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Sudden Gains: How Important Are They During Exposure and Response Prevention for Obsessive-Compulsive Disorder?

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Symptom reduction over the course of cognitive-behavioral therapy is not always distributed evenly across sessions. Some individuals experience a sudden gain, defined as a large, rapid, and stable decrease in symptoms during treatment. Although research documents a link between sudden gains and treatment for depression and anxiety, findings in the context of obsessive-compulsive disorder (OCD) treatment are mixed. The present study investigated the relationship between sudden gains and treatment outcome in 44 adults with OCD and addressed limitations of previous studies by measuring OCD symptoms dimensionally and comparing individuals who experience sudden gains to those who experience gradual gains of similar magnitude. Sudden gains were observed among 27% of participants, with highest rates among individuals with primary contamination symptoms. Participants who experienced a sudden gain had greater OCD symptom reductions at posttreatment (but not at follow-up), and this difference did not persist after controlling for gain magnitude. Thus, the importance of sudden gains during OCD treatment may be limited. Findings are discussed in light of inhibitory learning models of cognitive-behavioral therapy.

Keywords: sudden gains; obsessive-compulsive disorder; cognitive-behavioral therapy; exposure and response prevention

Obsessive -compulsive disorder (OCD) is among the most common and functionally impairing psychological conditions, with a lifetime prevalence rate of up to 3% in the general population (Adam, Meinlschmidt, Gloster, & Lieb, 2012; Ruscio, Stein, Chiu, & Kessler, 2010). Evidence from numerous studies supports the efficacy and effectiveness of cognitive-behavioral therapy (CBT) using exposure and response prevention (ERP), which involves repeated confrontation with internal and external obsessional cues (e.g., a dirty trash bin) without the use of compulsive rituals (e.g., excessive hand washing; Olatunji, Davis, Powers, & Smits, 2013). Although ERP has demonstrated superiority to other psychological interventions (Lindsay, Crino, & Andrews, 1997) and medication (Foa et al., 2005), about 50% of individuals continue to experience significant impairment after a full course of treatment (Loerinc et al., 2015; Springer, Levy, & Tolin, 2018). Accordingly, it is important to identify factors that predict treatment outcomes.

One potential prognostic indicator is a sudden gain, defined as a large, rapid, and stable decrease

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in symptoms during treatment. Tang and DeRubeis (1999), studying CBT for depression, were the first to report a relationship between sudden gains and treatment response. They quantified a sudden gain as a symptom reduction between two consecutive therapy sessions that is large (a) in absolute terms, (b) compared to severity before the gain, and (c) compared to fluctuations before and after the gain. They identified sudden gains in more than 50% of their patients and found that sudden gains were associated with greater overall improvements in depressive symptoms. Using similar criteria, researchers have replicated these findings in the treatment of depression (e.g. Hardy et al., 2005), social anxiety disorder (Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006), generalized anxiety disorder (Present et al., 2008), panic disorder (Clerkin, Teachman, & Smith-Janik, 2008), and posttraumatic stress disorder (Doane, Feeny, & Zoellner, 2010). A recent meta-analysis found a medium effect (Hedges's $g = .62$, SE = .09) for sudden gains as predictors of outcome for anxiety and depression (Aderka, Nickerson, Bøe, & Hofmann, 2012).

Only two studies to date, however, have examined sudden gains during the treatment of OCD. Combining data from two randomized controlled trials, Aderka and colleagues (2012) found sudden gains among 34.1% of 91 individuals receiving either cognitive therapy, exposure therapy, or either treatment in combination with fluvoxamine. Participants who experienced sudden gains reported fewer OCD symptoms posttreatment relative to those who did not, and this difference was maintained at 12-month follow-up. Seeking to replicate and extend these findings, Collins and Coles (2017) examined 27 patients undergoing ERP for OCD and found that 52% experienced a sudden gain, yet sudden gains did not predict OCD symptom reduction at posttreatment.

One important limitation of both OCD sudden gain studies is that they assessed OCD symptoms *globally*. Research indicates, however, that the substantial heterogeneity of OCD distills into four symptom dimensions (i.e., contamination, symmetry, responsibility for harm, and unacceptable thoughts; for a review see McKay et al., 2004). Further, these dimensions show differential responses to ERP: individuals with primarily contamination obsessions and washing/cleaning compulsions tend to fare better than those with other presentations of OCD, whereas those with primary obsessions concerning unacceptable/taboo topics (e.g., sex, immorality, blasphemy) are more likely to have attenuated outcomes (Kelley, Storch, Merlo, & Geffken, 2008). Accordingly, assessing sudden gains across various presentations of OCD may provide more precise and clinically useful information relative to relying on global severity measures.

Another key shortcoming of previous studies is that moderation analyses do not compare individuals who gain suddenly to those who gain gradually. That is, in the two studies on OCD treatment, outcomes for patients who experienced sudden gains were compared to those of *all* other participants in the same sample who did not undergo a sudden gain. Yet as Greenfield, Gunthert, and Haaga (2011) have pointed out, such a comparison group may include individuals who did not improve, or whose symptoms worsened. Therefore, findings suggesting a relationship between sudden gains and treatment outcome may be tautological, reflecting merely that *any* gains, rather than *sudden* gains, predict better outcome. Greenfield et al. suggest that it is more precise to compare outcomes for patients who experience sudden gains to those who experience similarly sizable, but *gradual*, improvement during treatment. In the current study, we built on existing research by taking Greenfield and colleagues' approach to evaluating sudden gains as outcome moderators in ERP for OCD.

Beyond symptom severity, sudden gains may also be associated with processes through which people have been shown to improve during OCD treatment. For example, depression treatment studies have found associations between cognitive changes and sudden gains (e.g., Tang & DeRubeis, 1999). However, studies on treatment for anxiety disorders have reported mixed results (e.g., Hofmann et al., 2006; Kelly, Roberts, & Ciesla, 2005; Norton, Klenck, & Barrera, 2010), and neither of the previous OCD sudden gains studies examined whether sudden gains with ERP were associated with changes in cognitive-behavioral processes. Two notable psychological constructs related to OCD are obsessive beliefs and psychological flexibility. Common obsessive beliefs include (a) exaggerated estimates of threat and responsibility, (b) intolerance of uncertainty and imperfection, and (c) appraisals of normally occurring intrusive thoughts as significant, threatening, and needing to be controlled (Tolin, Woods, & Abramowitz, 2003). As ERP is thought to promote learned safety (i.e., fear extinction) by violating threat-based expectancies (e.g., Craske et al., 2008), and previous research indicates that obsessive beliefs are modified as a result of ERP (e.g., Whittal, Thordarson, & McLean, 2005), examining whether sudden gains during ERP are associated with greater changes in obsessive beliefs may help shed light on cognitive change during ERP.

Relatedly, increasing psychological flexibility is an important aim of ERP (e.g., Craske et al., 2008;

Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014; Jacoby & Abramowitz, 2016; Twohig et al., 2018). In this way, ERP overlaps with acceptance-based therapies that aim to increase one's willingness to experience unwanted private experiences such as obsessions, anxiety, and fear (e.g., Twohig et al., 2015). Psychological *in*flexibility refers to the tendency to resist or avoid unpleasant thoughts and emotions and is thought to motivate maladaptive behaviors (e.g., avoidance, compulsive rituals) that maintain obsessional distress (see Chawla & Ostafin, 2007). Given ERP's focus on breaking avoidance cycles and preventing the use of compulsive rituals to promote symptom improvement, sudden gains during ERP for OCD may also be predictive of increased psychological flexibility.

The present study was designed to contribute to the burgeoning literature on sudden gains during ERP for OCD. Specifically, we (a) examined sudden gains in different presentations of OCD, (b) conservatively controlled for gain magnitude in our outcome moderator analyses, and (c) examined sudden gains as predictors of change in conceptually relevant constructs (i.e., obsessive beliefs and psychological flexibility). We used data from a two-site treatment study in which 44 individuals with OCD completed a full course of ERP (Twohig et al., 2018). On the basis of previous research, we predicted that (a) approximately 50% of individuals would experience a sudden gain, (b) the rates of sudden gains would be highest among participants with primary OCD symptoms related to contamination and lowest among those with primary OCD symptoms related to unacceptable thoughts, (c) individuals who experience a sudden gain would report significantly greater OCD symptom reduction at posttreatment and 6-month follow-up than those without a sudden gain, even when controlling for the gain magnitude, and (d) individuals who experience a sudden gain would report significantly greater changes in obsessive beliefs and psychological flexibility at posttreatment and follow-up than those without a gain. We also explored whether demographic characteristics (i.e., gender, age, current medication treatment), OCD symptom severity, baseline obsessive beliefs, psychological flexibility, and/or treatment condition predicted sudden gains.

Method

PARTICIPANTS

Participants were 44 adults (28 female) between the ages of 18 and 56 (M = 27.19, SD = 8.22) who completed a 16-session trial of manualized ERP treatment for OCD. Fifty-eight adults enrolled in the parent trial, 3 dropped out before beginning treatment, 9 dropped out before the posttreatment session, and 2 were missing session-by-session data. The sample was 80% (n = 35) White, 7% (n = 3) Hispanic, and 5% (n = 2) African American. All participants received a DSM-IV diagnosis of OCD according to the Mini International Neuropsychiatric Interview 5.0 (MINI 5.0), and primary OCD symptoms were determined using the Dimensional Obsessive-Compulsive Scale (DOCS). Each participant's primary OCD symptom dimension was determined by identifying the DOCS subscale with the highest pretreatment score: contamination (n = 12), responsibility for harm (n = 15), unacceptable thoughts (n = 21), and symmetry (n = 5). Nine participants were assigned two primary OCD symptoms because two DOCS subscales had equally high scores. Approximately half of the sample had at least one secondary DSM-IV diagnoses (52.3%; n = 23), and the most common were mood (n = 14) and anxiety disorders (n = 10). Exactly half of the sample reported taking psychotropic medication during treatment, which was admissible in the parent trial as long as doses remained stable from one month before starting treatment through the end of the study. Of the 22 participants using medication, the majority reported taking selective serotonin reuptake inhibitors (n = 13).

MEASURES

Dimensional Obsessive-Compulsive Scale (DOCS; Abramowitz et al., 2010)

The 20-item self-report DOCS assesses the severity of four empirically supported OCD symptom dimensions: contamination, responsibility for harm, symmetry/ordering, and unacceptable thoughts. Each dimension has its own subscale containing five items (rated 0 to 4, anchors change) that assess the following five severity parameters: time occupied by obsessions and rituals, avoidance behavior, associated distress, functional interference, and difficulty disregarding the obsessions and refraining from the compulsions. DOCS subscale scores range from 0 to 20, have shown excellent reliability and sensitivity to the effects of treatment in clinical samples, and demonstrate good convergent validity with other measures of OCD symptoms (Abramowitz et al., 2010). Participants completed the DOCS at pretreatment, posttreatment, 6-month follow-up, and at the beginning of each treatment session.

Given that the DOCS includes subscales that may not pertain to every participant's symptom presentation, total scores on this measure could suppress its sensitivity to treatment (Abramowitz et al., 2010). We therefore used the procedures described in

Abramowitz et al. (2010) for determining each participant's most relevant DOCS subscale, called the "DOCS main" symptom score (as previously described), and computing DOCS main symptom scores at each session. In cases where two or more subscales tied for the highest score at pretreatment (i.e., the patient had multiple "main" symptoms), subsequent DOCS main scores were computed as the mean of the corresponding subscale scores.

Acceptance and Action Questionnaire-II (AAQ-II; Bond et al., 2011)

The AAQ-II is a 10-item scale that assesses psychological flexibility. Participants rate their agreement with each of the statements (e.g., "I'm afraid of my feelings") on a 1 (*never true*) to 7 (*always true*) scale, such that higher scores indicate lower psychological flexibility. The AAQ-II has demonstrated good psychometric properties and good convergent, discriminant, and incremental validity (Bond et al., 2011; Fledderus, Oude Voshaar, Ten Klooster, & Bohlmeijer, 2012). Participants completed the AAQ at pretreatment, posttreatment, and follow-up.

Obsessive Beliefs Questionnaire (OBQ; Obsessive Compulsive Cognitions Working Group, 2005)

The OBQ is a 44-item self-report instrument that measures dysfunctional (i.e., obsessive) beliefs hypothesized to underlie OCD symptoms. It contains three subscales: (a) threat overestimation and responsibility (OBQ-RT; 16 items), (b) perfectionism and need for certainty (OBQ-PC; 16 items), and (c) importance and control of thoughts (OBQ-ICT; 12 items). Participants rate items on a Likert scale ranging from 1 (*disagree very much*) to 7 (*agree very much*). The instrument has demonstrated good validity, internal consistency, and test-retest reliability (Obsessive Compulsive Cognitions Working Group, 2005). Participants completed the OBQ at pretreatment, posttreatment, and follow-up.

PROCEDURE

Treatment

Data for this investigation were drawn from an OCD treatment study examining the effects of adding components of Acceptance and Commitment Therapy (ACT) to ERP (Twohig et al., 2018). Participants completed 16 twice-weekly, 2-hour sessions of individualized treatment for OCD at one of two sites: University of North Carolina at Chapel Hill (UNC; n = 21) and Utah State University (USU; n = 23) and were randomly assigned (at each site) to receive either traditional ERP or ERP + ACT. The number of in-session therapist-supervised exposure trials was equal across both conditions. Treatment was delivered by doctoral-level therapists and advanced clinical psychology doctoral students who received training in the treatment protocols, adhered to detailed treatment manuals, and received supervision from doctoral-level clinical psychologists with expertise in the treatment of OCD.

Sessions 1–3 in both treatment conditions included information gathering, psychoeducation, and treatment planning. Sessions 4–16 involved exposure exercises with instructions to practice exposures out-of-session ("homework") and refrain from rituals. Session 16 also addressed treatment termination and relapse prevention. The centerpiece of both treatment conditions was ERP, and in the ACT + ERP condition, metaphors drawn from ACT were included before, during, and after each exposure trial to reinforce the concepts central to this approach (e.g., defusion from obsessional thoughts, acceptance of unwanted thoughts and anxiety, and the importance of ERP to one's values). Analyses indicated no between-group differences in outcome for OCD symptoms, obsessive beliefs, or psychological flexibility (Twohig et al., 2018); therefore, all treatment completers were included in the present analyses regardless of study condition.

Definition of Sudden Gains

We used the criteria proposed by Tang and DeRubeis (1999) to identify sudden gains in our sample. These criteria require that the change between sessions is large in absolute terms, compared to severity before the gain, and compared to fluctuations before and after the gain, and were operationalized as follows:

Criterion A. As in previous research (e.g., Collins & Coles, 2017), we used the reliable change index (Jacobson & Truax, 1991) to identify gains that were large in absolute terms. We divided the difference scores between consecutive sessions by the standard error of the difference and compared this value to 1.96. Values larger than 1.96 would be unlikely to occur due to chance alone, and thus satisfy Criterion A.

Criterion B. As defined by Tang and DeRubeis (1999), the difference scores between consecutive sessions must represent at least 25% of the pre-gain score to satisfy Criterion B.

Criterion C. To determine if a gain is large relative to fluctuations before and after the gain, we conducted independent sample t-tests to compare the three before-gain and after-gain DOCS main scores. Replicating the sudden gains calculations of Hardy and colleagues, we used cutoffs to determine whether difference scores constituted sudden gains.

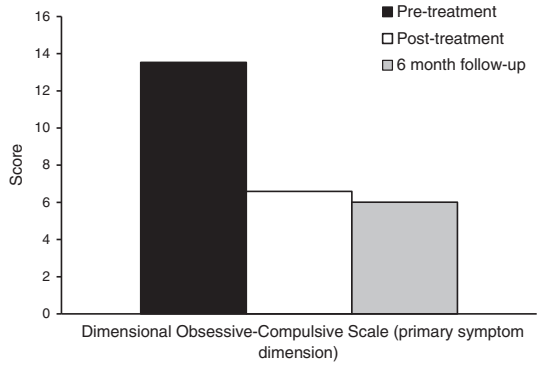

FIGURE 1 Pretreatment, posttreatment, and follow-up mean scores for the full sample (N = 44).

Gains between two consecutive sessions met criterion C if $t \geq 2.50$ ($t \geq 3.00$ if only two pre-gain or after-gain scores were available). If only one pre-gain score was available, sudden gains were not identified. Therefore, gains occurring immediately after Session 1 were not included in analyses.

Results

TREATMENT RESPONSE AND FREQUENCY OF SUDDEN GAINS

As shown in Figure 1, treatment was associated with a substantial overall reduction in OCD symptoms from pre- to posttreatment. On average, scores on the DOCS main symptom scale decreased by 51%, which is consistent with rates of improvement observed in other studies of ERP for OCD (Eddy, Dutra, Bradley, & Westen, 2004). Moreover, improvements were maintained at 6-month follow-up.

Using the criteria previously defined, we identified 12 participants (27.3% of the sample) who experienced a sudden gain during treatment. The average sudden gain magnitude was 5.58 points on the DOCS main (SD = 2.12). The average total symptom reduction for the full sample from pre- to posttreatment was 7.02 points on the DOCS main (SD = 3.76), and the average total symptom reduction for participants who experienced a sudden gain was 9.25 points on the DOCS main (SD = 3.91). Thus, for participants who experienced sudden gains, such gains accounted for an average of 60.3% of total symptom reduction. First gains occurred after Session 3 (n = 3), after Session 4 (n = 2), after Session 6 (n = 3), after Session 9 (n = 1), after Session 10 (n = 2), and after Session 12 (n = 1).

SUDDEN GAINS BY OCD SYMPTOM DIMENSION

Of the 12 participants whose primary OCD symptoms concerned contamination, 6 (50%) experienced a sudden gain. Of the 21 participants with primary OCD symptoms related to unacceptable thoughts, 5 (23.8%) experienced a sudden gain. Of the 15 participants with primary OCD symptoms related to responsibility for harm, 2 (13.3%) experienced a sudden gain. Finally, of the 5 participants with primary OCD symptoms related to symmetry, 1 (20%) experienced a sudden gain. Although it would be desirable to conduct a chi-square test to statistically compare the frequencies of sudden gains across OCD symptom presentations, the data are nonindependent as some participants were part of multiple DOCS main groups. In addition, multiple cell counts failed to meet the minimum required 5 observations, precluding chi-square analyses.

SUDDEN GAINS AS A MODERATOR OF TREATMENT OUTCOME

Full Sample

Pretreatment, posttreatment, and follow-up DOCS main scores for the full sample by sudden gain status are presented in Table 1. There were no significant pretreatment differences in OCD symptoms between participants who experienced sudden gains and those who did not ($p > .05$).

To test our hypotheses that sudden gains would predict superior outcomes, we conducted a series of 2 (time: pre, post/follow-up) × 2 (sudden gain: yes, no) ANOVAs with DOCS main as the dependent variable. Pairwise deletion was implemented to account for missing data at follow-up. We found a

Table 1
Pretreatment, Posttreatment, and Follow-up Mean Scores (standard deviations) for Participants With and Without a Sudden Gain (N = 44)

	Sudden Gain			No Sudden Gain		
	Pretreatment	Posttreatment	Follow-up	Pretreatment	Posttreatment	Follow-up
DOCS main	14.42 (2.84)	5.17 (3.32)	5.25 (3.81)	13.19 (2.66)	7.13 (3.09)	6.28 (3.79)
AAQ-II	30.25 (9.19)	21.08 (8.41)	19.00 (8.56)	29.88 (8.07)	25.13 (8.74)	23.14 (8.48)
OBQ	196.58 (48.16)	121.67 (36.51)	116.86 (28.49)	198.28 (48.20)	152.31 (51.76)	136.37 (53.53)

Note. DOCS main = Dimensional Obsessive-Compulsive Scale (primary symptom dimension), AAQ-II = Acceptance and Action Questionnaire-II, OBQ = Obsessive Beliefs Questionnaire

significant main effect of time indicating overall improvement in DOCS main symptoms at both posttreatment, $F(1, 41) = 165.20$, $p < .001$, $\eta_p^2 = .80$, and at follow-up, $F(1, 37) = 136.53$, $p < .001$, $\eta_p^2 = .79$. There was also a significant time by sudden gain interaction effect from pre- to posttreatment, such that participants who experienced a sudden gain had greater average symptom reduction than did those without a sudden gain, $F(1, 41) = 6.64$, $p = .014$, $\eta_p^2 = .14$. This interaction effect, however, was not significant from pretreatment to follow-up, $F(1, 36) = 2.84$, $p = .10$, $\eta_p^2 = .07$. These findings indicate that sudden gains are associated with greater symptom reduction at posttreatment but not at follow-up.

Matched Pairs

To control for the gain magnitude during treatment, as suggested by Greenfield and colleagues (2011), each of the 12 participants who experienced a sudden gain was matched with a participant from the remaining pool of 32 patients who did not experience a sudden gain, but who (a) had a pretreatment DOCS main score within one point, (b) achieved a comparable gain magnitude in DOCS main scores between the first treatment session and at the post-gain session (within three points), and (c) had the same main OCD symptom. Although matches on the first two criteria were available for all cases, only 4 cases could also be matched on the third criterion. Pretreatment, posttreatment, and follow-up scores for the sample of matched pairs are presented in Table 2.

A series of 2 (time: pre, post/follow-up) × 2 (sudden gain: yes, no) ANOVAs with DOCS main as the dependent variable were computed to examine whether sudden gains moderated outcome using this matched comparison group. These analyses indicated no significant time by sudden gain status interactions on the DOCS main scores at pre- and posttreatment, $F(1, 22) = .762$, $p = .39$, $\eta_p^2 = .03$, or at follow-up, $F(1, 20) = 1.48$, $p = .24$, $\eta_p^2 = .07$. This suggests that sudden gains were not significantly associated with outcome when controlling for gain magnitude.

Table 2
Pretreatment, Posttreatment, and Follow-up DOCS Main Scores (Standard Deviations) for the Sample of Matched Pairs ($n = 24$)

	Sudden Gain		No Sudden Gain	
Assessment	*M*	*SD*	*M*	*SD*
Pretreatment	14.42	2.84	13.83	3.24
Posttreatment	5.17	3.32	5.96	2.54
Follow-up	5.25	3.81	6.88	4.10

Note. DOCS main = Dimensional Obsessive-Compulsive Scale (primary symptom dimension)

Sudden Gains Predicting Changes in Obsessive Beliefs and Psychological Flexibility

Pretreatment, posttreatment, and follow-up scores on the OBQ and AAQ-II for the full sample by sudden gain status are presented in Table 1. A series of 2 (time: pre, post/follow-up) × 2 (sudden gain: yes, no) ANOVAs with DOCS main as the dependent variable were computed to examine whether sudden gains were associated with change on these measures. Pairwise deletion was implemented to account for missing data at follow-up. These analyses indicated no significant time by sudden gain status interactions for the OBQ total scores from pre- to posttreatment, $F(1, 39) = 2.43$, $p = .13$, $\eta_p^2 = .13$, or from pretreatment to follow-up, $F(1, 30) = .004$, $p = .95$, $\eta_p^2 < .001$. There were also no significant time by sudden gain status interactions for the OBQ-RT scores from pre- to posttreatment, $F(1, 39) = 3.40$, $p = .07$, $\eta_p^2 = .08$, or from pretreatment to follow-up, $F(1, 29) = 1.73$, $p = .20$, $\eta_p^2 = .06$. Similarly, there were no significant time by sudden gain status interactions for the OBQ-PC scores from pre- to posttreatment, $F(1, 38) = .16$, $p = .70$, $\eta_p^2 = .004$, or from pretreatment to follow-up, $F(1, 29) = .34$, $p = .56$, $\eta_p^2 = .01$. Finally, there were no significant time by sudden gain status interactions for the OBQ-ICT scores from pre- to posttreatment, $F(1, 39) = .74$, $p = .39$, $\eta_p^2 = .02$, or from pretreatment to follow-up, $F(1, 30) = 2.94$, $p = .10$, $\eta_p^2 = .09$.

There were no interactions for the AAQ-II from pre- and posttreatment, $F(1, 42) = 1.96$, $p = .17$, $\eta_p^2 = .04$, or from pretreatment to follow up, $F(1, 37) = .67$, $p = .42$, $\eta_p^2 = .02$. Identical analyses were performed in the sample of matched pairs and no significant time by sudden gain status interactions were found. This indicates that change in OBQ and AAQ-II scores did not differ by sudden gain status.

PREDICTORS OF SUDDEN GAINS

Logistic regression analyses were conducted to examine the relationship between continuous independent variables and sudden gains, and Pearson chi-square analyses were conducted to examine the relationship between binary independent variables and sudden gains. DOCS main scores, obsessive beliefs, psychological flexibility, age, gender, and concurrent medication at baseline were not significantly associated with sudden gains (all $ps > .05$). However, 10 individuals who experienced a sudden gain were in the ERP condition, whereas two were in the ACT+ERP condition. A chi-square analysis

revealed that the difference in these frequencies was larger than what would be predicted, χ^2 (1) = 6.38, p = .01 (OR = 7.31).

Discussion

Numerous studies document sudden gains during the psychological treatment of depression and anxiety-related disorders, and some have found that such gains are associated with enhanced treatment outcome. To date, however, only two studies have examined this phenomenon in the treatment of OCD, and the findings are conflicting. Thus, the purpose of the present study was to shed additional light on sudden gains during ERP for OCD and build on the conceptual and methodological approaches used in previous research on this topic. Specifically, we examined the frequency of sudden gains, their relationship to OCD symptom presentation, and their relationship to treatment outcome at both posttreatment and follow-up after controlling for gain magnitude. Lastly, we examined whether sudden gains were related to changes in psychological constructs related to OCD.

Twenty-seven percent (n = 12) of the patients in our sample achieved sudden gains during ERP. This provided only partial support for our first hypothesis, as we had expected a greater frequency of sudden gains among individuals receiving CBT for OCD based on previous research (i.e., 34.1% in Aderka, Anholt, et al., 2012; Aderka, Nickerson, et al., 2012; 52% in Collins & Coles, 2017). In concert with our second prediction regarding OCD symptom presentation, participants with obsessions and compulsions primarily related to contamination achieved the highest rate of sudden gains (50%) in our sample. Yet contrary to our prediction, the lowest rate was found among those whose primary obsessions concerned responsibility for harm and mistakes (13.3%), not among individuals with primary unacceptable thoughts (23.8%) as we had expected. Importantly, the small number of individuals who experienced a sudden gain overall precludes definitive conclusions regarding dimensional differences. Moreover, only 27% of our sample had primary contamination symptoms, which is lower than the average rate of such symptoms in OCD treatment studies (e.g., 48% in a meta-analytic review by Ball et al., 1996). Thus, although Aderka et al. and Collins and Coles did not report the symptom presentations of their participants, the relatively low frequency of contamination symptoms (the presentation of OCD most associated with sudden gains) in our sample might account for the lower overall rates of sudden gains observed in the present study.

Contamination-related obsessions and compulsions are generally responsive to ERP (Abramowitz, Franklin, Schwartz, & Furr, 2003), yet this presentation of OCD might be particularly prone to sudden gains for two reasons. First, the ERP procedures for targeting fear-based expectancies related to contamination (e.g., "I will get sick if I touch a toilet") are generally straightforward for clinicians and patients to implement (e.g., touching toilets and refraining from hand washing). Second, in many instances, ERP is able to directly and immediately violate contamination-related fear expectancies, leading to swift fear extinction. That is, the patient can plainly observe that (a) feared consequences (e.g., contracting illness) are unlikely, and (b) feelings of fear are temporary and safe. Given that 50% of individuals with primary contamination symptoms did not achieve a sudden gain, however, it might be interesting to look more closely at the specific content of these individuals' obsessions. Perhaps their particular presentation of contamination fears and rituals complicated either the implementation of exposure or the ability to immediately violate fear expectations. For example, some patients have contamination obsessions in which feared consequences would not occur until some point in the more distant future (e.g., "I will get cancer in 30 years because of exposure to asbestos"), making it difficult to rapidly violate fear expectancies (for a discussion of how to implement ERP for such obsessions, see Abramowitz & Arch, 2014). Future research, however, is needed to establish whether sudden gains are consistently most common among OCD symptoms related to contamination.

We expected to find the lowest rates of sudden gains among participants whose obsessions primarily concerned unacceptable/taboo topics (e.g., sex, immorality, blasphemy), but instead found the lowest rate of sudden gains among responsibility for harm obsessions. In accord with our discussion above, both unacceptable thoughts and responsibility for harm obsessions can be challenging to target with ERP for a few reasons. First, obsessional fears (i.e., threat-based expectancies) typical of these presentations of OCD often relate to repugnant ideas and "unknowable" or long-term negative consequences (e.g., "I will go to hell when I die"), which cannot easily (if at all) be violated or disconfirmed with ERP. Thus, treatment of such symptoms must focus on violating expectations related to uncertainty (Abramowitz & Jacoby, 2015). In addition to challenging some clinicians who have less expertise using ERP for OCD, this process may simply proceed more gradually than violating threat overestimations related to more immediate, tangible outcomes. However, it is important to note that the limited number of participants in

some cells, as well as individuals endorsing symptoms associated with more than one symptom dimension, necessitate caution when drawing conclusions about differences in sudden gains across symptom dimensions.

Consistent with our third hypothesis and the findings of Aderka and colleagues (2012), individuals who experienced a sudden gain had larger pre- to posttreatment reductions in their primary OCD symptoms than those who did not. However, sudden gains were not predictive of outcome at follow-up, suggesting that the relevance of sudden gains to symptom reduction lessens over time after the completion of a course of ERP. Moreover, when participants who experienced a sudden gain were matched with those who experienced a gradual gain of similar magnitude, sudden gains were not associated with treatment outcome at posttreatment or follow-up. Thus, it was not the suddenness of the gain that was associated with improved outcome, but rather the magnitude of the gain.

It is possible that the reduced sample size used for our matched pairs analyses limited the power to detect a statistically significant effect, although the small effect sizes we found (η_p^2 = .03 at posttreatment and η_p^2 = .05 at follow-up) suggest that this was unlikely to be the case. It is also notable that our findings differ from those of Greenfield et al. (2011), who found significant differences between "sudden gainers" and "gradual gainers" in a diagnostically heterogeneous outpatient sample. However, important methodological differences between our study and theirs may account for this difference. Specifically, Greenfield et al. examined a diagnostically heterogeneous sample of outpatients treated in a psychotherapy training clinic, whereas all of our participants received an OCD diagnosis and were treated as part of a randomized controlled trial. It is possible that sudden gains are less relevant to outcome during manualized OCD treatment relative to the treatment of other psychological conditions in a general outpatient training clinic setting.

In contrast with our fourth hypothesis, there were no significant relationships between the experience of a sudden gain and changes in obsessive beliefs or psychological flexibility during treatment. Thus, although associated with overall decreases in obsessions and compulsions at posttreatment, rapid symptom reduction during ERP was not associated with changes in psychological constructs that have been shown to maintain these symptoms. This finding further undermines the relevance of sudden gains to OCD, given prior research suggesting that obsessive beliefs and psychological flexibility change over the course of treatment and are associated with symptom reduction (e.g., Twohig et al., 2018; Twohig, Hayes, & Masuda, 2006; Whittal et al., 2005). Our results are surprising because previous studies have shown relationships between cognitive-behavioral constructs and sudden gains (e.g., Tang & DeRubeis, 1999; Whittal et al., 2005). These inconsistencies may be explained by the specific constructs of interest in this study, which differ from the cognitive factors that were associated with sudden gains in previous work. It is important to note that these findings do not rule out the possibility that sudden gains were associated with psychological changes during and between individual sessions, or processes not assessed in the current study. Future research should examine changes in process measures preceding and co-occurring with sudden gains.

Our analyses of predictors of sudden gains revealed that patients who received ERP were more likely than those receiving ACT+ERP to experience a sudden gain. One explanation for this is the emphasis on habituation and anxiety reduction in traditional ERP. Specifically, relative to the ACT approach that encourages acceptance of unwanted thoughts and anxiety, traditional ERP may lead to more rapid decreases in reported symptoms. Moreover, our self-report measure of OCD symptoms, the DOCS, may be more sensitive to therapeutic change as promoted by the traditional ERP approach (i.e., reduction in obsessions) relative to the ACT approach.

The present findings should be considered in light of a number of study limitations. Notably, our data were collected in a randomized controlled trial and several design decisions necessarily prioritized internal over external validity, as is typical of such investigations. For example, individuals who had severe co-occurring disorders or previous experience with CBT for OCD were excluded from participating, therapists used detailed treatment manuals, and every case was supervised by a licensed clinical psychologist. These conditions likely do not reflect the level of diagnostic heterogeneity or treatment delivery in typical service settings. Finally, as noted, our sample size may have limited our power to detect effects, particularly in our reduced sample of matched pairs. As previously noted, our empirically supported decision to examine OCD symptoms dimensionally led to a small number of individuals in each primary symptom group, so results related to symptom dimensions must be interpreted with caution. Overall, our sample size tempers the conclusions that can be drawn, and replication in larger samples is warranted.

Despite these limitations, the present study adds to the existing literature on sudden gains during OCD treatment. Although sudden gains have been repeatedly linked to treatment outcome in depression and anxiety treatment, the present study casts doubt on their relevance to long-term outcome in OCD treatment. We agree with the sentiment expressed by Collins and Coles (2017) that the relationship between sudden gains and outcome during psychological treatment is an atheoretical investigation of an observed clinical phenomenon. Future research that empirically investigates theory-driven relationships may lead to more precise identification of treatment mechanisms and ultimately contribute to improving outcomes for individuals with OCD.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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