shown in Figure 2.2.

[1] One row per individual and one column for each repeated measure.

```
# Fit bivariate latent change score model
fit_bi_lcsm(data = data_bi_lcsm,
            var_x = c("x1", "x2", "x3", "x4", "x5"),
            var_y = c("y1", "y2", "y3", "y4", "y5"),
            model_x = list(alpha_constant = TRUE,
                           beta = TRUE,
                           phi = TRUE),
            model_y = list(alpha_constant = TRUE,
                           beta = FALSE,
                           phi = TRUE),
            coupling = list(xi_lag_yx = TRUE,
                            xi_lag_xy = TRUE))
```

Extract fit statistics and parameter estimates

To evaluate how well a particular model fits the underlying data, one option is to compare different fit statistics of competing models. The function `extract_fit()` can be used to extract commonly used fit statistics of multiple LCSMs with increasing complexity. Parameter estimates of the best fitting model can be extracted using the `extract_param()` function. Names and descriptions of the available parameters are presented in Appendix A, see Table A.1. Both of these functions make use of the R package *broom* (Robinson et al., 2020).

Plot simplified path diagrams

Simplified path diagrams can be visualised using the `plot_lcsm()` function of this package. This function is built on the *semPlot* package (Epskamp, 2019) and can give an overview of the modelling parameters that were chosen for a particular model. Figures 2.1 and 2.2 presented earlier in this chapter were both created using this function. An interactive illustration of these functions is available in the online application *shinychange*, see Figure A.2 in Appendix A.

Simulate data

The functions `sim_uni_lcsm()` and `sim_bi_lcsm()` can be used to simulate data to explore the effect of different parameters. An interactive illustration of these functions is available in the online application *shinychange*, see Figure A.3 in Appendix A.

2.2.4 Discussion

Analysing the longitudinal relationships between changes in two constructs may help to better understand how they unfold over time. LCSMs are a specific form of longitudinal structural equation models that allow to examine this question. In psychotherapy research this would allow to test how changes in a specific therapy processes (e.g., negative appraisals) are associated with subsequent changes in a treatment outcome measure (e.g., PTSD symptoms).

This chapter addressed a small subset of the specifications available using a latent change score modelling approach. A more detailed overview of methodological background and further adaptations can be found elsewhere (e.g. Grimm et al., 2017). It is also important to mention that the interpretation of results from LCSMs can be difficult, especially when multiple parameters (e.g., constant change and proportional change) are used to examine change over time (see Clark et al., 2018; Jacobucci et al., 2019). The aim of this chapter was to provide an overview of the *lcsm* package and the specific subset of latent change score modelling that will be used in this thesis.

2.3 *suddengains*: An R package for identifying sudden gains[2]

Given the potential significance of sudden gains reviewed in Chapter 1.6, examining such events specifically may be informative in understanding when and why such large improvements occur, which could help to improve existing interventions.

[2]The work presented in this chapter is based on published work: Wiedemann, M., Thew, G. R., Stott, R., & Ehlers, A. (2020). suddengains: An R package to identify sudden gains in longitudinal data. *PLOS ONE*. https://doi.org/10.1371/journal.pone.0230276

Rates of sudden gains within published clinical studies vary considerably (e.g., 17.8% to 52.2% of participants; Aderka et al., 2012), which may partly be due to differences in the methods used to identify them. However, such differences are hard to examine given that sufficient methodological details to permit a comparison are not always reported. In addition, some studies have raised concerns about the validity of sudden gains identified through current methods, demonstrating that they can be found in placebo interventions and simulated datasets (Vittengl et al., 2005; Vittengl et al., 2015). This suggests that not all gains reflect meaningful change or show a causal association with the intervention being studied. This highlights the need to examine the presence and strength of these associations and to consider if the current methods of identification can be refined. The *suddengains* R package is the first software program to offer explicit and reproducible methods to automatically identify sudden gains, which may be valuable in improving methodological reporting and consistency across studies. It may also facilitate closer examination of the methods used to identify sudden gains, to help improve their validity and ensure that they more accurately reflect meaningful events. This article aims to provide an accessible overview of how sudden gains are calculated, describe the principal functions of the package, and give instructions on how to use these with longitudinal data. It is hoped that using this package will facilitate improvements in the efficiency, reporting, and reproducibility of sudden gains research.

2.3.1 Identification of sudden gains

Tang and DeRubeis (Tang & DeRubeis, 1999; Tang et al., 2005) suggested the following three criteria to identify sudden gains:

1. The gain must be large in absolute terms. While this was originally operationalised as a decrease of at least 7 points on the Beck Depression Inventory (BDI; Beck & Steer, 1993b), subsequent studies have generally used the Reliable Change Index (RCI; Jacobson & Truax, 1991) to define an appropriate cutoff for other scales Stiles et al. (2003). Further details are discussed below.

2. The gain must be large in relative terms. This is defined as a drop of at least 25% of the previous score.
3. The gain must be large relative to symptom fluctuation. Originally an independent t test was proposed to compare the size of the sudden gain with symptom fluctuation before and after the gain. This method was controversial given the assumption of independence of the measurements before and after the gain is not met (Tang, 2015; Vittengl et al., 2005). Consequently the wording of this criterion was updated by Tang and colleagues (Tang, 2015; Tang et al., 2005), though the calculations remained the same: The difference between the mean scores of the three measurements before the gain (M_{pre}), and the three measurements after the gain (M_{post}), must be greater than the pooled standard deviation of these two groups multiplied by a critical value of 2.776 (i.e., the two-tailed t statistic for $\alpha = 0.05$ and $df = 4$). The formula for criterion 3 is therefore:

$$M_{pre} - M_{post} > \text{critical value} * \sqrt{\frac{(n_{pre} - 1) * SD_{pre}^2 + (n_{post} - 1) * SD_{post}^2}{n_{pre} + n_{post} - 2}} \quad (2.4)$$

The criteria used to identify sudden gains vary between studies. For example, some studies have used different methods to define a cutoff value for criterion one (Doane et al., 2010; Lutz et al., 2013), criterion two was not included in some studies because of concerns about the impact of different response scales and data suggesting it has little effect on the number of gains found (Hardy et al., 2005), and studies have used different methods to select a critical value for use in criterion 3 (Lutz et al., 2013; Zilcha-Mano, Eubanks, et al., 2019), see Equation (2.4).

Defining a cutoff for the first criterion

Tang and DeRubeis (1999) originally defined a 7 point cutoff on the BDI for the first criterion based on frequency distribution plots of session to session change scores on the BDI in clinical trials. The authors reported that 7 BDI points approximately

reflected one standard deviation in clinical samples (Tang, 2015). Stiles et al. (2003) noted that 7 BDI points was close to the reliable change value reported in Barkham et al. (1996) and therefore used the RCI formula to define a cutoff for a new measure. Subsequent studies have generally adopted this approach. Jacobson and Truax (1991) proposed the following formula to test whether the observed pre to post change on a measure reflects more than just fluctuation due to measurement error:

$$\frac{\text{pre - post}}{S_{\text{diff}}} = \text{RCI} \tag{2.5}$$

Following Jacobson and Truax (1991), reliable change on a measure is present when:

$$\frac{\text{pre - post}}{S_{\text{diff}}} > 1.96; \text{ therefore} \tag{2.6}$$

$$\text{reliable change} > 1.96 \times S_{\text{diff}}; \tag{2.7}$$

where S_{diff} is the standard error of the difference between pre and post scores. Using the standard error of measurement (S_E), S_{diff} can be expressed as:

$$S_{\text{diff}} = \sqrt{2 \times (S_E)^2}; \tag{2.8}$$

where S_E is calculated using the standard deviation of the control group or normal population s_1 and the test-retest reliability of the measure (r_{xx}):

$$S_E = s_1\sqrt{1 - r_{xx}}; \tag{2.9}$$

Some studies have adapted this formula following suggestions from Martinovich et al. (1996) by replacing the test-retest reliability with the internal consistency

(α) and replacing the standard deviation of the normal population (s_1) with the standard deviation of the clinical sample at baseline (SD_{pre}) so that all statistics can be extracted from the sample data (König et al., 2014). Note that the use of the test-retest reliability or internal consistency when calculating S_E makes the assumption that the scale being examined is unidimensional, and that these reliability estimates remain constant over time, and between individuals. Exploring the factor structure and measurement invariance of the scale may be appropriate to examine if these assumptions hold.

$$S_E = SD_{pre}\sqrt{1-\alpha} \tag{2.10}$$

In the sudden gains literature different approaches have been used to define a cutoff for the first criterion using the RCI formula. Some studies (Doane et al., 2010; Jun et al., 2013) have used the standard error of the difference (S_{diff}) while others (Lutz et al., 2013; Zilcha-Mano, Eubanks, et al., 2019) have used the reliable change value ($1.96 \times S_{diff}$). When defining a cutoff it is important to consider the statistical assumptions involved, and to ensure that this value reflects a meaningful change (large in absolute terms) that is realistic in a session-by-session context for the intervention.

Missing data

Missing data, for example where a participant does not provide data on one or more occasions, need to be considered carefully when identifying sudden gains for several reasons. Firstly, depending on the number and pattern of missing data points for an individual, it may not be possible to identify sudden gains, see Table 2.4. Specifically, in order to estimate the standard deviation values in criterion 3, at least two of the three measurements immediately prior to the gain must be present, as well as at least two of the three measurements immediately following the gain. Some researchers have suggested that methods used to replace missing values, such as *last observation carried forward* or *multiple imputation*, may not be appropriate when identifying

sudden gains given the potential for additional gains to be detected based on data that were not provided by participants (Shalom et al., 2018; Tang et al., 2007).

Secondly, where values are missing in the period around the potential sudden gain, two approaches have been described to evaluate the stability of the change. Following the updated version of the third criterion by Tang and colleagues (Tang, 2015; Tang et al., 2005) some studies have used a critical value of 2.776 across all session to session intervals to check the stability (Zilcha-Mano, Eubanks, et al., 2019). An alternative approach adjusts the critical values used in criterion 3, see Equation (2.4), based on the data that were available in the period around the potential sudden gain (Lutz et al., 2013): Where no data are missing $t_{(4;97.5\%)} > 2.776$; where one data point is missing either before or after the gain $t_{(3;97.5\%)} > 3.182$; and where one data point is missing both before and after the gain $t_{(2;97.5\%)} > 4.303$. This method has been adopted in some subsequent studies (Wucherpfennig, Rubel, Hofmann, et al., 2017) and Chapter 4.

It is important to understand the reasons for missing data and consider whether methods to handle missing data need to be employed both at the identification stage and in subsequent analyses (Rubin, 1976; Schafer & Graham, 2002). Further research to examine the impact of missing data and different methods to handle missing data when identifying sudden gains would be beneficial.

Terminology

The naming of specific sessions (or measurement points) around the gain follows the convention that the session immediately prior to the gain is session N (also known as the *pregain* session), and the session immediately after is session N+1 (or *postgain* session). Other sessions are referred to in relation to session N (e.g., N-2, N+3).

Reversals

According to Tang and DeRubeis (1999) a sudden gain is counted as reversed if 50% of the improvement made during the gain was lost at any subsequent point. For example, where the sudden gain represents a drop from 40 to 30 points, the

gain is classed as having reversed if a score of 35 or more is observed at any later session. As discussed in Wucherpfennig, Rubel, Hofmann, et al. (2017) a reversal might not necessarily be a stable phenomenon. These authors modified this criterion by suggesting that a stable reversal is present when a reversal is also classified as a sudden loss (see below).

Sudden losses

Although less frequently studied than sudden gains, sudden losses represent the inverse phenomenon, where a participant shows a large and stable increase of scores on the outcome variable. While some authors invert the three sudden gains criteria (Krüger et al., 2014; Lutz et al., 2013), others further adjust the percentage threshold of the second criterion, e.g. 33% (König et al., 2014).

2.3.2 Why is a package needed?

As indicated by the criteria above, identifying sudden gains requires the application of each of the three criteria to each session to session interval, and that this is performed for each individual in a given dataset. A large number of calculations and extensive manipulation of data is therefore involved, particularly in larger datasets. Doing these data manipulations manually (e.g., in spreadsheets) can be extremely time consuming and lead to errors. It also means that certain methodological decisions, such as determining the critical value for the third criterion, or handling of participants with multiple gains, may not be addressed sufficiently or in a consistent way across studies. It is hoped that the use of the *suddengains* package will provide faster and more accurate calculations, as well as offering a transparent and consistent method to address these methodological considerations.

2.3.3 Worked example

This demonstration uses a dataset `'sgdata'` that was created to illustrate the functions of this package. The data show self-report weekly questionnaire scores for 43 participants who have received psychological therapy for depression. The

intervention lasted for 12 sessions, and each participant completed a set of outcome measures at the beginning of each session, including the BDI and a fictional secondary measure assessing rumination (RQ).

Functions of the *suddengains* package

The *suddengains* package provides a set of functions to calculate the presence of sudden gains (and sudden losses) within a longitudinal dataset, and to provide basic plots and descriptive statistics of the gains. It can also extract scores on secondary outcome or process measures around the period of each gain. Output files (in SPSS, Excel, or CSV formats) arranged by individual gain, or by person can be generated for further analyses in other programs. This package is supplemented by an interactive web application (Chang et al., 2020) *shinygains* that illustrates the main functions of this package at https://milanwiedemann.shinyapps.io/shinygains (see Appendix A, Figures A.4 and A.5). As it allows users to explore and understand the impact of different methodological choices, it may be useful in planning sudden gains studies. Table 2.3 lists and describes the main functions.

Table 2.3: Main functions of the *suddengains* R package

Function	Description
Identify sudden gains	
`define_crit1_cutoff()`	Uses modified RCI formula to determine a cutoff value for criterion 1
`check_interval()`	Checks if a given interval is a sudden gain/loss
`identify_sg()`, `identify_sl()`	Identifies sudden gains/losses
Create datasets	
`create_bysg()`, `create_byperson()`	Creates a dataset with one row for each sudden gain/loss or one row for each person
`extract_values()`	Extracts values on a secondary measure around the sudden gain/loss
Describe sudden gains	
`describe_sg()`	Generates summary descriptive statistics
`plot_sg()`	Creates plots of the average sudden gain
`plot_sg_trajectories()`	Creates plots of individual case trajectories
Additional functions	
`plot_sg_intervals()`	Visualise data that can be investigated for sudden gains/losses
`select_cases()`	Selects cases to be included in the sudden gains analysis based on different criteria
`write_bysg()`, `write_byperson()`	Exports CSV, SPSS, Excel, or STATA files of the sudden gains datasets

Note. More details about each function can be found in the package documentation or using the `help()` function in R.

Table 2.4: Data patterns required to identify sudden gains

	x_{n-2}	x_{n-1}	x_n	x_{n+1}	x_{n+2}	x_{n+3}
Pattern 1	○	●	●	●	●	○
Pattern 2	○	●	●	●	○	●
Pattern 3	●	○	●	●	●	○
Pattern 4	●	○	●	●	○	●

Note. x_{n-2} to x_{n+3} represent any six consecutive measurement points within the dataset. The minimum number of data points that must be present (●) in order to investigate the interval from x_n to x_{n+1} as a potential sudden gain is four, arranged in one of the patterns shown. Note that the pregain (x_n) and postgain (x_{n+1}) data points must always be present. ○ represents missing data

Preparation of data

The data to be analysed for sudden gains are arranged in wide format i.e., one row per participant, and one column for each questionnaire score at each measurement point. A unique identifier variable also needs to be included. Some researchers have specified a minimum number of measurement points that must be present for participants to be included, to ensure that they received a sufficient amount of the intervention being studied (Tang & DeRubeis, 1999). Alternatively it may be of interest to analyse all cases whose data are distributed such that at least one interval can be examined for a potential sudden gain, see Chapter 4.1. For all three criteria to be applied there must be data present for at least two of the three data points prior to, and two of the three following, the interval to be examined, see Table 2.4. The optional `select_cases()` function can be used to identify samples of cases for analysis who fulfil such conditions, though researchers should consider whether these methods are appropriate for the aims of the study.

Identification of sudden gains

The `identify_sg()` function applies the sudden gains criteria as specified by the user to each session to session interval in the dataset. As shown below, the user specifies: `'data'`, the dataset to use in wide format; `'sg_crit1_cutoff'`, the cutoff value to use for criterion 1 (which can be entered manually or calculated using the `define_crit1_cutoff()` function); `'sg_crit2_pct'`, the percentage change value to use for criterion 2 (0.25 by default); `'sg_crit3'`, whether or not to apply the third criterion (`TRUE` by default); `'sg_crit3_alpha'`, the alpha value to use when calculating the criterion 3 critical value (0.05 by default); `'id_var_name'`, the name of the unique identifier variable within the dataset; and `'sg_var_list'`, a list of the variables representing the span of sessions to be analysed, which is sessions 1 to 12 in this example. By default all functions that identify sudden gains apply the adjustment of the critical value in Equation (2.4) as described by Lutz et al. (2013). To turn off this adjustment and instead apply a manually defined critical value across all session to session intervals, the argument `'sg_crit3_adjust = FALSE'` can be included and `'sg_crit3_critical_value'` specified. Additional options to customise this analysis are discussed in the package documentation. An alternative function, `identify_sl()`, is identical to `identify_sg()` but applies the criteria in the inverse direction to calculate sudden losses. The function `check_interval()` can be used to examine whether a specific session to session interval is a sudden gain/loss.

The output data frame shows each session to session interval, for example `sg_2to3` representing the interval between sessions two and three. Variables indicate whether each of the three criteria were met and therefore whether a sudden gain was observed for each interval. Sudden gains are indicated by a value of 1, see Table 2.5. Examining this interval in our example data, we see that only `id` = 10 meets all three criteria, for `id` = 2 none of the three criteria can be tested, for `id` = 18 only the third criterion can not be tested, for all other participants at least one criterion is not met.

To permit further analysis of our data, we wish to obtain an output dataset containing both the original data and the newly identified sudden gains. As

Table 2.5: Illustration of identifying sudden gains

id	sg_crit1_2to3	sg_crit2_2to3	sg_crit3_2to3	sg_2to3
1	FALSE	FALSE	FALSE	FALSE
2	*NA*	*NA*	*NA*	*NA*
10	TRUE	TRUE	TRUE	TRUE
12	TRUE	FALSE	FALSE	FALSE
18	FALSE	FALSE	*NA*	*NA*
23	FALSE	FALSE	TRUE	FALSE

Note. For the variables testing the three sudden gains criteria, referred to by `crit1`, `crit2`, and `crit3` in the variable names `TRUE` indicates that the criterion is met, while `FALSE` indicates the criterion is not met. *NA* indicates that a particular criterion could not be tested for a sudden gain due to missing data.

participants may experience more than one gain, as in the present example, and to allow for different subsequent analyses, the package provides two options for output datasets: The `create_bysg()` function creates a dataset structured with one row per sudden gain, and the `create_byperson()` function creates a dataset structured with one row per person, indicating whether or not they experienced a sudden gain. The `'tx_start_var_name'` and `'tx_end_var_name'` arguments are used to specify the start and end of treatment (tx) variables, and `'sg_measure_name'` specifies the name of the measure used to calculate sudden gains.

```
# Identify sudden gains in measures from "bdi_s1" to to "bdi_s12"
# and create dataset with one row for each sudden gain
create_bysg(data = sgdata,
            sg_crit1_cutoff = 7,
            sg_crit2_pct = 0.25,
            sg_crit3 = TRUE,
            id_var_name = "id",
            tx_start_var_name = "bdi_s1",
            tx_end_var_name = "bdi_s12",
            sg_var_list = c("bdi_s1", "bdi_s2", "bdi_s3",
                            "bdi_s4", "bdi_s5", "bdi_s6",
```

Table 2.6: Description of variables created by the *suddengains* package

Variable name	Variable label
`id_sg`	Unique ID variable for every identified sudden gain / loss
`sg_crit123`	Indicates whether all applied sudden gain criteria were met (No = 0; Yes = 1)
`sg_session_n`	Pregain session number
`sg_freq_byperson`	Frequency of sudden gains / losses per person
`sg_bdi_2n`	Pre-pre-pre gain session score (N-2)
`sg_bdi_1n`	Pre-pre gain session score (N-1)
`sg_bdi_n`	Pre-gain session score (N)
`sg_bdi_n1`	Post-gain session score (N+1)
`sg_bdi_n2`	Post-post gain session score (N+2)
`sg_bdi_n3`	Post-post-post gain session score (N+3)
`sg_magnitude`	Raw magnitude of sudden gain
`sg_bdi_tx_change`	Total change during treatment
`sg_change_proportion`	Proportion of total change represented by the sudden gain
`sg_reversal_value`	Reversal value
`sg_reversal`	Indicates whether the reversal value was met at any point in treatment following the sudden gain (No = 0; Yes = 1)

Note. The variable names listed includeing `_bdi_` will reflect the name of the measure specified in the `sg_measure_name` arguement.

```
                        "bdi_s7", "bdi_s8", "bdi_s9",
                        "bdi_s10", "bdi_s11", "bdi_s12"),
          sg_measure_name = "bdi")
```

The new variables created by the `create_bysg()` and `create_byperson()` functions are described in Table 2.6. To continue working in another program (e.g., SPSS, STATA, Excel) the functions `write_bysg()` and `write_byperson()` can be used to export the datasets created in R (R Core Team, 2020) as *.sav*, *.dta*, *.xlsx*, or *.csv* files.

Analysis of sudden gains

In this example, we have calculated sudden gains based on depression scores using the BDI. In analysing these gains, we are interested in how rumination scores on the fictional RQ measure change around the period of the sudden gains in

depression. The `extract_values()` function extracts the RQ values from the three sessions before (N-2, N-1, N) and the three sessions after (N+1, N+2, N+3) each depression sudden gain. In the dataset that gets returned by this function we refer to these sessions as sg_bdi_**2n**, sg_bdi_**1n**, sg_bdi_**n**, sg_bdi_**n1**, sg_bdi_**n2**, and sg_bdi_**n3**, respectively. This function can be applied to either the `bysg` or `byperson` dataset. By default the extracted values will be added as new variables to the dataset used. Here we demonstrate applying this function to the `bysg` dataset, as shown in the code below. First, the RQ variables are added to the `bysg` dataset. Second, the `extract_values()` function is applied. Note that the list of RQ variables included in the `'extract_var_list'` argument must match those used for the `'sg_var_list'` argument used previously in the `create_bysg()` function. This means that the number of variables in these lists has to be identical and measured at the same time points. The output data frame can be saved as a new object, or the existing `bysg` object can be overwritten, as in this example. The RQ scores now in the `bysg` dataset can be examined, for example to look at the temporal relationship between changes in rumination and changes in depression symptoms.

The `describe_sg()` function provides descriptive statistics about the sudden gains based on the variables from the `bysg` and `byperson` datasets. For the present example, this function indicates that 16 of the 43 participants experienced a sudden gain, and 9 experienced more than one gain, leading to a total of 26 sudden gains within the data. Information on the mean gain magnitude and reversals is also provided.

The `plot_sg()` function plots the average sudden gain, and can be used to show the primary or secondary outcome measure data. The `'sg_pre_post_var_list'` argument specifies the pregain and postgain variables to be plotted, namely sessions N-2 to N+3. This function is built using the R package *ggplot2* (Wickham, 2016) and additional *ggplot2* functions can be added to the plot. It is also possible to plot the average gain magnitude of different groups (e.g., two treatment arms in a trial) in one figure by using the optional `'group'` argument.

An additional function, `plot_sg_trajectories()`, is available to plot the trajectories of a selection of individual cases within the dataset. This function can be paired with a filter command, for example `filter()` from the R package *dplyr* (Wickham, François, et al., 2020), to visualise trajectories of specific groups of participants. For example, all participants with more than one sudden gain, or all participants with a sudden gain between sessions 3 and 4.

Measuring the performance of the *suddengains* package

As described above, identifying sudden gains can take a long time if not fully automated. If the data is structured in an appropriate way (i.e., one participant per row and one repeated measure per column), sudden gains can be identified using the *suddengains* R package. To evaluate the computing time it takes to identify sudden gains, a benchmark test was conducted using the R package *microbenchmark* (Version: 1.4-7; Mersmann, 2019). Sudden gains were identified 100 times in nine datasets with varying sample sizes (100, 500, 1000) and repeated measurements (5, 10, 15). The average computing times indicate that it takes less than one second to identify sudden gains in a dataset with 100 participants and 10 repeated measurements (see Figure 2.4). The computing time to identify sudden gains increases with lager sample sizes and more repeated measurements. These results suggest that, using the *suddengains* R package, it would take less than 6 minutes[3] to identify sudden gains in all 50 studies that were reported in a recent meta-analysis by Shalom and Aderka (2020).

2.3.4 Discussion

The analysis of sudden gains and losses provides a detailed examination of within-participant changes during the course of an intervention, and may help to understand individual processes of change. The *suddengains* package aims to facilitate the computation of gains, which can be laborious and error-prone. It also aims to

[3]This calculation is based on the total number of participants $N = 6355$ and the average number of sessions across all studies $M = 15.11$ ($SD = 5.47$) reported in Shalom and Aderka (2020).

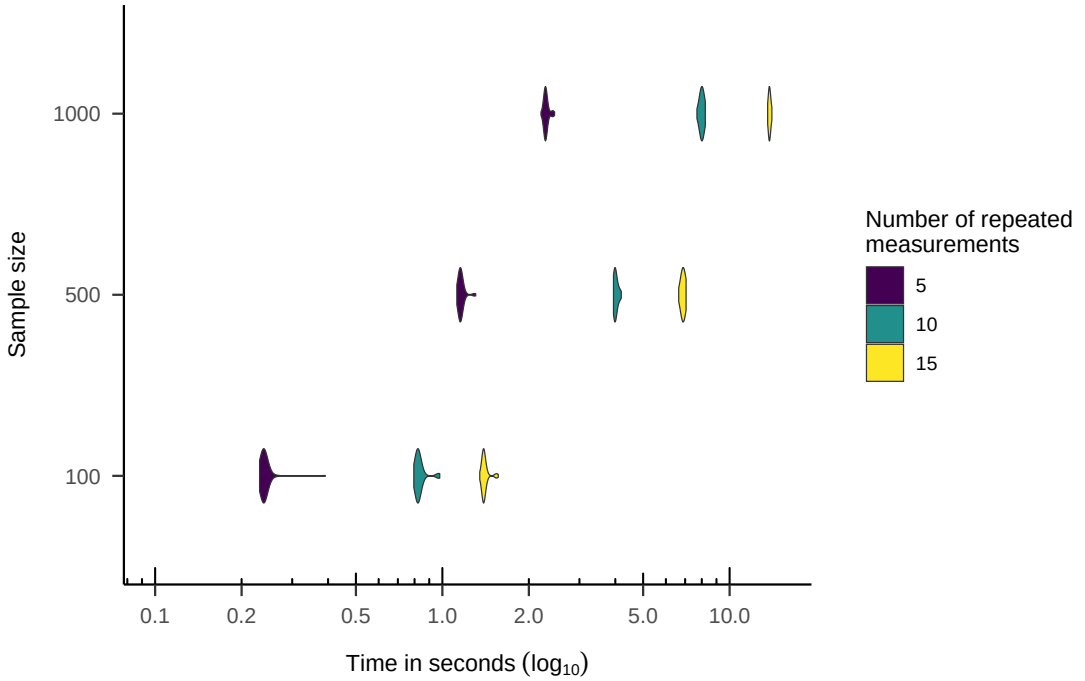


Figure 2.4: Benchmark of the *suddengains* package comparing differences in data input.

address common methodological issues, for example by allowing adjustments to the critical value for the third criterion in the presence of missing data, and by highlighting participants with multiple gains.

Limitations of the package include the fact that more substantial adaptations to the standard criteria cannot currently be implemented, though as the underlying code is publicly available, researchers may wish to use this in combination with other tools for further development work. Second, while the package may significantly increase the speed and accuracy of calculations, it cannot and should not substitute considered methodological thinking. In particular, users should consider carefully the appropriateness of the methods selected within each function, including related assumptions and limitations. Lastly it should be emphasised that sudden gains and losses identified by applying a set of mathematical criteria are not necessarily related to the effects of the intervention being studied, and that further investigation would be required to establish the presence and strength of evidence for a causal relationship.

More analytically advanced methods to detect changes exist within the field

of clinical psychology (for a review see Eubanks-Carter et al., 2012) and other disciplines, with implementations of these methods available through other R packages. For example multiple change point analysis (e.g., Lindeløv, 2020), indicator saturation analysis (e.g., Pretis et al., 2018), or interrupted time series analysis (e.g., Miratrix, 2020) can provide more information about specific aspects of the sudden change (e.g., the rate of change before and after the change point). Some of these methods require more repeated measures than commonly available in clinical trials with weekly measurements and might therefore not always be appropriate. Furthermore, these methods have not been developed to specifically detect meaningful changes in psychological constructs during therapy and would therefore have to be applied with caution and should be compared to sudden gains (Tang & DeRubeis, 1999) to evaluate potential differences in results.

3

Longitudinal processes of clinical improvement

3.1 Changes in cognitive processes and coping strategies

3.1.1 Aims

As discussed in Chapter 1.5, there are currently few studies that investigated how changes in cognitive processes and coping strategies are associated with subsequent changes in symptoms during cognitive therapy for posttraumatic stress disorder. Based on Ehlers and Clark's (2000) cognitive model of PTSD, the present study investigates changes in trauma-related negative appraisals, trauma memory characteristics, and cognitive and behavioural coping mechanisms over the course of the therapy, and how they temporally relate to changes in PTSD symptoms. Following previous research, the first aim was to test whether changes in PTSD symptoms are preceded by changes in trauma-related negative appraisals. While not definitive evidence of causality, such temporal precedence would suggest that change in trauma-related negative appraisals predicts change in PTSD symptoms. In line with Ehlers and Clark's model, we also tested how changes in trauma memory characteristics (flashback quality of memories and disorganised mode of

remembering the traumatic experience) and coping mechanisms (safety behaviours and unhelpful responses to intrusions, i.e., suppression, rumination, and numbing) relate to changes in PTSD symptoms. Testing the predictions by Ehlers and Clark's (2000) cognitive model of PTSD we hypothesised that changes in these PTSD process measures precede changes in PTSD symptoms. Finally, we investigated whether improvements in any predictors identified are also related to recovery.

3.1.2 Methods

Participants

Patients met criteria for PTSD as assessed by the Structured Clinical Interview for DSM-IV (First et al., 1997). This study is a secondary analysis of data drawn from a cohort study of 343 consecutive patients who were treated with CT-PTSD in routine clinical care (Ehlers, Wild, et al., 2020). Patients started treatment in a National Health Service outpatient clinic in South London between June 2009 and March 2013. Ethical approval was granted by the local research ethics committee.

For the analyses presented in this study, patients had to have available data for at least 5 sessions on both measures of interest (PTSD symptoms and the respective process measure - see the 'Measures' section below for more details) to be included in each set of analyses. A total sample of $n = 217$ patients were included in the analyses for this study, see Table 3.1 for patient characteristics.

Table 3.1: Demographic and clinical characteristics

	Sample 2 ($n = 217$)		
Variable	n	%	M (SD)
Age in years	217		37.47 (10.91)
Months since main traumatic event	216		53.38 (80.64)
Weekly treatment sessions	217		11.04 (4.32)
Gender			
Female	120	55.3%	
Male	97	44.7%	
Relationship			
Married/Cohabiting	86	39.6%	
Divorced/Separated/Widowed	23	10.6%	
Never married	100	46.1%	
No information	8	3.7%	

Table 3.1 continued

	Sample 2 (n = 217)		
Variable	n	%	M (SD)
Ethnicity			
Black	51	23.5%	
Caucasian	141	65.0%	
Indo-Asian	11	5.1%	
Other	14	6.5%	
Education			
University	68	31.3%	
A-levels	30	13.8%	
GCSE	48	22.1%	
Other	29	13.4%	
No information	42	19.4%	
Employment			
Employed/Self-employed	103	47.5%	
Sick leave	12	5.5%	
Disability/Retired	10	4.6%	
Unemployed	69	31.8%	
Student	9	4.1%	
No information	14	6.5%	
Type of main traumatic event			
Interpersonal violence	135	62.2%	
Accident or disaster	44	20.3%	
Death or harm to others	27	12.4%	
Other	11	5.1%	
Comorbid depression			
No	106	48.8%	
Yes	111	51.2%	
Comorbid anxiety			
No	108	49.8%	
Yes	109	50.2%	

Note. n = Number of available responses for each variable. % = Percentage of total sample included in this study. GCSE = General Certificate of Secondary Education.

Treatment

All patients underwent a course of CT-PTSD (Ehlers et al., 2005). Patients received on average 11.09 (SD = 4.27, Range = 5 to 33) weekly sessions. CT-PTSD aims to reduce the participant's sense of current threat by (1) changing problematic meanings of the trauma and its consequences, (2) elaborating and updating the memories of the trauma with information that gives them a less threatening meaning,

(3) discriminating triggers of intrusive memories, and (4) changing behaviours and cognitive processes that maintain PTSD. The therapy is tailored to each participant based on the individual case formulation, with the relative weights given to each treatment procedure differing between the individuals. Treatment usually started with the individual formulation, reclaiming your life assignments and the memory updating procedure. For comparability with Kleim et al. (2013) and to reduce the overall rate of missing data, only responses from the questionnaires filled in during the initial 10 weeks of the therapy were used for the current analysis.

Therapists

The therapists were qualified clinical psychologists, psychiatrist or nurse therapists or trainees in these professions. All therapists had completed at least basic training in cognitive behaviour therapy and a workshop on CT-PTSD. The majority of patients were treated by staff therapists. All cases were discussed in weekly supervision meetings and trainees also received individual case supervision.

Measures

All measures of PTSD symptoms and process measures were completed by the patients before they attended treatment sessions. The time frame of the questionnaires was the past week. For the current analysis we used mean scores across all items of each questionnaire to aid the interpretation of the therapeutic improvements and reduce the variance of the scores to facilitate the estimation of parameters. The item wordings for all therapy process measures that were used in this study are available at https://oxcadatresources.com/.

PTSD symptoms. The Posttraumatic Diagnostic Scale (PDS; Foa et al., 1997) assessed the PTSD symptoms specified in DSM-IV (American Psychiatric Association, 2000). Patients were asked to rate how much they were bothered by each of the 17 symptoms in the past week on a scale from 0 (*Not at all*) to 3 (*5 or more times a week*). The internal consistency at baseline was Cronbach's $\alpha = .89$.

Negative appraisals. Negative trauma-related appraisals were assessed with a short 20-item version of the Posttraumatic Cognitions Inventory (PTCI; Ehlers, 2020). Patients rated how much they agreed with the statements representing a range of cognitive themes: vulnerable self, self-criticism, overgeneralised danger, preoccupation with unfairness, perceived permanent change, alienation, hopelessness and negative view of body, each from 1 (*Totally disagree*) to 7 (*Totally agree*). The internal consistency at baseline was Cronbach's $\alpha = .91$.

Memory disorganisation. Disorganisation of patients' trauma memories was assessed using a 5-item version of the Trauma Memory Questionnaire (TMQ; adapted from Halligan et al., 2003). Patients rated the extent of the disorganisation of their memories of the traumatic experiences on 5 items ranging from 0 (*Not at all*) to 4 (*Very strongly*). The internal consistency at baseline was Cronbach's $\alpha = .84$.

Flashback memories. Patients reported characteristics of their intrusive trauma memories on the Unwanted Memories Questionnaire (UMQ; adapted from Hackmann et al., 2004). Patients were asked to report the perceived nowness, disjointedness, vividness, distress and ease of triggering of their main intrusions, each ranging from 0 (*Not at all*) to 100 (*Very strongly*). The scores of this measure were divided by 10 to facilitate parameter estimation in our analyses. The internal consistency at baseline was Cronbach's $\alpha = .82$.

Responses to intrusions. Unhelpful responses to intrusions were assessed with a short 12-item version of the Responses to Intrusions Questionnaire (RIQ-S; adapted from Clohessy & Ehlers, 1999; Murray et al., 2002). Patients were asked to rate to what extent items measuring suppression, rumination, and emotional numbing applied to them on a scale from 0 (*Never*) to 3 (*Always*). The internal consistency at baseline was Cronbach's $\alpha = .81$.

Safety behaviours. Safety behaviours were assessed using a short 7-item version of the Safety Behaviours Questionnaire (SBQ-S; adapted from Dunmore et al., 1999, 2001). Patients were asked to indicate how often they take extra precautions a scale from 0 (*Never*) to 3 (*Always*). The internal consistency at baseline was Cronbach's $\alpha = .85$.

Statistical analysis

All analyses were performed in R (Version 4.0.2; R Core Team, 2020) through R Studio IDE (Version 1.3.1073; RStudio Team, 2020). All LCSMs were estimated using the R package *lavaan* (Version 0.6.7; Rosseel, 2012) and model syntax was generated using the R package *lcsm* (Version 0.1.3; Wiedemann, 2020, see Chapter 2.2). Survival analyses were conducted using the R package *survival* (Version 3.1-12; Therneau, 2020).

Univariate latent change score models (LCSM) were used to estimate the change for each construct separately and bivariate LCSM were used to explicitly test the longitudinal associations between changes in therapy process measures and PTSD symptoms during treatment (Grimm et al., 2012; McArdle, 2009). To interpret changes in the mean score of PTSD symptoms and process measures, we assumed that these measures represent the same construct at each treatment session (i.e., longitudinal measurement invariance). First, univariate LCSMs were fit for PTSD symptoms and each therapy process measure separately to determine how each construct changed during treatment independently. Taking into account previous findings about early changes in symptoms and cognitive processes during cognitive therapies for PTSD (e.g., Kleim et al., 2013; Macdonald et al., 2011) and the therapy techniques used predominantly in early versus later sessions of CT-PTSD, we allowed changes in PTSD symptoms and all process measures to vary between the first (Sessions 1 to 5) and second (Sessions 5 to 10) part of therapy.

Next, bivariate LCSMs were used to evaluate the temporal associations between changes in PTSD symptoms and each cognitive process separately. The best fitting univariate LCSM for each construct was selected and lagged coupling parameters

were added between the constructs to test the hypothesised effect that changes in PTSD symptoms (ΔPTSD symptoms$_{(t)}$) are determined by prior changes in each cognitive process (ΔCognitive process$_{(t-1)}$), here described by parameter $\xi_{\text{lag}_{xy}}$ (ΔCognitive process$_{(t-1)} \rightarrow \Delta$PTSD symptoms$_{(t)}$). In order to contrast this with the alternative explanation – that changes in PTSD symptoms drive changes in each cognitive process - we also tested the reverse relationship described as parameter $\xi_{\text{lag}_{yx}}$ (ΔPTSD symptoms$_{(t-1)} \rightarrow \Delta$Cognitive process$_{(t)}$) and a bidirectional relationship by adding both parameters $\xi_{\text{lag}_{xy}}$ and $\xi_{\text{lag}_{yx}}$. In order to simplify the model interpretation and permit its full identification, several restrictions were imposed on the univariate and bivariate LCSMs following methodological recommendations (Grimm et al., 2017) and similar clinical studies (Hawley et al., 2017). These included fixing autocorrelations within constructs and covariances of residuals between constructs across time. Lagged coupling parameters were set to equal throughout therapy suggesting that improvement in process measures would predict improvement in symptoms similarly, except for when the therapy content justified that this effect may act differently during specific parts of the treatment. Because most memory updating work was conducted in the early phase of treatment and is different to further memory work conducted in the later phases of therapy (Ehlers, 2015; Ehlers et al., 2005), we allowed the cross-lagged coupling effects between changes in memory characteristics and PTSD symptoms to vary during the first and second part of therapy. Simplified path diagrams illustrating differences in modelling strategies between memory characteristics and other PTSD therapy process measures are presented in Figures 3.1C and 3.1D. Given some data missingness, all models were estimated using the Full Information Maximum Likelihood (FIML) estimator. We conducted likelihood ratio tests for competing models that were nested and also considered different types of absolute and comparative fit indices to determine the best fitting univariate and bivariate LCSMs: Models with smaller values on the Akaike Information Criterion (AIC; Akaike, 1974) and Bayesian Information Criterion (BIC) indicate better model fit, values ≥ 0.95 on the Comparative Fit Index (CFI; Bentler, 1990) and Tucker-Lewis Index (TLI; Tucker & Lewis, 1973) suggest

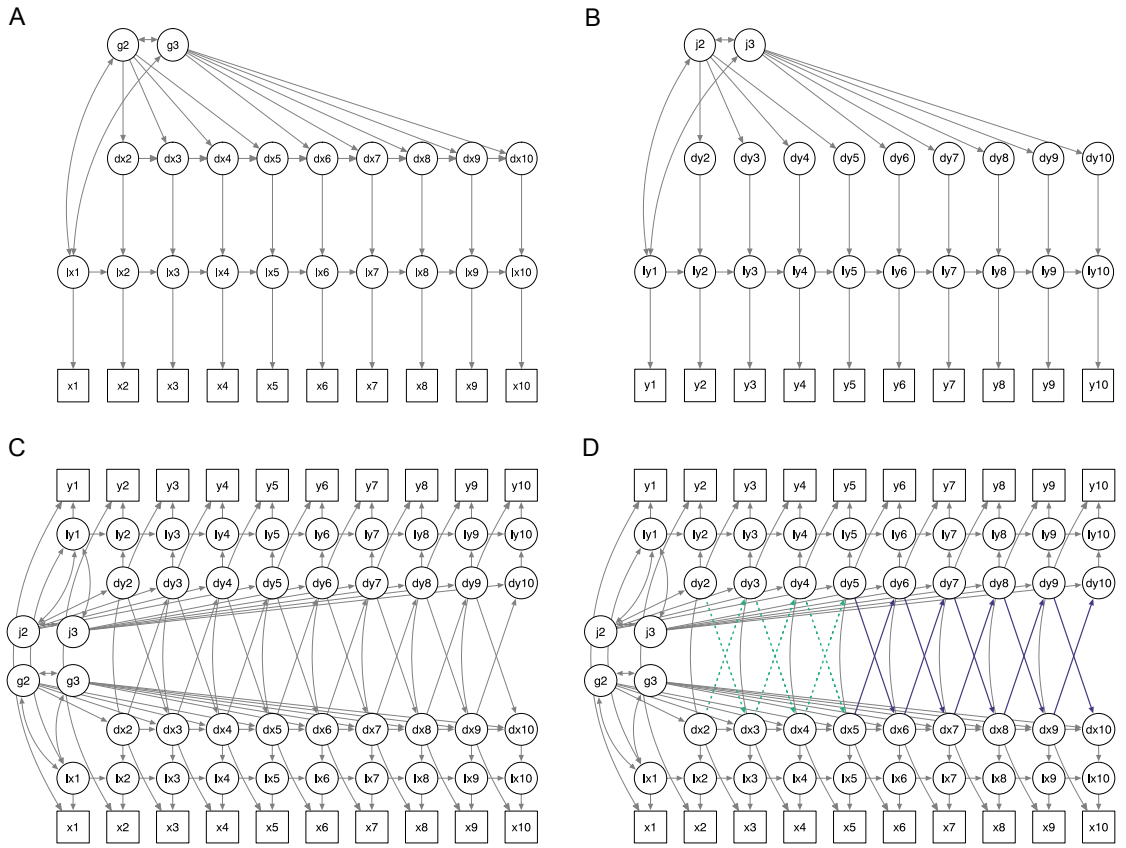


Figure 3.1: Simplified path diagrams for univariate and bivariate LCSMs. Univariate LCSMs (A) including and (B) not including autoregressions of change scores. Bivariate LCSMs (C) restricting coupling parameters over the entire treatment and (D) restricting coupling parameters for the first (dotted green line) and second (solid purple line) part of treatment. Squares = Observed variables; Circles = Latent variables; Single-headed arrows = Regressions; Double-headed arrows = Covariance. 'x' (PTSD symptoms) and 'y' (Process measures) represent the measured variables, the prefix 'l' indicates the latent construct and the prefix 'd' indicates latent change scores. 'g' and 'j' represent constant change factors.

good model fit, and values ≤ 0.10 on the root mean square error of approximation (RMSEA; Steiger & Lind, 1980) suggest adequate fit.

Finally, we investigated the extent to which changes in the cognitive and behavioural processes shown to precede symptom change in the LCSM analyses were associated with recovery from PTSD, rather than just a subsequent improvement in symptoms. To this end we conducted survival analyses using Cox Proportional Hazard Models, looking at whether session-by-session improvements in these cognitive and behavioural processes are associated with PTSD recovery. Significant

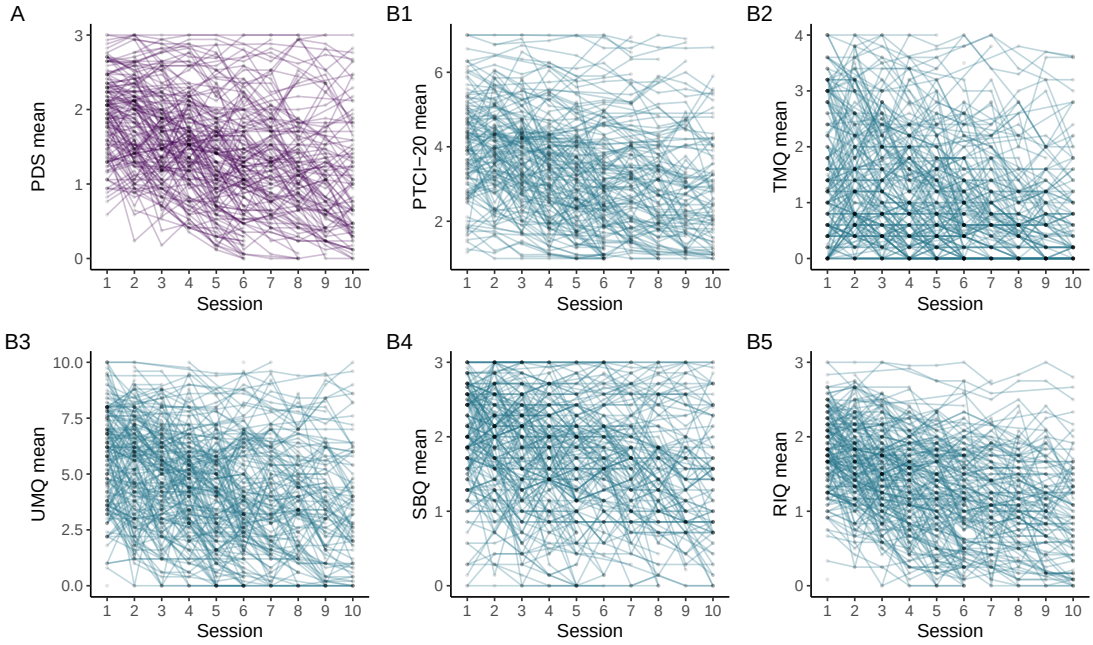


Figure 3.2: Observed individual trajectories in PTSD symptoms and process measures during therapy for 70% of the sample. PDS = PTSD symptoms; PTCI-20 = Negative appraisals; RIQ = Responses to intrusions; SBQ = Safety behaviours; TMQ = Disorganised memories; UMQ = Flashback memories.

processes from separate Cox Proportional Hazard Models were also analysed in a joint model to explore whether they are acting through the same or different pathways. Session scores of the process measures were entered into the models as time-dependent covariates. PTSD recovery was operationalised as showing reliable improvement[1] and scoring less than 18 points on the PDS (see Ehring et al., 2007). Data for each participant was considered until the first occurrence of the 'recovery'.

3.1.3 Results

Changes in PTSD symptoms and process measures during therapy

Given different patterns of questionnaire completion among the patients the sample size varies slightly between the analyses ($n_{\text{PDS–PTCI}} = 212$; $n_{\text{PDS–SBQ}} = 211$; $n_{\text{PDS–RIQ}} = 215$; $n_{\text{PDS–UMQ}} = 204$; $n_{\text{PDS–TMQ}} = 212$). Mean scores of PTSD symptoms and all process measures decreased over the first ten therapy sessions (see Figure 3.2).

[1]Defined following Foa et al. (1997) and Foa et al. (2002).

Table 3.2: Parameter estimates for chosen univariate LCSM of PTSD symptoms

	PDS		
Parameter	EST	SE	p
Initial status mean (γ_{lx1})	1.92	0.04	$< .001$
Initial status variance (σ^2_{lx1})	0.29	0.03	$< .001$
Observed scores variance (σ^2_{ux})	0.06	0.00	$< .001$
Constant change 1 mean (α_{g2})	-0.10	0.02	$< .001$
Constant change 2 mean (α_{g3})	-0.06	0.02	.005
Constant change 1 variance (σ^2_{g2})	0.01	0.00	.004
Constant change 2 variance (σ^2_{g3})	0.00	0.00	.060
Initial status with constant change 1 ($\sigma_{g2,lx1}$)	-0.01	0.00	.195
Initial status with constant change 2 ($\sigma_{g3,lx1}$)	-0.01	0.00	.090
Constant change 1 with 2 ($\sigma_{g2,g3}$)	0.00	0.00	.396
Autoregression of change scores (ϕ_x)	0.38	0.16	.020

Note. EST = Estimated parameter; SE = Standard error; PDS = PTSD symptoms.

Parameter estimates for all univariate LCSMs are presented in Tables 3.2 and 3.3, model fit statistics can be found in Appendix B (see Table B.1). For all measures, the greatest improvements occurred in the initial five weeks of therapy (Constant change 1 mean: α_{g2}), with a slower improvement afterwards (Constant change 2 mean: α_{g3}). Patients varied significantly in their change scores during the first (Constant change 1 variance: σ^2_{g2}) and second (Constant change 2 variance: σ^2_{g3}) part of therapy on all measures. Univariate latent change score models also suggested that patients with high scores in therapy processes showed slower improvement in these processes during the first ($\sigma_{g2,lx1}$) or second part ($\sigma_{g3,lx1}$) of therapy[2] – we did not find evidence for this effect for PTSD symptoms. The best fitting model for PTSD symptoms also suggested that changes in PTSD symptoms were significantly correlated with subsequent changes in PTSD symptoms (Autoregression of change scores: ϕ_x), i.e., patients with large improvements in symptoms at a certain session also showed large improvements during the following sessions.

[2]Pretreatment levels of negative appraisals (PTCI-20) and disorganised memory (TMQ) were significantly correlated with changes in corresponding processes during the first and second part of therapy; pre-treatment flashback memories (UMQ) only correlated with changes in subsequent flashbacks during the first part of therapy; and responses to intrusions (RIQ) and safety behaviours (SBQ) only correlated with changes in the second part of therapy.

Table 3.3: Parameter estimates for chosen univariate LCSMs of process measures

	PTCI-20			TMQ			UMQ			RIQ			SBQ		
Parameter	EST	SE	p	EST	SE	p	EST	SE	p	EST	SE	p	EST	SE	p
Initial status mean (γ_{lx1})	4.07	0.09	< .001	1.58	0.08	< .001	5.93	0.16	< .001	1.78	0.04	< .001	2.02	0.05	< .001
Initial status variance (σ^2_{lx1})	1.60	0.15	< .001	1.25	0.10	< .001	4.22	0.47	< .001	0.28	0.03	< .001	0.41	0.05	< .001
Observed scores variance (σ^2_{ux})	0.23	0.02	< .001	0.16	0.01	< .001	1.21	0.12	< .001	0.07	0.01	< .001	0.09	0.01	< .001
Constant change 1 mean (α_{g2})	-0.22	0.02	< .001	-0.09	0.02	< .001	-0.39	0.04	< .001	-0.13	0.01	< .001	-0.08	0.01	< .001
Constant change 2 mean (α_{g3})	-0.14	0.01	< .001	-0.11	0.01	< .001	-0.32	0.03	< .001	-0.08	0.01	< .001	-0.08	0.01	< .001
Constant change 1 variance (σ^2_{g2})	0.06	0.01	< .001	0.04	0.01	< .001	0.20	0.04	< .001	0.01	0.00	< .001	0.01	0.00	< .001
Constant change 2 variance (σ^2_{g3})	0.02	0.01	< .001	0.02	0.00	< .001	0.07	0.02	< .001	0.01	0.00	< .001	0.01	0.00	< .001
Initial status with constant change 1 ($\sigma_{g2,lx1}$)	-0.08	0.03	.001	-0.12	0.02	< .001	-0.28	0.11	.011	-0.01	0.01	.119	-0.01	0.01	.454
Initial status with constant change 2 ($\sigma_{g3,lx1}$)	-0.04	0.02	.042	-0.06	0.02	< .001	-0.01	0.08	.862	-0.01	0.00	.038	-0.01	0.01	.038
Constant change 1 with 2 ($\sigma_{g2,g3}$)	0.01	0.01	.008	0.00	0.00	.386	0.03	0.02	.134	0.00	0.00	.015	0.00	0.00	.777

Note. EST = Unstandardised estimated parameter; SE = Standard error; PTCI-20 = Negative appraisals; TMQ = Disorganised memories; UMQ = Flashback memories; RIQ = Responses to intrusions; SBQ = Safety behaviours. - indicates parameter was not estimated.

Associations between changes in PTSD symptoms and process measures during therapy

Parameter estimates for all bivariate LCSMs are presented in Table 3.4 and model fit statistics are shown in Appendix B (see Table B.2). For all models the covariances of residuals between PTSD symptoms and the process measures (σ_{su}) were significant. Also the covariances of the intercepts between PTSD symptoms and process measures ($\sigma_{ly1,lx1}$) were significant in all models, indicating that patients who report higher levels of PTSD symptoms at the beginning of treatment also show higher scores in all PTSD process measures.

Table 3.4: Parameter estimates for chosen bivariate LCSMs

	PTCI-20 - PDS			TMQ - PDS			UMQ - PDS			RIQ - PDS			SBQ - PDS		
Parameter	EST	SE	p	EST	SE	p	EST	SE	p	EST	SE	p	EST	SE	p
Covariance of residuals x and y (σ_{su})	0.04	0.01	$< .001$	0.03	0.01	$< .001$	0.10	0.01	$< .001$	0.02	0.00	$< .001$	0.02	0.00	$< .001$
Covariance of initial status x and y ($\sigma_{ly1,lx1}$)	0.53	0.06	$< .001$	0.28	0.04	$< .001$	0.80	0.09	$< .001$	0.23	0.03	$< .001$	0.22	0.03	$< .001$
Session 1 to 10 (Δ)															
ΔCognitive Process$_{t-1} \rightarrow \Delta$PTSD$_t$ ($\xi_{\text{lag}_{xy}}$)	0.52	0.24	.031	-	-	-	-	-	-	1.09	0.35	.002	0.85	0.15	$< .001$
ΔPTSD$_{t-1} \rightarrow \Delta$Cognitive Process$_t$ ($\xi_{\text{lag}_{yx}}$)	0.60	0.56	.277	-	-	-	-	-	-	0.10	0.13	.469	-	-	-
Session 1 to 5 (Δ_1) **and 5 to 10** (Δ_2)															
Δ_1Cognitive Process$_{t-1} \rightarrow \Delta_1$PTSD$_t$ ($\xi_{1\ \text{lag}_{xy}}$)	-	-	-	0.13	0.05	.013	0.35	0.12	.004	-	-	-	-	-	-
Δ_2Cognitive Process$_{t-1} \rightarrow \Delta_2$PTSD$_t$ ($\xi_{2\ \text{lag}_{xy}}$)	-	-	-	0.06	0.09	.533	0.48	0.16	.003	-	-	-	-	-	-
Δ_1PTSD$_{t-1} \rightarrow \Delta_1$Cognitive Process$_t$ ($\xi_{1\ \text{lag}_{yx}}$)	-	-	-	0.72	0.11	$< .001$	1.40	0.85	.099	-	-	-	-	-	-
Δ_2PTSD$_{t-1} \rightarrow \Delta_2$Cognitive Process$_t$ ($\xi_{2\ \text{lag}_{yx}}$)	-	-	-	0.75	0.16	$< .001$	0.18	0.98	.850	-	-	-	-	-	-

Note. Parameter estimates of lagged associations between changes in process measures and PTSD symptoms. EST = Unstandardised estimated parameter; SE = Standard error; PDS = PTSD symptoms; PTCI-20 = Negative appraisals; TMQ = Disorganised memories; UMQ = Flashback memories; RIQ = Responses to intrusions; SBQ = Safety behaviours. - indicates parameter was not estimated.

Negative appraisals - PTSD symptoms. Work on negative appraisals is closely linked with many other aspects of the therapy and is therefore an important component of every session in CT-PTSD. Assuming that the relationship between changes PTSD symptoms and negative cognitions is similar throughout treatment, we fixed cross-lagged effects as equal over time. The best fitting model included bidirectional coupling parameters (χ^2 = 410, CFI = .960, TLI = .963, RSMEA = .069, AIC = 4,465, BIC = 4,549). Changes in negative appraisals predicted changes in PTSD symptoms in the following session ($\xi_{lag_{xy}} = 0.52$, $SE = 0.24$, $p = .031$). In contrast, changes in PTSD symptoms did not significantly predict subsequent changes in negative appraisals ($\xi_{lag_{yx}} = 0.60$, $SE = 0.56$, $p = .277$).

Disorganised recall and flashback quality - PTSD symptoms. During the early phases of CT-PTSD patients are asked to give an account of the whole trauma from the beginning to the end either in imaginal reliving or a moment-by-moment trauma narrative and start updating the worst moments. Therapeutic techniques in the later stages of treatment involve work on memory triggers and a site visit and may have different effects on disorganisation (Ehlers et al., 2005). We therefore allowed the lagged coupling effects between disorganised memories and PTSD symptoms to vary between the early sessions (1 to 5) and subsequent sessions (5 to 10). The best fitting model included piecewise bidirectional coupling parameters (χ^2 = 364, CFI = .964, TLI = .966, RSMEA = .061, AIC = 4,214, BIC = 4,304). Lagged coupling effects indicated that changes in disorganised memories predicted subsequent changes in PTSD symptoms for the early sessions of therapy ($\xi_{1\ \mathrm{lag}_{xy}} = 0.13$, $SE = 0.05$, $p = .013$), but not during subsequent sessions. Lagged coupling effects in the other direction indicated that changes in PTSD symptoms predicted subsequent changes in disorganised memories during the early as well as subsequent sessions of therapy ($\xi_{1\ \mathrm{lag}_{yx}} = 0.72$, $SE = 0.11$, $p < .001$, $\xi_{2\ \mathrm{lag}_{yx}} = 0.75$, $SE = 0.16$, $p < .001$).

Similarly, we allowed the lagged coupling effects between flashback qualities and PTSD symptoms to vary between the early sessions (1 to 5) of therapy and

subsequent sessions (5 to 10). The best fitting model included piecewise bidirectional coupling parameters (χ^2 = 356, CFI = .965, TLI = .967, RSMEA = .061, AIC = 6,664, BIC = 6,754). Lagged coupling effects indicated that changes in flashback memories predicted subsequent changes in PTSD symptoms in the early sessions ($\xi_{1\ \mathrm{lag}_{xy}} = 0.35$, $SE = 0.12$, $p = .004$) and later sessions ($\xi_{2\ \mathrm{lag}_{xy}} = 0.48$, $SE = 0.16$, $p = .003$) during therapy. At the same time, there were no significant effects of changes in PTSD symptoms on changes in flashback memories at any interval of the CT-PTSD.

Responses to intrusions - PTSD symptoms. Work on unhelpful responses to intrusion (suppression, ruminations, numbing) is a key goal throughout treatment, therefore cross-lagged coupling effects were set to equal over time. The best fitting model included cross-lagged coupling effects (χ^2 = 373, CFI = .965, TLI = .968, RSMEA = .062, AIC = 2,414, BIC = 2,498). Changes in responses to intrusions predicted subsequent changes in PTSD symptoms in the following session ($\xi_{lag_{xy}} = 1.09$, $SE = 0.35$, $p = .002$). In contrast, changes in PTSD symptoms did not significantly predict subsequent changes in responses to intrusions ($\xi_{lag_{yx}} = 0.10$, $SE = 0.13$, $p = .469$).

Safety behaviours - PTSD symptoms. Therapeutic interventions aiming to reduce unhelpful safety behaviours are implemented throughout treatment, therefore cross-lagged coupling effects were set to equal over time. The best fitting model included only the coupling effect of $\Delta\text{Safety behaviours}_{t-1} \rightarrow \Delta\text{PTSD symptoms}_{t}$ (χ^2 = 389, CFI = .959, TLI = .963, RSMEA = .065, AIC = 3,064, BIC = 3,144). Changes in safety behaviours were significantly associated with changes in PTSD symptoms in the following session ($\xi_{lag_{xy}} = 0.85$, $SE = 0.15$, $p < .001$). Adding the reverse relationship $\xi_{\mathrm{lag}_{yx}}$ ($\Delta\text{PTSD symptoms}_{(t-1)} \rightarrow \Delta\text{Safety behaviours}_{(t)}$) to the model did not improve the fit, indicating that there is no evidence for an effect of changes in PTSD symptoms predicting subsequent changes in safety behaviours.

PTSD recovery prediction

Analyses using separate Cox Proportional Hazard Models for each process measure suggested that individual session-by-session improvements on all process measures were significant predictors of recovery from PTSD. Odds ratios (OR) of recovery decreased with higher levels of negative appraisals (OR = 1.040, 95%CI: 1.032 to 1.048), flashback memories (OR = 1.007, 95%CI: 1.006 to 1.009), disorganised memories (OR = 1.095, 95%CI: 1.054 to 1.137), responses to intrusions (OR = 1.098, 95%CI: 1.069 to 1.128), and safety behaviours (OR = 1.146, 95%CI: 1.109 to 1.185). These results suggest that patients who did not improve on these process measures had less chance to recover from PTSD during the first 10 sessions of therapy.

In a joint model incorporating all significant predictors, negative appraisals (OR = 1.030, 95%CI: 1.018 to 1.042), flashback memories (OR = 1.004, 95%CI: 1.002 to 1.006), and unhelpful safety behaviours (OR = 1.059, 95%CI: 1.015 to 1.104) remained significant, suggesting that their effects on the PTSD symptoms operate though – at least partly – distinct pathways. The OR estimates suggest that a one point improvement in therapy process measures was associated with an increase in the odds of recovery, for example a one point improvement on the PTCI was associated with an increase in in the odds of recovery from PTSD by 1.030. The variance inflation factors (VIF) of the process measures in the multivariate survival model were in the acceptable range ($VIF_{PTCI} = 2.08$, $VIF_{TMQ} = 1.40$, $VIF_{UMQ} = 1.73$, $VIF_{RIQ} = 2.24$, $VIF_{SBQ} = 1.54$) indicating that the model did not violate the assumptions of multicollinearity.

3.1.4 Discussion

This study investigated whether key processes hypothesised by Ehlers and Clark's (2000) cognitive model for PTSD are relevant for driving clinical improvement during CT-PTSD in routine clinical care. Our overall findings were that changes in negative appraisals, memory characteristics, as well as unhelpful cognitive and behavioural coping strategies are driving subsequent changes in PTSD symptoms.

For disorganised memories we only found evidence for this effect early in therapy, while a reverse relationship was found throughout therapy. These findings extend prior research on therapeutic processes in CT-PTSD (Kleim et al., 2013) and demonstrate that the theory-derived cognitive processes that CT-PTSD aims to change play a key role in PTSD symptom improvements during therapy (Ehlers & Clark, 2000; Ehlers et al., 2005). In addition to the finding that changes in these therapeutic processes are driving subsequent changes in PTSD symptoms, we also found that improvements in all of these therapy processes individually predicted PTSD recovery. A multivariate analysis including all therapy process measures suggested that improvements in negative appraisals, flashback memories, and unhelpful safety behaviours each had a unique effect on PTSD recovery.

Our finding that trauma-related negative appraisals precede changes in PTSD symptoms are consistent with assumptions of cognitive models of PTSD (e.g., Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1992) and in line with the majority of studies investigating this relationship during treatment (for a review see Brown et al., 2018). Identifying and modifying trauma-related negative appraisals has been proposed as a central therapeutic aim in different forms of psychological therapies for PTSD (Schnyder et al., 2015). Importantly, our results showed no evidence for a reverse or bidirectional relationship between PTSD symptoms and appraisals in our sample, replicating Kleim et al.'s (2013) findings.

One study found evidence for a reciprocal relationship between appraisal change and PTSD improvement in a randomised controlled trial of participants with comorbid PTSD and alcohol dependence (McLean, Su, et al., 2015). Discrepancies may be due to differences in the clinical samples. Furthermore, it is possible that the longer time intervals between the assessments in McLean, Su, et al. (2015) allowed for enough changes in PTSD symptoms and negative appraisals to result in a bidirectional relationship. Following Kleim et al. (2013), this study provides further evidence for the importance of addressing trauma-related negative cognitions in CT-PTSD in routine clinical care.

In line with our hypothesis and earlier research investigating pre- to post-treatment changes in different aspects of the trauma narrative (e.g., Mundorf & Paivio, 2011) we found evidence that changes in trauma memory disorganisation through the elaboration of what happened during the trauma in the first sessions of therapy drove improvements in PTSD symptoms. Contrary to our hypothesis, we also found evidence that changes in PTSD symptoms were driving subsequent changes in memory disorganisation throughout therapy. This would suggest that improvements in some aspects of memory disorganisation are driven by previous improvements in PTSD symptoms. A possible explanation may be that reductions in symptoms include the reduction in cognitive avoidance, which may allow patients to engage more with their trauma memories. This may facilitate further improvements in memory disorganisation in later parts of the treatment and explain the bidirectional relationship we found in our sample. To our knowledge this is the first study to investigate lagged effects between disorganised trauma memories and PTSD symptoms during psychological therapy for adults with PTSD. Our results provide initial evidence for a bidirectional effect and suggest that this effect may vary during different phases of therapy, for example, memory disorganisation appears to drive symptom change when it is directly targeted in sessions using techniques that facilitate memory elaboration such as imaginal reliving and writing a moment-by-moment narrative.

Extending earlier research showing that specific flashback characteristics of intrusive trauma memories improved during therapy (e.g., Hackmann et al., 2004; Speckens et al., 2006), we found that changes in these characteristics are driving subsequent changes in PTSD symptoms throughout therapy. Reductions in flashback characteristics of unwanted memories were associated with subsequent improvements in PTSD symptoms, with a similar effect during the initial five sessions of therapy and subsequent sessions. In contrast to our memory disorganisation results, we did not find evidence for a reverse relationship of PTSD symptom reduction on flashback qualities. This suggests that both the early and later work targeting

intrusive trauma memories in CT-PTSD drove PTSD symptom change throughout treatment in our sample.

In CT-PTSD unhelpful coping strategies are usually addressed in behavioural experiments and through discussions of their advantages and disadvantages (see Ehlers et al., 2005). In line with previous prospective studies that provided evidence for the importance of suppression, rumination, and intentional numbing in the development of PTSD (e.g., Beierl et al., 2019; Kleim et al., 2012) our results provide initial evidence that changes in unhelpful responses to intrusions drive subsequent changes in PTSD symptoms during CT-PTSD. Similarly, our results suggest that dropping unhelpful safety behaviours drives subsequent changes in PTSD symptoms. This is in line with prospective studies (e.g., Beierl et al., 2019; Dunmore et al., 2001; Ehring et al., 2008) and extends previous evidence from a PTSD treatment study (Goodson & Haeffel, 2018). Taken together, the results not only highlight the importance of cognitive processes in clinical improvement, but also highlight the key role of behavioural changes as suggested by cognitive models of PTSD.

In line with earlier studies demonstrating that some patients experience significant improvements early during therapy, we also found that PTSD symptoms and therapy process measures improved more during the first part of therapy compared to the second part (Kleim et al., 2013; Macdonald et al., 2011). The slower improvements during the second part of therapy may in part be explained by floor effects as a significant subgroup had minimal symptoms and in part by complex cases, for examples those with multiple traumas, comorbidities or social problems, required more treatment sessions as their treatment had to focus on other issues besides the traumas. However, our results also highlight that patients varied significantly in their changes during both parts of therapy (σ^2_{g2}, σ^2_{g3}) suggesting that patients improved via different trajectories and that there may be particular subgroups that need further investigation. A study by Schumm et al. (2013) specifically investigated whether creating subgroups can help to explain differences in how participants experience symptom changes during treatment in 207 treatment seeking veterans with PTSD receiving cognitive processing therapy. The authors

applied latent class analysis and growth mixture modelling to define latent classes and found that demographic variables and pretreatment symptom severity predicted latent class membership, which in turn was associated with treatment outcomes. Although we used different methods, our results are at least partly in line with results by Schumm et al. (2013) – higher scores in all process measures were associated with slower improvements in these measures.

Strengths and limitations

Strengths of this study include that our sample was ethnically diverse and included different trauma types. Therapists with different levels of expertise delivered the therapy in routine clinical care, increasing the generalisability of our findings. We were able to test all key processes of clinical improvement hypothesised by Ehlers and Clark's (2000) cognitive model of PTSD. Although the PTSD therapy processes were correlated with each other as well as with PTSD symptoms we found evidence for lagged effects in LCSM analyses. Furthermore, our results from survival analyses suggested that improvements in all theory-derived processes were related to recovery and those in negative appraisals, flashback memories, and safety behaviours showed distinct pathways to recovery. The use of weekly assessment during treatment allowed for a detailed examination of change processes during treatment, however other time intervals and forms of data collection should be explored if important changes in therapy processes are thought to occur during shorter or longer time periods.

This study also has several limitations. First, this study used self-report measures to assess PTSD symptoms and therapy process measures. Second, the current sample size and analytical method did not allow for a combined analysis of PTSD symptoms and all therapy process measures. Third, because this study investigated the therapy processes suggested by Ehlers and Clark's (2000) cognitive model of PTSD which is the basis of the case formulation in CT-PTSD, some of the investigated processes may be treatment specific. The results may also be specific to PTSD as the primary outcome measure in this study. Non-specific or common factors (e.g., therapeutic

alliance) and alternative outcome measures (e.g., quality of life) would also be of interest to explore in further studies (e.g., Bredemeier et al., 2020). Although the statistical models fit the data well, other variables not measured in this study may have influenced the results and alternative models are also possible. Fourth, like any analysis that investigates changes in constructs over time, this study assumed that the construct that is being measured is the same across treatment sessions. While this study considers nonlinear trajectories of symptoms and the time interval between sessions was mainly consistent, new advances in methods allow researchers to incorporate differences in time between sessions in the estimation of parameters and should be explored when the time between sessions varies (Driver et al., 2017; Voelkle et al., 2018). Although the measures have been shown to be reliable and informative in assessing improvements in symptoms and therapy processes during treatment, the assumption of longitudinal measurement invariance should be tested statistically in larger samples (e.g., Stochl et al., 2020), especially because simulation studies have shown that model estimates may be less accurate and the type 1 error rate can increase when this assumption is not met (Xu et al., 2020). Given the short time frame of 10 weekly measurements in our study and the common use of these scales in longitudinal research studies we believe this assumption is reasonable.

Conclusions

Overall, the results of this study provide further evidence that cognitive and behavioural processes suggested by models of PTSD play a key role in driving symptom improvement during CT-PTSD in routine care and highlight potential starting points to improve existing therapies.

4

Sudden symptom improvements

4.1 Cognitive processes associated with sudden gains[1]

4.1.1 Aims

Chapter 1.6 reviewed sudden gain studies in psychological treatments for PTSD and Chapter 1.6.1 highlighted that it is currently not known how changes in cognitive processes are associated with sudden gains in PTSD symptoms. This study aims to investigate how changes in cognitive processes drawn from Ehlers and Clark's (2000) cognitive model of PTSD are associated with sudden gains in PTSD symptoms.

The present study investigated sudden gains in two large clinical samples of patients with PTSD treated with CT-PTSD in routine clinical care, using the same criteria and including a matched control group. The first aim was to replicate findings that patients who experience a sudden gain during therapy report better outcomes at the end of treatment and at follow-up compared with all patients who did not experience a sudden gain (Hypothesis 1). The second aim was to investigate

[1]The work presented in this chapter is based on published work: Wiedemann, M., Stott, R., Nickless, A., Beierl, E. T., Wild, J., Warnock-Parkes, E., Grey, N., Clark, D. M., & Ehlers, A. (2020). Cognitive processes associated with sudden gains in cognitive therapy for posttraumatic stress disorder in routine care. *Journal of Consulting and Clinical Psychology.* https://doi.org/10.1037/ccp0000488

processes associated with the occurrence of sudden gains. We hypothesised that compared with matched patients who did not experience a sudden gain, patients with sudden gains would show a greater change in negative appraisals and flashback memories during the sudden gain (Hypothesis 2) and greater change in negative appraisals and flashback memories before the sudden gain (Hypothesis 3). Baseline predictors of sudden gains and group differences in changes in cognitive processes after the sudden gains were also explored.

4.1.2 Methods

Participants

This study is a secondary analysis of data drawn from studies investigating the effectiveness of CT-PTSD in routine clinical care. Two cohorts of consecutive patients with PTSD treated in a specialist outpatient clinic for anxiety disorders serving an inner-city population characterised by above-average rates of social deprivation and crime and a greater proportion of ethnic minorities than the national average were treated with CT-PTSD. Patients met the criteria for PTSD according to the Structured Clinical Interview for DSM-IV (SCID; First et al., 1997), and PTSD was their main problem. The SCID was administered by trained clinical psychologists. Outcome measures (pretreatment and last-session symptom scores) were available for all patients, including dropouts (14% and 16% respectively), and results are reported by Ehlers et al. (2013) for Sample 1 ($N = 330$) and by Ehlers, Wild, et al. (2020) for Sample 2 ($N = 343$). Ethical approval was granted by the local research ethics committee.

The present study included the patients from these consecutive cohorts who provided sufficient week-to-week data to apply Tang and DeRubeis's (1999) sudden gains criteria – that is, at least two of three scores prior to a potential gain must be present, as well as at least two of three scores following a potential gain (Sample 1, $N = 248$; Sample 2, $N = 234$). Patient characteristics for each sample are presented in Table 4.1.

Table 4.1: Demographic and clinical characteristics for both samples

	Sample 1 (n = 248)			Sample 2 (n = 234)		
Variable	n	%	M (SD)	n	%	M (SD)
Age in years	248		38.90 (11.23)	234		37.82 (11.14)
Months since main traumatic event	238		37.61 (57.94)	232		52.34 (78.45)
Weekly treatment sessions	248		11.55 (4.63)	233		10.81 (4.35)
Gender						
Female	143	57.7%		131	56.0%	
Male	105	42.3%		103	44.0%	
Relationship						
Married/Cohabiting	87	35.1%		92	39.3%	
Divorced/Separated/Widowed	46	18.5%		28	12.0%	
Never married	108	43.5%		106	45.3%	
No information	7	2.8%		8	3.4%	
Ethnicity						
Black	64	25.8%		56	23.9%	
Caucasian	138	55.6%		150	64.1%	
Other	46	18.5%		28	12.0%	
Education						
University	71	28.6%		69	29.5%	
A-levels	37	14.9%		30	12.8%	
GCSE	69	27.8%		53	22.6%	
Other	54	21.8%		37	15.8%	
No information	17	6.9%		45	19.2%	
Employment						
Employed/Self-employed	93	37.5%		109	46.6%	
Student	12	4.8%		10	4.3%	
Sick leave	34	13.7%		13	5.6%	
Disability/Retired	22	8.9%		12	5.1%	
Unemployed	73	29.4%		76	32.5%	
No information	14	5.6%		14	6.0%	
Type of main traumatic event						
Interpersonal violence	144	58.1%		147	62.8%	
Accident or disaster	51	20.6%		47	20.1%	
Death or harm to others	23	9.3%		28	12.0%	
Other	30	12.1%		12	5.1%	
Comorbid depression						
No	124	50.0%		111	47.4%	
Yes	124	50.0%		123	52.6%	
Comorbid anxiety						
No	137	55.2%		114	48.7%	
Yes	111	44.8%		120	51.3%	

Note. n = Number of available responses for each variable. % = Percentage of total sample included in this study. GCSE = General Certificate of Secondary Education.

Treatment

Patients received a course of CT-PTSD (Ehlers et al., 2005) based on Ehlers and Clark's (2000) cognitive model of PTSD. The treatment aims to reduce the patient's sense of current threat by changing problematic meanings of the trauma and its consequences; elaborating and updating the memories of the trauma with information that gives them a less threatening meaning at present; discriminating triggers of intrusive memories; and changing behaviours and cognitive processes that maintain PTSD, such as rumination and safety behaviours. For a detailed description of the treatment see Chapter 1.3.

Therapists were qualified (i.e., had completed their professional training in clinical psychology, psychiatry, or as a nurse therapist and were registered health professionals) or trainees in these professions. Therapists received training in CT-PTSD (a 2-day workshop followed by case supervision) and attended weekly supervision throughout the studies to ensure treatment fidelity.

The number of sessions depended on the number of traumas and comorbidities to be addressed, usually up to 12 weekly sessions if treatment addressed one or two index traumas and up to 24 sessions if treatment addressed more than two traumas. On average, patients received 11.55 ($SD = 4.63$) weekly treatment sessions in Sample 1 and 10.81 ($SD = 4.35$) sessions in Sample 2. If patients were taking psychotropic medication, they had to be on a stable dose for at least 1 month before starting therapy and were asked to stay on that dose for the duration of the treatment.

Measures

Patients completed the following measures of established reliability and validity at the beginning of every treatment session. Two thirds also completed symptom measures at follow-up ($M = 280$ days after treatment). The measures for Sample 2 assessed the same concepts as Sample 1 but were updated due to a change in clinic procedures.

PTSD symptoms. Both samples completed the Posttraumatic Diagnostic Scale (PDS; Foa et al., 1997) to assess PTSD symptom severity. The PDS is a reliable and validated 17-item self-report measure of the PTSD symptoms (PDS; Foa et al., 1997) specified in the DSM-IV (American Psychiatric Association, 2000). Patients rated the extent to which they were bothered by each of the 17 symptoms during the last week on a scale from 0 (*Not at all*) to 3 (*5 or more times a week/almost always*). The internal consistency at baseline was Cronbach's $\alpha = .85$ in Sample 1 and $\alpha = .89$ in Sample 2. A cutoff of 18 has been found to best predict a PTSD diagnosis (Ehring et al., 2007). Independent ratings of PTSD symptoms were also conducted by trained clinicians experienced in diagnosing PTSD for a subsample using the PTSD Symptom Scale Interview (PSS-I; Foa et al., 1993) at the beginning and end of treatment. The internal consistency at baseline was Cronbach's $\alpha = .83$ in Sample 1 and $\alpha = .89$ in Sample 2.

Depression symptoms. To assess the severity of depressive symptoms, Sample 1 completed the Beck Depression Inventory (BDI; Beck & Steer, 1993a), a 21-item self-report measure with high reliability and validity, Cronbach's α at baseline = .90. A score of 17 or above indicates moderate depression, and a score of 30 or above indicates severe depression. Sample 2 completed the reliable and validated Patient Health Questionnaire–9 (PHQ-9; Kroenke et al., 2001), Cronbach's α at baseline = .91. A score of 10 or above suggests a diagnosis of depression.

Anxiety symptoms. To assess the severity of anxiety symptoms, Sample 1 completed the Beck Anxiety Inventory (BAI; Beck & Steer, 1993b), a 21-item self-report measure of anxiety symptoms with high reliability and validity, Cronbach's α at baseline = .93. Sample 2 completed the Generalised Anxiety Disorder 7-item scale (GAD-7; Spitzer et al., 2006). A score of 16 or above indicates moderate anxiety, and a score of 26 or above indicates severe anxiety. The internal consistency of the GAD-7 at baseline was Cronbach's $\alpha = .90$. A score of 8 or above suggests clinical anxiety.

Negative trauma-related appraisals. Patients completed short versions of the Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999). This self-report measure of negative appraisals asks respondents to indicate their agreement with statements indicating negative appraisals about the self, others, and self-blame that are characteristic of patients with PTSD on a scale from 1 (*Totally disagree*) to 7 (*Totally agree*). Sample 1 completed a 22-item version (PTCI-22; see Kleim et al., 2013), Cronbach's α at baseline = .92, and Sample 2 completed a revised 20-item version (PTCI-20; see Ehlers, 2020), Cronbach's α at baseline = .91. The short versions were developed from the items that had the highest factor loadings and best discrimination between people with and without PTSD.

Flashback memories. Patients reported the degree of flashback-like qualities of their unwanted memories of the trauma on the Unwanted Memories Questionnaire (UMQ; adapted from Hackmann et al., 2004). Sample 1 completed a 4-item version of the scale (UMQ-4) and reported the degree of perceived nowness, disjointedness, vividness, and distress of their main intrusions, each on a scale between 0 (*Not at all*) and 100 (*Very strongly*), Cronbach's α at baseline = .62. Sample 2 completed a revised 5-item version (UMQ-5; Ehlers, Beierl, et al., 2020) that contained one further item about easy triggering of intrusive memories by many different cues from a study by Halligan et al. (2003), Cronbach's α at baseline = .84.

Data analyses

Identification of sudden gains. Sudden gains were based on patient scores on the PDS and were defined following the three criteria described by Tang and DeRubeis (1999). The R package *suddengains* (Version 0.4.0; Wiedemann, Thew, et al., 2020) developed in Chapter 2.3 was used to identify sudden gains in both samples. We included PDS scores from the baseline assessment and 12 weekly scores to identify sudden gains between Sessions 2 and 10. Following previous PTSD sudden gains studies (Doane et al., 2010; Jun et al., 2013; Krüger et al., 2014) a cutoff value for the first criterion was defined as the standard error of the difference

from the Reliable Change Index (RCI; Jacobson & Truax, 1991) as calculated by Foa et al. (2002), resulting in a cutoff value of 6.15 on the PDS for both samples. The standard error of the difference was computed using the test–retest reliability (r_{xx}) of .83 and standard deviation of a nonclinical sample (s_1) of 10.54 of the PDS as reported by Foa et al. (1997). The standard error of measurement for the scale ($S_E = 4.35$) was computed using $S_E = s1 \times \sqrt{1 - r_{xx}}$. The standard error of the difference ($S_{\text{diff}} = 6.15$) was computed using $S_{\text{diff}} = \sqrt{2 \times (S_E)^2}$ by Foa et al. (2002). More details describing the method used to define a cutoff are described in Chapter 2.3. A sudden gain was identified between Session N (pregain session) and Session N+1 (postgain session) according to the following three criteria:

1. The decrease between two consecutive scores on the PDS was at least 6.15 (PDS_{N} - $\text{PDS}_{\text{N-1}} \geq 6.15$). This change represents 12.06% of the total range on the PDS (0 - 51),
2. PDS scores decreased by at least 25% relative to the pregain score (PDS_{N} - $\text{PDS}_{\text{N+1}} \geq .25 \times \text{PDS}_{\text{N}}$), and
3. The pooled standard deviation between the mean PDS score of three sessions (or two sessions if three were not available) before the sudden gain (Sessions N-2, N-1, and N) and after the sudden gain (Sessions N+1, N+2, and N+3) was greater than the following critical values, which were adjusted for missingness based on t values from the two-sample t test: $t_{(4;97.5\%)} > 2.776$; $t_{(3;97.5\%)} > 3.182$; $t_{(2;97.5\%)} > 4.303$.

If patients experienced more than one sudden gain, the earliest gain was selected for all further analyses. The stability of gains was assessed in two ways. Following Tang and DeRubeis (1999), a sudden gain was coded as reversed when at least 50% of the magnitude of the sudden gain was lost at any point later in treatment. Following Wucherpfennig, Rubel, Hollon, et al. (2017), a stable reversal was coded when a reversal also met the criteria for a sudden loss. Sudden losses are defined as the inverse criteria of sudden gains (i.e., parallel criteria to sudden gains for symptom deterioration).

Matching procedure. Mahalanobis distance matching, including the propensity score, was used to select matched patients without sudden gains. This method reduces the group differences between patients with and without sudden gains while selecting pairs of patients who are similar based on a list of covariates (Rosenbaum & Rubin, 1985). The following 10 variables were selected as covariates for the matching procedure: age; gender (male; female); months since the main index trauma; type of trauma (interpersonal violence; accidents or disasters; harm to others; other); comorbid depression (yes; no); and baseline scores of PTSD, depression, and anxiety symptoms and negative appraisals and flashback memories. Propensity scores were calculated using logistic regression with sudden gain status (yes; no) as the dependent variable and all selected covariates as predictors. Following recommendations by Rosenbaum and Rubin (1983) and Stuart (2010), a 1:1 matching approach was used. Patients were matched on the Mahalanobis distance within calipers of 0.25 to decrease the within-pair differences (Rosenbaum & Rubin, 1985). The R package *MatchIt* (Version 3.0.2; Ho et al., 2011) was used to perform the matching. Each matched patient was assigned a 'matched session' with the same pregain session number as the sudden gains patient they were matched with. Two datasets were created for each sample: (a) a 'by person' dataset including all patients with sufficient week-to-week data (Sample 1: $n = 248$; Sample 2: $n = 234$) and (b) a 'matched' dataset including all patients with sudden gains and matched patients without sudden gains (Sample 1: $n = 152$; Sample 2: $n = 174$).

Statistical analysis. All analyses were performed in R (Version 3.5.2; R Core Team, 2020) through RStudio IDE (Version 1.1.463; RStudio Team, 2020). A significance criterion of $\alpha = .05$ was set for all analyses. All linear mixed-effect models were estimated using the R package *nlme* (Version 3.1.137; Pinheiro et al., 2020) with the maximum-likelihood (ML) estimator. The R code for all analyses can be found at https://osf.io/dgt8x/.

The relationship between sudden gains and primary (PTSD symptoms) and secondary (depression and anxiety symptoms) treatment outcomes were analysed

by fitting linear mixed-effect models to account for repeated measures over time. To estimate the effect of the sudden gains at the end of treatment and follow-up, time (categorical), group (all patients with sudden gains and all patients without sudden gains), and the interaction between time and group were included as fixed effects. Baseline scores of the dependent variable were entered as a covariate. Random intercepts were estimated to account for measurements taken from the same individual. Contrasts were specified to test for the effect of sudden gains on the primary outcome. Cohen's d was computed as a standardised effect size of sudden gains on treatment outcome by dividing the adjusted mean difference by the pooled standard deviation at baseline.

Univariate logistic regression models were used to test whether patient characteristics (age, gender, and months since trauma), baseline psychopathology (PTSD symptoms, depression symptoms, anxiety symptoms, and diagnosis of comorbid depression), or baseline cognitive processes (negative appraisals, flashback memories) showed an association with the occurrence of sudden gains. To test the overall predictive effect of all predictors, multivariate logistic regression models were run. The assumption of linearity of the logit was met for all continuous variables.

Differences in changes in the process variables before, during, and after sudden gains/matched sessions between the groups were analysed, fitting one linear mixed-effect model for each process variable using the matched datasets. For all variables, five scores around the sudden gain (N-2, N-1, N, N+1, N+2) were extracted to investigate changes in four between-session intervals around the sudden gain (N-2 to N-1, N-1 to N, N to N+1, N+1 to N+2). The model included the scores of the process variable as the dependent variable and time (N-2, N-1, N, N+1, N+2) and group (all patients with sudden gains and all matched patients) as fixed effects. Time was treated as a categorical variable to allow maximum flexibility in the way the outcome changed over time. This approach allowed us to estimate the change in outcome between any two sessions. The interaction between time and group was modelled as a fixed factor to allow the estimation of the difference between groups in the change in outcome for each interval. Random intercepts were estimated to

account for measurements taken from the same individual. Contrasts were specified to test for within- and between-groups differences in changes in the process variables during the time intervals around the sudden gain. Estimates of differences between the time intervals within the sudden gains group are labelled as δ_1, and those within the matched control group are labelled as δ_2. The estimates of the difference between the two groups are labelled as Δ_3. The assumption of normality of the residuals was confirmed visually for all outcomes.

The estimates of the group differences from both samples were meta-analysed to obtain pooled estimates of the changes in process variables for each analysed time interval around the sudden gain. Individuals were assumed to be drawn from the same population. Therefore, a fixed-effects model was run using the R package *metafor* (Version 2.0.0; Viechtbauer, 2010) to estimate the pooled effect based on the adjusted Standardised Mean Difference (SMD; Hedges & Olkin, 1985). The SMD was calculated based on the estimated difference within the sudden gains group (δ_1) and the matched group (δ_2) based on the following equation:

$$\begin{aligned} \text{SMD} &= \frac{\delta_1 - \delta_2}{S} \text{ where} \\ \text{S} &= \sqrt{\frac{(n_{\text{SG}} - 1) \times {\text{SD}_{\text{diff}_1}}^2 + (n_{\text{Control}} - 1) \times {\text{SD}_{\text{diff}_2}}^2}{n_{\text{SG}} + n_{\text{Control}} - 2}} \text{ where} \\ \text{SD}_{\text{diff}_{1,2}} &= \text{SD}(x_t - x_{t+1}) \text{ for } \text{SG}_{(1)} \text{ and } \text{control}_{(2)} \text{ group.} \end{aligned}$$

The standard deviation for the sudden gains group and the matched control group was calculated from the difference scores of the investigated interval using the raw data. The n was based on the number of patients for which a difference score was available for the investigated interval.

4.1.3 Results

Frequency and characteristics of sudden gains

A total of 1,459 and 1,254 between-session intervals were investigated for sudden gains in Samples 1 and 2, respectively. Following the three criteria by Tang and DeRubeis (1999), 76 out of 248 patients (30.65%) experienced a total of 83 sudden

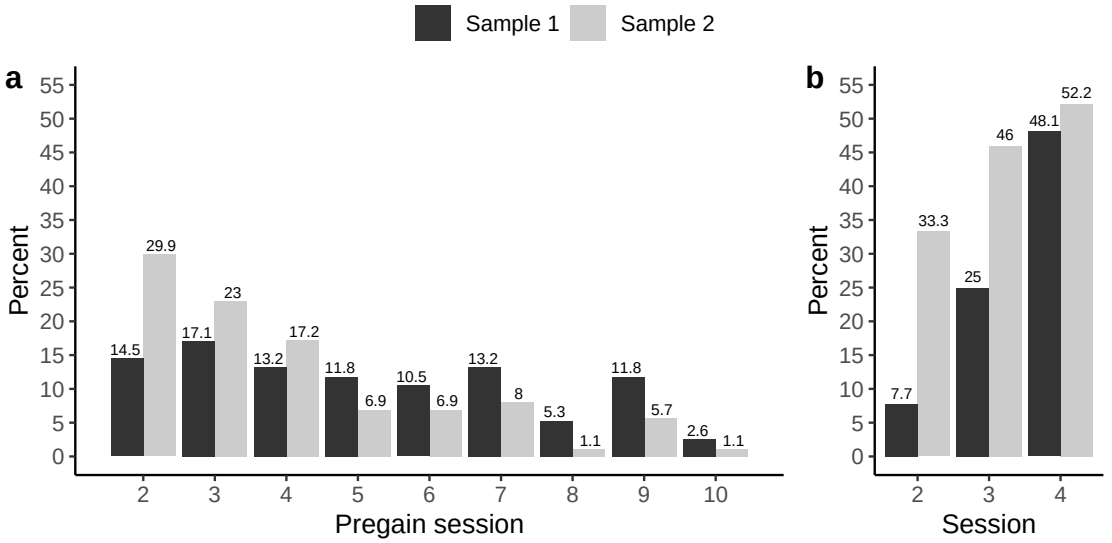


Figure 4.1: (a) Distribution of pregain sessions in percent for all sudden gains in Sample 1 and 2. Percentages are based on the number of patients who experienced sudden gains in each sample respectively (Sample 1: $n = 76$; Sample 2: $n = 87$). (b) Percentage of sessions early in treatment with memory updating procedure.

gains between Sessions 2 and 10 (Median = 5, Mode = 3) in Sample 1. In Sample 2, 87 out of 234 patients (37.18%) experienced a total of 100 sudden gains between Sessions 2 and 10 (Median = 3, Mode = 2). The distribution of the pregain session numbers is presented in Figure 4.1a and showed that sudden gains tended to occur earlier in Sample 2 compared with Sample 1. This may be related to the fact that a core treatment procedure, updating trauma memories, was on average conducted earlier in treatment in the second cohort, in line with guidance by the treatment developers (see Figure 4.1b).

Multiple gains were experienced by 6 patients (2.42%) in Sample 1 (5 patients experienced two sudden gains; 1 patient experienced three sudden gains) and 11 patients (4.70%) in Sample 2 (9 patients experienced two sudden gains; 2 patients experienced three sudden gains). In total, 13 sudden gainers (17.11%) lost 50% of the improvement made during the sudden gain at some point later in treatment (Tang & DeRubeis, 1999) in Sample 1, and 10 (11.49%) in Sample 2, but most of these (92.11% in Sample 1 and 96.55% in Sample 2) regained the improvement made during the sudden gain by the end of treatment. No sudden gainer in Sample 1 and 3 sudden gainers (3.45%) in Sample 2 experienced a stable reversal

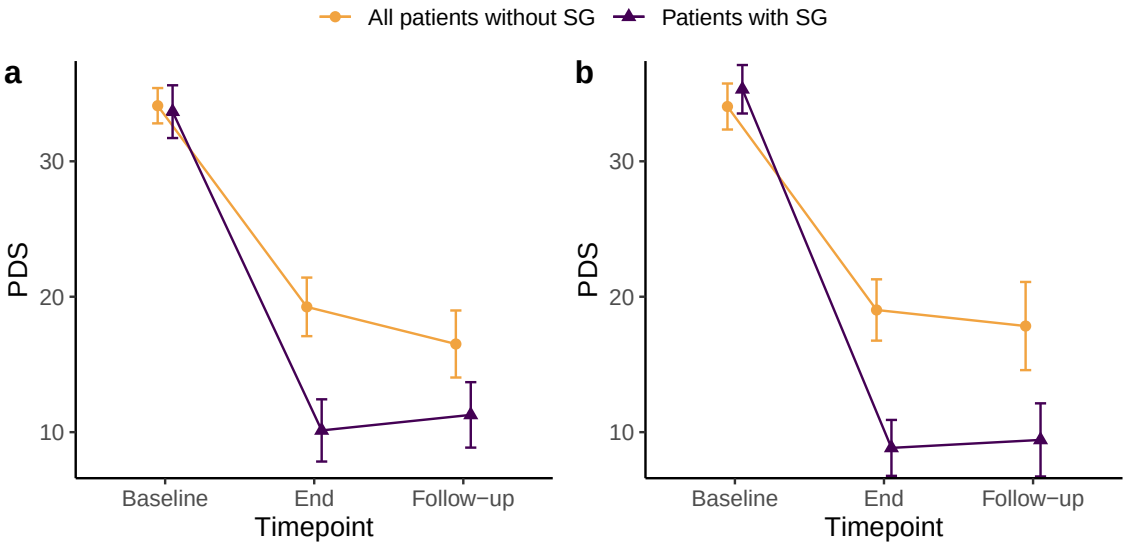


Figure 4.2: Mean PTSD severity for patients with and without sudden gains in (a) Sample 1 and (b) Sample 2. Error bars represent 95% confidence intervals.

(see Wucherpfennig, Rubel, Hollon, et al., 2017). We repeated all analyses after excluding patients who met the criteria for a stable reversal, and the results did not differ. The average sudden gain was $M = 12.30$ ($SD = 4.44$) points on the PDS in Sample 1 and $M = 12.11$ ($SD = 3.83$) in Sample 2.

Sudden gains and treatment outcomes

In both samples, patients with sudden gains reported significantly lower PTSD, depression, and anxiety symptoms at the end of treatment than patients without sudden gains (see Table 4.2). The same result was found for the two subsamples of patients in each cohort ($n_{S1} = 79$, $n_{S2} = 80$) for whom interviewer-assessed PTSD symptoms (PSS-I) were obtained. The differential effect in outcomes between the groups remained significant at follow-up. The mean PTSD symptom severity for patients with and without sudden gains at baseline, the end of therapy, and follow-up is illustrated in Figure 4.2.

Baseline predictors of sudden gains

In Sample 1, the multivariate logistic regression model including only statistically significant predictors of the univariate models suggests that higher age and the

Table 4.2: Primary and secondary treatment outcomes for all patients with and without sudden gains

	Unadjusted mean (SD)				Adjusted difference*		
Measure / Time	*n*	SG	*n*	No SG	Mean [95% CI]	*d* [95% CI]	*p*
PTSD (Self-report)							
S_1: Baseline	76	33.66 (8.66)	172	34.10 (8.72)			
S_1: End	76	10.13 (10.22)	172	19.25 (14.47)	-8.81 [-10.40, -7.22]	1.02 [0.73, 1.30]	< .001
S_1: FU	67	11.27 (10.09)	105	16.51 (12.94)	-6.54 [-8.22, -4.86]	0.75 [0.44, 1.07]	< .001
S_2: Baseline	84	35.41 (8.22)	142	34.04 (10.33)			
S_2: End	87	9.46 (10.54)	147	19.02 (14.00)	-11.04 [-12.60, -9.47]	1.15 [0.86, 1.43]	< .001
S_2: FU	59	10.23 (11.14)	74	17.83 (14.29)	-10.18 [-12.01, -8.36]	1.06 [0.69, 1.42]	< .001
PTSD (Interviewer)							
S_1: Baseline	32	31.35 (8.16)	47	30.35 (9.01)			
S_1: End	32	11.09 (11.34)	47	16.78 (14.11)	-6.37 [-11.56, -1.76]	0.70 [0.23, 1.16]	.016
S_2: Baseline	42	35.30 (7.11)	38	33.34 (8.48)			
S_2: End	42	8.52 (7.71)	38	15.79 (13.36)	-8.18 [-12.61, -3.75]	0.71 [0.25, 1.16]	< .001
Depression†							
S_1: Baseline	76	26.29 (12.17)	172	28.06 (11.76)			
S_1: End	76	10.85 (10.68)	172	18.05 (14.14)	-6.12 [-7.61, -4.62]	0.51 [0.24, 0.79]	< .001
S_1: FU	66	11.11 (10.14)	102	14.36 (12.10)	-3.34 [-4.93, -1.76]	0.28 [-0.03, 0.59]	.035
S_2: Baseline	85	17.08 (6.40)	142	16.39 (7.25)			
S_2: End	87	4.53 (5.59)	146	10.50 (7.94)	-6.22 [-7.12, -5.32]	0.90 [0.62, 1.17]	< .001
S_2: FU	59	5.94 (6.70)	73	9.99 (8.45)	-4.54 [-5.61, -3.46]	0.65 [0.30, 1.01]	< .001
Anxiety†							
S_1: Baseline	74	25.87 (13.31)	167	29.78 (13.79)			
S_1: End	76	8.07 (9.85)	172	17.11 (15.71)	-6.90 [-8.60, -5.20]	0.50 [0.23, 0.78]	< .001
S_1: FU	67	9.40 (10.94)	102	13.29 (13.53)	-4.18 [-5.97, -2.40]	0.31 [-0.01, 0.61]	.019
S_2: Baseline	85	14.46 (5.32)	143	14.16 (5.52)			
S_2: End	87	3.78 (4.23)	145	8.50 (6.76)	-4.83 [-5.59, -4.08]	0.89 [0.61, 1.17]	< .001
S_2: FU	59	5.04 (5.11)	74	8.27 (6.92)	-3.65 [-4.55, -2.75]	0.67 [0.32, 1.02]	< .001

Note. S_1 = Sample 1 (n = 248). S_2 = Sample 2 (n = 234). End = End of treatment. FU = Follow-up. SG = Sudden gain. d = Between-group standardised effect size. *The difference is adjusted for baseline scores. †Measures for depression (Sample 1 = BDI, Sample 2 = PHQ-9) and anxiety (Sample 1 = BAI, Sample 2 = GAD-7) differed between the samples.

absence of comorbid major depression predicted the occurrence of sudden gains (see Table 4.3 legend). For age, the odds of experiencing a sudden gain increased by a factor of 1.03, 95% CI [1.01, 1.06], for each year increase in age. For patients with comorbid major depression, the odds of experiencing a sudden gain were 0.45, 95% CI [0.24, 0.81]. However, these results did not replicate in Sample 2. Results from explorative analyses suggested that the association with age in Sample 1 might be driven by three outliers in the sudden gains group aged around 80 years (see Appendix C, Figure C.1). In Sample 2, no significant baseline predictors of sudden gains were found. See Table 4.3 for detailed results of the univariate and multivariate

logistic regression models investigating baseline predictors of sudden gains.

Table 4.3: Logistic regression analyses predicting the occurrence of sudden gains

	Sample 1: Univariate†		Sample 1: Multivariate†		Sample 2: Univariate†		Sample 2: Multivariate†	
Term	OR [95% CI]	p	OR [95% CI]	p	OR [95% CI]	p	OR [95% CI]	p
Demographic predictors								
Age	1.03 [1.01, 1.06]	.007	1.04 [1.01, 1.07]	.010‡	1.00 [0.98, 1.02]	.938	1.00 [0.97, 1.02]	.817
Gender	0.94 [0.54, 1.62]	.819	1.20 [0.62, 2.34]	.589	0.95 [0.56, 1.62]	.848	1.10 [0.61, 2.00]	.742
Months since trauma	1.00 [0.99, 1.00]	.135	1.00 [0.99, 1.00]	.201	1.00 [1.00, 1.00]	.629	1.00 [1.00, 1.00]	.918
Baseline psychopathology								
PTSD symptoms	0.99 [0.96, 1.03]	.712	1.04 [0.98, 1.10]	.192	1.02 [0.99, 1.05]	.300	1.05 [0.99, 1.11]	.110
Depression symptoms	0.99 [0.96, 1.01]	.282	1.00 [0.95, 1.06]	.911	1.01 [0.98, 1.06]	.471	1.02 [0.93, 1.11]	.649
Anxiety symptoms	0.98 [0.96, 1.00]	.042	0.97 [0.94, 1.00]	.076‡	1.01 [0.96, 1.06]	.688	0.96 [0.87, 1.06]	.475
Comorbid depression	0.39 [0.22, 0.68]	.001	0.46 [0.21, 0.96]	.041‡	1.02 [0.60, 1.74]	.942	1.13 [0.58, 2.24]	.714
Baseline cognitive processes								
Negative cognitions	0.99 [0.98, 1.00]	.210	1.00 [0.98, 1.02]	.979	1.00 [0.99, 1.01]	.890	1.00 [0.98, 1.01]	.661
Flashback memories	0.99 [0.97, 1.00]	.171	1.00 [0.98, 1.02]	.744	1.00 [0.99, 1.01]	.908	0.99 [0.97, 1.01]	.266

Note. †Logistic regression model. Sudden gains (0 = no, 1 = yes). Gender (0 = male, 1 = female). ‡A multivariate model using only the significant predicturs from the univariate model gave the following results: Age: OR = 1.03, 95% CI [1.01, 1.06], $p = .012$; Anxiety symptoms: OR = 0.99, 95% CI [0.96, 1.01], $p = .269$; Comorbid depression: OR = 0.45, 95% CI [0.24, 0.81], $p = .009$.

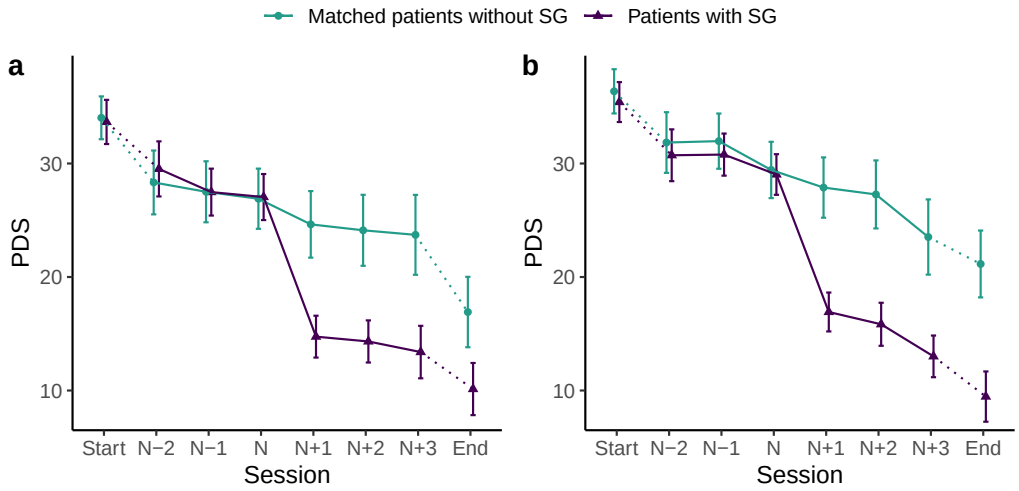


Figure 4.3: Average change in PTSD symptoms (PDS) around the sudden gain/matched session. Error bars represent 95% confidence intervals.

Cognitive processes associated with sudden gains

When analysing processes around sudden gains, patients with sudden gains were compared with matched patients who were similar in relevant patient characteristics, symptoms, and cognitive processes at baseline. Figure 4.3 illustrates that the PTSD symptom trajectory was very similar for both groups in both samples up to the session before the sudden gain/matched session and different afterwards.

All baseline variables were well balanced between the sudden gains and matched groups for all continuous variables, mean differences in PDS baseline scores (Sample 1 = 0.36, Sample 2 = 0.95), mean difference in months since main index trauma (Sample 1 = 0.64, Sample 2 = 1.64), mean differences in treatment length (Sample 1 = 0.18, Sample 2 = 0.40 sessions), and identical for all categorical variables. Figure 4.3 shows the average change in PTSD symptoms around the sudden gain/matched session for both samples. The average sudden gain represented a marked change from the otherwise similar symptom trajectory in the two groups up to the point of the sudden gain. Explorative analyses suggest that both groups in both samples showed a similar degree of improvement from the postgain session or the corresponding matched session to the end of therapy.

Baseline correlations between PTSD, depression, and anxiety symptoms as well as cognitive process measures, were medium to high and statistically significant

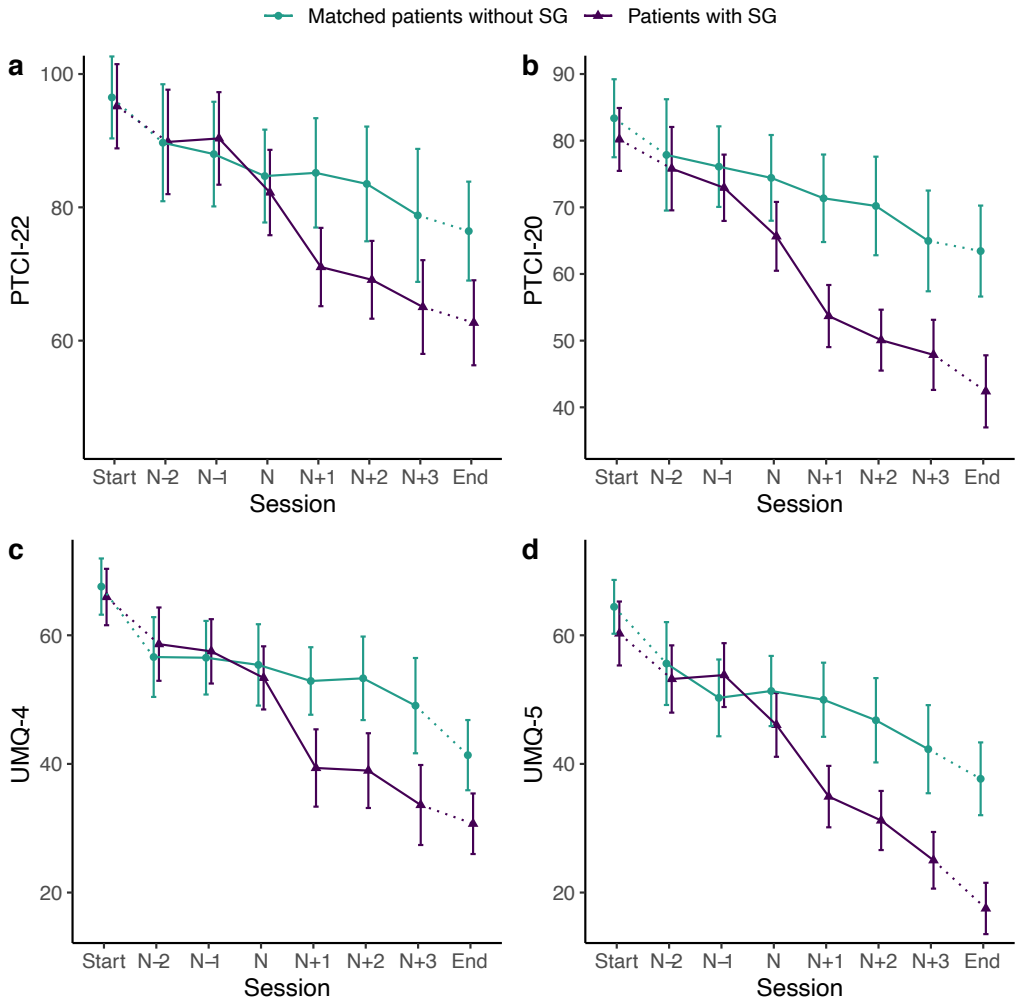


Figure 4.4: Average change in negative appraisals (Sample 1: PTCI-22; Sample 2: PTCI-20) and flashback memories (Sample 1: UMQ-4; Sample 2: UMQ-5) around the sudden gain/matched session. Error bars represent 95% confidence intervals.

(see Appendix C, Tables C.1 and C.2). Figure 4.4 shows the average change in negative appraisals and flashback memories around the sudden gain/matched session. Within- and between-group changes are presented in Table 4.4.

During the sudden gain (N to N+1), in both samples, the sudden gains group showed large and statistically significant decreases in cognitive processes, which were larger than those observed in the matched control group for both negative appraisals and flashback memories. The pooled estimates of Samples 1 and 2 for change in negative appraisals (β = -0.71, 95% CI [-0.96, -0.45], $p < .001$) and flashback memories (β = -0.58, 95% CI [-0.84, -0.31], $p < .001$) during sudden gains showed significant differences between the sudden gains and matched groups

Table 4.4: Estimated changes in negative appraisals and flashback memories in the time intervals around the sudden gain

	Sudden gains group		Matched group		Group difference	
Measure / Time interval	δ_1 (SE)	p	δ_2 (SE)	p	Δ_3 (SE)	p
S_1: Negative appraisals						
N–2 to N-1	1.36 (2.01)	.500	-1.60 (2.09)	.445	2.95 (2.90)	.309
N–1 to N	-6.08 (1.85)	.001	-3.03 (1.92)	.114	-3.05 (2.66)	.252
N to N+1	-10.90 (1.80)	<.001	-0.39 (1.89)	.837	-10.51 (2.61)	<.001
N+1 to N+2	-3.05 (1.80)	.091	-2.51 (1.98)	.205	-0.53 (2.68)	.842
S_2: Negative appraisals						
N–2 to N–1	-5.14 (2.18)	.018	-0.71 (2.36)	.762	-4.42 (3.22)	.169
N–1 to N	-7.40 (1.94)	<.001	-2.89 (2.14)	.176	-4.51 (2.88)	.117
N to N+1	-12.12 (1.93)	<.001	-2.90 (2.11)	.169	-9.22 (2.86)	.001
N+1 to N+2	-3.63 (1.92)	.058	-3.16 (2.19)	.150	-0.48 (2.91)	.870
S_1: Flashback memories						
N–2 to N–1	-2.12 (2.45)	.386	0.25 (2.61)	.924	-2.37 (3.58)	.508
N–1 to N	-3.96 (2.28)	.082	-2.60 (2.40)	.278	-1.36 (3.31)	.680
N to N+1	-14.18 (2.29)	<.001	-3.63 (2.35)	.122	-10.54 (3.28)	.001
N+1 to N+2	-1.81 (2.43)	.457	-0.41 (2.41)	.864	-1.40 (3.42)	.683
S_2: Flashback memories						
N–2 to N–1	-1.83 (2.31)	.430	-5.23 (2.54)	.039	3.41 (3.43)	.321
N–1 to N	-8.09 (2.06)	<.001	-1.54 (2.29)	.501	-6.56 (3.08)	.033
N to N+1	-11.26 (1.98)	<.001	-3.11 (2.11)	.140	-8.15 (2.89)	.005
N+1 to N+2	-3.80 (1.98)	.056	-3.18 (2.20)	.148	-0.62 (2.96)	.835

Note. For each time interval the estimated changes were compared within (δ_1, δ_2) and between (Δ_3) groups.

(Figures 4.5 and 4.6). In the interval before the sudden gain (N-1 to N), negative appraisals already showed decreases in the sudden gains group for both samples, whereas the decreases in the matched groups were non-significant.

For flashback memories, a significant decrease in the sudden gains group was found in Sample 2 and a trend in Sample 1, whereas the decreases in matched controls were non-significant. The pooled estimates for the group differences in change in cognitive processes preceding sudden gains showed a significant group difference for negative appraisals, β = -0.27, 95% CI [-0.53, -0.02], p = .038, and the same effect size, but that was not statistically significant for flashback memories, β = -0.27, 95% CI [-0.54, 0.01], p = .059, indicating greater cognitive change in the sudden gains group (Figures 4.5 and 4.6) before the sudden gain. For the postgain session, no significant changes were found in negative appraisals or

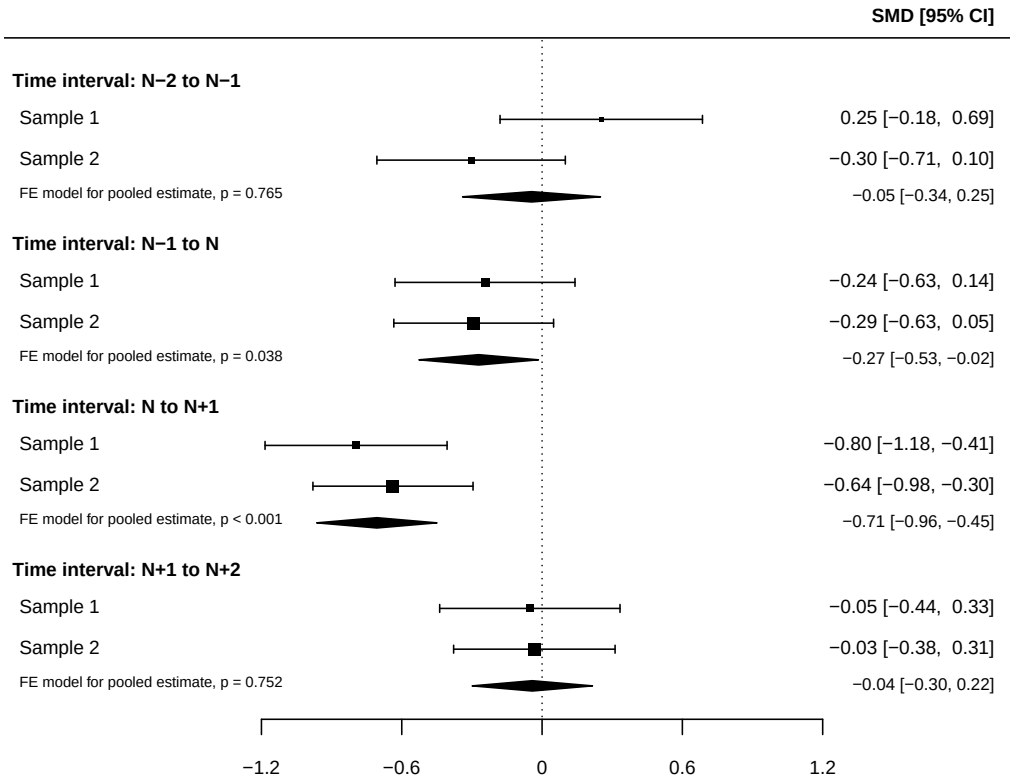


Figure 4.5: Forest plot of the group difference in changes in negative appraisals. Negative numbers indicate greater change in the sudden gains group; positive numbers indicate greater change in the matched patients without sudden gains. The point sizes are proportional to the precision of the estimates. SMD = Standardised Mean Difference; FE = Fixed Effect.

flashback memories for either group in either sample.

4.1.4 Discussion

This study investigated change processes around sudden gains during an empirically validated treatment for PTSD in routine clinical practice in two samples of consecutive cases and found that a substantial subgroup of around one third of patients showed large improvements in PTSD symptoms from one session to the next. In line with the first hypothesis, sudden gains were associated with better treatment outcomes in both samples, as measured by both self-reported and interviewer-rated PTSD-symptom severity. This replicates previous findings with other psychological therapies for PTSD (e.g., Aderka, Appelbaum-Namdar, et al., 2011; Kelly et al., 2009; König et al., 2014; Krüger et al., 2014). To analyse change

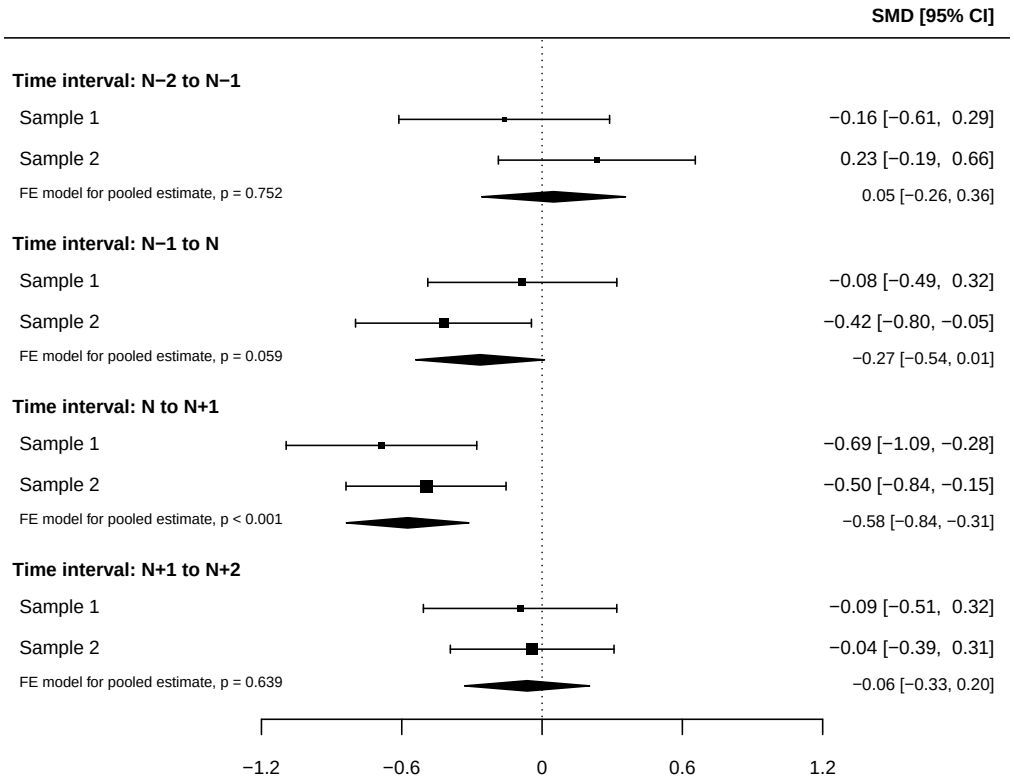


Figure 4.6: Forest plot of the group difference in changes in flashback memories. Negative numbers indicate greater change in the sudden gains group; positive numbers indicate greater change in the matched patients without sudden gains. The point sizes are proportional to the precision of the estimates. SMD = Standardised Mean Difference; FE = Fixed Effect.

processes around sudden gains, this study compared changes between patients with sudden gains and matched patients without sudden gains. In line with the second hypothesis, patients who experienced a sudden gain in PTSD symptoms showed large concurrent improvements in cognitive processes thought to maintain PTSD [negative appraisals and flashback memories; Ehlers and Clark (2000)]. In line with the third hypothesis, pooled estimates across both samples showed that negative appraisals had already decreased in the session prior to sudden gains to a larger extent than for matched patients before the corresponding matched session, and there was also a trend for a greater decrease in trauma flashback memories.

Sudden gains occurred in a similar proportion of patients in both samples (30.65% and 37.18%), with a similar average magnitude of the sudden gain (M = 12.30, SD = 4.44 and M = 12.11, SD = 3.83). These results are similar to

previous studies in PTSD (e.g., 22%, König et al., 2014; 25%, Krüger et al., 2014) and other disorders (37%, Aderka et al., 2012). Although a minority of patients with sudden gains met the Tang and DeRubeis (1999) criterion for a subsequent loss of 50% of the gain (reversal), most of these regained the improvements made during the sudden gain by the end of therapy, suggesting that reversals were mainly temporary deteriorations. Only three sudden gainers in Sample 2 experienced a stable reversal that met the criteria for a sudden loss. There was an interesting difference between the samples in that more patients experienced sudden gains early in treatment in Sample 2 compared with Sample 1 (see Figure 4.1), which paralleled the earlier use of the updating-memory procedure in Sample 2. This might indicate that starting to work on the trauma memory early in treatment facilitates large improvements in some patients.

No evidence for consistent baseline predictors of sudden gains was found across the samples. In contrast to Vittengl et al. (2005), we did not find that the baseline severity of the sudden gains outcome measure (PDS) predicts sudden gains in PTSD. Similar to other studies (e.g., Hunnicutt-Ferguson et al., 2012; Vittengl et al., 2005), we did not find evidence that cognitive processes at the beginning of treatment predict the occurrence of sudden gains, suggesting that processes during therapy are more important in the occurrence of sudden gains than patient characteristics or symptomatology before the treatment.

In line with some other sudden gains studies in depression (Tang & DeRubeis, 1999; Tang et al., 2005), this study also found evidence for cognitive changes prior to the sudden gain (see Table 4.4, δ_1 for negative appraisals from N-1 to N). However, matched patients without sudden gains also experienced non-significant decreases. This highlights the importance of a control group when analysing processes around sudden gains. Although the observed group differences with effect sizes of -0.24 and -0.29 did not reach significance within each sample, the meta-analysis suggested greater changes in appraisals in the sudden gains groups, -0.27, 95% CI [-0.53, -0.02], $p = .038$ (see Figure 4.5). Similar effects for group differences were obtained for flashback memories, with a pooled estimate of -0.27, which was not statistically

significant. Thus, there was some support for Hypothesis 3, although the effects were small. Three other studies did not find evidence for significant cognitive changes preceding sudden gains in individual samples of other disorders (Andrusyna et al., 2006; Bohn et al., 2013; Hofmann et al., 2006), suggesting overall small effects. Larger samples or pooling data across samples may be a way to further investigate the effect we found in this study. The observation that PTSD symptoms and cognitive-process variables are correlated with each other at baseline (see Appendix C, Tables C.1 and C.2) does not explain this pattern of findings.

This study also found further evidence for simultaneous changes of cognitive processes with the sudden gain in PTSD symptoms, supporting Hypothesis 2. These findings might partly be explained by the correlations between symptoms and cognitive processes in this sample. Our results show evidence that these concurrent changes are preceded by changes in cognitions.

Strengths and limitations

This study investigated the processes associated with sudden gains in two large clinical samples of patients with PTSD treated in routine clinical care with an empirically validated psychological treatment who completed weekly symptom and process measures. The large samples allowed for an advanced matching approach to create control groups of similar patients without sudden gains. The statistical modelling approach ensured a detailed analysis of potential process variables leading up to the gain, during the gain, and after the gain. Further, this is the first study of sudden gains to report identifying sudden gains using a fully automated approach and sharing the code publicly. A more detailed discussion of the benefits of transparent research practices and replication studies in the psychological sciences can be found elsewhere (Nosek et al., 2015; Open Science Collaboration, 2015; Tackett et al., 2017).

The limitations of this study include the variations in measures across the samples that reflected changes in clinic procedures. The internal reliability of the measure assessing flashback memories in Sample 1 was low (UMQ-4; Cronbach's

α at baseline = .62). However, because similar results for changes in flashback memories in Sample 2 were obtained with an improved measure (UMQ-5, Cronbach's α at baseline = .84), the findings appear to be valid. However, the measure only contained one item measuring the disjointedness of memories and did not assess other potentially relevant aspects of memory disorganisation, so the effect may have been underestimated. In addition, all measures assessing changes around sudden gains were patient self-reports, and other data, such as ratings of videotapes, were not available. Furthermore, the standard criteria used to identify sudden gains may yield some false positives. In a data simulation study, Vittengl et al. (2015) found that some sudden gains are due to random symptom fluctuation during therapy. Thomas and Persons (2013) argue that some sudden gains represent the largest and most stable change occurring in a gradual course of change.

Conclusions

From a methodological perspective, the present results highlight the importance of a control group when analysing processes associated with sudden gains. Whereas this and other studies (e.g., Wucherpfennig, Rubel, Hofmann, et al., 2017) assigned matched sessions based on the pregain session of the matched sudden gains patient, alternative methods also need to be explored. For example, taking the session with the largest gain in patients without sudden gains as the matched session may be a sensible alternative when analysing processes around sudden gains. Smaller intervals of measuring symptom and process variables would allow a more accurate identification of the point during the week at which the sudden gains occurred and also the identification of processes that precede and follow the gain more closely in time.

This study showed, in two independent, consecutive samples, that sudden gains occur in about a third of patients treated with CT-PTSD and reliably predict better treatment outcomes. There were no reliable baseline predictors of sudden gains, suggesting that they can occur in a wide range of patients. When sudden gains occur, they are associated with broad changes in cognitive processes. These

findings provide a better understanding of how CT-PTSD works, especially in patients with sudden gains.

4.2 Patterns of individual symptom changes during sudden gains

4.2.1 Aims

As discussed in Chapter 1.6.2, it is currently not known which individual symptoms change during sudden gains in PTSD symptom severity. A better understanding of the patterns of symptom change during sudden gains in PTSD symptoms may help to further understand individual differences between patients and guide further research questions.

The primary aim of the present study is to explore changes in depression and anxiety severity around PTSD sudden gains identified in Chapter 4.1. We hypothesised that patients with sudden gains will show greater change in depression and anxiety symptoms during the PTSD sudden gain compared to matched patients who did not experience a sudden gain. Given the heterogeneity of PTSD and high rates of comorbidity with depression and anxiety, the second aim of the present study was to examine whether the analysis of individual PTSD, depression, and anxiety symptoms changes can improve our understanding of patterns of symptom change during sudden gains in PTSD symptoms. In patients who experienced sudden gains, we aimed (1) to quantify improvements in individual PTSD, depression, and anxiety symptoms around the time of PTSD symptom gains and (2) to explore potential subcategories of sudden gains in PTSD symptoms by taking into account the degree of simultaneous changes in depression and anxiety.

4.2.2 Methods

Participants

This study is a secondary analysis of two studies of consecutive cases ($N_1 = 330$; $N_2 = 343$) investigating the effectiveness of cognitive therapy for PTSD in routine

clinical care (Ehlers et al., 2013; Ehlers, Wild, et al., 2020). The patients' primary diagnosis was PTSD, as determined by the Structured Clinical Interview for *DSM-IV* (SCID; First et al., 1997), as this was the current edition of DSM when the patients included in this study were treated. Patients who provided sufficient data to apply Tang and DeRubeis's (1999) sudden gains criteria were included in this study (n_1 = 248; Sample 2, n_2 = 234). A detailed description of the patients included in this study can be found in Chapter 4.1, for patient characteristics see Table 4.1. Ethical approval was granted by the local research ethics committee.

Treatment

Patients received a course of cognitive therapy for PTSD (CT-PTSD; Ehlers et al., 2005) based on Ehlers and Clark's (2000) cognitive model of PTSD. The treatment aims to reduce the patient's sense of current threat by changing problematic meanings of the trauma and its consequences, elaborating and updating the memories of the trauma with information that gives them a less threatening meaning at present, discriminating triggers of intrusive memories, and changing behaviours and cognitive processes that maintain PTSD, such as rumination and safety behaviours. Therapists were qualified or trainee clinical psychologists, psychiatrists or nurse therapists with a range of experience in treating PTSD. On average, patients received $M = 11.55$ ($SD = 4.63$) treatment sessions in Sample 1 and $M = 10.81$ ($SD = 4.35$) sessions in Sample 2. More details can be found in Ehlers et al. (2013) and Chapter 4.1.

Measures

Patients completed the following measures before each treatment session. Item descriptions for each measure are presented in Appendix C (see Tables C.3 to C.7). Because of copyright restrictions we only provide a short description of the content of each item to assist interpretation of the results. The depression and anxiety measures differed between Sample 1 and 2 due to a change in procedures for the respective NHS services.

PTSD symptoms. Both patient cohorts completed the Posttraumatic Diagnostic Scale (PDS; Foa et al., 1997) to assess PTSD symptom severity according to the DSM-IV criteria. The PDS is a reliable and validated 17-item self-report measure (Foa et al., 1997) assessing reexperiencing (4 items), avoidance (7 items), arousal (6 items) symptoms as specified in the DSM-IV (American Psychiatric Association, 2000). Patients rated the extent to which they were bothered by each of the 17 symptoms during the last week on a scale from 0 (*Not at all*) to 3 (*5 or more times a week/almost always*). See Table C.3 for a description of individual PDS items.

Depression symptoms. To assess depressive symptoms, Sample 1 completed the 21-item Beck Depression Inventory (BDI; Beck & Steer, 1993a) and Sample 2 completed Patient Health Questionnaire 9-item scale (PHQ-9; Kroenke et al., 2001). Items on the BDI ranged from 0 to 3 with different response categories for each item and from 0 (*Not at all*) to 3 (*Nearly every day*) on the PHQ-9. The internal consistency at baseline was Cronbach's $\alpha = .90$ on the BDI and $\alpha = .91$ on the PHQ-9. See Tables C.4 and C.5 for a short description of individual BDI and PHQ-9 items.

Anxiety symptoms. To assess anxiety symptoms Sample 1 completed the 21-item Beck Anxiety Inventory (BAI; Beck & Steer, 1993b) and Sample 2 completed the Generalised Anxiety Disorder 7-item scale (GAD-7; Spitzer et al., 2006). Patients were asked to indicate how much they were bothered by each symptom during the past week from 0 (*Not at all*) to 3 (*Severely – it bothered me a lot*) on the BDI and from 0 (*Not at all*) to 3 (*Nearly every day*) on the GAD-7. The internal consistency at baseline was Cronbach's $\alpha = .93$ on the BAI and $\alpha = .90$ on the GAD-7. See Tables C.6 and C.7 for a short description of individual BAI and GAD-7 items.

Data analyses

All analyses were performed in R (Version 4.0.2; R Core Team, 2020) through RStudio IDE (Version 1.3.1073; RStudio Team, 2020). We made considerable use of R packages from the *tidyverse* (Version 1.3.0; Wickham et al., 2019) for structuring

and visualising data. Similar to the research design in Chapter 4.1 each analysis was conducted separately within each sample.

Identification of PTSD sudden gains. Sudden gains in PTSD symptoms were identified as described in Chapter 4.1 using the R package *suddengains* developed in Chapter 2.3. Following the three criteria described by Tang and DeRubeis (1999) a sudden gain was identified between session N (pregain session) and N+1 (postgain session) when: (1) the decrease between two consecutive scores on the PDS was at least 6.15 point on the PDS, (2) PDS scores decreased by at least 25% relative to the pregain score, and (3) the pooled standard deviation between the mean PDS score of three sessions (or two, if three were not available) before the sudden gain and after the sudden gain was greater than the following critical values. The earliest gain was selected if patients experienced more than one sudden gain.

Changes in depression and anxiety severity around PTSD gains. Changes in depression and generalised anxiety severity around PTSD gains were assessed using the same matched samples and methods described in Chapter 4.1. Matched patients without sudden gains were selected using Mahalanobis distance matching including the propensity score (Rosenbaum & Rubin, 1985) using a 1:1 matching procedure and were implemented using the R package *MatchIt* (Version 3.0.2; Ho et al., 2011). Each matched patient was assigned a 'matched session' with the same pregain session number as the patient with a sudden gain they were matched with.

Differences in changes in comorbid symptom severity before (N-2 to N-1 and N-1 to N), during (N to N+1), and after (N-1 to N+2) sudden gains and matched sessions between the groups were analysed using linear mixed effect models. Differences between the time intervals are labelled δ_1 within the sudden gains group, δ_2 within the matched control group and differences between the two groups are labelled as Δ_3. The assumption of normality of the residuals was confirmed visually for all outcomes. A fixed effects model was run using the R package *metafor* (Version 2.0.0;

Viechtbauer, 2010) to estimate the pooled effect across both samples in this study based on the adjusted Standardised Mean Difference (SMD; Hedges & Olkin, 1985).

Characteristics of individual symptom changes around PTSD gains. Individual item-by-item scores around the sudden gain were extracted using the package *suddengains* (Version 0.4.4; Wiedemann, Thew, et al., 2020, see Chapter 2.3). To understand how many symptoms improved on average during sudden gains we visualised the underlying probability distribution of the number of item-by-item improvements for each measure in density plots. For PTSD symptoms we then explored how sudden gains are experienced across the PTSD symptom categories according to the DSM-IV criteria (reexperiencing, avoidance, arousal). For PTSD, depression, and generalised anxiety symptom measures we quantified how many patients with sudden gains improved on individual items during the sudden gain (N to N+1). We also computed changes on all items immediately prior (N-1 to N) and following (N+1 to N+2) the gain. To guide interpretation of the improvements around the time of the gain we also compared pretreatment scores between all patients with and without sudden gains. Differences in item-by-item improvements from start to end of treatment between these groups were also explored to investigate whether patients with sudden gains showed larger improvements on individual symptoms by the end of treatment.

Examination of possible sudden gains subcategories. To explore whether sudden gains in PTSD symptoms can be divided into different subcategories depending on whether or not depression and generalised anxiety symptoms change simultaneously we visualised item-by-item changes for all measures. A heatmap was developed to facilitate the exploration of potential subcategories.

4.2.3 Results

Changes in depression and anxiety severity associated with sudden gains on posttraumatic stress disorder symptoms

During the sudden gain (N to N+1) in PTSD symptoms, depression and anxiety severity showed significant decreases in the sudden gains group in both samples, see Table 4.5 for estimated within- (δ_1, δ_2) and between-group (Δ_3) changes. There were significant differences between patients with sudden gains and matched controls without sudden gains in the degree of change in both depression and anxiety severity, with significant pooled estimates as shown in Figures 4.7 and 4.8 (Depression severity: β = -0.90, 95%CI [-1.15, -0.66], $p < .001$; Anxiety severity, β = -0.72, 95% CI [-0.96, -0.47], $p < .001$). The average change in depression and anxiety severity around the sudden gain and matched session is shown in Appendix C, see Figure C.2.

There were no significant group differences (Δ_3) in the degree of change in depression or generalised anxiety severity before the sudden gain in either sample. However, the pooled estimates for both samples together suggest that generalised anxiety (β = -0.24, 95% CI [-0.47, 0.00], p = .050) but not depression (β = -0.09, 95% CI [-0.33, 0.14], p = .431) tended to show a greater decrease prior to sudden gains compared to the corresponding session in the matched group (see time interval N-1 to N in Figures 4.7 and 4.8). In the postgain session no statistically significant changes were found in depression or generalised anxiety severity in either group of the two samples.

Characteristics of individual symptom changes in the sudden gains group

Number of symptoms improving before, during, and after sudden gains. The estimated probability distribution illustrates the number of symptoms that improved before (yellow), during (green) and after (blue) the sudden gain as density plots (see Figure 4.9). Most patients with sudden gains reported improvements on 8 to 12 symptoms on the 17-item PDS during the sudden gain (N to N+1) in both samples. The number of items with improvements immediately before (N-1 to N) and after (N+1 to N+2) the gain was considerably lower. For depressive and generalised

Table 4.5: Estimated changes in depression and generalised anxiety severity in the time intervals around the sudden gain

	Sudden gains group		Matched group		Group difference	
Measure / Time interval	δ_1 (SE)	p	δ_2 (SE)	p	Δ_3 (SE)	p
S_1: Depression (BDI)						
N–2 to N-1	0.15 (0.81)	.850	-0.92 (0.84)	.274	1.07 (1.16)	.358
N–1 to N	-1.65 (0.76)	.031	-1.27 (0.81)	.115	-0.37 (1.11)	.735
N to N+1	-5.81 (0.76)	< .001	0.29 (0.81)	.718	-6.10 (1.11)	< .001
N+1 to N+2	-0.34 (0.76)	.659	-1.98 (0.84)	.018	1.65 (1.13)	.145
S_2: Depression (PHQ-9)						
N–2 to N–1	-1.07 (0.53)	.043	-0.46 (0.56)	.409	-0.61 (0.77)	.424
N–1 to N	-1.90 (0.47)	< .001	-1.41 (0.50)	.004	-0.48 (0.69)	.480
N to N+1	-4.21 (0.47)	< .001	-0.86 (0.51)	.089	-3.35 (0.69)	< .001
N+1 to N+2	-0.67 (0.47)	.152	0.03 (0.52)	.957	-0.70 (0.70)	.321
S_1: Anxiety (BAI)						
N–2 to N–1	0.87 (1.06)	.411	-0.51 (1.10)	.640	1.38 (1.52)	.364
N–1 to N	-1.53 (0.99)	.122	-0.23 (1.05)	.825	-1.29 (1.44)	.370
N to N+1	-6.83 (0.98)	< .001	-1.61 (1.07)	.133	-5.22 (1.45)	< .001
N+1 to N+2	-0.56 (0.99)	.573	-0.83 (1.10)	.451	0.28 (1.48)	.852
S_2: Anxiety (GAD-7)						
N–2 to N–1	0.18 (0.47)	.701	-0.63 (0.49)	.203	0.81 (0.68)	.236
N–1 to N	-2.01 (0.42)	< .001	-0.93 (0.44)	.035	-1.08 (0.61)	.075
N to N+1	-3.56 (0.41)	< .001	-0.81 (0.45)	.070	-2.75 (0.61)	< .001
N+1 to N+2	-0.56 (0.41)	.178	-0.21 (0.47)	.645	-0.34 (0.62)	.582

Note. S_1 = Sample 1; S_1 = Sample 2. For each time interval before (N-2 to N-1 and N-1 to N), during (N to N+1), and after (N+1 to N+2) the estimated changes were compared within (δ_1, δ_2) and between (Δ_3) groups.

anxiety symptoms, in Sample 1, most sudden gains patients improved on 2 to 6 symptoms on the 21-item depression measure (BDI) and 2 to 8 items on the 21-item anxiety measure (BAI). In Sample 2, the bimodal density distributions indicated that some patients with sudden gains improved only on few items (PHQ-9: 1 to 3; GAD-7: 0 to 2) while others showed improvements on more items (PHQ-9: 4 to 6; GAD-7: 4 to 7). Exploratory correlation analyses showed that there was a moderate correlation between the number of items that improved during the sudden gain and symptom severity immediately prior to the sudden gain, suggesting that patients who reported more severe PTSD, depression, and generalised anxiety symptoms also reported improvements on more items during the sudden gain (Sample 1: $r_{\text{PDS}} = 0.54$, $p = < .001$; $r_{\text{BDI}} = 0.60$, $p = < .001$; $r_{\text{BAI}} = 0.63$, $p = < .001$; Sample 2:

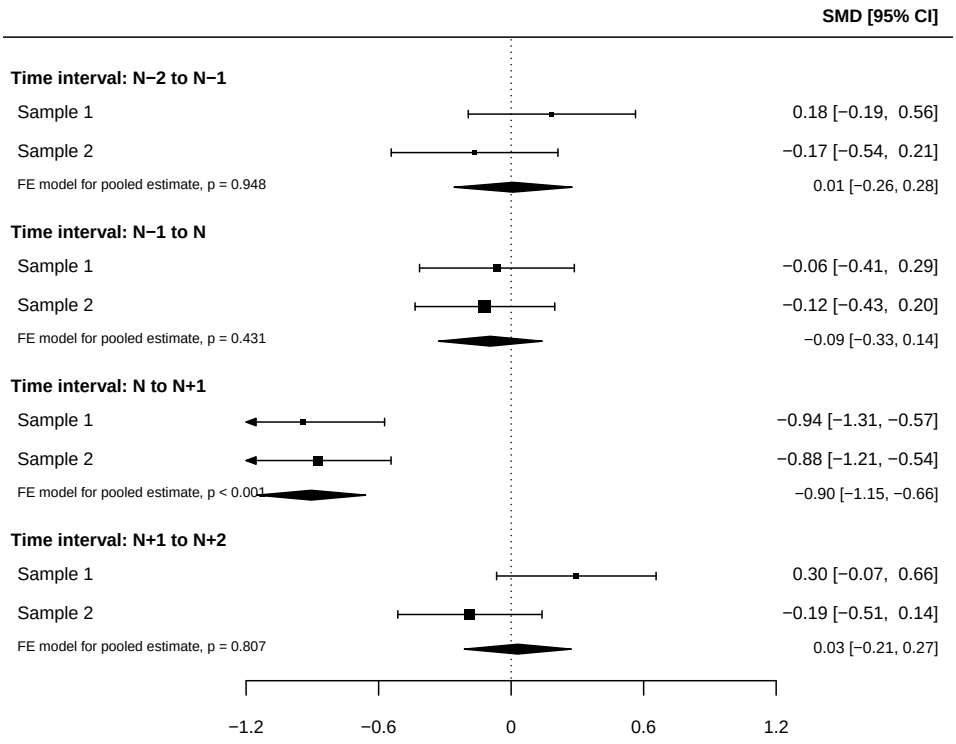


Figure 4.7: Forest plot of the difference between the sudden gains and matched control groups in changes in depression symptoms before, during and after the PTSD sudden gain. Negative numbers indicate greater change in the sudden gains group; positive numbers indicate greater change in the matched patients without sudden gains. The point sizes are proportional to the precision of the estimates. SMD = Standardised Mean Difference; FE = Fixed Effect.

$r_{\text{PDS}} = 0.40$, $p = < .001$; $r_{\text{PHQ-9}} = 0.60$, $p = < .001$; $r_{\text{GAD-7}} = 0.63$, $p = < .001$).

Patterns of PTSD symptom improvements during sudden gains. In Sample 1, every combination of PDS item improvements during the sudden gain was unique. This means that no patient experienced the same combination of item-by-item improvements as any other patient. In Sample 2, two patients experienced improvements on all 17 items of the PDS, all other combinations of item improvements were unique. Most patients in Sample 1 ($n = 70$, 92%) and Sample 2 ($n = 82$, 94%) improved in all three DSM-IV symptoms categories (*avoidance*, *arousal*, and *intrusion symptoms*). Six (8%) patients in Sample 1

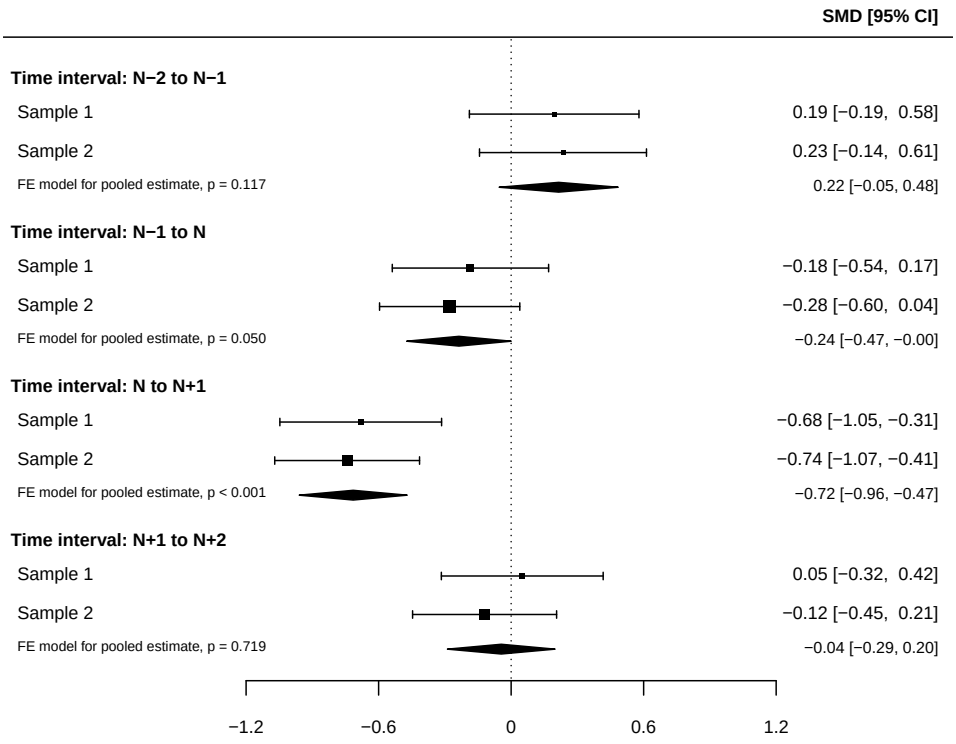


Figure 4.8: Forest plot of the difference between the sudden gains and matched control groups in changes in generalised anxiety symptoms before, during and after the sudden gain. Negative numbers indicate greater change in the sudden gains group; positive numbers indicate greater change in the matched patients without sudden gains. The point sizes are proportional to the precision of the estimates. SMD = Standardised Mean Difference; FE = Fixed Effect.

and five (6%) patients in Sample 2 only improved in two of the three categories (see Appendix C, Figure C.3).

Improvements in PTSD, depression, and anxiety symptoms during the sudden gain. For each individual item around the time of the gain we calculated the percentage of patients with sudden gains who improved to explore the individual patterns of changes in PTSD symptoms during sudden gains (see Figures 4.10A and 4.11A). These changes are reported in context of the overall item changes during treatment (see Figures 4.10B1 and 4.11B1) and pretreatment mean scores of patients with and without sudden gains (see Figures 4.10B2 and 4.11B2).

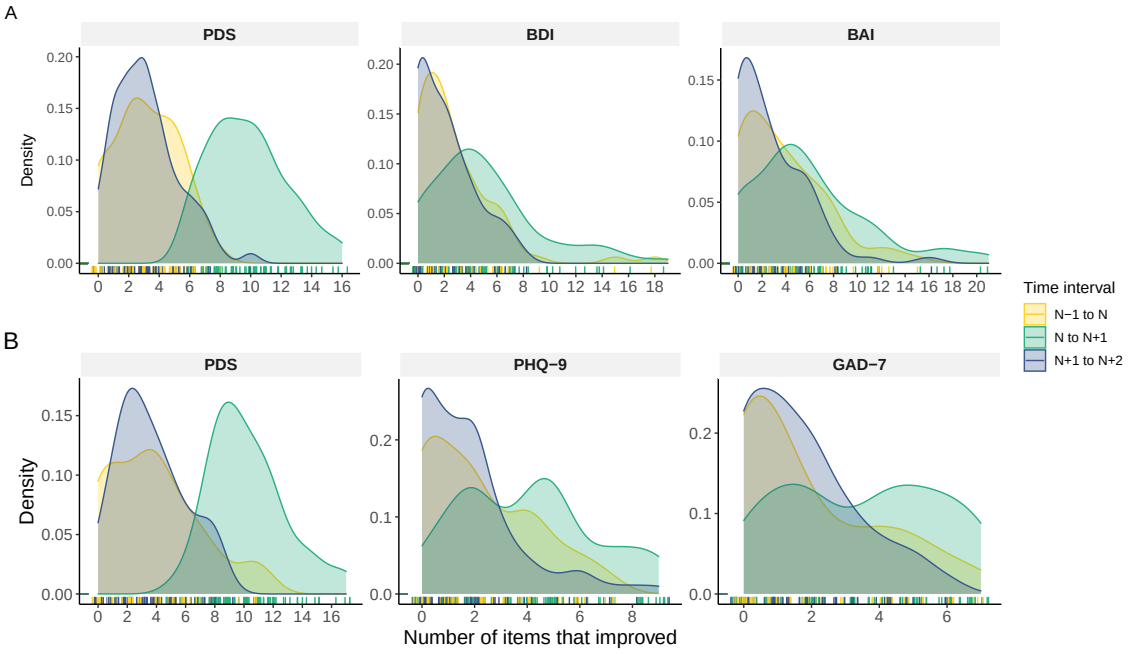


Figure 4.9: Probability distribution of the number of items that improved around sudden gains in PTSD symptoms on each measure for (A) Sample 1 and (B) Sample 2. PDS = PTSD symptoms; BDI and PHQ-9 = Depression symptoms; BAI and GAD-7 = Generalised anxiety symptoms. Vertical lines above the x-axis show each observation.

The results showed that patients who experienced a sudden gain improved in a broad range of PTSD symptoms during the sudden gain. The items on the PDS that improved in the lowest proportions of sudden gainers during the sudden gain were item 2 ('Nightmares') and item 8 ('Difficulty remembering') in both samples and item 11 ('Emotionally numb') in Sample 1 as well as item 3 ('Reliving the traumatic event') in Sample 2. Improvements on all other PDS items were experienced by more than half of the patients during the sudden gain, most notably items 6 ('Avoidance of thoughts'), 13 ('Trouble falling or staying asleep'), and 14 ('Feeling irritable') in Sample 1 as well as items 4 ('Emotionally upset'), 7 ('Avoidance of reminders'), and 17 ('Jumpy or easily startled') in Sample 2.

Patients with sudden gains in PTSD symptoms also experienced improvements in a broad range of depression and generalised anxiety symptoms during the sudden gain. In Sample 1, 5.3% to 38.2% of sudden gainers showed improvements during the PTSD gain on individual depressive symptoms (BDI) and between 13.2% to 38.2% on generalised anxiety symptoms (BAI). In Sample 2, the percentage of patients

who improved on individual PHQ-9 items during the sudden gain ranged from 24.1% to 55.2% and from 43.7% to 54.0% on generalised anxiety symptoms (GAD-7).

Items of the depression measures that most patients improved on during the PTSD gain in Sample 1 (BDI) were items 1 ('Sadness', 38.2%), 16 ('Insomnia', 34.2%), 2 ('Pessimism', 32.9%), and 4 ('Loss of pleasure', 28.9%). In Sample 2 (PHQ-9), most patients with sudden gains improved on items 3 ('Sleep problems', 55.2%), 4 ('Tired or little energy', 54.0%) 6 ('Feeling bad about yourself', 54.0%), and 1 ('Little interest or pleasure', 51.7%). A broad spectrum of generalised anxiety symptoms improved during the PTSD gains, most notably feeling nervous, anxious, or afraid, and trouble relaxing – in Sample 1, BAI items 9 (Terrified or afraid, 38.2%), 5 ('Fear of worst happening', 36.8%), 10 ('Nervous', 35.5%), and 4 ('Unable to relax', 36.8%) and in Sample 2, GAD-7 items 1 ('Feeling nervous, anxious', 52.9%) and 3 ('Worrying about different things', 52.9%), and 4 ('Trouble relaxing', 54.0%).

Comparing PTSD, depression, and anxiety symptom improvements between patients with and without sudden gain by the end of therapy. For each individual PTSD symptom we compared the proportions of patients with and without sudden gains who had improved by the end of treatment (see Figures 4.10B1 and 4.11B1). The largest group differences that were consistent across both samples were observed on item 13 ('Trouble falling or staying asleep', Sample 1: 28.6% and Sample 2: 34.2%), item 2 ('Nightmares', Sample 1: 31.2% and Sample 2: 32.6%), and item 9 ('Loss of interest', Sample 1: 30.9% and Sample 2: 33.8%). The percentages indicate the percentage difference in patients with and without sudden gains who improved on each of these items (e.g., in Sample 1, 28.6% more patients with sudden gains improved on item 13 compared to patients who did not experience a sudden gain). The smallest difference was observed on item 8 ('Difficulty remembering', Sample 1: 4.9% and Sample 2: 4.9%), suggesting that the extent of improvement was very similar between patients with and without sudden gains.

For the depression measures, the largest differences were observed on item 17 on the BDI ('Tiredness of fatigue'; Sample 1: 29.2%) and item 3 on the PHQ-9

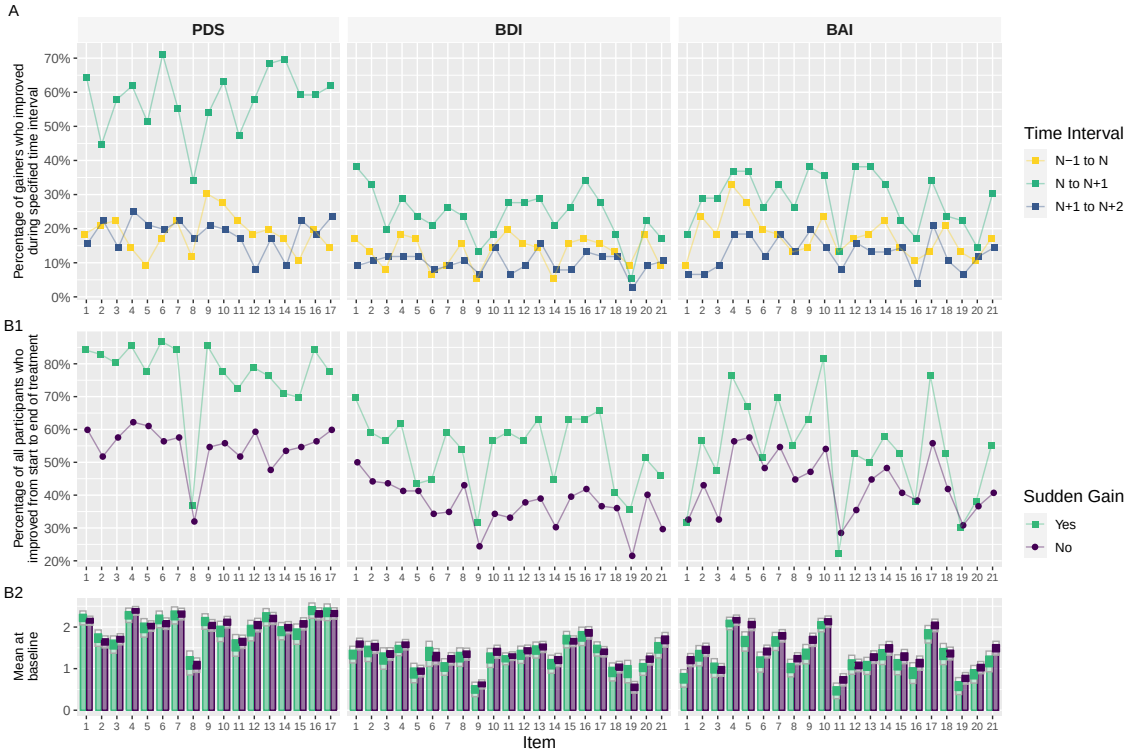


Figure 4.10: Panel A: Percentage of patients with sudden gains who improved on each item before (N-1 to N), during (N to N+1), and after (N+1 to N+2) the sudden gain in Sample 1; Panel B1: Percentage of patients with and without sudden gains who improved on each item from start to end of treatment; Panel B2: Item mean at the start of treatment for all patients.

('Sleep problems'; Sample 2: 32.4%). For generalised anxiety measures, the largest differences were observed on item 10 on the BAI ('Nervous'; Sample 1: 27.5%) and item 1 on the GAD-7 ('Feeling nervous, anxious; Sample 2: 33.4%).

Examination of subcategories of sudden gains

A heatmap was created to visualise item-by-item changes during sudden gains in PTSD symptoms together with changes in depression and generalised anxiety symptoms. After a visual inspection of the heatmap the following subcategories of PTSD gains were developed: '*generalised gains*' describe sudden gains in PTSD symptoms together with reliable improvement in depression or generalised anxiety symptoms, '*specific gains*' describe sudden gains in PTSD symptoms and no reliable improvement in depression or generalised anxiety symptoms. This category is further subdivided because the lack of reliable improvement in these measures has

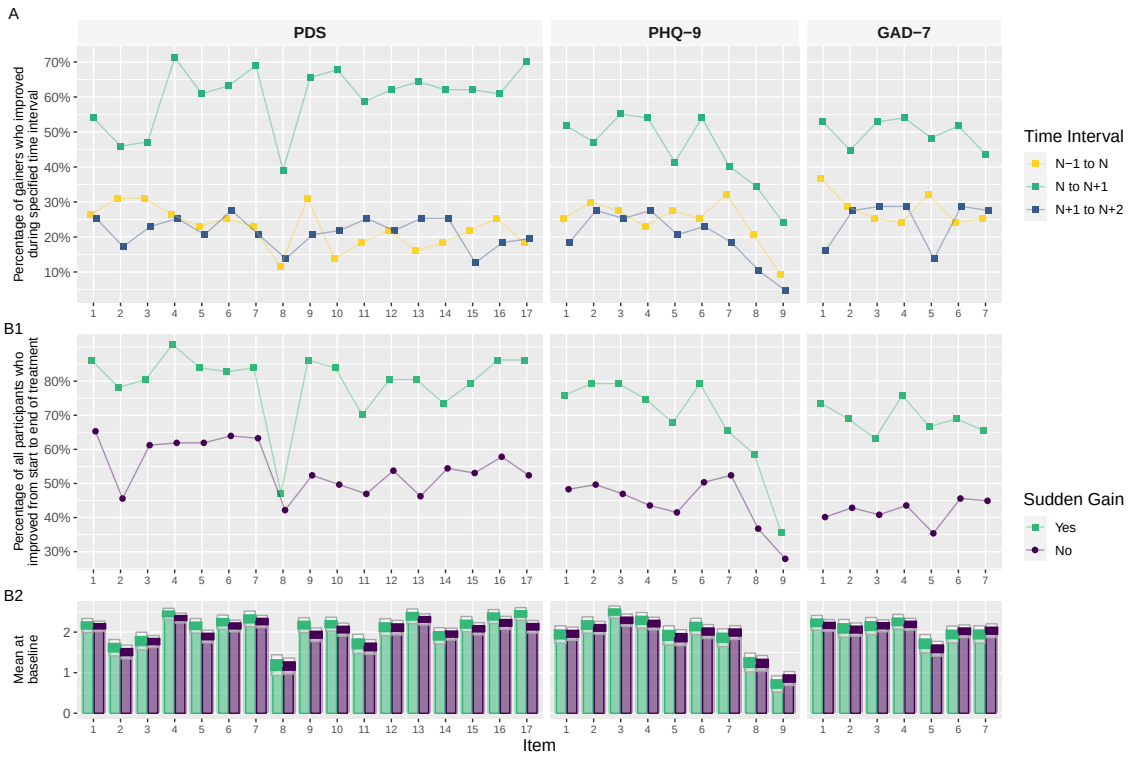


Figure 4.11: Panel A: Percentage of patients with sudden gains who improved on each item before (N-1 to N), during (N to N+1), and after (N+1 to N+2) the sudden gain in Sample 2; Panel B1: Percentage of patients with and without sudden gains who improved on each item from start to end of treatment; Panel B2: Item mean at the start of treatment for all patients.

different meanings for people with comorbid generalised anxiety and depression. For example, those who already score in the nonclinical range before the sudden gain in PTSD symptoms could not be expected to show a symptom change large enough to meet the criterion for reliable improvement. Thus, we distinguished between a specific gain in PTSD when generalised anxiety and depression were already in the non-clinical range immediately prior to the sudden gain ('*specific gains without clinical comorbidity*', '*Specific 1*' in Figure 4.12) and a truly specific sudden gain in patients with comorbid generalised anxiety or depression ('*specific gains with clinical comorbidity*', '*Specific 2*' in Figure 4.12). '*Mixed gains*' describe sudden gains in PTSD symptoms and reliable deterioration in at least one comorbid symptom measure. In Sample 1, patients were considered to be in the clinical range of depression and generalised anxiety with a score in the moderate range or above on the BDI ($\geq$ 20) and BAI ($\geq$ 15) (Beck & Steer, 1993a, 1993b). Following

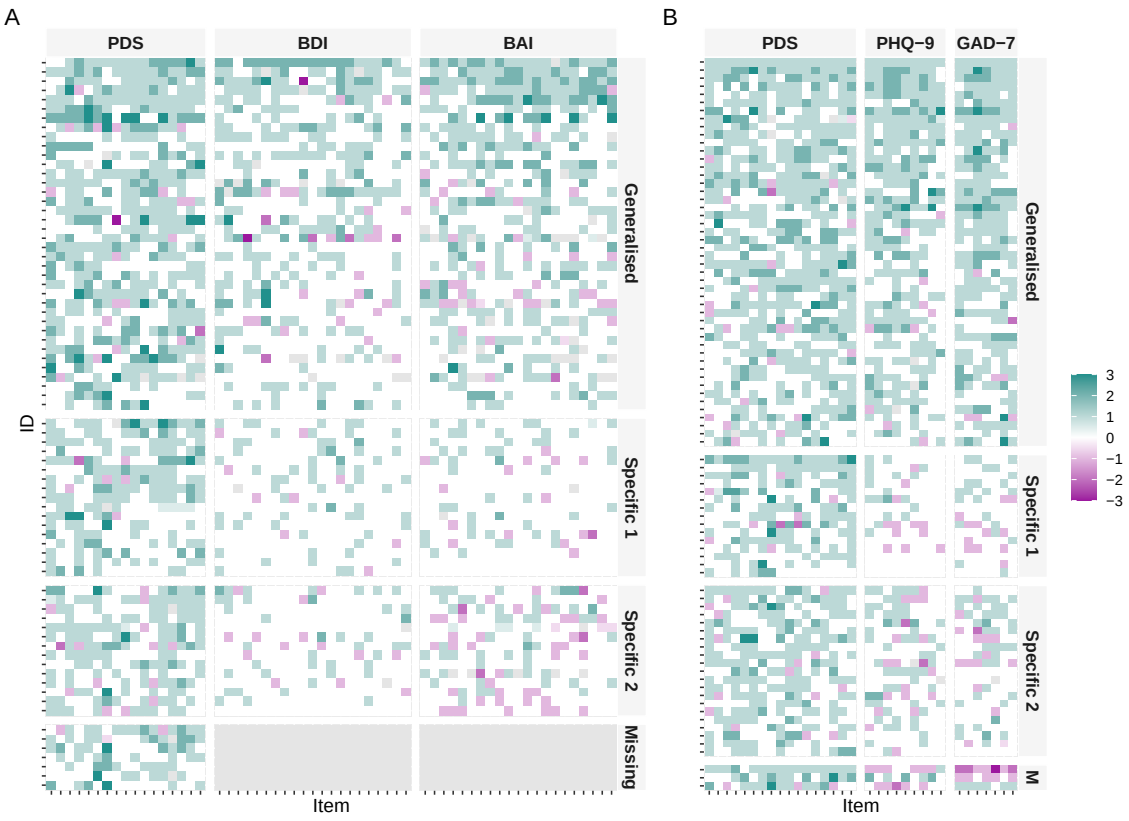


Figure 4.12: Heatmap of item-by-item changes for PTSD, depression, and anxiety measures during the sudden gain in PTSD symptoms (N to N+1) in (A) Sample 1 and (B) Sample 2. Improvement is visualised in green, no change in white, deterioration in pink, and missing values in grey. M = Mixed gains. PDS = PTSD symptoms; BDI and PHQ-9 = Depression symptoms; BAI and GAD-7 = generalised anxiety symptoms. Specific 1 = Specific gain without clinical comorbidity; Specific 2 = Specific gain with clinical comorbidity.

Jacobson and Truax (1991) improvements of 7 or greater were considered as reliable for both measures.[2] In Sample 2, patients were considered to be in the clinical range for depression with a score of 10 or above on the PHQ-9 and generalised anxiety with a score of 8 or above on the GAD-7 (Kroenke et al., 2001; Spitzer et al., 2006). Improvements greater than 5 on the PHQ-9 (McMillan et al., 2010), and 4 on the GAD-7 (The National Collaborating Centre for Mental Health, 2020) were considered as reliable.

Taking into account improvements in depression and generalised anxiety symp-

[2]Standard deviations of a nonclinical sample and the test-retest reliabilities (r_{tt}) were taken from Barkham et al. (1996) for the BDI ($SD_{\text{nonclinical}} = 4.46$; $r_{tt} = 0.75$), and from Osman et al. (2002) for the BAI ($SD_{\text{nonclinical}} = 5.05$; $r_{tt} = 0.75$).

toms during PTSD gains we found that $n = 38$ (50.0%) in Sample 1 and $n = 48$ (55.2%) in Sample 2 experienced a generalised gain. The majority of patients with generalised gains were in the clinical range on the depression or generalised anxiety measure immediately prior to the PTSD gain (Sample 1: $n = 33$, 86.8%; Sample 2: $n = 45$, 93.8%). Furthermore, a significant subgroup of $n = 17$ (22.4%) in Sample 1 and $n = 15$ (17.2%) in Sample 2 were in the non-clinical range in depression and generalised anxiety symptoms before the sudden gain and thus experienced a specific gain without clinical comorbidity.

Only 14 (18.4%) patients in Sample 1 and $n = 21$ (24.1%) in Sample 2 experienced specific gains in PTSD symptoms only, despite reporting clinical comorbidity in depression or anxiety symptoms immediately prior to the sudden gain in PTSD symptoms. The remaining patients in Sample 2 had either mixed gains, $n = 3$ (3.4%) or in Sample 1 missing data in the depression and generalised anxiety measures, $n = 7$ patients (9.2%).

To explore differences in the trajectories of patients with sudden gains and comorbidity who showed a generalised versus a specific gain we supplemented our analyses and visualised the average change in PTSD, depression, and generalised anxiety symptoms around the gain for these groups (see Figure 4.13). Five patients (13.2%) in Sample 1 and three patients (6.2%) in Sample 2 who experienced generalised gains were excluded from these analyses because they were not in the clinical range of depression or generalised anxiety prior to the sudden gain in PTSD symptoms.

As expected, patients with generalised gains who were in the clinical range of depression or generalised anxiety prior to the sudden gain also showed sudden and stable improvements in depression and generalised anxiety symptoms. Patients with specific gains who were in the clinical range of depression or generalised anxiety symptoms showed more gradual and consistent improvement around the time of PTSD gain, particularly for depression symptoms (see Figure 4.13c and 4.13d).

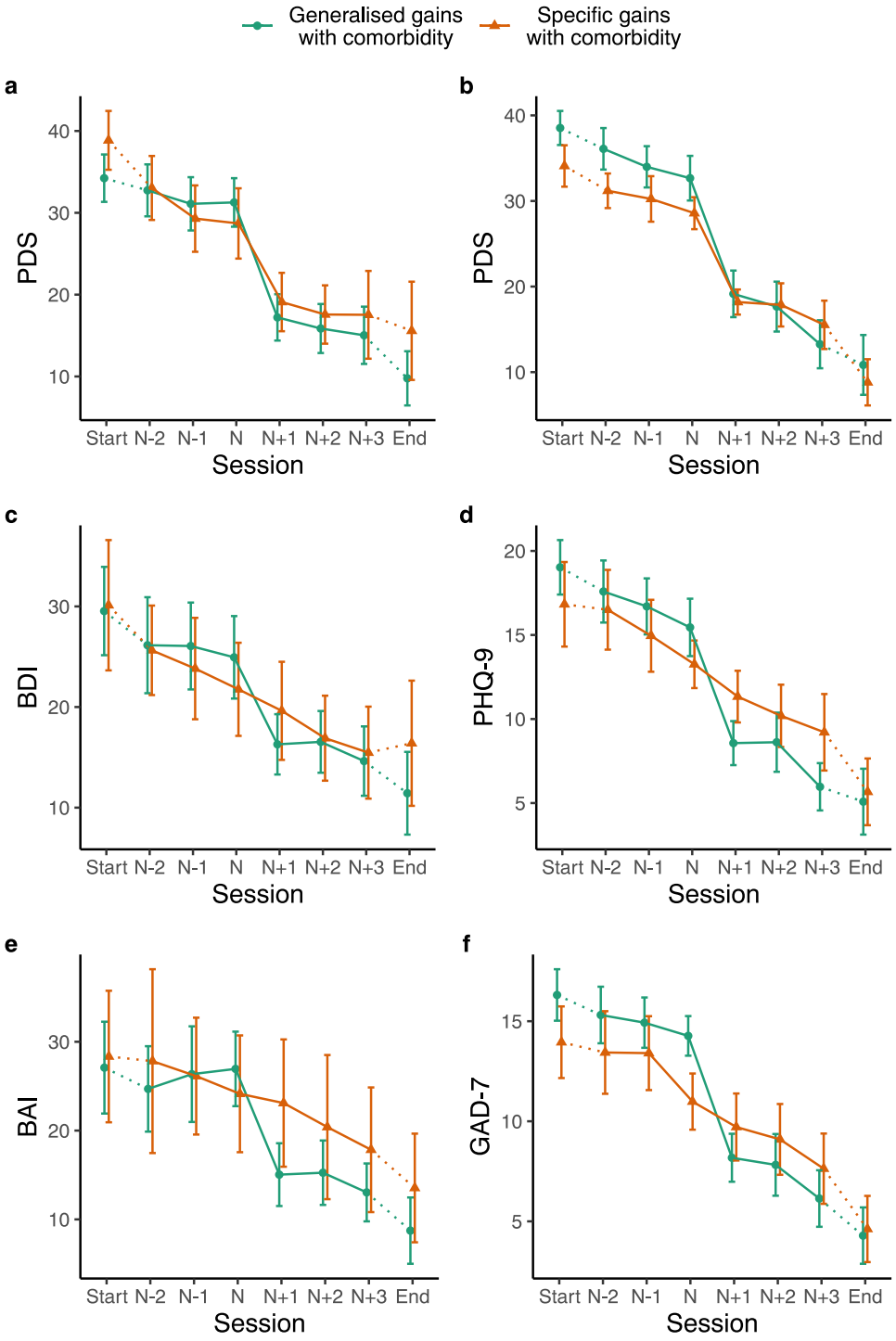


Figure 4.13: Average change in PTSD, depression, and generalised anxiety severity for groups of patients with generalised (green) or specific (orange) with clinical comorbidity around the sudden gain for Sample 1 (a, c, e) and Sample 2 (b, d, f). Error bars represent 95% confidence intervals.

4.2.4 Discussion

This study explored changes in individual symptoms of PTSD, depression, and generalised anxiety during sudden gains in PTSD symptoms during trauma-focused cognitive therapy for PTSD. In line with our first hypothesis, patients who experienced a sudden gain in PTSD symptoms showed large concurrent improvements in depression and generalised anxiety symptoms that were larger than for matched sessions of matched controls without a sudden gain. Pooled estimates across both samples suggest that patients with sudden gains reported larger improvements in generalised anxiety symptoms immediately prior to the sudden gain compared to matched controls. This parallels findings of improvements in cognitive processes preceding sudden gains reported in Chapter 4.1 and may indicate that reductions in negative cognitions and anxiety may facilitate the effects of interventions leading to sudden improvements in PTSD symptoms, such as trauma memory updating or behavioural experiments.

When investigating patterns of change for individual symptoms during the sudden gains, we found that most patients experienced broad improvements across all DSM-IV PTSD symptoms. Although most patients experienced unique patterns of individual PTSD symptom improvements during the sudden gain, the majority of gains were characterised by improvements in the majority of the 17 PTSD symptoms across the three DSM-IV symptom categories (see Figure C.3). These results suggest that sudden gains were generally experienced across the whole range of PTSD symptoms. Our results also demonstrate that patients differ as to whether sudden gains in PTSD symptoms are accompanied by simultaneous changes in depression or generalised anxiety symptoms. Some of these differences (e.g., the number of items that improve during the sudden gain) may be associated to the symptom severity immediately prior to the sudden gain.

In line with findings from psychometric (Armour et al., 2016) and network (Birkeland et al., 2020) studies we also found that changes in trauma amnesia (PDS item 8) were not experienced by many patients during the gain, or from

the beginning to the end of therapy. Mean levels at baseline further suggest that trauma amnesia was not a prominent symptom for many patients in our samples. This suggests that not all items are equally good indicators for assessing PTSD symptom severity and consequently may also not be informative for identifying sudden improvements in symptom severity. The amnesia item has also been criticised on theoretical grounds and has been subject to much debate (Berntsen & Rubin, 2014; Rubin et al., 2008). Further research should examine whether the identification of sudden symptom changes can be improved by taking into account more information than the sum score of one measure.

Although nightmares (PDS item 2) did not seem to improve in many patients during the sudden gain itself, by the end of treatment there was a difference in the mean of this item between patients with and without sudden gains in both samples. This suggests that patients with sudden gains improved more on this item compared to patients without sudden gains and that the better treatment outcomes in patients who experienced sudden gains may partly be driven by larger improvements in nightmares. Improvements in generalised anxiety symptoms during the sudden gain were mainly characterised by changes in feeling nervous, anxious, afraid, or restless. These feelings could be related to PTSD arousal or avoidance symptoms, a general feeling of anxiety, or appraisals of overgeneralised danger. The observation that sudden gainers tended to show greater improvements in generalised anxiety severity immediately prior to the gain compared to matched patients cannot be explained by a correlation between these measures and suggests that a reduction in generalised anxiety symptoms may facilitate large improvements in PTSD symptoms.

Sudden gains in PTSD symptoms were also accompanied by marked improvements in individual generalised anxiety and depression symptoms in comparison to the intervals preceding or following the gain. The vast majority of patients in Sample 1 ($n = 52$, 68.4%) and Sample 2 ($n = 69$, 79.3%) experienced generalised improvements in PTSD symptoms and depression or generalised anxiety symptoms, or specific improvements in PTSD symptoms in the context of non-clinical levels of generalised anxiety and depression before the sudden gain. A significant minority

experienced only reliable improvements in PTSD symptoms during the sudden gain whilst having clinically significant depressive or generalised anxiety beforehand. These patients showed gradual improvements in depression and anxiety around the sudden gain in PTSD symptoms

Interestingly a small minority of three patients reported reliable deteriorations in comorbid symptom severity together with a sudden gain in PTSD symptoms. This finding was unexpected and different to all other gains identified in both samples of this study. This pattern may be due to events unrelated to the trauma or therapy, but we could not find a consistent pattern that may help to explain this finding.

Our results suggest that while sudden gains in CT-PTSD treatments were experienced across the full range of PTSD symptoms, there was some heterogeneity. Of the patients with clinically significant depression or generalised anxiety, the majority experienced gains that generalised to the comorbid symptoms, but in a specific subset they remained limited to PTSD symptoms (*specific gains with clinical comorbidity*). Exploratory analyses suggested that patients who were in the clinical range of depression or generalised anxiety symptoms but experienced only specific PTSD gains, showed a gradual improvement in comorbid symptoms that overall seemed to be comparable in magnitude to patients who experienced generalised gains, particularly in depression symptoms (see Figures 4.13c and 4.13d).

These results need to be further explored but suggest that there may be different patterns of improvement in comorbid symptoms during PTSD gains. It is therefore possible that different psychological processes and therapeutic techniques are facilitating different subtypes of sudden gains and maintaining their positive clinical effects. For example, when comorbid depression or anxiety are addressed early in treatment, it is also possible that patients already improved in comorbid symptoms by the time a sudden gain in PTSD symptoms is experienced.

Limitations

Given the little knowledge about individual symptoms changes during sudden gains, this study was exploratory and findings need to be validated. Similarly, our method

for grouping patterns of sudden gains should be explored further and tested regarding its utility in improving our understanding of sudden gains. All measures used in this study are self-reported symptom measures. Sudden changes in positive processes like the therapeutic alliance may also contribute to facilitating or maintaining sudden symptom improvements and should also be explored (cf. Zilcha-Mano, Eubanks, et al., 2019). Although we only interpreted findings that replicated across both of our samples, further research is needed to validate these conclusions. In interpreting the findings of this study, we need to take into account that this chapter considered symptom changes in intervals of one week; more frequent measures may be needed to further investigate the results of this study. For example in patients who experienced generalised gains, we currently cannot be sure of the temporal sequence of changes in PTSD, depression, and generalised anxiety symptoms.

Conclusions

This study explored individual symptom changes during sudden gains in PTSD symptoms and highlighted that patients may experience different patterns of symptom changes. Further research should examine if these different patterns can be replicated and whether potential differences are clinically relevant for a better understanding and conceptualisation of sudden gains.

5 General discussion

5.1 Processes of change during CT-PTSD

5.1.1 Summary of key results

This thesis aimed to investigate how key processes hypothesised to maintain PTSD by Ehlers and Clark's (2000) cognitive model for PTSD (negative appraisals, trauma memory qualities and unhelpful cognitive and behavioural coping strategies) are related to clinical improvement during CT-PTSD in routine clinical care. CT-PTSD aims to reduce the patient's sense of current threat by targeting these three factors. Change in negative appraisals is promoted by guided discovery and behavioural experiments throughout treatment. This work is closely linked to the updating memories procedure, which aims to elaborate and update the worst memories of the trauma with information that gives them a less threatening meaning. Trigger discrimination training aims to reduce reexperiencing symptoms by detecting idiosyncratic triggers and learning to discriminate between 'Then' (cues during traumatic event) and 'Now' (cues in a new and safe context). A virtual or, if possible, in vivo site visit of the place where the trauma happened completes the memory updating, trigger discrimination, and cognitive restructuring work. Dropping unhelpful behaviours and cognitive processes is promoted by discussing

their advantages and disadvantages as well as conducting behavioural experiments.

Processes of change during CT-PTSD were examined by analysing (1) longitudinal associations between changes in therapy processes and changes in PTSD symptom severity and (2) sudden symptom improvements.

Chapter 3 tested whether session-by-session changes in the theory-derived cognitive processes explain subsequent changes in PTSD symptoms during CT-PTSD. We found that changes in PTSD symptoms were preceded by changes in negative appraisals, flashback quality of unwanted memories, unhelpful responses to intrusions such as rumination and safety seeking behaviours throughout treatment, but not vice versa. Survival analyses showed that changes in these factors were also predictive of recovery from PTSD. For changes in trauma memory disorganisation we found a bidirectional association suggesting that changes in PTSD symptoms are both driving and following changes in memory disorganisation.

Chapter 4.1 investigated how changes in negative appraisals and flashback quality of unwanted memories are associated with sudden gains in PTSD symptom severity. The results showed that patients with sudden gains reported better treatment outcomes in PTSD symptom severity, depression, and anxiety at the end of therapy and follow-up than those without sudden gains. During sudden gains, those with sudden gains reported greater changes in both cognitive factors than matched patients without sudden gains. Meta-analyses of the two samples suggest that negative appraisals, and to a smaller degree flashback quality of unwanted memories, had already decreased in the session prior to sudden gains in PTSD symptoms compared with matched patients.

Chapter 4.2 extended these results by examining patterns of individual PTSD, depression, and anxiety symptom improvements in patients who experienced sudden gains in PTSD symptom severity. The results suggest that patients experienced different patterns of sudden symptom improvements, while showing similar overall treatment outcomes. Although most patients who experienced a sudden gain in PTSD symptoms also reported clinically significant improvements in comorbid depression or anxiety symptoms during the gain. A smaller subgroup of patients

showed a more gradual improvement in comorbid symptoms around the time of the sudden gain in PTSD symptoms.

5.1.2 Theoretical implications

In this section, I will discuss to what extent the findings of Chapters 3 and 4.1 are in line with theoretical predictions from Ehlers and Clark's (2000) cognitive model of PTSD (see Figure 1.1), other psychological models of PTSD and previous experimental and prospective studies of trauma survivors. Previous studies have consistently found that negative appraisals, trauma memory qualities and unhelpful cognitive and behavioural coping strategies predict PTSD over and above initial symptom severity (e.g., Ehlers et al., 1998; Ehring et al., 2008; Kleim et al., 2007) and thus contribute to the maintenance of PTSD symptoms. If these factors maintain PTSD, then reducing or reversing them in therapy should lead to improvements in symptoms. A way to test this hypothesis is to investigate whether change in these factors precedes symptom change in therapy (Kazdin, 2007, 2009). Few studies to date have tested how changes in these factors are related to clinical improvement during treatment, and the present findings add to this literature.

The role of *trauma-related negative appraisals* in maintaining PTSD plays a central role in cognitive models of PTSD (e.g., Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1992) and has received most attention in psychological treatment research. In line with these models of PTSD, the findings presented in this thesis provide further evidence that changes in negative appraisals preceded changes in PTSD symptoms (Chapter 3). Furthermore, there was some evidence that sudden gains in PTSD symptoms were preceded by improvements in negative appraisals (Chapter 4.1). This suggests that cognitive change drives subsequent symptom change in CT-PTSD. The results of Chapter 3 replicated an earlier study of CT-PTSD (Kleim et al., 2013) and are consistent with the majority of studies using other psychological treatments (for a review see Brown et al., 2018). These findings are also consistent with the large predictive power of appraisals in prospective studies of trauma survivors (e.g., Beierl et al., 2019; Dunmore et al.,

2001; Ehring et al., 2008; Freeman et al., 2013; Kleim et al., 2007) and experimental findings (e.g., Sachschal et al., 2019).

Ehlers and Clark's (2000) model as well as other cognitive models of PTSD (e.g., Brewin, 2014; Foa & Riggs, 1993; Resick & Schnicke, 1992), further suggested that specific memory characteristics are relevant in the development and maintenance of PTSD. Ehlers and Clark (2000) and Brewin (2014) particularly emphasise the 'nowness' and sense of 'reliving' *unwanted trauma memories* that are retrieved involuntarily when matching triggers are present. Michael et al. (2005) found that the nowness of intrusions predicts PTSD prospectively over and above what can be predicted from intrusive memories. This 'flashback quality' of intrusions is now recognised as a central symptom of PTSD in ICD-11 (World Health Organization, 2018). Extending earlier research showing that flashback characteristics of intrusive trauma memories improved during therapy (e.g., Hackmann et al., 2004; Speckens et al., 2006), Chapter 3 found that changes in these flashback characteristics precede subsequent changes in PTSD symptoms throughout therapy. There was also a trend for a greater decrease in trauma flashback memories in the treatment session prior to sudden gains in PTSD symptoms (Chapter 4.1). These results extend the evidence from prospective longitudinal studies of assault survivors (e.g., Michael et al., 2005) that flashback qualities predict chronic PTSD symptoms.

Another feature of trauma memories highlighted by models of PTSD is the *disorganisation of voluntary recall* (e.g., Brewin, 2014; Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1992). Prospective longitudinal studies of accident survivors (e.g., Ehring et al., 2008; Harvey, 2000) and assault survivors (e.g., Halligan et al., 2003) as well as experimental analogue studies of student volunteers (e.g., Halligan et al., 2002) support the role of trauma memory disorganisation (for a review see Brewin, 2014) or memory disjointedness (Kleim et al., 2008; Sachschal et al., 2019). However, some authors have reported a series of negative findings (e.g., Rubin et al., 2016) and methodological differences may play a role (see Ehlers et al., 2012). Overall, the evidence for memory disjointedness of the worst moments of the trauma is more consistent than for a global disorganisation (e.g., Evans et al., 2007;

Jelinek et al., 2010). Some (e.g., Foa et al., 1995), but not all studies (e.g., Bedard-Gilligan et al., 2017) have found that trauma memories become more organised with trauma-focused treatment. Much of this evidence comes from patients' narratives of the trauma, and it is not always clear whether indicators of disorganisation, such as gaps or inconsistencies in the account, are due to disorganised memories or avoidance of distressing details. Furthermore, the studies focused on pre to post changes and did not assess session-by-session changes and longitudinal associations in changes between trauma memory disorganisation and PTSD symptoms during treatment. Chapter 3 therefore also examined how changes in trauma memory disorganisation are related to clinical improvement during CT-PTSD. The results suggested that changes in trauma memory disorganisation drove improvements in PTSD symptoms in the first sessions of therapy that focus on revisiting the trauma memory in detail. Contrary to our hypothesis, we also found evidence that changes in PTSD symptoms drove subsequent changes in memory disorganisation throughout therapy. This would suggest that improvements in some aspects of memory disorganisation are driven by previous improvements in PTSD symptoms, especially during later stages of therapy. Our results provide initial evidence for a bidirectional effect and suggest that this effect may vary during different phases of therapy. Memory disorganisation appears to drive symptom change early in therapy when it is directly targeted using techniques that facilitate memory elaboration such as imaginal reliving and writing a moment-by-moment narrative. Some of the techniques that have a more prominent role in later sessions (e.g., behavioural experiments and site visits) directly target some key symptoms of PTSD (such as avoidance and hypervigilance) but the way in which they do that may help patients to further access information that is useful for elaborating the trauma memory and making it more coherent.

In line with Ehlers and Clark's (2000) model and prospective studies that provided evidence for the importance of *unhelpful responses to intrusive memories* (i.e., thought suppression, rumination, and intentional numbing) in the development of PTSD in trauma survivors (e.g., Beierl et al., 2019; Ehlers et al., 1998; Kleim et al., 2012) the results presented in Chapter 3 provide initial evidence that changes

in unhelpful responses to intrusions are associated with subsequent changes in PTSD symptoms during CT-PTSD. Similarly, our results suggest that dropping unhelpful safety behaviours drives subsequent changes in PTSD symptoms, in accordance with longitudinal prospective studies (e.g., Beierl et al., 2019; Dunmore et al., 2001; Ehring et al., 2008) and extends previous evidence from a PTSD treatment study (Goodson & Haeffel, 2018).

In summary, this thesis presents further evidence supporting the maintenance factors of PTSD specified in Ehlers and Clark's (2000) model, while also showing that targeting these maintaining factors during treatment is related to subsequent clinical improvement as predicted by the underpinning theory and treatment protocol (see Figure 1.2). Importantly, the results in Chapter 3 suggest that, with the exception of trauma memory disorganisation, changes in therapy processes predicted subsequent changes in PTSD symptoms, but not vice versa.

Overall, the results support Ehlers and Clark's (2000) model, but also other theories that propose a role of appraisals, memory features and unhelpful coping in PTSD (e.g., Brewin, 2014; Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1992). In line with a general discussion of the role of memory disorganisation in PTSD in the field (e.g., Brewin, 2016; Rubin et al., 2016), the only concept that did not show a unidirectional effect on subsequent symptom change was a measure of memory disorganisation. Further work identifying the best way to assess this concept is needed. This may require further refinement. For example, Ehlers et al. (2004) suggested that for explaining reexperiencing, the disjointedness of the memory for the worst moments is most relevant. There is some evidence for this hypothesis (Evans et al., 2007; Jelinek et al., 2010; Sachschal et al., 2019).

5.1.3 Clinical implications

The results presented in this thesis have several clinical implications. While the results support the hypothesis that the therapeutic procedures in CT-PTSD have the desired effect of changing symptoms via their effects on appraisals, memory characteristics and coping strategies, these effects may not be unique to CT-PTSD

and other treatments of PTSD may work through the same processes. For example, Brown et al's (2018) meta-analysis suggested that changes in negative appraisals drive change in PTSD symptoms in several trauma-focused treatments. Such results may pave the way to further improvements in therapy procedures that particularly focus on maximizing change in appraisals, memory features and unhelpful coping. They may also help therapists tailor their treatment to the degree to which each of these factors is present in an individual patient. For example, in patients who are preoccupied with the unfairness of the trauma and ruminate excessively about it, but have relatively few flashbacks, focusing on rumination in treatment and measuring progress with the Response to Intrusion Questionnaire used in this study may lead to the fastest progress in treatment. For patients whose life is dominated by flashback memories, the updating memories and trigger discrimination procedures may be the best way to start therapy as they are likely to lead to fast changes in the nowness of memories and reductions in involuntary reexperiencing.

If the processes identified in this study replicate in future sudden gains studies, the results could indicate the importance of maximising cognitive change to promote symptom change in PTSD. This could be achieved by focusing early in therapy on the individual meanings of the trauma that lead to a sense of current threat. For example, the updating-memories procedure used in the cohort studies for this purpose was associated with sudden gains in therapy as early as Session 2 (see Chapter 4.1, Figure 4.1).

The findings of this thesis suggest that monitoring changes in therapy process measures during treatment in routine clinical care may be useful to inform treatment procedures aiming to maximise the change in these processes and subsequently PTSD symptoms.

5.1.4 Looking beyond PTSD symptoms

This thesis highlighted that a substantial subgroup of patients with PTSD showed concurrent large improvements in PTSD symptoms, negative appraisals, and

flashback characteristics of intrusive trauma memories from one treatment session to the next (Chapter 4.1).

Many people with PTSD have comorbid disorders, especially comorbid depression and anxiety are very common (Kessler, 1995; Kessler et al., 2005). The therapeutic procedures of CT-PTSD can be expected to change depression and general anxiety for several reasons. Restructuring excessive negative appraisals will include appraisals that are also involved in depression (negative appraisals of the self, e.g., 'I am worthless') and anxiety (e.g., 'I cannot trust anyone'). Reclaiming your life assignments have some similarities with behavioural activation, which is an effective treatment for depression. Reduction of avoidance and safety behaviours overlaps with treatments for anxiety disorders. These commonalities would suggest that concurrent changes in PTSD, depression, and anxiety symptoms are likely.

Chapter 4.2 investigated whether sudden improvement in PTSD symptoms generalises to concurrent improvements in comorbid depression or generalised anxiety symptoms. This was the case for the majority of patients. The findings also suggested that reductions in anxiety preceded sudden gains in PTSD symptoms. It is possible that these changes in anxiety facilitated their engagement and ability to benefit from the therapeutic interventions, e.g., engagement with and processing of trauma memories or behavioural experiments. Explorative analyses further suggested that patients who did not experience large concurrent improvements in comorbid symptoms together with the sudden gain in PTSD symptoms improved in a more gradual way. There might be a difference between patients whose anxiety and depression are a consequence of the PTSD symptoms, compared to patients who were experiencing anxiety or depression prior to developing PTSD. This distinction could be tested in future research and might be important to consider when analysing changes in secondary outcome measures around sudden gains in PTSD symptoms.

Overall, these results suggest that when patients make large improvements in PTSD symptoms, they also tend to show large improvements in comorbid symptoms, suggesting that CT-PTSD procedures address comorbid depression and anxiety symptoms. In line with these results, randomised controlled trials

(Ehlers et al., 2005; Ehlers et al., 2003; Ehlers et al., 2014) show large pre-post changes in measures of anxiety and depression.

5.1.5 Limitations and future research

The studies reported in this thesis have a number of limitations that should be considered in future research. Although the studies presented in this thesis build on previous prospective and experimental research and the statistical methods address important aspects that are required to establish a causal relationship (e.g., matched control group in Chapters 4.1 and 4.2 and testing for temporal precedence in Chapters 3, 4.1, and 4.2), the data used in this thesis was observational and causality can therefore not be inferred from these results. For example, our data did not include a randomised control group or an experimental manipulation of the hypothesised therapeutic processes (Cuijpers et al., 2019; Kazdin, 2007). Furthermore, it is possible that changes in unmeasured variables account for changes in both the therapy process and PTSD symptom severity. However, taking into account the strong theoretical background and previous empirical evidence, the results provide evidence in favour of a causal interpretation of the therapy processes.

While this thesis provides evidence that processes hypothesised by the Ehlers and Clark's (2000) are driving clinical improvement, it needs to be further explored whether changes in these processes are related to the therapeutic techniques designed to target them. As the content of treatment sessions depends on the individual case formulation, the heterogeneity of the session content as well as the data available did not allow us to address this question in this thesis. Different forms of treatments with a session-to-session protocol and possibilities to extract detailed information about therapeutic technique would provide an opportunity to address this important question. Internet delivered treatments provide a promising opportunity as the content is delivered more consistently across patients in comparison to face-to-face therapies. Furthermore, internet programmes could be designed to collect data on therapeutic techniques or session content without requiring additional work from patients or therapists.

All therapy processes measures in this thesis were self-reports assessed in weekly time intervals. It would be helpful to monitor changes in therapy processes during shorter time intervals to investigate the temporal relationships between therapeutic intervention, therapy processes, and symptom change more closely. One possibility would be intensive daily treatment protocols with diaries of the processes of interest (Woodward et al., 2017). Different forms of data collection (e.g., text analyses of written trauma narratives to examine the memory disorganisation, see Bedard-Gilligan et al., 2017) could be also be explored in future studies.

Some negative cognitions are now part of the DSM-5 diagnosis the results of this thesis and other research suggests that changes in all items are not always simultaneous. Analyses of subcategories of symptoms or individual items may help to further improve our knowledge of processes of change in psychological therapies (e.g., Blanken et al., 2019; McNally, 2016). To reduce overlap between measures of DSM-5 symptoms and cognitive therapy processes, some measures may need to be adapted in future studies when investigating longitudinal associations to avoid an overlap between the constructs. However, as this thesis used a symptom scale that is based on DSM-IV, this problem does not apply to the present results. Therapy process measures may need to be further refined to ensure that they capture meaningful changes that can be used to evaluate processes of change during therapy.

Looking only at the medium to large effect sizes of sudden gains on treatment outcome (Shalom & Aderka, 2020), it is unclear whether this effect is driven by the majority of patients with sudden gains or only by a subgroup. The clinical importance of sudden gains for individual patients who experience sudden gains is less clear and only few studies have investigated this question. Some studies reported the percentage of patients who recovered with and without sudden gains (e.g., Lemmens et al., 2016; Tang & DeRubeis, 1999; Wucherpfennig, Rubel, Hofmann, et al., 2017; Wucherpfennig, Rubel, Hollon, et al., 2017). In those studies that have investigated the recovery rates, patients with sudden gains showed a higher recovery rate than patients without sudden gains, however these results are mostly broken down into only two categories (*recovered* vs. *nonrecovered*). A more detailed

investigation including a finer range of categories (e.g., *deteriorated*, *unchanged*, *improved*, *recovered*) suggested by Jacobson and Truax (1991), or other approaches to examine the clinical importance of sudden gains for individual patients is needed.

Future research on processes of change in psychological therapies should also address more directly why therapy does not work for everyone. As much as we need to understand how treatments work, it is also important to investigate the processes involved in drop-out or non-recovery. The choice of the methods used in this thesis are primarily addressing how therapy works. While it is likely that our results are also in parts relevant for understanding why therapy does not work, investigating drop-out or non-recovery would require different methods and different inclusion criteria of participants.

Different forms of psychological treatments have been developed for PTSD. While they all share some similarities, there are also differences in the underpinning theoretical frameworks (Schnyder et al., 2015). It will be important to bring together the findings of studies investigating processes of change in with different treatments approaches (e.g., dysfunctional thinking in CBT for depression: Cristea et al., 2015) and to extend reviews of the role of negative appraisals to include other therapy processes (e.g., Brown et al., 2018; Gómez de La Cuesta et al., 2019; Woud et al., 2017). More transparent reporting of the analyses might make it easier to compare the methods and results across studies.

5.2 The value of free and open source research software

The development of free and open source research software has the potential to increase the quality of current psychotherapy research (for reviews see Ince et al., 2012; Lowndes et al., 2017; Mislan et al., 2016). Not only does this make it possible to evaluate the methods that are underlying the statistical methods, but it also allows other researchers to extend the functionality for their own research. A range of free and open source software tools have enabled the development of the R packages

presented in this thesis (Chapters 2.2 and 2.3). Furthermore, all analyses reported in this thesis as well as the writing[1] were done using free and open source software tools.

Both packages and the accompanying interactive online applications (*shinygains*: https://milanwiedemann.shinyapps.io/shinygains; *shinychange*: https://milanwiedemann.shinyapps.io/shinychange) that were developed as part of this thesis are already being used by other researchers. Since the first stable version of the *suddengains* package was published on the Comprehensive R Archive Network (CRAN) package repository in May 2019 it has been downloaded 10184 times. The *lcsm* package was first published on CRAN in June 2020 and has been downloaded 4076 times[2].

The R package *suddengains* has already facilitated further independent research of large routine clinical care datasets (Ladwa et al., 2020) and clinical trials (Mechler et al., 2020; Warbrick, 2020). The development of the package has also led to a collaboration in which we developed a new method that aims to test whether sudden gains occur above and beyond chance in a given sample (Lorenzo-Luaces et al., 2020). It should also be noted that we were made aware of a coding error very early during the development of the *suddengains* package by independent researchers which made it possible for us to address the error before publication. In this case, openly sharing analytic code and developing research software has not only facilitated new collaborations and work to improve existing methods but also helped us to detect and correct coding errors early.

Although both R packages are released on CRAN and the tests that are implemented in the packages suggest that they are working correctly, it is possible that some errors are currently undetected. More specific tests need to be developed for both R packages in the future to ensure that they work as intended. Furthermore, while the functionality of both packages covers a wide range of possible specifications, some features are still missing. For example, in the *lcsm* package it is currently not possible to specify multiple indicators (e.g., multiple items of a symptom or therapy

[1]This thesis was written using R Markdown (Xie et al., 2018), the `oxforddown` template (Lyngs, 2019), and the R package *papaja* (Aust & Barth, 2020).

[2]The download count for both R packages was last updated on February 17, 2021.

process measure) at repeated time points (for a demonstration of this method see Kievit et al., 2018). In the current version it is only possible to specify a summary statistic (e.g., the sum or mean of multiple indicators) at each time point. Although the *suddengains* package covers most adaptations of the original criteria by Tang and DeRubeis (1999), some less commonly used adaptations (e.g., Kelly et al., 2007; Kelly et al., 2005) are currently not implemented. This means that not all published adaptations of the original sudden gain criteria can be replicated in the current version, but it is possible to add more features in upcoming releases of the package. The open development of these R packages on GitHub allows other researchers to report errors or suggest new features. All currently known issues as well as planned features of both packages are available online and can be amended by other researchers (*lcsm*: https://github.com/milanwiedemann/lcsm/issues; *suddengains*: https://github.com/milanwiedemann/suddengains/issues).

Sharing analytic scripts or developing research software is only one step towards making psychotherapy research more transparent and reproducible. I argue that it is relatively easy to implement sharing analytic code compared to some other open research practices that would require more detailed planning in advance (e.g., pre-registration) or careful consideration how to protect sensitive information about individuals (e.g., sharing data). Reviewers, editors, and founding bodies play an important role in encouraging and incentivising this and other open research principles in the planning, conducting, and reporting of psychological treatment research (e.g., Nosek et al., 2015).

Overall, it is anticipated that both packages developed in this thesis will permit a faster and more transparent examination of processes of change in psychological therapies, and that these packages could provide valuable tools to explore how these methods could be improved further. It is hoped that the work presented in this thesis assists other psychotherapy researchers to incorporate some open research practices in their work, for example sharing analytic code or engaging in the development of research software by suggesting or adding new features to R packages.

5.3 Conclusion

Evidence-based treatments for PTSD can have a significant positive impact on many patients' lives (e.g., Cusack et al., 2016; Kline et al., 2018; Mavranezouli et al., 2020). A continuous evaluation of the theoretical models underpinning psychological treatments is important to ensure that psychological treatments are rooted in empirical science. The present thesis aimed to contribute to the emerging findings on the role of the factors maintaining PTSD specified by Ehlers and Clark (2000), which also partly overlap with those suggested by other authors (e.g., Brewin, 2014; Foa & Riggs, 1993; Resick & Schnicke, 1992). The results in this thesis largely supported that changes in these factors are driving symptom change during therapy. For negative appraisals, it has already been established that these results hold up for other trauma-focused therapies. It remains to be investigated whether this also applies to the other factors investigated in this thesis and whether they are also found for nontrauma-focused psychological therapies. To improve the quality of research and current psychological therapies, it will be important to address the clinical and methodological gaps in an open and collaborative way by bringing together expertise from different disciplines.

Appendices

A

Development of R packages

Table A.1: Complete list of parameters available in the *lcsm* package

Parameter	Symbol	Description
Construct X		
`gamma_lx1`	γ_{lx1}	Mean of latent true scores x (Intercept)
`sigma2_lx1`	σ^2_{lx1}	Variance of latent true scores x
`sigma2_ux`	σ^2_{ux}	Variance of observed scores x
`alpha_g2`	α_{g2}	Mean of change factor (g2)
`alpha_g3`	α_{g3}	Mean of change factor (g3)
`sigma2_g2`	σ^2_{g2}	Variance of change factor (g2)
`sigma2_g3`	σ^2_{g3}	Variance of change factor (g3)
`sigma_g2lx1`	σ_{g2lx1}	Covar: Change factor (g2) with initial true score x (lx1)
`sigma_g3lx1`	σ_{g3lx1}	Covar: Change factor (g3) with initial true score x (lx1)
`sigma_g2g3`	σ_{g2g3}	Covar: Change factors within construct x
`beta_x`	β_x	Proportional change factor of construct x
`phi_x`	ϕ_x	Autoregression of change scores x
Construct Y		
`gamma_ly1`	γ_{ly1}	Mean of latent true scores y (Intercept)
`sigma2_ly1`	σ^2_{ly1}	Variance of latent true scores y
`sigma2_uy`	σ^2_{uy}	Variance of observed scores y
`alpha_j2`	α_{j2}	Mean of change factor (j2)
`alpha_j3`	α_{j3}	Mean of change factor (j3)
`sigma2_j2`	σ^2_{j2}	Variance of change factor (j2)
`sigma2_j3`	σ^2_{j3}	Variance of change factor (j3)
`sigma_j2ly1`	σ_{j2ly1}	Covar: Change factor (j2) with initial true score y (ly1)
`sigma_j3ly1`	σ_{j3ly1}	Covar: Change factor (j3) with initial true score y (ly1)
`sigma_j2j3`	σ_{j2j3}	Covar: Change factors within construct y
`beta_y`	β_y	Proportional change factor of construct y
`phi_y`	ϕ_y	Autoregression of change scores y
Coupling X & Y		
`sigma_su`	σ_{su}	Covar: Residuals x with y
`sigma_ly1lx1`	σ_{ly1lx1}	Covar: Intercepts x with y
`sigma_g2ly1`	σ_{g2ly1}	Covar: Change factor x (g2) with initial true score y (ly1)

Table A.1 continued

Parameter	Symbol	Description
`sigma_g3ly1`	σ_{g3ly1}	Covar: Change factor x (g3) with initial true score y (ly1)
`sigma_j2lx1`	σ_{j2lx1}	Covar: Change factor y (j2) with initial true score x (lx1)
`sigma_j3lx1`	σ_{j3lx1}	Covar: Change factor y (j3) with initial true score x (lx1)
`sigma_j2g2`	σ_{j2g2}	Covar: Change factors y (j2) with x (g2)
`sigma_j2g3`	σ_{j2g3}	Covar: Change factors y (j2) with x (g3)
`sigma_j3g2`	σ_{j3g2}	Covar: Change factors y (j3) with x (g2)
`delta_con_xy`	$\delta_{\text{con}_{xy}}$	Change score x (t) determined by true score y (t)
`delta_con_yx`	$\delta_{\text{con}_{yx}}$	Change score y (t) determined by true score x (t)
`delta_lag_xy`	$\delta_{\text{lag}_{xy}}$	Change score x (t) determined by true score y (t-1)
`delta_lag_yx`	$\delta_{\text{lag}_{yx}}$	Change score y (t) determined by true score x (t-1)
`xi_con_xy`	$\xi_{\text{con}_{xy}}$	Change score x (t) determined by change score y (t)
`xi_con_yx`	$\xi_{\text{con}_{yx}}$	Change score y (t) determined by change score x (t)
`xi_lag_xy`	$\xi_{\text{lag}_{xy}}$	Change score x (t) determined by change score y (t-1)
`xi_lag_yx`	$\xi_{\text{lag}_{yx}}$	Change score y (t) determined by change score x (t-1)

Note. Covar = Covariance. More details for each parameter can be found in the package documentation using `help(sim_uni_lcsm)` or `help(sim_bi_lcsm)`.

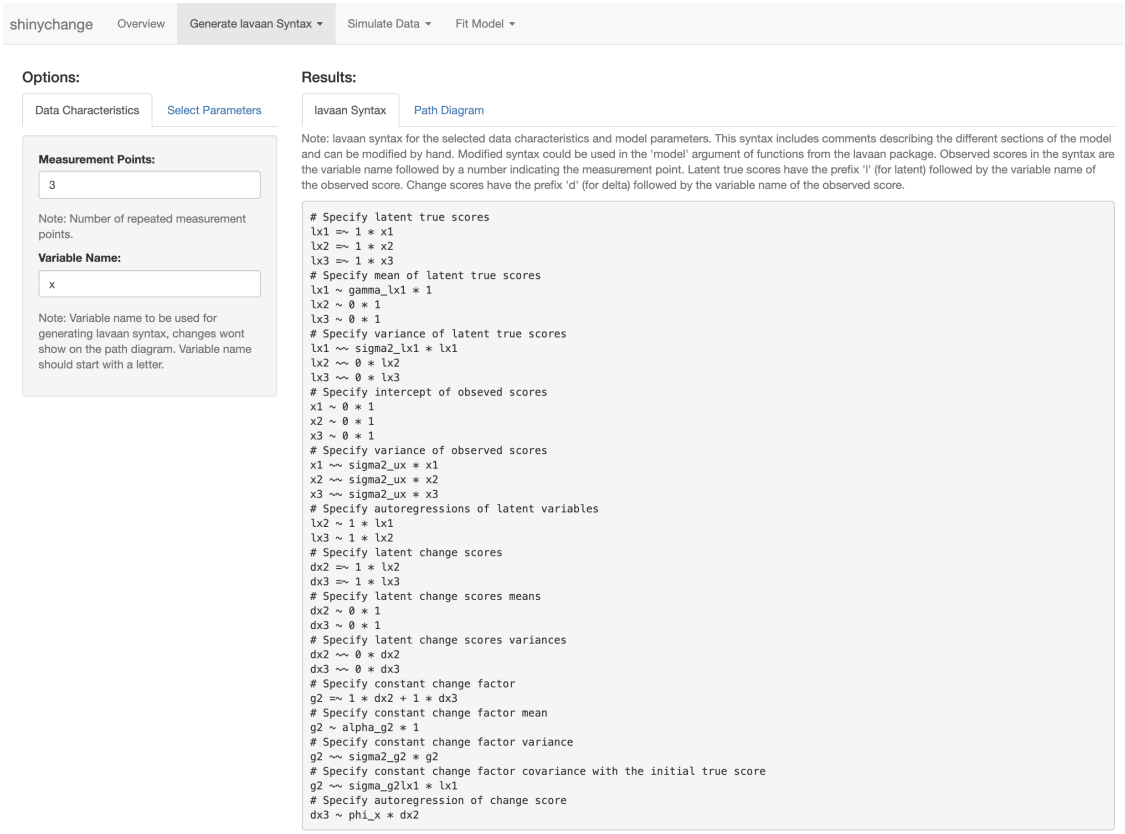


Figure A.1: Generate *lavaan* syntax of a univariate LCSM using *shinychange*.

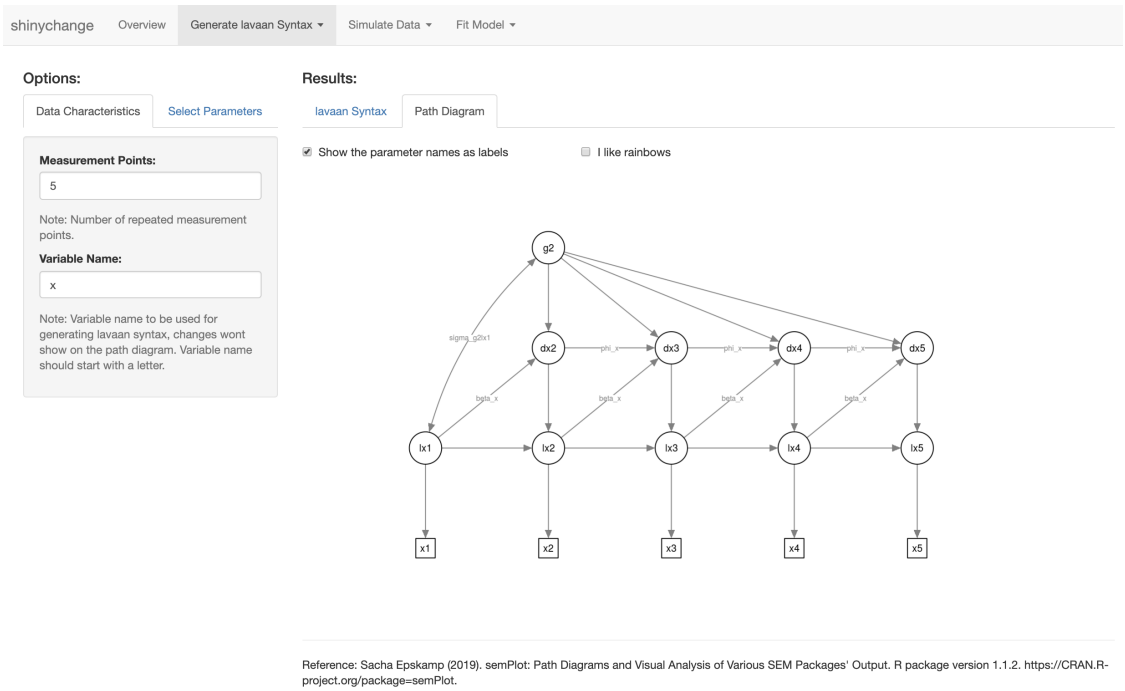


Figure A.2: Plot simplified path diagram of a univariate LCSM using *shinychange*.

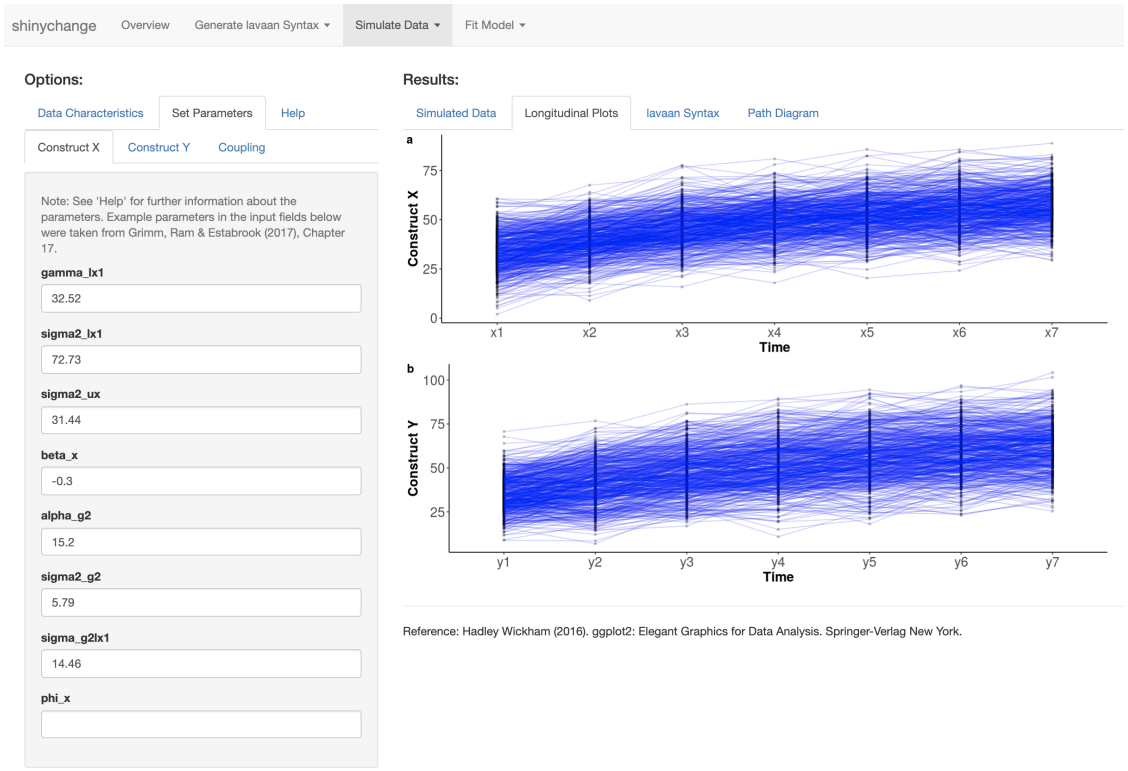


Figure A.3: Simulate and visualise data based on bivariate LCSM parameters using *shinychange*.

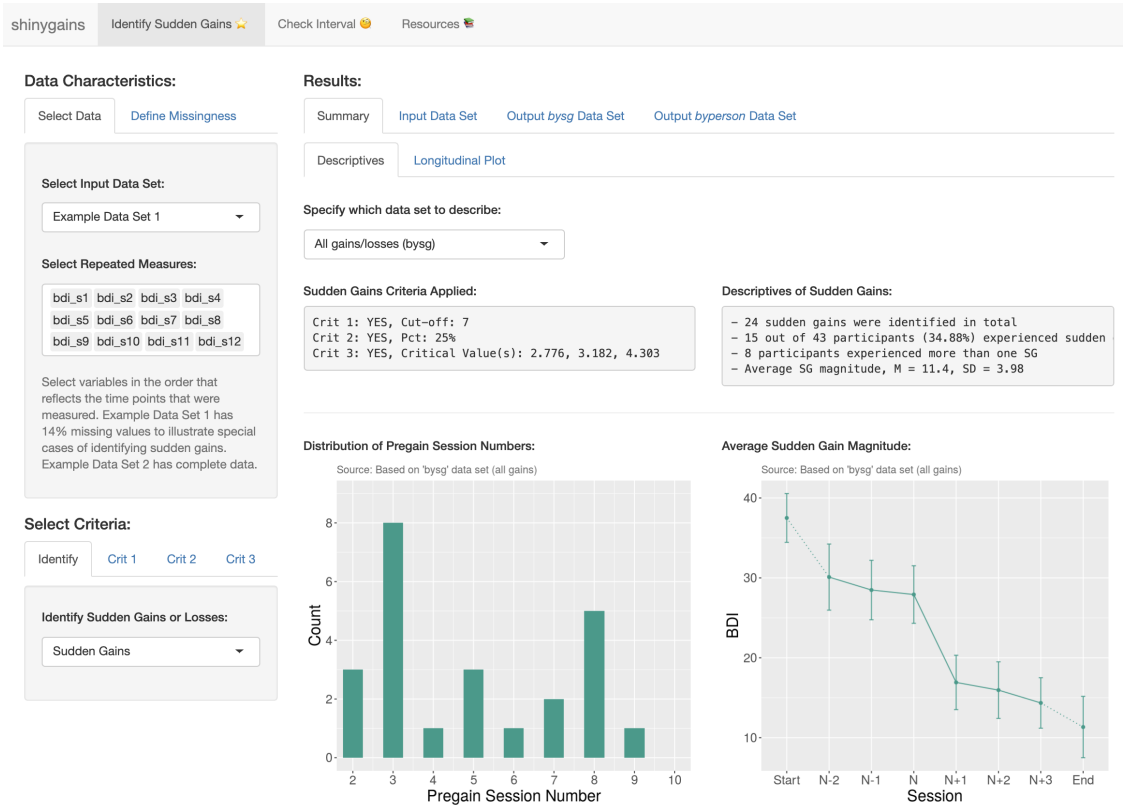


Figure A.4: Demonstration of identifying sudden gains using *shinygains*.

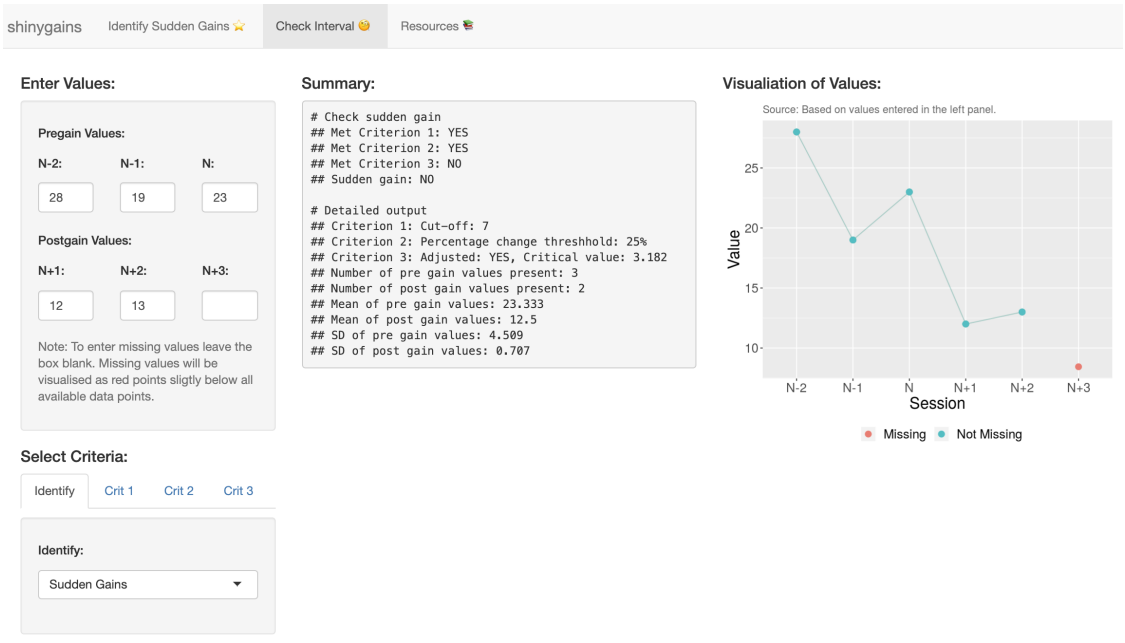


Figure A.5: Testing for a sudden gain using *shinygains*.

B

Longitudinal processes of change

Table B.1: Fit statistics for univariate LCSMs

Model	χ^2	Parameters	AIC	BIC	CFI	TLI	RMSEA
PTSD symptoms (PDS)							
No change	1,546	3	2,900	2,910	.326	.510	.336
Constant change	189	6	1,550	1,570	.941	.955	.102
Piecewise constant change	128	10	1,496	1,530	.967	.973	.079
Piecewise constant change + AR †	119	11	1,490	1,527	.970	.975	.076
Negative appraisals (PTCI-20)							
No change	1,168	3	4,629	4,639	.507	.643	.290
Constant change	183	6	3,650	3,670	.945	.958	.100
Piecewise Constant change †	124	10	3,599	3,632	.969	.975	.077
Piecewise constant change + AR	121	11	3,598	3,635	.970	.975	.077
Responses to intrusions (RIQ)							
No change	1,232	3	2,661	2,671	.382	.552	.296
Constant change	162	6	1,598	1,618	.946	.958	.090
Piecewise constant change †	117	10	1,560	1,594	.967	.973	.072
Piecewise constant change + AR	116	11	1,561	1,598	.967	.973	.073
Safety behaviours (SBQ)							
No change	787	3	2,560	2,570	.621	.725	.235
Constant change	171	6	1,949	1,969	.942	.955	.095
Piecewise constant change †	132	10	1,919	1,952	.959	.967	.082
Piecewise constant change + AR	132	11	1,920	1,957	.959	.966	.083
Disorganised memories (TMQ)							
No change	898	3	3,788	3,798	.574	.691	.252
Constant change	233	6	3,129	3,149	.911	.932	.118
Piecewise constant change †	146	10	3,049	3,083	.954	.962	.088
Piecewise constant change + AR	143	11	3,048	3,085	.955	.962	.088
Flashback memories (UMQ)							
No change	902	3	6,679	6,689	.455	.605	.258
Constant change	166	6	5,949	5,968	.931	.947	.094
Piecewise constant change †	131	10	5,922	5,955	.951	.960	.082
Piecewise constant change + AR	129	11	5,922	5,959	.951	.959	.083

Note. χ^2 = Chi square; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA = Root Mean Square Error of Approximation. AR = Autoregression of change scores. † indicates selected model.

Table B.2: Fit statistics for bivariate LCSMs

Model	χ^2	Parameters	AIC	BIC	CFI	TLI	RMSEA
Negative appraisals (PTCI-20) - PTSD symptoms (PDS)							
No Coupling	644	23	4,695	4,772	.915	.922	.100
ΔNegative appraisals$_{(t-1)} \rightarrow \Delta$PTSD symptoms$_{(t)}$	420	24	4,473	4,554	.958	.962	.070
ΔPTSD symptoms$_{(t-1)} \rightarrow \Delta$Negative appraisals$_{(t)}$	435	24	4,487	4,568	.956	.959	.072
Bidirectional Coupling†	410	25	4,465	4,549	.960	.963	.069
Responses to intrusions (RIQ) - PTSD symptoms (PDS)							
No Coupling	620	23	2,657	2,735	.914	.922	.096
ΔResponses to intrusions$_{(t-1)} \rightarrow \Delta$PTSD symptoms$_{(t)}$	374	24	2,413	2,494	.965	.968	.062
ΔPTSD symptoms$_{(t-1)} \rightarrow \Delta$Responses to intrusions$_{(t)}$	429	24	2,468	2,549	.954	.957	.071
Bidirectional Coupling†	373	25	2,414	2,498	.965	.968	.062
Safety behaviours (SBQ) - PTSD symptoms (PDS)							
No Coupling	520	23	3,193	3,270	.931	.936	.085
ΔSafety behaviours$_{(t-1)} \rightarrow \Delta$PTSD symptoms$^{\dagger}_{(t)}$	389	24	3,064	3,144	.959	.963	.065
ΔPTSD symptoms$_{(t-1)} \rightarrow \Delta$Safety behaviours$_{(t)}$	418	24	3,093	3,174	.953	.957	.070
Bidirectional Coupling	389	25	3,066	3,150	.959	.962	.065
Disorganised memories (TMQ) - PTSD symptoms (PDS)							
No Coupling	477	23	4,319	4,396	.939	.944	.078
ΔDisorganised memories$_{(t_{1,2}-1)} \rightarrow \Delta$PTSD symptoms$_{(t_{1,2})}$	407	25	4,252	4,336	.954	.958	.068
ΔPTSD symptoms$_{(t_{1,2}-1)} \rightarrow \Delta$Disorganised memories$_{(t_{1,2})}$	368	25	4,214	4,298	.963	.966	.061
Piecewise Bidirectional Coupling†	364	27	4,214	4,304	.964	.966	.061
Flashback memories (UMQ) - PTSD symptoms (PDS)							
No Coupling	595	24	6,897	6,976	.911	.918	.096
ΔFlashback memories$_{(t_{1,2}-1)} \rightarrow \Delta$PTSD symptoms$_{(t_{1,2})}$	384	26	6,689	6,776	.959	.962	.066
ΔPTSD symptoms$_{(t_{1,2}-1)} \rightarrow \Delta$Flashback memories$_{(t_{1,2})}$	365	26	6,671	6,757	.963	.966	.062
Piecewise bidirectional coupling†	356	27	6,664	6,754	.965	.967	.061

Note. χ^2 = Chi Square; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA = Root Mean Square Error of Approximation. † indicates selected model.

C

Sudden symptom improvements

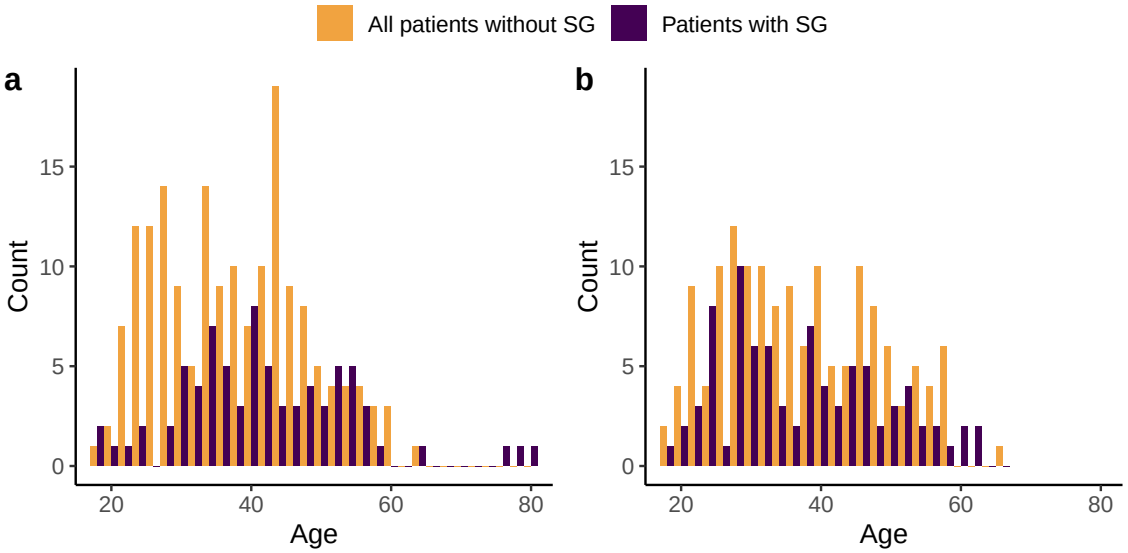


Figure C.1: Age distribution of patients with and all patients without sudden gains in Sample 1 (a) and Sample 2 (b).

Table C.1: Correlations between symptoms and cognitive processes in Sample 1

	PDS	PTCI-22	UMQ-4	BDI	BAI
PDS	-				
PTCI-22	.68	-			
UMQ-4	.46	.43	-		
BDI	.66	.77	.47	-	
BAI	.60	.57	.40	.63	-

Note. All Pearson correlation coefficients were statistically significant at $p < .001$, $n = 248$.

Table C.2: Correlations between symptoms and cognitive processes in Sample 2

	PDS	PTCI-20	UMQ-5	PHQ-9	GAD-7
PDS	-				
PTCI-20	.66	-			
UMQ-5	.62	.46	-		
PHQ-9	.75	.58	.52	-	
GAD-7	.72	.58	.49	.82	-

Note. All Pearson correlation coefficients were statistically significant at $p < .001$, $n = 234$.

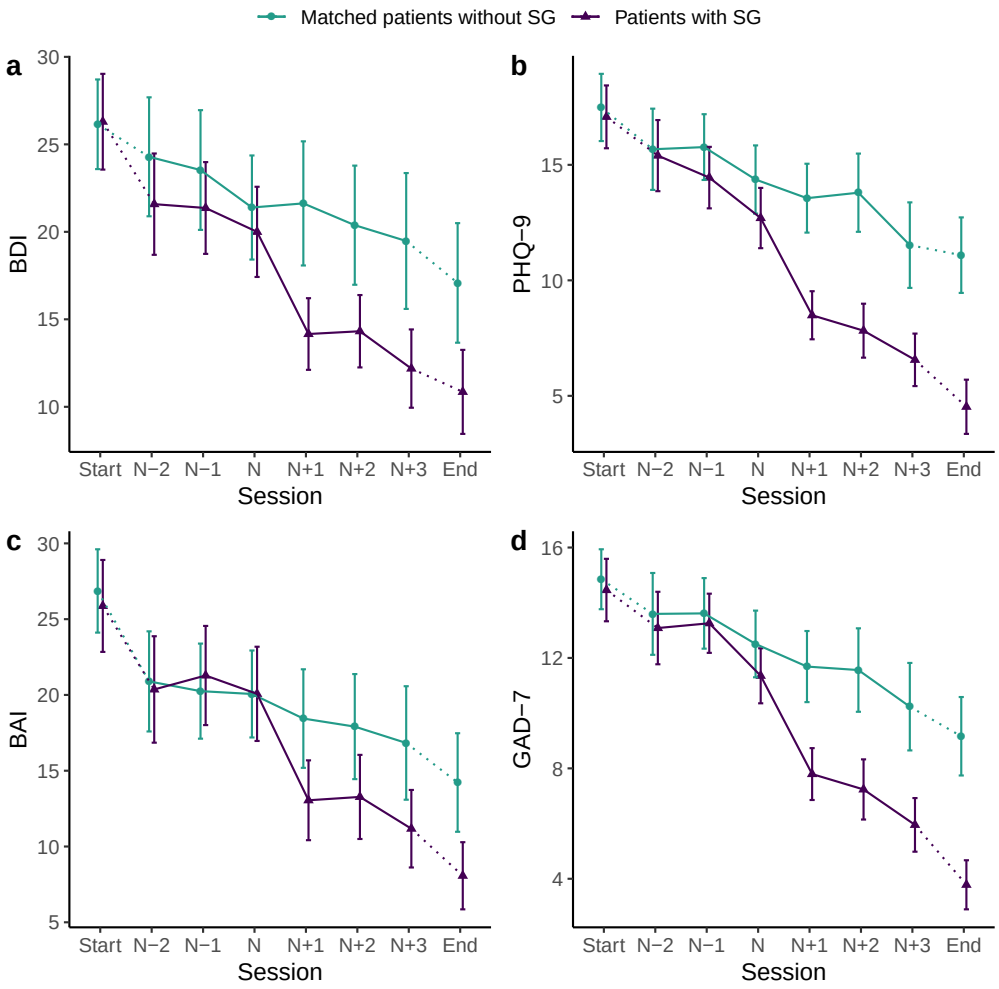


Figure C.2: Average change in depression and anxiety severity around the PTSD sudden gain session versus matched session for controls in Sample 1 (a, c) Sample 2 (b, d). Error bars represent 95% confidence intervals.

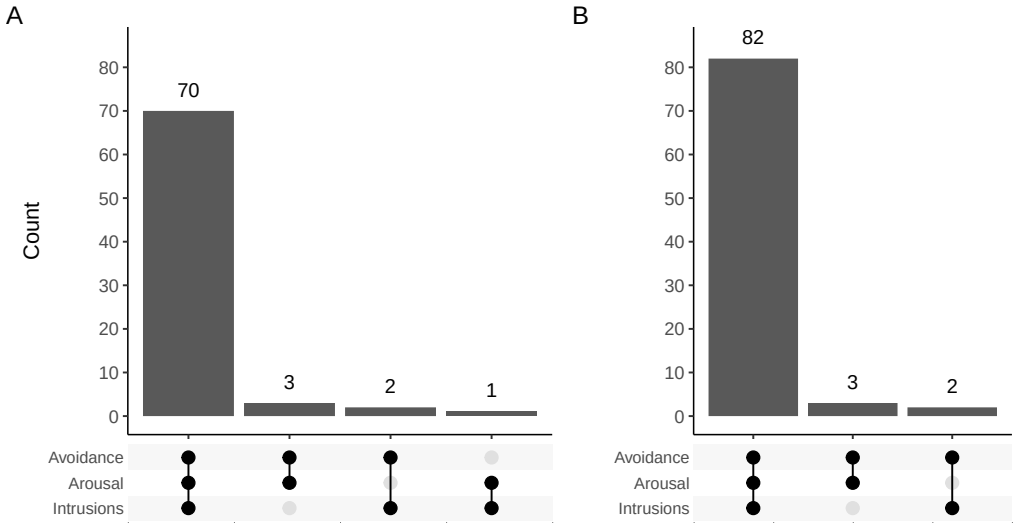


Figure C.3: Number of sudden gainers with improvements on PDS subscales for (A) Sample 1, $n = 76$ and (B) Sample 2, $n = 87$.

Table C.3: Item descriptions of the Posttraumatic Stress Disorder Scale (PDS)

Item	Description	Subscale
1	Upsetting thoughts	Intrusions
2	Nightmares	Intrusions
3	Reliving the traumatic event	Intrusions
4	Emotionally upset	Intrusions
5	Physical reactions	Arousal
6	Avoidance of thoughts	Avoidance
7	Avoidance of reminders	Avoidance
8	Difficulty remembering	Avoidance
9	Loss of interest	Avoidance
10	Distant	Avoidance
11	Emotionally numb	Avoidance
12	Future hopes or plans will not come true	Avoidance
13	Trouble falling or staying asleep	Arousal
14	Feeling irritable	Arousal
15	Trouble concentrating	Arousal
16	Overly alert	Arousal
17	Jumpy or easily startled	Arousal

Note. Response categories range from 0 (*Not at all*) to 3 (*5 or more times a week/almost always*).

Table C.4: Item descriptions of the Beck Depression Inventory (BDI)

Item	Description
1	Sadness
2	Pessimism
3	Sense of failure
4	Loss of pleasure
5	Guilty feelings
6	Punishment feelings
7	Self-dislike
8	Self-accusation
9	Suicidal thoughts
10	Crying
11	Irritability
12	Social withdrawal
13	Indecisiveness
14	Change in body image
15	Difficulty working
16	Insomnia
17	Tiredness of fatigue
18	Loss of appetite
19	Loss of weight
20	Somatic preoccupation
21	Loss of interest in sex

Note. Response categories range from 0 to 3 with different wordings for each item.

Table C.5: Item descriptions of the Patient Health Questionnaire 9-item scale (PHQ-9)

Item	Description
1	Little interest or pleasure
2	Feeling down, depressed, or hopeless
3	Sleep problems
4	Tired or little energy
5	Poor appetite or overeating
6	Feeling bad about yourself
7	Trouble concentrating
8	Moving or speaking so slowly or being so fidgety or restless
9	Suicidality

Note. Response categories range from 0 (*Not at all*) to 3 (*Nearly every day*).

Table C.6: Item descriptions of the Beck Anxiety Inventory (BAI)

Item	Description
1	Numbness or tingling
2	Feeling hot
3	Wobbliness in legs
4	Unable to relax
5	Fear of worst happening
6	Dizzy or lightheaded
7	Heart pounding / racing
8	Unsteady
9	Terrified or afraid
10	Nervous
11	Feeling of choking
12	Hands trembling
13	Shaky / unsteady
14	Fear of losing control
15	Difficulty in breathing
16	Fear of dying
17	Scared
18	Indigestion
19	Faint / lightheaded
20	Face flushed
21	Hot / cold sweats

Note. Response categories range from 0 (*Not at all*) to 3 (*Severely – it bothered me a lot*).

Table C.7: Item descriptions of the Generalised Anxiety Disorder 7-item scale (GAD-7)

Item	Description
1	Feeling nervous, anxious
2	Not able to control worrying
3	Worrying about different things
4	Trouble relaxing
5	Being so restless
6	Easily annoyed or irritable
7	Feeling afraid

Note. Response categories range from 0 (*Not at all*) to 3 (*Nearly every day*).

D
Publications

Chapters 1.6.1 and 4.1 are based on published work:

Wiedemann, M., Stott, R., Nickless, A., Beierl, E. T., Wild, J., Warnock-Parkes, E., Grey, N., Clark, D. M., & Ehlers, A. (2020). Cognitive processes associated with sudden gains in cognitive therapy for posttraumatic stress disorder in routine care. *Journal of Consulting and Clinical Psychology*. https://doi.org/10.1037/ccp0000488

Chapter 2.3 is based on published work:

Wiedemann, M., Thew, G. R., Stott, R., & Ehlers, A. (2020). suddengains: An R package to identify sudden gains in longitudinal data. *PLOS ONE*. https://doi.org/10.1371/journal.pone.0230276

The work presented in Chapter 2.3 has led to a collaboration. The manuscript is currently under-review and available as a preprint:

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