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SHORT COMMUNICATION

Predictors of treatment response for cognitive behaviour therapy for prolonged grief disorder

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ABSTRACT

Background: Prolonged grief disorder (PGD) causes significant impairment in approximately 7% of bereaved people. Although cognitive behaviour therapy (CBT) has been shown to effectively treat PGD, there is a need to identify predictors of treatment non-response.
Methods: PGD patients (*N* = 80) were randomly allocated to receive 10 weekly two-hour group CBT sessions and (a) four individual sessions of exposure therapy or (b) CBT without exposure. PGD was assessed by self-report measures at baseline, post-treatment (*N* = 61), and six-months (*N* = 56) after treatment.
Results: Post-treatment assessments indicated that greater reduction in grief severity relative to pretreatment levels was associated with being in the CBT/Exposure condition, and lower baseline levels of self-blame and avoidance. At follow-up, greater grief symptom reduction was associated with being in the CBT/Exposure condition and lower levels of avoidance.
Conclusions: These patterns suggest that strategies that target excessive self-blame and avoidance during treatment may enhance response to grief-focused cognitive behaviour therapy.

Predictores de la respuesta al tratamiento con terapia cognitivo conductual para el trastorno de duelo prolongado

Antecedentes: El trastorno por duelo prolongado (PGD, por sus siglas en inglés) causa un deterioro significativo en aproximadamente el 7% de las personas en duelo. Aunque se ha demostrado que la terapia cognitivo conductual (TCC) es efectiva para tratar el PGD, existe una necesidad de identificar factores predictivos de la falta de respuesta al tratamiento.
Método: Los pacientes con PGD (*N* = 80) fueron asignados al azar para recibir 10 sesiones semanales de TCC grupales de 2 horas y (a) 4 sesiones individuales de terapia de exposición o (b) TCC sin exposición. El PGD se evaluó mediante medidas de auto-reporte en línea base, post-tratamiento (*N* = 61) y 6 meses después del tratamiento (*N* = 56).
Resultados: Las evaluaciones post-tratamiento indicaron que una mayor reducción en la gravedad del duelo en relación con los niveles pre-tratamiento se asoció con estar en la condición de TCC con exposición y con niveles basales más bajos de culpa a sí mismo y evitación. En el seguimiento, una mayor reducción de los síntomas de duelo se asoció con estar en la condición de TCC con exposición y con menores niveles de evitación.
Conclusiones: Estos patrones sugieren que estrategias dirigidas a la excesiva culpa a sí mismo y a la evitación durante el tratamiento pueden mejorar la respuesta a la terapia cognitivo conductual centrada en el duelo.

延长哀伤障碍的认知行为治疗的治疗反应预测因子

背景：延长爱上障碍（PGD）让大约7%的丧亲者承受严重痛苦。虽然已经证明认知行为疗法（CBT）可有效治疗PGD，但仍需要确定无治疗反应的预测因子。
方法：PGD患者（*N*= 80）随机分配接受10周2小时团体CBT治疗和（a）4个单独的暴露治疗或（b）CBT但无暴露治疗。在基线期、治疗后（*N*= 61）和6个月（*N*= 56）后通过自我报告评估PGD。
结果：治疗后评估表明，相对于治疗前水平哀伤程度的降低与CBT/暴露条件有关，也和基线期的自责和回避水平较低相关。在随访期，更严重的哀伤症状与CBT/暴露条件和较低的回避水平相关。
结论：这些模式表明，在治疗过程中针对过度自责和回避的方案，可能会增强以哀伤中心的CBT的治疗反应。

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关键词
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HIGHLIGHTS

- Many patients with prolonged grief disorder do not respond to cognitive behaviour therapy.
- In this trial poor treatment response was predicted by self-blame and avoidance tendencies.
- Strategies that address these factors may enhance treatment for prolonged grief disorder.

There is increasing recognition that prolonged grief disorder (PGD) represents a major public health issue, affecting approximately 7% of bereaved people (Kersting et al., 2011). Initial definitions of PGD described the disorder as persistent yearning for the deceased and range of associated psychological

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problems (Prigerson et al., 2009). These conceptualizations were subsequently developed in the preparation of ICD-11 to propose a new diagnosis describing persistent and problematic grief responses (Maercker et al., 2013). The ICD-11 has recently announced the final diagnostic description of PGD that is arguably broader than the initial descriptions and defines the disorder as persistent and pervasive longing for the deceased that is associated with emotional pain that can be reflected in an array of difficulties (e.g. guilt, anger, difficulty accepting the death, numbing, identity loss, difficulty engaging in activities). A major rationale for the introduction of this diagnosis was to identify people with PGD because it has been shown to be associated with many psychological, social, somatic and functional problems (Prigerson et al., 2009).

Most of the controlled trials of PGD have involved some form of exposure therapy that required emotional processing of the death. Exposure-based techniques usually involves reliving the time the person experienced the bereavement, which may involve hearing of the death, being with the deceased at the time of death or possibly associated distressing events (e.g. burial). The first controlled trial showed that CBT was significantly superior in reducing PGD symptoms relative to interpersonal therapy (Shear, Frank, Houck, & Reynolds, 2005). A subsequent trial found that providing exposure prior to cognitive restructuring was more effective than providing these two strategies in the reverse order (Boelen, de Keijser, van Den Hout, & van Den Bout, 2007). Reinforcing the role of exposure was a study that reported that CBT that involved exposure therapy of death memories led to greater treatment outcomes than CBT without exposure (Bryant et al., 2014, 2017). Further evidence of the efficacy of therapies involving exposure-based techniques has come from other controlled trials (Eisma et al., 2015; Rosner, Bartl, Pfoh, Kotoucova, & Hagl, 2015; Shear et al., 2016). The mechanisms underpinning exposure-based approaches are not well understood at this time, although it is possible that emotionally processing affectively laden memories of the loss can reduce avoidance of key memories and facilitate acceptance of the death and reframing of core cognitions that may be prolonging the severe grief response (Bryant et al., 2014).

Across these studies, 50–70% of patients responded positively to treatment (e.g. 51%, Shear et al., 2005; 50%, Boelen et al., 2007; 70%, Stroebe et al., 2014). This pattern points to the need for better understanding of the factors associated with symptom reduction. Delineation of factors associated with symptom change shed light on how treatment can be improved to enhance response. In one study, no evidence was found that treatment outcome was associated with

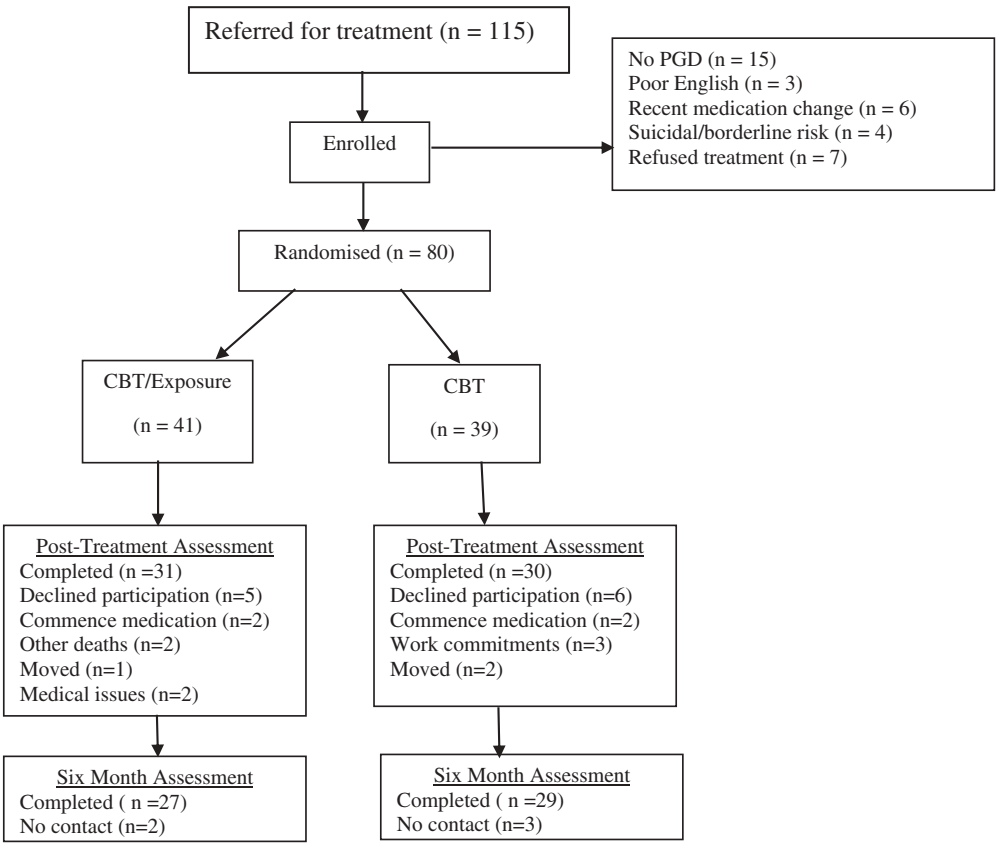

Figure 1. Participant flow in the study.

race, age, gender, time since loss or relationship to the deceased; although poorer response tended to be linked to violent death or child death despite these associations not reaching statistical significance (Shear et al., 2005). To explore this issue further, we investigated factors associated with symptom change in one previous trial (Bryant et al., 2014). This trial found that CBT that included exposure to memories of the death led to greater reductions in PGD symptoms than CBT without exposure; it should be noted that although this relative gain was maintained at two years follow-up, both CBT conditions displayed marked reductions in PGD symptoms at two years relative to pretreatment levels (Bryant et al., 2017). Based on evidence that PGD may be associated with relationship to the deceased and violent death type (Nakajima, Ito, Shirai, & Konishi, 2012), we entered these factors into the predictive model. There is also evidence that maladaptive appraisals about the loss is associated with worse grief reactions (Boelen, van Den Bout, & van Den Hout, 2003), providing a rationale for this factor as a potential predictor. Finally, there are theoretical (Shear et al., 2007) and empirical (Boelen, van Den Bout, & van Den Hout, 2006) indications that avoidance maintains prolonged grief and may be associated with symptom change during treatment.

1. Method

1.1. Participants

Trial participants were patients treated at the UNSW Traumatic Stress Clinic between 17 September 2007 and 7 June 2010. Patients had experienced bereavement at least 12 months earlier and satisfied criteria for PGD. Patients were excluded if they had a history of psychosis, current substance dependence, borderline personality disorder, severe suicidal risk, inability to converse in English, or aged less than 17 years of age or more than 70 years of age. Participants completed written informed consent approved by the UNSW Human Research Ethics Committee (trial registration: ACTRN12609000229279). Participant characteristics of the initial sample are presented in Bryant et al. (2014). The participant characteristics of the sample are reported in Table 1.

Table 1. Participant characteristics.

	CBT	CBT/Exposure (n = 41)	Test (n = 39)
Age, mean years	51.0 ± 14.4	54.8 ± 9	F_{78} = 1.3, p = .18
Years since death	4.00 ± 3.39	3.62 ± 3.10	F_{78} = .53, p = .60
Education, mean years	13.6 ± 2.6	13.3 ± 2.9	F_{78} = 0.5, p = .65
Gender			χ^2 = 1.13, p = .29
Male	10%	17%	
Female	90%	83%	
Employed	75%	73%	χ^2 = .04, p = .84
Education, mean years	13.71 ± 2.76	13.27 ± 2.93	F_{78} = .63, p = .53
Relationship to deceased			χ^2 = 4.23, p = .52
Partner	27%	33%	
Child	27%	35%	
Parent	34%	23%	
Other	12%	9%	
Death type			χ^2 = .76, p = .86
Sudden illness	23%	18%	
Chronic illness	55%	54%	
Accident	15%	15%	
Suicide	9%	13%	

1.2. Procedures

Participants were randomized by an individual independent of the study. Eighty patients were randomized into the study and were allocated to either CBT/Ex (n = 41) or CBT (n = 39); the type of deaths included death of parents (29%), partners (30%), children (31%) or others (19%). The causes of death included sudden illness (21%), chronic illness (54%), accident (14%) or suicide (11%). A total of 61 participants (76%) completed the post-treatment assessment and 56 patients (70%) completed the six-month follow-up assessment (see Figure 1). Post-treatment assessments were conducted by independent clinicians who were unaware of the treatment condition of participants. Blindness was maintained by ensuring that clinicians who conducted assessments did not have access to (a) participant notes or (b) condition allocation of participants.

1.2.1. Treatment conditions

Therapy comprised 10 x weekly two-hour group sessions as well as four x weekly one-hour individual sessions that were conducted by Masters-level clinical psychologists. Patients in each treatment condition participated in group sessions dedicated to that condition. Treatment manuals are available from RAB on request.

1.2.2. CBT/ Exposure

Session 1 comprised education about grief. Session 2 addressed the rationales for the treatment components. Sessions 3, 4 and 5 included cognitive restructuring that addressed themes related to the grief. Session 6 addressed strategies on managing rumination. This session also included a letter writing task in which participants expressed issues they wished to communicate to the deceased. Session 7 continued with cognitive challenging, letter writing to the deceased and also commenced facilitation of positive memories in which participants described memories of positive experiences with the deceased. Session 8 continued letter writing, facilitation of positive memories and initiated steps for new goals and activities. Session 9 focused on identification of future goals. This strategy was continued in

Session 10, which also developed relapse prevention strategies for high risk times. Following group Session 2, participants participated in four weekly one-hour individual therapy sessions which focused on imaginal exposure to memories of the death. Participants recounted their memories of the circumstances of the death for 40 minutes. Patients were instructed to conduct the exposure at least once between sessions for homework.

1.2.3. CBT

The group therapy was identical to the treatment provided in the CBT/Exposure condition. The treatment conditions differed in that in the four weekly one-hour individual sessions, participants in CBT were invited to discuss anything they wished in a nondirective manner. Audiotapes of 20% of sessions were rated for treatment fidelity and quality; good adherence to protocol and high treatment quality was achieved.

1.2.4. Measures

Complicated Grief Assessment (CGA; Zhang, El-Jawahri, & Prigerson, 2006) is a clinician administered semi-structured interview for assessing problematic grief reactions, which was used to diagnose PGD and determine eligibility for entry into the study.

Inventory of Complicated Grief (Prigerson et al., 1995) is a self-report measure for assessing problematic persistent grief symptoms. The ICG assesses for the presence of separation distress (Criterion A) and other symptoms including a difficulty accepting the death, numbness, bitterness, difficulty engaging in life, and a sense of purposelessness and meaninglessness. The ICG was used to provide a continuous measure of grief symptoms and was the primary treatment response measure.

Posttraumatic Cognitions Inventory (PTCI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999) is a 36-item self-report scale that yields three factors, including negative cognitions about self, negative cognitions about the world and self-blame. The PTCI possesses good internal consistency (alpha = .97) and discriminant ability to differentiate people with and without PTSD (sensitivity = .78, specificity = .93) (Foa et al., 1999).

Impact of Events-Revised (IES-R; Weiss & Marmar, 1997) is a self-report questionnaire that indexes an individual's subjective response to a traumatic event and consists of three subscales: intrusions, avoidance and hyperarousal. The current study utilized the Avoidance subscale, which comprises eight items that are answered on a 5-point Likert scale (0 = *Not at all*, 4 = *Extremely*). The internal consistency of the Avoidance subscale has been validated (coefficient alpha = .82) and it has good test-retest reliability (coefficient alpha = .79) for intervals less than one week (Sundin & Horowitz, 2002).

1.2.5. Data analysis

In contrast to the initial trial report that focused on linear mixed models to report outcomes, this study restricts analyses to those who completed assessments in order to determine predictive models. Symptom change was calculated by subtracting (a) post-treatment CGA scores from baseline scores and (b) follow-up CGA scores from baseline scores. To determine factors associated with symptom change, we calculated reduction of scores on the CGA at post-treatment relative to pretreatment levels. To factors associated with symptom change, we entered relationship to the deceased, type of death and treatment condition at Step 1 because of the demonstrated impact of these factors on development of PGD (Meert et al., 2011), PTCI subscale scores at Step 2 because of the role of cognitive appraisals on PGD (Boelen et al., 2003) and IES-Avoidance score at Step 3 (Boelen et al., 2006).

2. Results

2.1. Preliminary analyses

In terms of completion of the post-treatment assessment, 31 (75.6%) participants in the CBT/Exposure condition and 30 (76.9%) in the CBT condition completed the assessment following treatment. Planned comparisons of treatment completers and treatment drop-outs demonstrated that there were no differences between the two conditions on any pretreatment psychopathology, demographic or bereavement-related factors (see Table 1). In terms of meeting PGD diagnostic criteria at post-treatment, there was no difference between CBT/Exposure (5, 16.1%) and CBT (9, 30.0%) [χ^2 (N = 61) = 1.66, p = .20]. There was a greater reduction in PGD symptoms at post-treatment (relative to pretreatment levels) in CBT/Exposure (M = 20.4, SD = 12.9) than CBT (M = 7.4, SD = 15.3) [t(59) = 3.58, p = .001].

2.2. Factors associated with symptom change

In terms of factors associated with symptom change at post-treatment, the prediction model was significant, F (7, 48) = 3.81, p = .002). Greater reduction in grief severity was associated with being in the CBT/Exposure condition, lower PTCI Self-Blame scores and lower pre-treatment avoidance (see Table 2). Specifically, treatment condition (CBT/Exp) accounted for 18% of the variance, lesser self-blame accounted for 20% and lesser avoidance accounted for 9%. In terms of symptom change from pretreatment to follow-up, the prediction model was marginally significant, F(7, 43) = 2.08, p = .066). Greater reduction in grief was associated with being in the CBT/Exposure condition and lower pre-treatment avoidance.

Table 2. Summary of hierarchical regression models reduction in grief severity.

	B	SEB	*β*	*p*
Post-treatment				
Step 1:	−11.64	3.73	−3.71	.003
Treatment condition	1.61	2.09	.09	.445
Type of death	1.63	1.92	.10	.400
Relationship				
Step 2:	−.13	.11	−.20	.260
PTCI Self	−.11	.23	−.07	.631
PTCI World	.70	.34	.31	.042
PTCI Blame				
Step 3:	.96	.36	.34	.011
Avoidance				
Follow-up				
Step 1:	−9.28	3.84	−.34	.020
Treatment condition	−.26	2.24	−.02	907
Type of death	2.14	1.73	.16	.223
Relationship				
Step 2:	−.10	.11	.16	.429
PTCI Self	−.08	.24	.06	.733
PTCI World	.35	.33	.18	.300
PTCI Blame				
Step 3:	.71	.36	.29	.047
Avoidance				

Symptom change was calculated by subtracting (a) post-treatment CGA scores from baseline scores and (b) follow-up CGA scores from baseline scores. Post-treatment: Step 1 $R^2 = .16$, $\Delta R^2 = .16$. Step 2 $R^2 = .18$, $\Delta R^2 = .03$. Step 3 $R^2 = .26$, $\Delta R^2 = .07$. Follow-up: Step 1 $R^2 = .20$, $\Delta R^2 = .20$. Step 2 $R^2 = .26$, $\Delta R^2 = .06$. Step 3 $R^2 = .36$, $\Delta R^2 = .09$.

3. Discussion

The key findings to emerge from these analyses were that reduced symptom change at post-treatment was associated with greater self-blame and avoidance tendencies. This may suggest that the treatment strategies used in this study may not be ideally suited to people with marked self-blame or avoidance. Interestingly, prior cognitive behaviour studies of a variety of disorders have noted that more extreme catastrophic appraisals predict poor treatment response (Ehlers et al., 1998; Scholing & Emmelkamp, 1999). Cognitive models of PGD emphasize the role of maladaptive thoughts in the development and maintenance of the disorder (Maccallum & Bryant, 2013). Evidence regarding the role of self-blame in grief is somewhat mixed, with findings reporting that it is (Stroebe et al., 2014) and is not (Boelen & Spuij, 2008) associated with persistent grief reactions. One systematic review of 18 studies found that self-blame after bereavement was predictive of more severe psychopathology (Duncan & Cacciatore, 2015). It is possible that greater self-blame in PGD participants is associated with less symptom reduction because for many participants it may represent a core reason for the persistent grief reaction, and so excessive self-blame may limit response to treatment strategies. Although both CBT conditions in this programme included cognitive restructuring strategies that addressed self-blame, it is possible that these were not as successful in cases where self-blame was dominant. It is also possible that exposure does not impact self-blame to the extent that it does anxiety-related emotions (Gray et al., 2012); accordingly, elevated self-blame may not be associated with symptom reduction in treatments that involve exposure therapies. It is possible that strategies developed to reduce self-blame, including those targeted towards moral injury (e.g. self-forgiveness), may be appropriate for PGD (Gray et al., 2012). In this context, it is also interesting to note decreases in self-blame during psychotherapy for posttraumatic stress disorder predicts diminishment of posttraumatic stress symptoms (Schumm, Dickstein, Walter, Owens, & Chard, 2015).

The other key variables associated with reduced symptom reduction was avoidance. Avoidance is widely conceptualized in models of PGD as important in impeding processing emotions of the loss (Boelen et al., 2006; Stroebe & Schut, 2010) and is supported by evidence that avoidant strategies are influential in PGD (Eisma et al., 2013). It is possible that avoidance tendencies limit the extent to which participants were able to engage in emotional tasks during therapy, which restricted their capacity achieve optimal treatment outcomes.

We note several methodological limitations. First, our sample size was relatively small, thereby limiting the extent to which we could examine the differential predictors in each arm of the study. Relatedly, we did not report prediction of outcomes at the two-year follow-up because poor retention at this assessment and limited sample size precluded predictive analyses. Second, we used generic assessments of appraisals (PTCI) and avoidance (IES) which do not target grief-specific appraisals or avoidance strategies. Future studies could use targeted assessment tools, such as the Grief Cognitions Questionnaire (Boelen & Lensvelt-Mulders, 2005) and the Grief-Related Avoidance Questionnaire (Baker et al., 2016), may have yielded more specific information about appraisals and avoidance, respectively. Relatedly, we did not assess other cognitive processes that have been shown to limit treatment outcomes, such as ruminative thinking (Turner, Holtzman, & Mancl, 2007). Third, we recognize that this sample was limited in its range of types of death and relationship to the deceased; these factors are known to impact risk for PGD (Nakajima et al., 2012) and so they may also influence response to treatment. Fourth, we note that our measures of PGD pre-dated the release of the ICD-11 criteria and, accordingly, do not fully align with the ICD-11 PGD definition.

These limitations notwithstanding, this pattern of findings does suggest that treatment planning for patients with PGD needs to consider the levels of avoidance and catastrophic thinking present at the time of treatment. It is possible that specific strategies are

needed to address these issues in treating these PGD patients. For example, those with greater levels of avoidance may require more intensive imaginal or *in vivo* exposure to facilitate emotional processing. Additionally, targeted cognitive restructuring may be needed for those with elevated levels of catastrophic thinking to address entrenched patterns of maladaptive appraisals. As the evidence base increases on treating PGD, it is imperative that further knowledge is acquired regarding the factors that limit optimal treatment response.

Disclosure statement

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