



## Research report

## The relationship between interpersonal problems, negative cognitions, and outcomes from cognitive behavioral group therapy for depression

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## ABSTRACT

**Background:** Interpersonal functioning is a key determinant of psychological well-being, and interpersonal problems (IPs) are common among individuals with psychiatric disorders. However, IPs are rarely formally assessed in clinical practice or within cognitive behavior therapy research trials as predictors of treatment attrition and outcome. The main aim of this study was to investigate the relationship between IPs, depressogenic cognitions, and treatment outcome in a large clinical sample receiving cognitive behavioral group therapy (CBGT) for depression in a community clinic.

**Methods:** Patients ( $N=144$ ) referred for treatment completed measures of IPs, negative cognitions, depression symptoms, and quality of life (QoL) before and at the completion of a 12-week manualized CBGT protocol.

**Results:** Two IPs at pre-treatment, 'finding it hard to be supportive of others' and 'not being open about problems,' were associated with higher attrition. Pre-treatment IPs also predicted higher post-treatment depression symptoms (but not QoL) after controlling for pre-treatment symptoms, negative cognitions, demographics, and comorbidity. In particular, 'difficulty being assertive' and a 'tendency to subjugate one's needs' were associated with higher post-treatment depression symptoms. Changes in IPs did not predict post-treatment depression symptoms or QoL when controlling for changes in negative cognitions, pre-treatment symptoms, demographics, and comorbidity. In contrast, changes in negative cognitions predicted both post-treatment depression and QoL, even after controlling for changes in IPs and the other covariates.

**Limitations:** Correlational design, potential attrition bias, generalizability to other disorders and treatments needs to be evaluated.

**Conclusions:** Pre-treatment IPs may increase risk of dropout and predict poorer outcomes, but changes in negative cognitions during treatment were most strongly associated with improvement in symptoms and QoL during CBGT.

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## 1. Introduction

Interpersonal problems can be defined as recurrent difficulties in relating to others (Horowitz et al., 1993) and encompass a broad range of possible problems, including finding it hard to show affection or socialize with others, being too controlling of others, or subjugating one's own needs by excessive attempts to please others. Interpersonal functioning is intrinsically linked with psychological well-being and interpersonal difficulties are common complaints from individuals seeking psychological assistance (Horowitz and Vitkus, 1986). Research suggests that including measures of interpersonal functioning in therapeutic settings is

clinically informative (Cain et al., 2012; Horowitz et al., 1988; Whisman and Uebelacker, 2003). Individual differences in interpersonal problems can predict differences in development of therapeutic alliance (Muran et al., 1994), therapy processes and outcomes (Gurtman, 1996; Horowitz et al., 1993; Mohr et al., 1990), as well as long term prognosis (Cain et al., 2012). Evidence from this research suggests that accounting for interpersonal functioning may improve treatment efficacy and patient outcomes in psychotherapy. Despite this, IPs are not routinely measured in clinical practice (Hatfield and Ogles, 2004). An enhanced understanding of the relationship between IPs and symptoms before, during, and after treatment could inform prognosis, illuminate mechanisms of change, and highlight opportunities to enhance treatment efficacy.

It has been argued that models of depression must incorporate an understanding of the interactional process between a person who is depressed and the social context within which they exist

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(Coyne, 1976a; Joiner and Coyne, 1999). The experience of IPs predisposes people to depression, and depressed mood is likely to precipitate or exacerbate interpersonal difficulties such as infrequent social engagement, lack of positive reinforcement from others, and deficiencies in interpersonal style during group interactions (Barrett and Barber, 2007; Joiner, 2002; Youngren and Lewinsohn, 1980). Depressed individuals are likely to experience a range of symptoms (e.g. fatigue, concentration difficulties), cognitions (e.g. hopelessness) and behaviors (e.g. withdrawal) that contribute to difficulties sustaining positive and meaningful interpersonal relationships. Depressed people may find that over time their friends and family withdraw from them, thereby removing the benefits of social support and exacerbating IPs (Coyne, 1976b; Joiner et al., 1992; Strack and Coyne, 1983).

While the relationship between psychopathology and interpersonal problems has clearly been established, Cognitive Behavioral Therapy (CBT) has been criticized for overlooking the importance of clients' interpersonal functioning as an area for possible intervention (e.g. Coyne & Gotlib, 1983; Goldfried & Castonguay, 1993; Robins & Hayes, 1993). CBT, within individual (CBIT) or group (CBGT) formats, is an efficacious and relatively low cost treatment for depression (Burlingame et al., 2004; Butler et al., 2006; Morrison, 2001), but not all patients benefit (Burlingame et al., 2004). Identification of patient characteristics associated with poor outcomes could highlight additional avenues for intervention, thereby increasing treatment effectiveness and reducing vulnerability to relapse. Indeed, attending to patients' interpersonal difficulties during CBT is associated with positive change at the end of treatment (Hayes et al., 1996), which suggests CBT may be more efficacious when interpersonal functioning is a direct treatment consideration.

A recent study used the Inventory of Interpersonal Problems Circumplex Scale (IIP-C; Alden et al., 1990) to evaluate changes in interpersonal problems during 16–20 sessions of cognitive therapy in a large sample of depressed adult outpatients (Renner et al., 2012). The IIP (Horowitz et al., 1988) assesses problems in interpersonal relationships across a range of domains and has a number of derivative forms, which have been used to assess the relationship between IPs and therapeutic outcome. Renner and colleagues found that the majority of the sample reported problems with social avoidance and non-assertiveness before treatment and, consistent with previous research (Vittengl et al., 2003), they found that the mean scores for interpersonal distress were reduced after treatment. These findings suggest that although cognitive therapy does not directly target interpersonal problems it may still be effective in improving interpersonal functioning. Other studies have found IPs to be predictive of poor treatment outcomes in clinical samples with generalized anxiety disorder (GAD, Borkovec et al., 2002) and depression (Hardy et al., 2001), particularly difficulties being socially involved. Similarly, in a prospective, non-intervention longitudinal study over a 10 year period Cain et al. (2012) found that depressed individuals with a submissive interpersonal style experienced more chronic symptoms and poorer functioning than those within one of five alternative interpersonal styles (extraverted, dominant, arrogant, cold, or unassuming). Recent research has identified interpersonal subtypes in patients with Generalised Anxiety Disorder GAD (Przeworski et al., 2011) and there is evidence that combining CBT with Interpersonal Processing Treatment provides additional therapeutic benefits (Newman et al., 2008). However, one recent study found the addition of interpersonal processing techniques to CBT did not significantly improve outcomes for GAD patients overall, but the authors suggested that interpersonal processing techniques may still be advantageous for some types of clients with GAD (Newman et al., 2011). Research into IPs most strongly associated with treatment outcomes is therefore needed to guide

future studies of integrative cognitive behavioral and interpersonal treatment protocols. Intervention studies to date have also suffered from several limitations that need to be addressed.

First, many studies have used relatively small sample sizes, which may affect the reliability and generalizability of the findings. Second, although treatment is informed by CBT protocols, the absence of manualized treatments may result in differences in session content between clients that could obscure the true impact of IPs on outcomes. Third, some studies of changes in IPs during the course of cognitive therapy have not reported the association between changes in IPs and changes in depression symptoms (e.g. Renner et al., 2012), leaving the relationship between these factors unclear. Fourth, only outcomes for individual treatment with trained clinicians, who are well equipped to manage challenging interpersonal styles, have been evaluated. It is plausible that IPs would have a greater impact on treatment outcomes in group-based treatments that require interpersonal interactions with other group members. Although standard CBGT for depression generally does not explicitly target IPs as an area for direct intervention, IPs may adversely affect clients' ability relate to other group members, thereby limiting their engagement in the process of group therapy and ultimately increasing the likelihood of treatment attrition or poorer outcomes. Finally, it is also important to demonstrate that the assessment of IPs provides predictive utility above and beyond other factors such as demographics, comorbidity, and the mechanisms targeted in CBGT such as changes in negative cognitions.

The first aim of this study was to determine the strength of the relationships between pre-treatment IPs, negative cognitions, depression symptoms, and quality of life (QoL) in a large clinical sample with major depressive disorder, and to explore clinical and demographic factors associated with IPs. It was expected that more severe IPs would be associated with more severe depression symptoms and negative cognitions, and poorer QoL. The second aim was to examine the relationship between pre-treatment IPs and treatment attrition during a 12-week course of manualized CBGT. Manualized treatments reduce the influence of therapeutic variations on treatment outcome and increase the capacity to detect differences associated with patient characteristics. It was hypothesized that more severe IPs would interfere with therapy engagement, thereby increasing the likelihood of dropout. The third aim was to identify IPs associated with less improvement in symptoms and QoL, as it was expected that IPs would attenuate treatment gains. Based on previous research, IPs consistent with a submissive interpersonal style (e.g., problems with being sociable and assertive) were expected to be most strongly associated with poorer outcomes. The final aim was to determine whether improvements in negative cognitions and IPs would independently predict post-treatment depression symptoms and QoL.

## 2. Method

### 2.1. Participants

Participants ( $N=144$ , 68.1% women) were consecutive referrals to a community based specialist mental health clinic by health practitioners (general practitioners, psychiatrists, psychologists) for a unipolar depressive disorder with a mean age of 38.56 years ( $SD=13.69$ , range=18–73). Inclusion criteria for the treatment group were (a) a diagnostic and statistical manual of mental disorders (DSM-IV, American Psychiatric Association, 2000) unipolar depressive disorder, (b) no current active suicidal intent (suicidal ideation or history were not exclusion criteria), and (c) no psychotic or bipolar affective disorder. DSM-IV diagnoses were determined using the mini international neuropsychiatric

interview (MINI, Lecrubier et al., 1997; Sheehan et al., 1997, 1998), which was administered by experienced diagnosticians with doctoral or masters-level clinical psychology qualifications. Clinicians are routinely video-taped and observed by a more senior clinician for between 12 (doctorate) and 24 (masters) months after their qualifications. Diagnoses are discussed and discrepancies resolved at weekly supervision. In addition, diagnoses are presented and discussed at clinical review and peer supervision meetings. Principal diagnoses were major depressive disorder ( $n=127$ , 88.2%) or dysthymia ( $n=17$ , 11.8%). Ninety-five (66.0%) patients met criteria for at least one additional disorder, and 36 (25.0%) met criteria for at least two additional disorders. The most common comorbid disorders were GAD ( $n=41$ ), social phobia ( $n=32$ ), dysthymia ( $n=28$ ), and panic disorder/agoraphobia ( $n=14$ ). Most patients were born in Australia (71.5%), followed by Europe/United Kingdom (15.3%), Asia (3.5%) and North America (3.5%). Half (51.0%) were employed, 36.9% were single, 41.1% were married or in a live in relationship, 19.9% were separated or divorced, and 2.1% were widowed. High school was the highest qualification for 34.6%, whereas 13.1% did not complete high school, 19.2% had a trade qualification, and 19.2% had a tertiary education. Most (82.5%) reported taking medication for their presenting problem. Most (92%) had been on their medication for more than 1 month, 78% for at least 2 months, 71% for at least 3 months, and 50% for at least 9 months.

## 2.2. Measures

### 2.2.1. Inventory of interpersonal problems (IIP-32, Barkham et al., 1996)

The IIP-32 is a relatively brief 32-item measure with eight subscales reflecting different IPs. The IIP-32 subscales (4 items each) have demonstrated adequate internal consistency in outpatient and non-clinical samples (Barkham et al., 1996). McEvoy et al. (in press) recently confirmed that Barkham et al.'s (1996) eight-factor structure of the IIP-32 was robust and highly internally reliable across clinical samples with anxiety and depressive disorders, and eating disorders. The IIP-32 subscales are also associated with symptoms of anxiety, depression, and eating disorders (Lampard et al., 2011; McEvoy et al., in press). This version of the IIP-32 (there are around 10 versions of the IIP in the literature) was used instead of circumplex versions to maximize clinical utility. Whilst the circumplex versions do provide useful theoretical and clinical information (see Renner et al., 2012), they tend to be more complex for clinicians to calculate and interpret than the IIP-32 subscale scores. For instance, scoring the circumplex versions require several steps, including computation of raw scores, calculating a general factor score by averaging the individual's eight octant scores, ipsatizing the octant scores by subtracting that individual's general factor score from each raw octant score, then combining ipsatized octant scores to form vectors (see Locke, 2011, for a detailed explanation). The version derived from factor analysis used in this study simply requires clinicians to divide total scores by the number of items in each subscale (i.e., 4). It is noteworthy that the 32-item circumplex version (IIP-Short Circumplex, IIP-SC) shares only 13 items with the 32-item version derived by factor analysis used in this study (IIP-32), so the circumplex methodology cannot be used to score the IIP-32. Our primary aim was to identify IIP-32 subscales associated with treatment attrition and outcome to complement information derived from circumplex versions. Four of the subscales begin with the stem "hard to be" and four begin with "too". The "Hard to be" scales refer to difficulty being sociable, assertive, involved, and supportive. The "too" scales refer to being too open, caring, aggressive and dependent. In this study, Cronbach's alphas demonstrated good internal reliability for the whole scale (0.81)

and acceptable to excellent internal reliability for each subscale: hard to be sociable (0.86), hard to be assertive (0.80), hard to be involved (0.74), hard to be supportive (0.83), too open (0.79), too caring (0.73), too aggressive (0.82), too dependent (0.69).

### 2.2.2. Cognitions checklist (CCL; Beck et al., 1987)

The CCL consists of 14 items measuring common cognitive themes associated with depression and 12 items addressing cognitive content associated with anxiety. Only the depression subscale was used for this study (CCLD). Participants indicate the frequency of each thought on a five-point scale ranging from 'never' (0) to 'always' (4). The CCL has good convergent and discriminant validity in regards to measures of depression and anxiety symptoms (Beck et al., 1987), and within community clinics its brevity is an advantage over many alternative measures. Cronbach's alpha for the CCLD in the current study was 0.91.

### 2.2.3. Beck anxiety inventory (BAI; Beck et al., 1988)

The BAI consists of 21 items and measures the severity of anxiety symptoms over the previous week. Reliability and validity are established; internal consistency reliability coefficients range from 0.85 to 0.94, with a 1-week test-retest reliability coefficient of 0.75 (Beck et al., 1988). Cronbach's alpha in the current study was 0.93.

### 2.2.4. Beck depression inventory (BDI-II; Beck et al., 1996)

The BDI-II is a 21-item measure of depression symptoms experienced during the previous fortnight. Factor analytic studies of the BDI-II provide evidence for both a total score and two factor scores representing cognitive and somatic dimensions (Beck et al., 1996). Internal consistency ( $\alpha=0.92$ ) and test-retest reliability ( $r=0.93$  over 1 week) are established (Beck et al., 1996), and evidence for construct validity has been demonstrated (e.g. Dozois et al., 1998; Osman et al., 2004). Support for convergent and discriminant validity has also been reported (Steer et al., 1997). Cronbach's alpha in the current study was 0.89.

### 2.2.5. Quality of life enjoyment and satisfaction questionnaire – short form (Q-LES-Q, Endicott et al., 1993)

The Q-LES-Q short form is a 14-item self-report instrument deriving from the General Activities Scale of the original 93-item Q-LES-Q. The Q-LES-Q short form includes items on various areas of daily functioning such as work, physical health, social relationships, family relationships, ability to function in daily life, and overall well being. The total score is the sum of items expressed as a percentage of the maximum score, with lower scores indicating poorer QoL. The Q-LES-Q short form has good test-retest reliability, internal consistency, and construct and criterion validity (Rapaport et al., 2007; Ritsner et al., 2002). The scale explains variance beyond that accounted for by symptom scales (Hope et al., 2009). Cronbach's alpha in the current study was 0.83.

## 2.3. Procedure

All patients completed the IIP-32, CCL, Q-LES-Q, BDI-II, and BAI prior to their clinical assessment as part of the standard admission protocol and again at the last group session. The MINI was administered at the initial clinical assessment and patients meeting all inclusion criteria were offered a place in the next available group. Previous research found that the CBGT protocol used in this study is effective and compares well to international benchmarks (McEvoy and Nathan, 2007). The program focuses on depressive symptoms but, given the high rates of comorbidity between anxiety and depressive disorders, the strategies are also discussed with reference to anxiety symptoms. Patients diagnosed with



principal anxiety disorders but with prominent depressive features were also included in the groups, but most (more than 80%) had a principal depressive diagnosis and only these patients are included in this study.

All patients attended the same group program comprising of 10 two-hour weekly sessions, which is based on Beck's (1979) depression manual and Barlow and Craske's (1994) anxiety manual. The core components of the program are: (a) psychoeducation about depression and anxiety, (b) de-arousal techniques including slow breathing, (c) behavioral activation tasks, (d) exposure tasks, and (e) cognitive restructuring. None of the content explicitly focused on interpersonal issues, although patient-driven treatment goals may have had interpersonal contexts (e.g., to re-engaged in previously enjoyed social or sporting activities). Between 8 and 10 patients commenced each group, and treatment integrity was encouraged by a structured and very detailed therapist manual containing an agenda, detailed content outline for each session, therapist instructions, and patient handouts. All groups are facilitated by one experienced masters- or doctoral-level clinical psychologist and one clinical psychologist trainee. Therapist training in the protocol involved co-facilitation of at least one group with a more experienced therapist, along with weekly supervision from another more senior clinical psychologist in the service. Patients were encouraged to maintain a stable medication regime during the group program, but this was not a requirement for continued treatment. The process of receiving informed written consent for using patients' data for research purposes was approved by the Area Health Service's Mental Health Human Research Ethics Committee (Registration number 2013-13).

#### 2.4. Data analysis

Pearson bivariate correlation coefficients were calculated to test the first hypothesis, that more severe IPs would be associated with more severe depression, more negative cognitions, and poorer QoL. To test our second hypothesis, that more severe IPs would be associated with dropout, independent-samples *t*-tests were used to compare treatment completers and dropouts on IPs, negative cognitions, symptoms (depression and anxiety), and QoL. A follow-up univariate ANOVA comparing IPs across completers and dropouts, whilst controlling for pre-treatment negative cognitions, was used to guard against the possibility that differences in IPs were simply a consequence of more negative cognitions leading to an excessively pessimistic assessment of IPs. To ensure that the intervention was effective, paired-samples *t*-tests were then used to test for significant changes in IPs, negative cognitions, symptoms, and QoL (completer and intention to treat, ITT). Cohen's *d* indexed effect sizes. A repeated-measures ANCOVA with Time (pre- vs. post-treatment) as a between-subjects variable, and BDI-II and CCLD change scores as covariates, was used to test whether IIP-32 total changes simply reflected changes in mood and negative thinking.

Hierarchical multiple linear regression (HMLR) analyses were then used to test the third and fourth hypotheses, that IPs would be associated with an attenuation of treatment gains (hypothesis 3) and that changes in negative cognitions and IPs would independently predict post-treatment depression symptoms and QoL (hypothesis 4). An alpha of 0.05 was used for all analyses. *T*-tests, ANOVAs, regression analyses generally had 80% power to detect at least medium effect sizes, although the relatively low dropout rate meant that comparisons between completers and dropouts were powered to detect large effect sizes (Cohen, 1992). Missing value analysis revealed a non-significant Little's (1988) chi-square,  $\chi^2(293)=312.85$ ,  $p=0.20$ , thus not ruling out the null hypothesis that data were missing completely at random. All available data

were therefore used for each analysis as missing data were unlikely to significantly bias parameter estimates.

### 3. Results

#### 3.1. Correlations between interpersonal problems, negative cognitions, symptoms, and quality of life

At pre-treatment, 3 (2.1%), 7 (4.9%), 1 (0.7%), and 2 (1.4%) patients failed to provide BDI-II, BAI, CCLD, and Q-LES-Q data, respectively. At post-treatment, the corresponding numbers were 51 (35.4%), 50 (34.7%), 44 (30.6%), and 45 (31.3%). All patients provided pre-treatment IIP-32 data, but 60 (41.7%) did not provide post-treatment data. Reasons for missing post-treatment data included discontinuation with the group, failure to attend the last treatment session, and/or failure to return the questionnaire package.

Table 1 shows that pre-treatment IIP-32 total score was significantly correlated with pre-treatment BDI-II, CCLD, and Q-LES-Q scores, suggesting that more IPs were associated with more severe depression symptoms, more negative cognitions, and poorer QoL. The IIP-32 total score was not significantly associated with the BAI ( $r=0.16$ ,  $p=0.06$ ). The hard to be sociable, too aggressive, hard to be involved, and too dependent subscales were significantly and positively associated with the BDI-II, whereas only the too aggressive subscale was significantly and positively associated with the BAI. The CCLD was significantly associated with all IIP-32 subscales except the Too Open subscale. The Q-LES-Q was significantly associated with the hard to be sociable and hard to be involved subscales, such that higher scores were associated with lower QoL.

#### 3.2. Relationship between interpersonal problems, negative cognitions, and dropout

Thirty-two (22.2%) patients were coded as treatment dropouts. Reasons for dropout included being unhappy with the group format ( $n=1$ ), difficulties with cognitive behavior therapy (CBT,  $n=1$ ), gaining employment ( $n=5$ ), and non-mutual termination

**Table 1**

Pearson bivariate correlation coefficients between pre-treatment IIP-32 scores, depression, anxiety, quality of life, and age.

	BDI-II	BAI	CCLD	Q-LES-Q	Age
BDI-II	–	0.60***	0.62***	–0.64***	–0.18*
BAI	–	–	0.36***	–0.39***	–0.14
CCLD	–	–	–	–0.50***	–0.16
Q-LES-Q	–	–	–	–	0.08
IIP-32 total	0.34***	0.16	0.49***	–0.22**	–0.07
Hard to be sociable	0.27***	0.09	0.43***	–0.33***	0.01
Hard to be assertive	0.12	–0.02	0.20*	–0.03	0.17*
Too aggressive	0.25**	0.22*	0.32**	–0.16	–0.26**
Too open	–0.15	–0.04	–0.11	0.02	0.13
Too caring	0.13	0.06	0.20*	0.02	–0.09
Hard to be supportive	0.11	0.11	0.18*	–0.08	0.08
Hard to be involved	0.21*	0.09	0.28**	–0.21*	0.10
Too dependent	0.22**	0.09	0.30***	–0.06	–0.30***

Note: BDI-II=Beck depression inventory, BAI=Beck anxiety inventory, CCLD=Cognitive checklist depression subscale, Q-LES-Q=Quality of life enjoyment and satisfaction questionnaire, IIP-32=Inventory of interpersonal problems-32.

(\*)  $p<0.05$

(\*\*)(\*)  $p<0.01$

(\*\*)(\*)(\*)  $p<0.001$

**Table 2**  
Comparisons between completers and dropouts on clinical variables.

Pre-treatment	Completers		Dropouts		Test statistics	<i>d</i>
	<i>n</i>	Mean ( <i>SD</i> )	<i>n</i>	Mean ( <i>SD</i> )		
BDI-II	109	29.79 (10.69)	32	32.97 (10.49)	<i>t</i> (139)=1.49	0.30
BAI	107	19.36 (12.65)	30	22.43 (10.76)	<i>t</i> (135)=1.22	0.26
CCLD	111	28.41 (10.52)	32	33.28 (10.87)	<i>t</i> (141)=2.29*	0.46
Q-LES-Q	110	42.98 (12.06)	32	38.12 (16.69)	<i>t</i> (140)=−1.83	0.34
IIP-32 total	112	1.70 (0.50)	32	1.93 (0.56)	<i>t</i> (142)=2.25*	0.53
Hard to be social		2.13 (0.97)		2.38 (1.11)	<i>t</i> (142)=1.24	0.24
Hard to be assertive		2.08 (0.90)		2.27 (0.84)	<i>t</i> (142)=1.09	0.22
Too aggressive		1.38 (0.91)		1.60 (0.75)	<i>t</i> (142)=1.24	0.27
Too open		1.63 (0.94)		1.27 (0.86)	<i>t</i> (142)=−1.98*	−0.4
Too caring		2.01 (0.92)		1.88 (0.90)	<i>t</i> (142)=−0.63	−0.14
Hard to be supportive		1.01 (0.93)		1.46 (1.05)	<i>t</i> (142)=2.35*	0.45
Hard to be involved		1.60 (0.97)		1.93 (0.95)	<i>t</i> (142)=1.86	0.34
Too dependent		1.83 (0.88)		2.06 (0.91)	<i>t</i> (142)=1.32	0.26

Note. BDI-II=Beck depression inventory, BAI=Beck anxiety inventory, CCLD=Cognitive checklist depression subscale, Q-LES-Q=Quality of life enjoyment and satisfaction questionnaire, IIP-32=Inventory of interpersonal problems-32.

(\*)  $p<0.05$

**Table 3**  
Completer and intention-to-treat (ITT) means (standard deviations), test statistics and effect sizes for symptom measures and IIP-32 total and subscales.

	Pre	Post	Post	Completer		ITT	
				Test statistics	<i>d</i>	Test statistics	<i>d</i>
		Completer	ITT				
BDI-II	30.51 (10.69)	18.02 (13.17)	23.56 (14.54)	<i>t</i> (89)=8.60**	1.05	<i>t</i> (140)=7.56**	0.55
BAI	20.03 (12.29)	13.13 (11.69)	15.95 (12.41)	<i>t</i> (88)=4.47**	0.58	<i>t</i> (135)=4.24**	0.33
CCLD	29.50 (10.76)	19.57 (12.54)	23.64 (13.41)	<i>t</i> (98)=7.35**	0.85	<i>t</i> (142)=6.81**	0.48
Q-LES-Q	41.89 (13.34)	53.71 (20.32)	48.98 (20.14)	<i>t</i> (96)=4.95**	0.70	<i>t</i> (141)=4.75**	0.42
IIP-32 total	1.75 (0.52)	1.36 (0.55)	1.55 (0.60)	<i>t</i> (83)=6.88**	0.73	<i>t</i> (143)=6.20**	0.36
Hard to be sociable	2.19 (1.00)	1.61 (1.00)	1.87 (1.06)	<i>t</i> (83)=5.71**	0.58	<i>t</i> (143)=5.31**	0.31
Hard to be assertive	2.12 (0.89)	1.92 (0.97)	1.98 (0.93)	<i>t</i> (83)=2.91*	0.22	<i>t</i> (143)=2.86*	0.15
Too aggressive	1.43 (0.88)	0.98 (0.66)	1.18 (0.77)	<i>t</i> (83)=4.90**	0.58	<i>t</i> (143)=4.64**	0.30
Too open	1.55 (0.93)	1.70 (0.80)	1.61 (0.85)	<i>t</i> (83)=−1.40	−0.17	<i>t</i> (143)=−1.40	−0.07
Too caring	1.98 (0.92)	1.71 (0.98)	1.77 (0.93)	<i>t</i> (83)=4.16**	0.28	<i>t</i> (143)=4.00**	0.23
Hard to be supportive	1.11 (0.98)	0.66 (0.69)	0.94 (0.93)	<i>t</i> (83)=3.02*	0.54	<i>t</i> (143)=3.00*	0.18
Hard to be involved	1.65 (0.97)	1.32 (0.95)	1.51 (1.00)	<i>t</i> (83)=2.79*	0.34	<i>t</i> (143)=2.75*	0.14
Too dependent	1.88 (0.89)	1.44 (0.84)	1.68 (0.90)	<i>t</i> (83)=3.71**	0.51	<i>t</i> (143)=3.60**	0.22

Note: Pre=pre-treatment, Post=post-treatment, ITT=Intention to treat, *d*=Cohen's *d*, BDI-II=Beck depression inventory-II, BAI=Beck anxiety inventory, CCLD=Cognitive checklist depression scale, Q-LES-Q=Quality of life enjoyment and satisfaction questionnaire, IIP-32=Inventory of interpersonal problems-32.

\*  $p<0.01$   
\*\*  $p<0.001$

where the reason for discontinuation was unknown ( $n=25$ ). Independent-samples *t*-tests showed that dropouts had a higher pre-treatment IIP-32 total and CCLD scores than completers, but did not significantly differ from completers on the BDI-II, BAI, or Q-LES-Q (Table 2). Completers scored significantly higher on too open, and lower on hard to be supportive, compared to dropouts. A follow-up univariate ANOVA controlling for CCLD scores found that the difference between dropouts and completers on the hard to be supportive subscale remained significant,  $F(1,140)=3.96$ ,  $p<0.05$ ,  $d=0.41$ . In contrast, once CCLD was taken into account completers and dropouts no longer significantly differed on the too open subscale,  $F(1, 140)=3.06$ ,  $p=0.08$ ,  $d=0.35$ .

### 3.3. Treatment outcomes

Table 3 provides completer and intention-to-treat (ITT) means (standard deviations) at pre- and post-treatment, as well as effect sizes. Missing post-treatment data were replaced with data from the last observation. Paired-samples *t*-tests demonstrated significant improvements on completer and ITT BDI-II, BAI, CCLD, and Q-LES-Q scores. Effect sizes were moderate to large (Cohen, 1988). The IIP-32 total score significantly reduced and the effect size was

large for completers. All subscales significantly reduced during treatment, with the exception of the Too Open subscale. To determine whether change in IIP-32 total scores simply reflected a change in mood and negative thinking, a repeated-measures ANCOVA was run with Time (pre- vs. post-treatment) as a between-subjects variable, BDI-II and CCLD change scores as covariates, and IIP-32 total change score as the dependent variable. The main effect of Time was significant,  $F(1, 68)=7.78$ ,  $p<0.01$ , partial  $\eta^2=0.10$ , suggesting that the change in self-reported IPs was not simply a consequence of a change in mood state or negative thinking.

### 3.4. Relationship between pre-treatment IIP-32 and negative cognitions, and outcome

HMLR analyses were conducted separately for post-treatment BDI-II and Q-LES-Q scores to identify if pre-treatment IIP-32 subscale scores could predict post-treatment symptoms and QoL. Given that the primary question was whether the IIP-32 could provide useful prognostic information above and beyond demographic and clinical indicators, pre-treatment symptoms were entered in step 1, demographics (age, gender) and the presence

**Table 4**

HMLRs with pre-treatment depression symptoms, demographics, comorbidity, and pre-treatment IIP-32 subscales predicting post-treatment depression symptoms.

Criterion	Step	Predictors	$\Delta R^2$	Test statistics				
				B	SE B	$\beta$	t	Part r
Post-BDI-II	1	Pre-BDI-II	0.22***	0.61	0.12	0.47	5.03***	0.47
	2	Pre-BDI-II	0.01	0.61	0.13	0.47	4.77*(*)	0.46
		Age	–	0.05	0.09	0.05	0.5	0.05
		Gender	–	–0.59	2.83	–0.02	–0.21	–0.02
		Comorbidity	–	0.84	2.54	0.03	0.33	0.03
	3	Pre-BDI-II	0.13*(*)	0.49	0.15	0.38	3.28*(*)	0.19
		Age	–	0.02	0.1	0.02	0.24	0.02
		Gender	–	0.51	2.85	0.02	0.18	0.02
		Comorbidity	–	–1.86	2.55	–0.07	–0.73	–0.07
		Pre-CCLD	–	–0.01	0.16	–0.01	–0.01	0.01
		Hard to be sociable	–	0.04	1.45	0.01	0.03	0.01
		Hard to be assertive	–	3.62	1.64	0.24	2.21(*)	0.20
		Too caring	–	3.16	1.45	0.23	2.17(*)	0.19
		Too dependent	–	0.25	1.58	0.02	0.16	0.01

Note: Pre = pre-treatment, Post = post-treatment, BDI-II = Beck depression inventory, CCLD = Cognitive checklist depression scale.

(\*)  $p < 0.05$ (\*) (\*)  $p < 0.01$ (\*) (\*) (\*)  $p < 0.001$ 

of comorbid disorders in step 2, and pre-treatment CCLD and IIP-32 subscale scores in step 3. To maximize power and appropriately balance Type I and Type II error rates, only IIP-32 subscales with significant ( $p < 0.05$ ) bivariate correlations with post-treatment symptoms were included in step 3. Pre-treatment hard to be assertive, too caring, hard to be sociable, and too dependent subscale scores were significantly correlated with post-treatment BDI-II so were included in the HMLR analyses (Table 4). Pre-treatment Hard to be Assertive and Too Caring subscale scores explained unique variance in post-treatment BDI-II scores above and beyond pre-treatment BDI-II and CCLD scores, demographics, and comorbidity (1 = no comorbid disorder, 2 = comorbid disorder). Pre-treatment Too Caring and Too Dependent subscales were significantly correlated with post-treatment Q-LES-Q scores, but the Hard to be Sociable and Too Aggressive subscales were also entered in step 3 for the Q-LES-Q because they only just fell short of statistical significance ( $ps = 0.06$ ). However, steps 2 and 3 failed to explain unique variance in post-treatment Q-LES-Q scores ( $ps > 0.05$ ) so these findings are not reported.

### 3.5. Changes in negative cognitions, IPs, depression symptoms and QoL

BDI-II change scores were significantly correlated with CCLD ( $r = 0.72$ ,  $p < 0.001$ ) and IIP-32 ( $r = 0.50$ ,  $p < 0.001$ ) change scores. Q-LES-Q change scores were also significantly and negatively correlated with BDI-II ( $r = -0.64$ ,  $p < 0.001$ ), CCLD ( $r = -0.57$ ,  $p < 0.001$ ), and IIP-32 ( $r = -0.32$ ,  $p < 0.01$ ) change scores. HMLR was used to determine whether CCLD and IIP-32 changes during treatment were independently associated with post-treatment BDI-II and Q-LES-Q scores (Table 5). Demographics (age, gender), the presence of comorbidities, and pre-treatment scores (BDI-II or Q-LES-Q) were entered in step 1, IIP-32 total change scores were entered in step 2, and CCLD change scores were entered in step 3. In a second HMLR steps 2 and 3 were reversed. For the BDI-II, pre-treatment BDI-II scores were significantly associated with post-treatment BDI-II in all three steps. IIP-32 change scores were also associated with post-treatment BDI-II in step 2 but not when entered in step 3 ( $p = 0.08$ ). In contrast, CCLD change scores were associated with post-treatment BDI-II when entered in step 2 or 3. The BDI-II includes items assessing cognitive symptoms of depression, which may have artificially inflated its relationship with the CCLD, so the models were re-run with only the BDI-II somatic

subscale. The pattern of findings was similar, so is not reported. For the Q-LES-Q, pre-treatment Q-LES-Q was associated with post-treatment Q-LES-Q in all three steps, IIP-32 change scores were associated with post-treatment Q-LES-Q only in step 2, and CCLD change scores were associated with post-treatment Q-LES-Q when entered in step 2 or 3.<sup>1</sup>

## 4. Discussion

Integrative theoretical models emphasize the role that interpersonal context plays in triggering, maintaining, and/or exacerbating depression symptoms (Joiner, 2002), but few CBT trials have investigated the relationship between IPs and treatment outcome. The first aim of this study was to determine the strength of the relationship between pre-treatment IPs, negative cognitions, depression symptoms and QoL in a clinical sample with principal major depressive disorder. As hypothesized, more severe IPs were associated with more severe depression symptoms, more negative cognitions, and poorer QoL. More specifically, finding it difficult to be sociable and involved with others, and being too aggressive and dependent, were associated with more severe depression symptoms. Being too aggressive was also associated with more anxiety symptoms. Finding it hard to be sociable or involved with others were the only IIP-32 subscales associated with poorer QoL. The cross-sectional nature of these associations precludes causal conclusions. It may be that IPs increase depressive and anxiety symptoms or that cognitive (e.g., negative thoughts about oneself, past experiences, and future expectations), somatic (e.g., lethargy, agitation), and behavioral (e.g., withdrawal,

<sup>1</sup> For completeness the analyses were re-run with the IIP-32 subscales instead of the IIP-32 total score. When predicting post-treatment total BDI-II, pre-treatment BDI-II explained unique variance in all steps but age, sex, and comorbidities did not. The CCLD added significant explanatory power when added in step 2 ( $\Delta R^2 = 0.38$ ,  $p < 0.001$ ) or 3 ( $\Delta R^2 = 0.17$ ,  $p < 0.001$ ). IIP-32 subscale scores only added significant explanatory power when added in step 2 ( $\Delta R^2 = 0.26$ ,  $p < 0.01$ , Hard to be Sociable subscale only, Part  $r = -0.17$ ,  $p = 0.03$ ) but not in step 3 ( $\Delta R^2 = 0.05$ ,  $p = 0.39$ ). When predicting BDI-II somatic subscale, entering the IIP-32 subscales in step 2 added significant explanatory power to the whole model ( $\Delta R^2 = 0.27$ ,  $p < 0.01$ , Hard to be Sociable subscale only, Part  $r = -0.26$ ,  $p = 0.01$ ), but not in step 3 ( $\Delta R^2 = 0.02$ ,  $p = 0.99$ ). When predicting post-treatment QoL, pre-treatment QoL and CCLD change predicted post-treatment QoL in all steps of the model but age, sex, and comorbidities did not. Adding IIP-32 subscales in steps 2 ( $\Delta R^2 = 0.14$ ,  $p = 0.13$ ) or 3 ( $\Delta R^2 = 0.07$ ,  $p < 0.45$ ) failed to add unique explanatory power.

**Table 5**  
HMLRs with demographics, comorbidity, pre-treatment depression symptoms, changes in interpersonal problems, and changes in negative cognitions predicting post-treatment depression and quality of life.

Criterion	Predictors	$\Delta R^2$	Statistics				
			B	SE B	$\beta$	t	Part r
Post BDI-II	Step 1	0.27***					
	Age		0.11	0.11	0.11	0.99	0.10
	Gender		0.45	3.29	0.02	0.14	0.01
	Comorbidity		2.40	2.99	0.09	0.80	0.09
	Pre BDI-II		0.70	0.15	0.52	4.74***	0.50
	Step 2a	0.19***					
	Age		0.04	0.10	0.04	0.43	0.04
	Gender		-0.36	2.87	-0.01	-0.13	-0.01
	Comorbidity		1.28	2.61	0.05	0.49	0.05
	Pre BDI-II		0.67	0.13	0.50	5.22***	0.48
	IIP-32 total $\Delta$		-13.59	2.88	-0.44	-4.72***	-0.43
	Step 2b	0.39***					
	Age		-0.02	0.08	-0.02	-0.22	-0.02
	Gender		-0.66	2.28	-0.02	-0.29	-0.02
	Comorbidity		2.71	2.07	0.10	1.31	0.08
	Pre BDI-II		0.69	0.10	0.52	6.78***	0.49
	CCLD $\Delta$		-0.76	0.09	-0.64	-8.58***	-0.47
	Step 3a (3b)	0.22*** (0.02)					
	Age		-0.03	0.08	-0.03	-0.33	-0.02
	Gender		-0.8	2.24	-0.03	-0.36	-0.03
	Comorbidity		2.28	2.04	0.08	1.12	0.08
Post Q-LES-Q	Pre BDI-II		0.68	0.10	0.51	6.79***	0.49
	IIP-32 total $\Delta$		-4.75	2.62	-0.15	-1.81	-0.13
	CCLD $\Delta$		-0.66	0.10	-0.56	-6.54***	-0.47
	Step 1	0.15*					
	Age		-0.06	0.17	-0.04	-0.32	-0.04
	Gender		-1.41	5.04	-0.03	-0.28	-0.03
	Comorbidity		-5.98	4.60	-0.14	-1.30	-0.14
	Pre Q-LES-Q		0.63	0.19	0.35	3.25**	0.35
	Step 2a	0.09**					
	Age		0.01	0.17	0.01	0.05	0.01
	Gender		0.70	4.84	0.02	0.14	0.02
	Comorbidity		-5.11	4.38	-0.12	-1.17	-0.12
	Pre Q-LES-Q		0.61	0.18	0.35	3.33**	0.34
	IIP-32 total $\Delta$		14.45	4.84	0.31	2.99**	0.31
	Step 2b	0.23***					
	Age		0.08	0.15	0.05	0.55	0.05
	Gender		0.76	4.34	0.02	0.17	0.02
	Comorbidity		-6.92	3.94	-0.16	-1.75	-0.16
	Pre Q-LES-Q		0.71	0.17	0.40	4.27***	0.39
	CCLD $\Delta$		0.90	0.17	0.50	5.25***	0.48
	Step 3a (3b)	0.15*** (0.01)					
	Age		0.09	0.15	0.06	0.58	0.05
	Gender		1.08	4.39	0.02	0.25	0.02
	Comorbidity		-6.64	3.99	-0.16	-1.67	-0.15
	Pre Q-LES-Q		0.70	0.17	0.40	4.17***	0.39
	IIP-32 total $\Delta$		3.41	5.14	0.07	0.66	0.06
	CCLD $\Delta$		0.83	0.20	0.46	4.12***	0.38

Note: Pre = pre-treatment, Post = post-treatment, BDI-II = Beck depression inventory, IIP-32 = Inventory of interpersonal problems-32, Q-LES-Q = Quality of life enjoyment and satisfaction questionnaire,  $\Delta$  = change from pre- to post-treatment. Steps 2a and 3a included IIP-32 total change score in the second step, whereas steps 2b and 3b included CCLD change score in the second step.

(\*)  $p < .05$

(\*\*)(\*)  $p < .01$

(\*\*)(\*\*)(\*)  $p < .001$

avoidance) symptoms adversely impact on interpersonal relations, although these relationships are most likely to be reciprocal.

The second aim of this study was to examine the relationship between pre-treatment IPs and treatment attrition. It was expected that more severe IPs would interfere with engagement in group therapy, thereby increasing the likelihood of dropout. This hypothesis was partially supported, with treatment dropouts scoring higher on the Hard to be Supportive subscale, but lower on the Too Open subscale, compared to completers. Completers and discontinuers did not significantly differ on pre-treatment depression, anxiety, or QoL. The fact that dropouts scored more highly on the Hard to be Supportive subscale, even after controlling for differences in negative cognitions, suggests that the more

difficulty they had being supportive of others' needs the less likely they were to continue with the group. It is tempting to speculate that patients high on the Hard to be Supportive subscale would be more likely to complete individual treatment, where they do not need to attend to others' needs. In contrast to the hypothesis, those with higher scores on the Too Open subscale were more likely to complete treatment. The most likely explanation for this finding is that preparedness to share personal information may have different impacts within different contexts. Regular over-disclosure within friendships or partnerships may be detrimental, whereas sharing personal information, experiences, and concerns within a therapeutic group is likely to optimize learning, engagement with the group, and ultimately investment in the program. Moreover,



therapists will have fewer opportunities to help patients apply the treatment strategies to their personal circumstances, and overcome idiosyncratic obstacles to change, if they are reluctant to describe their experiences within the group context. It is noteworthy that completers and discontinuers no longer significantly differed on the Too Open subscale after controlling for the CCLD, which may suggest that the Too Open difference simply reflected differences in negative cognitions. However, the fact that the Too Open subscale was stable across treatment despite improvements in negative cognitions is inconsistent with this proposition. Our findings suggest, therefore, that patients who were able to be supportive to others and who tended to be more open about their own problems prior to attending the group were more likely to complete the program.

The third aim was to identify pre-treatment IPs associated with less improvement in symptoms and QoL. Consistent with previous research, CBGT resulted in significant and moderate to large improvements in depression symptoms and QoL (McEvoy and Nathan, 2007). Also consistent with previous research of individual CT for depression (e.g., Renner et al., 2012) and GAD (Newman et al., 2011), CBGT was also associated with significant and moderate (ITT) to large (completer) reductions in IPs. Moreover, these reductions were broad, with significant improvements in seven of the eight IIP-32 subscales. The ability to predict treatment outcome from IPs at pre-treatment could have considerable clinical utility in terms of case formulation, management, treatment-matching, and prognosis. The Hard to be Assertive and Too Caring subscales were the only pre-treatment IPs that explained unique variance in post-treatment depression symptoms after controlling for pre-treatment symptoms, negative cognitions, demographics, and comorbidity. It is plausible that difficulties with assertiveness would limit patients' ability to derive as much benefit from the group, if it interferes with their preparedness to discuss difficulties with their therapist, or to assert their own needs, opinions, and experiences within the group. Consistent with this notion, the Too Caring subscale measures a tendency to subjugate one's own needs (e.g., I put other people's needs before my own too much. It is hard to attend to my own welfare when someone else is needy), which may further undermine learning in relation to one's own problems and experiences. These findings are consistent with previous research demonstrating associations between a submissive interpersonal style and poorer treatment outcome (Hardy et al., 2001) and greater depression chronicity (Cain et al., 2012).

The Dominance Behavioral System (DBS) provides a theoretical account of a biologically based system that guides drive and energy to pursue power (dominance motivation), dominance behaviors, and subordination (Gilbert, 2000; Sloman, 2000; see Johnson et al., 2012, for a review). This account suggests that submissive behaviors including escape can be functionally adaptive, in that they signal a lack of competition, thereby reducing the likelihood of ongoing fighting, punishment, or conflict, and may elicit helping behaviors from others. Depression is thought to ensue when individuals are unable to terminate the submissive behavioral style. Our findings are consistent with the DBS account, given that submissive styles were associated with treatment attrition or 'escape' (lower on the Too Open subscale), or ultimately less progress in treatment (higher on the Hard to be Assertive and Too Caring subscales). Individual treatment offers the flexibility for therapists to formulate and address problematic interpersonal styles at each phase of therapy, either through exploration of the therapeutic relationship or by teaching specific social skills (e.g., assertiveness training). Findings from this study suggest that prior to commencing group treatment, patients identified as having submissive interpersonal styles may benefit from prior assertiveness training. Alternatively, including a

module into the group program that explicitly targets submissive behaviors may improve retention and outcomes.

The final aim of this study was to determine whether changes in IPs were associated with improvements in depression symptoms and QoL above and beyond pre-treatment symptoms, changes in negative cognitions, and demographic and clinical factors. Change in IPs did explain unique variance in changes in depression symptoms and QoL when controlling for age, gender, comorbidity, and pre-treatment symptoms. However, when changes in negative cognitions were entered into the model IPs just fell short of significantly adding unique explanatory power for post-treatment depression symptoms, and IPs no longer explained unique variance in QoL once negative cognitions were taken into account. Moreover, changes in IPs failed to explain additional variance in depression or QoL when entered in the model after changes in negative cognitions. The main implication of these findings is that cognitive shift during CBGT appears to be most strongly associated with symptom reduction and improvement in QoL than changes in IPs. Together with the earlier findings, it appears that while some *pre-treatment* IPs were useful predictors of attrition and post-treatment symptoms, the principal mechanism of *change* in CBGT in this study was consistent with cognitive theory. This finding is perhaps unsurprising given that the CBGT program did not directly target IPs. Future treatments targeting the most dysfunctional IPs identified in this study may indeed provide additive benefit. It is noteworthy, however, that research on anxiety and depressive disorders has not unequivocally supported the notion that CBT and interpersonal interventions actually alter distinct mechanisms.

Newman et al. (2011) found no difference between CBT with and without interpersonal interventions on IPs for individuals with GAD. Hoffart et al. (2009) also found that CBT and interpersonal psychotherapy (IPT) had similar impacts on cognitive (e.g., estimated probability and costs of negative social events), behavioral (e.g., safety behaviors), and interpersonal (e.g., perceived acceptance by others) processes for individuals with social anxiety disorder (SAD). Other SAD research has shown that both cognitive therapy (CT) and IPT are superior to waitlist control, and equally effective for comorbid depression symptoms, but that CT is superior to IPT for SAD symptoms (Stangier et al., 2011). There is evidence that CBT for depression has more specific effects on cognitive mechanisms than IPT (Quilty et al., 2008), and our study found larger effect sizes of CBGT on negative thoughts and mood than IPs. However, contrary to expectations, one study found that CBT more effectively treated depressed individuals with dysfunctional interpersonal attachments than IPT (McBride et al., 2006). Few studies have directly compared the impact of CBT and IPT on interpersonal processes, but research is currently underway to better understand the mechanisms of change of these treatments (Lemmens et al., 2011). It is likely that complex reciprocal relationships exist between IPs, negative cognitions, and depression symptoms.

This study has several limitations. First, this is the first treatment study we are aware of to use this version of the IIP-32, which has been found to have a robust, invariant factor structure and good internal reliability (Barkham et al., 1996; McEvoy et al., *in press*). There are numerous versions of the IIP, most of which use a circumplex approach. We used the short version derived from factor analysis to maximize clinical utility. Although our findings converge with evidence from the circumplex framework, in terms of adverse impacts of a submissive interpersonal style, it is difficult to make direct comparisons to previous circumplex research so it is important that our findings are replicated. Second, our findings may not generalize to other treatments or principal disorders. Third, this study did not investigate relationships between IPs and interpersonal processes within treatment sessions. Future research



exploring the relationships between pre-existing IPs, therapeutic alliance, and group cohesion would be well placed to more specifically determine how IPs may interfere with interpersonal processes within group therapy. Fourth, it also must be acknowledged that our self-report measures may have been susceptible to mood congruent effects, such that higher levels of depression impacted on perceptions of IPs, thereby inflating the strength of these relationships. However, this cannot provide a complete explanation of our findings because the correlation between IPs and QoL remained significant when controlling for BDI-II scores, and the IIP-32 was associated with treatment dropout, whereas depression, anxiety, and QoL were not. Additionally, total IIP-32 score significantly reduced during treatment when controlling for change in CCLD and BDI-II change scores. If self-reported IPs were entirely explicable by mood state, these independent effects would not be expected. Nonetheless, future research using multi-method approaches to assessing IPs would be useful. Fifth, whilst the primary aims of this study were to identify IPs associated with attrition and completer outcomes, it is still important to note that post-treatment data were not available for a substantial minority of participants. To the degree that a systematic attrition bias was present, our completer findings may not be representative of the whole pre-treatment sample. Sixth, the correlational design precludes causal conclusions. RCTs comparing the impact of CBGT and IPT on cognitive, behavioral, interpersonal, and emotional factors over time would be better placed to identify reciprocal and independent causal relationships between purported mechanisms of change. Finally, our ability to make causal conclusions was limited by the lack of a control group, because we could not isolate the influence of the intervention itself from time (e.g., regression to the mean, spontaneous remission) and other non-specific effects (e.g., attention from the clinician).

This study found that CBGT for major depression was associated with improvements in depression symptoms and negative cognitions, as well as broad improvements in IPs and QoL. IPs may be particularly important in terms of treatment attrition, with those who find it difficult to support others or disclose personal information being at highest risk of dropout. Individuals who find it hard to be assertive and who tend to subjugate their own needs may find it most difficult to actively participate in group therapy, thus achieving less symptom relief. Clinicians may need to more actively manage patients who endorse these IPs before or during group therapy, or offer them individual treatment, if outcomes are to be optimized.

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#### Conflict of interest

None to declare.

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