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Intraindividual Variability in Symptoms Consistently Predicts Sudden Gains: An Examination of Three Independent Datasets

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Objective: Sudden gains are robust predictors of outcome in psychotherapy. However, previous attempts at predicting sudden gains have yielded inconclusive findings. The aim of the present study was to examine a novel, transdiagnostic, transtherapeutic predictor of sudden gains that would replicate in different settings and populations. Specifically, we examined intraindividual variability in symptoms. ***Method:*** We examined data from a randomized controlled trial (RCT) of prolonged exposure therapy for posttraumatic stress disorder (PTSD) in children and adolescents ($n = 63$), an RCT of cognitive and behavioral therapies for obsessive–compulsive disorder (OCD) in adults ($n = 91$), and psychodynamic therapy delivered under routine clinical conditions in a naturalistic setting for diverse disorders ($n = 106$). In all 3 data sets, we examined whether a measure of variability in symptoms occurring during the first sessions could predict sudden gains. ***Results:*** Variability in symptoms was found to be independent of total change during treatment. Variability in symptoms significantly predicted sudden gains in all 3 data sets and correctly classified 81.0%, 69.2%, and 76.9% of individuals to sudden gain or nonsudden gain status, respectively. ***Conclusions:*** The present study represents the first examination of variability in symptoms as a predictor of sudden gains. Findings indicated that sudden gains are significantly predicted by intraindividual variability in symptoms, in diverse settings, contexts, and populations. Advantages of this predictor, as well as clinical and research implications are discussed.

What is the public health significance of this article?
Previous predictors of sudden gains in psychotherapy have been inconsistent. The present study indicates that variability in symptoms may represent a promising transdiagnostic and transtherapeutic predictor of sudden gains that cuts across contexts and populations.

Keywords: sudden gains, symptom variability, transdiagnostic, transtherapeutic, processes of change

Sudden gains are rapid reductions in symptoms that occur between consecutive treatment sessions (Tang & DeRubeis, 1999). First defined by Tang and DeRubeis (1999), sudden gains occur when a reduction in symptoms meets three criteria: the reduction is (1) large in absolute terms; (2) represents a 25% drop in symptoms; and (3) is stable (i.e., the symptom levels in the three sessions following the gain are significantly lower than the symptom levels preceding the gain). Sudden gains were initially investigated in cognitive therapy for depression among adults, but have since been found in a range of treatments, disorders, and across the life span (see Aderka, Nickerson, Bøe, & Hofmann, 2012 for a review). Individuals who experience sudden gains during treatment have been consistently found to have better posttreatment outcomes compared with individuals who do not experience sudden gains (Aderka, Nickerson, et al., 2012). Moreover, even when compared with individuals without sudden gains who significantly improve during treatment, individuals with sudden gains have better outcomes (e.g., Aderka, Appelbaum-Namdar, Shafran, & Gilboa-Schechtman, 2011; Greenfield, Gunthert, & Haaga, 2011). Longer term outcomes of individuals with sudden gains are less robust, resulting in smaller but significant average effect sizes

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(Aderka, Nickerson, et al., 2012; although see Vittengl, Clark, & Jarrett, 2005 for a study in which individuals with sudden gains had poorer long-term outcomes).

Whereas sudden gains' predictive utility and ubiquity have been established, findings regarding the processes leading to sudden gains have been mixed and little is known about the causes of sudden gains. For instance, Tang and DeRubeis (1999) found higher levels of cognitive changes in sessions preceding sudden gains compared with control sessions, and suggested that cognitive changes may lead to sudden gains. Cognitive changes were found to precede sudden gains in three additional studies (Cavallini & Spangler, 2013; Norton, Klenck, & Barrera, 2010; Tang, DeRubeis, Beberman, & Pham, 2005). However, many other studies failed to find support for cognitive changes preceding sudden gains (Andrusyna, Luborsky, Pham, & Tang, 2006; Bohn, Aderka, Schreiber, Stangier, & Hofmann, 2013; Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006; Hunnicutt-Ferguson, Hoxha, & Gollan, 2012; Vittengl et al., 2005). In addition, Tang, Luborsky, and Andrusyna (2002) hypothesized that sudden gains would be more frequent in cognitive therapy compared with supportive-expressive therapy as the former directly targets cognitions. However, they found that sudden gains had similar magnitudes, affected a similar percentage of patients, and occurred at about the same time in both treatments, failing to provide support for cognitive changes leading to sudden gains.

The mixed findings regarding cognitive changes leading to sudden gains have led some researchers to suggest that different treatment-specific mechanisms may cause sudden gains in different treatments (Andrusyna et al., 2006). For instance, cognitions may lead to sudden gains in cognitive therapy but other processes may lead to sudden gains in therapies based on different theoretical orientations. In support of this, a study of sudden gains in supportive-expressive therapy indicated that the accuracy of therapist interpretations (a treatment-specific mechanism of change in that treatment) predicted sudden gains (Andrusyna et al., 2006). However, other studies have failed to find evidence for treatment-specific processes. For instance, Hofmann, Schulz, Meuret, Moscovitch, and Suvak (2006) as well as Bohn, Aderka, Schreiber, Stangier, and Hofmann (2013) found no cognitive changes preceding sudden gains in cognitive behavior therapy (CBT) of social anxiety disorder. This is despite the fact that cognitive change has been established as a significant mediator of change in CBT for social anxiety disorder (Hofmann, 2004). Moreover, sudden gains have been identified even in the absence of treatment (Kelly, Roberts, & Bottonari, 2007; Krüger et al., 2014), and among individuals receiving pill placebo (Vittengl et al., 2005). Importantly, sudden gains in pill placebo were similar in characteristics (magnitude, timing, frequency) to those found in active psychological treatments (Vittengl et al., 2005). These findings raise questions regarding specific mechanisms as causes of sudden gains.

Some researchers have suggested that sudden gains are the result of processes or events that occur outside of weekly therapy sessions. For instance, Hardy et al. (2005) examined positive and negative life events and found no relationship with sudden gains. Similarly, other researchers failed to find support for life events leading to sudden gains (Kelly et al., 2007). Hofmann et al. (2006) also proposed that sudden gains may be a result of nontreatment-related processes. These researchers pointed to the statistical phenomenon of regression to the mean as a possible reason for sudden gains occurrence. Specifically, in their sample, pretreatment symptom levels significantly predicted sudden gains occurrence such that individuals with higher levels of pretreatment symptoms were more likely to subsequently experience sudden gains. Such an effect was also found in another study examining sudden gains in depression (Vittengl et al., 2005). However, most sudden gains studies find no relationship between pretreatment severity and sudden gain occurrence (e.g., Aderka, Anholt, et al., 2012; Aderka et al., 2011; Jun, Zoellner, & Feeny, 2013; Stiles et al., 2003). An additional nontreatment-related process that may be related to sudden gains is the Hawthorne effect (also sometimes referred to as the Trial Effect or Observer Effect; McCarney et al., 2007). This effect describes improvement in participants resulting from inclusion in research (as opposed to being treated naturalistically) and is commonly thought to be a result of the attention of researchers or measurement during research. However, studies on sudden gains in naturalistic settings have repeatedly identified sudden gains (e.g., Lutz et al., 2012) and this does not support the Hawthorne effect as a core process accounting for sudden gain occurrence. Thus, the evidence regarding extratherapeutic processes leading to sudden gains is currently not compelling.

Other factors have been proposed to lead to sudden gains but have failed to garner empirical support. These include reductions in hopelessness and dysfunctional attitudes, and increases in self-efficacy (Kelly et al., 2007), increases in self-esteem (Kelly, Roberts, & Ciesla, 2005), strengthening of the therapeutic relationship (Lutz et al., 2012), and low pretreatment levels of negative cognitions as well as adaptive pretreatment interpersonal functioning (Vittengl et al., 2005). Other factors such as feelings of hope have received mixed support such that one study found hope to predict sudden gains (Abel, Hayes, Henley, & Kuyken, 2016) whereas another did not (Kelly et al., 2007). There have been several additional predictors of sudden gains found only in a single study (e.g., therapist competence in case-conceptualization in Abel et al., 2016; clients' age in Jun et al., 2013). However, no consistent predictors of sudden gains have been found in the literature.

Using a different approach to investigate sudden gains, two studies have used Monte Carlo data simulation techniques (Thomas & Persons, 2013; Vittengl, Clark, Thase, & Jarrett, 2015). These studies simulated treatment data using gradual slopes of change and random error around these slopes. Vittengl, Clark, Thase, and Jarrett (2015) found that their simulated data sets contained similar proportions of sudden gains to those reported in the literature. Thus, when gradual reductions are combined with downward errors (i.e., errors in the direction of fewer symptoms), large fluctuations may occur and a sudden gain may be detected. Thomas and Persons (2013) arrived at similar conclusions following their own Monte Carlo simulation. In addition, other researchers have suggested that sudden gains may be the result of large fluctuations in symptoms (Stiles et al., 2003; Vittengl et al., 2005). For instance, Stiles et al. (2003) suggested that sudden gains are simply the largest and most extreme among many fluctuations in symptom levels during treatment. Thus, variability in symptoms may represent a promising predictor of sudden gains in treatment data sets.

The aim of the present study was to examine intraindividual (i.e., within-person) variability in symptoms as a predictor of sudden gains in treatment data (as opposed to computer-generated data). Specifically, we hypothesized that individuals with greater symptom variability would be more likely to experience sudden gains. To ensure that this predictor is robust and can be generalized we examined intraindividual variability in three independent datasets: A randomized controlled trial (RCT) of prolonged exposure therapy for posttraumatic stress disorder (PTSD) in children and adolescents, an RCT of cognitive and behavioral therapies for obsessive–compulsive disorder (OCD) in adults, and psychodynamic therapy delivered under routine clinical conditions in a naturalistic setting for diverse disorders. Such an examination in diverse treatments, samples, and disorders can help establish intraindividual variability in symptoms as a trans-diagnostic and trans-therapeutic predictor of sudden gains.

Method

Description of Datasets

The first dataset (Dataset I) examined in the present study included data from a RCT of prolonged exposure treatment for children and adolescents with PTSD (Gilboa-Schechtman et al., 2010). A complete account of the characteristics of sudden gains in that trial and their relationship to outcome has been published (Aderka et al., 2011). The second dataset (Dataset II) was obtained from two RCTs of cognitive, behavioral and pharmacological treatments for adults with OCD (van Balkom et al., 1998; van Oppen et al., 1995). A complete account of the sudden gains in these RCTs can be found in Aderka, Anholt et al. (2012). The third dataset (Dataset III) was derived from a study of psychodynamic treatment for adults in a naturalistic setting (Atzil-Slonim et al., 2018). As data on sudden gains from this sample have not been previously published, information on characteristics of sudden gains and their prediction of outcome appears in the results section in order to facilitate comparison with the sudden gains literature. For more information on datasets and overlap with previously published studies see Appendix.

Participants

Dataset I. Participants were 63 children and adolescents who sought treatment for PTSD at a large public clinic in Israel. Ages ranged from 8 to 17, mean age was 11.9 (SD = 3.2) and 37 participants (58.7%) were female. Of the total sample, 38 participants (60.3%) were children and 25 (39.7%) were adolescents. All participants were diagnosed with primary PTSD according to criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 2000). Complete information on study participants can be found in Gilboa-Schechtman et al. (2010).

Dataset II. Participants were 91 individuals who sought treatment for OCD in the Netherlands. Ages ranged from 19 to 64, mean age was 35.9 (SD = 11.0) and 51 participants (56.0%) were female. All participants were diagnosed with primary OCD using the Anxiety Disorder Interview Schedule-Revised (Di Nardo, O'Brien, Barlow, Waddell, & Blanchard, 1983).

Dataset III. The study included 106 participants who sought treatment for various disorders at a university-based clinic in Israel providing service to the community. Participants were diagnosed using the Mini International Neuropsychiatric Interview Version 5.0 (Sheehan et al., 1998). The majority (55%) of participants were diagnosed with mood disorders, 8.3% were diagnosed with anxiety disorders, and 36.7% had symptoms and distress that did not meet MINI criteria for Axis I disorders. Mean age was 38.0 (SD = 13.0) and 59 participants (55.6%) were female.

Treatment

Dataset I. Treatment was prolonged exposure (PE) therapy adapted for children and adolescents (Foa, Chrestman, & Gilboa-Schechtman, 2008). The treatment consisted of 12–15 sessions and included three modules. Therapists were clinical psychologists who underwent training in pediatric PE by Edna B. Foa prior to the treatment.

Dataset II. Participants in this dataset were randomized to one of three treatment conditions: cognitive therapy (CT: n = 30), exposure therapy (ET: n = 30), and either CT or ET plus pharmacotherapy (fluvoxamine: n = 31). Both CT and ET consisted of 16 weekly sessions. Therapists in psychological treatments were clinical psychologists who had ample experience in the use of cognitive and behavioral techniques, and psychiatrists or psychiatry residents administered fluvoxamine. Complete information on treatments provided in this study can be found elsewhere (Aderka, Anholt et al., 2012; van Balkom et al., 1998; van Oppen et al., 1995).

Dataset III. Treatment consisted of once or twice weekly sessions and was based on a short-term psychodynamic psychotherapy treatment model (Blagys & Hilsenroth, 2006; Shedler, 2010; Summers & Barber, 2010). Therapists were clinical psychology graduate students who received weekly supervision by senior clinicians. All treatment sessions were audiotaped for supervision purposes.

Because the treatment was provided in a naturalistic setting (clinic treating individuals in the community) there was a large variance in number of sessions (range = 7 to 47). Therefore, to equate the number of sessions between participants and to facilitate comparisons with previous studies, we used up to the first 20 sessions for each participant. Complete information on treatments provided in this study can be found in Atzil-Slonim et al. (2018).

Measures

Dataset I. Sudden gains in PTSD were calculated using the Child PTSD Symptom Scale (CPSS; Foa, Johnson, Feeny, & Treadwell, 2001) which was measured weekly prior to each treatment session.

Dataset II. Sudden gains in OCD were calculated using the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989) assessed weekly at every session.

Dataset III. Sudden gains in the naturalistic setting study were measured at every session using the Hopkins Symptom Checklist-short form (HSCL-11; Lutz, Tholen, Schürch, & Berking, 2006). This 11-item self-report inventory assesses symptomatic distress. It is a brief version of widely used SCL-90–R

(Derogatis, 1992). In addition, the Outcome Questionnaire 45 (OQ-45; Lambert et al., 1996) was administered at pretreatment and posttreatment. The OQ-45 is a 45-item self-report measure designed to assess patient outcomes during therapy in three dimensions: (1) subjective discomfort, (2) interpersonal relationships, and (3) social role performance. Finally, the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) was also administered at pretreatment and posttreatment. The BDI-II is a widely used 21-item self-report measure of depression symptoms severity.

Sudden Gains Criteria

Identification of sudden gains in all three datasets was based on the guidelines of Tang and DeRubeis (1999)—Criterion A: gains were large in absolute terms. The reliable change index (Jacobson & Truax, 1991) was used to arrive at a gain that was large in absolute terms. Changes of four points or greater in the CPSS were considered as fulfilling this criterion in the first dataset. In the second dataset changes of four points or greater in the Y-BOCS fulfilled the first criterion. In the third dataset changes of 0.35 points or greater in the HSCL fulfilled the first criterion. Criterion B: The gain's magnitude exceeded 25% of the pregain score. Criterion C: Postgain sessions had lower symptom levels compared with pregain sessions. In all datasets independent *t* tests were conducted between the three sessions before a sudden gain and the three sessions after a sudden gain. Importantly, the *t* tests used to operationalize this criterion have been criticized due to autocorrelation of the data which can inflate alpha values (see Vittengl et al., 2005 for a comprehensive discussion of this issue). Consistent with Vittengl, Clark, and Jarrett (2005) we chose to view the resulting *t*-values as descriptive criteria useful in identifying a reliable pattern of sudden gains, rather than as unbiased inferential indicators of population values. Characteristics of sudden gains in each of our three datasets appear in Table 1.

Analytic Strategy

To assess intraindividual variability in symptoms we first calculated change scores between consecutive sessions for each individual (e.g., Δ between Sessions 1 and 2, Δ between Sessions 2 and 3). Then, we used the absolute value of these change scores to avoid having changes in opposite directions cancel each other out. Put differently, we were interested in the total amount of change (fluctuations) along the course of treatment regardless of its directionality. Finally, we averaged the sum of these change scores (in absolute values) to obtain the average change per intersession interval. This served as the measure of variability in symptoms (i.e., fluctuations). The formula for variability was therefore

$$\sum_{s=1}^{s=N}\left(\frac{|\Delta_{s,\,s+1}|}{N}\right)$$

where N is the number of intersession intervals.

We opted to use this measure over standard deviation/variance as the latter is not sensitive to the order of observations. For instance, if we compare an intraindividual vector of 1, 1, 1, 2, 2, 2 and one of 1, 2, 1, 2, 1, 2, standard deviation is identical for both, but our measure would yield a value of 0.2 average change for the first vector and 1.0 for the second, thus capturing the greater variability in symptoms in the second vector. In order to enhance the reliability of our assessment of fluctuations we computed variability measures only when based on four or more intersession intervals. Thus, we only included individuals for whom at least four between-session deltas could be computed. This can occur when individuals have five consecutive measurements, or alternatively, when there are more measurements which are not completely consecutive (e.g., four consecutive measurements at one point and two additional measurements at another point in treatment). According to guidelines for data screening (Tabachnick & Fidell, 2013), outliers (at least three standard deviations above or below the mean) were excluded from analyses.

Table 1
Characteristics of Sudden Gains and Intraindividual Variability in Symptoms

Characteristics	Dataset I	Dataset II	Dataset III
SG characteristics			
Measure	CPSS	Y-BOCS	HSCL
SG frequency (%)	49.2	34.1	23.6
SG timing (Md)	5	5	7
Significantly predict outcome	Yes	Yes	Yes
SG reversal[a] (%)	16.1	19.4	44.0
Variability characteristics			
Mean (*SD*)	3.86 (1.60)	2.59 (1.57)	.25 (.15)
Range	1.44 to 8.75	.46 to 9.62	.02 to .79
Skewness of distribution	.86	1.13	.94
Kurtosis of distribution	.09	1.14	1.11
Correlation with change during	$r = .25$	$r = -.11$	$r = .07$
Treatment	$p = .07$	$p = .41$	$p = .56$
Correlation with SG magnitude	$r = .08$	$r = .20$	$r = .09$
	$p = .71$	$p = .34$	$p = .73$
Correlation with SG reversal	$r = -.01$	$r = .28$	$r = .25$
	$p = .98$	$p = .21$	$p = .23$

Note. SG = sudden gains.
[a] A reversal is defined as an erosion of 50% of the gain at any time in treatment following the gain.

We did not impute missing data in our datasets to facilitate comparison with previous sudden gains studies and inform the sudden gains literature. Moreover, we wanted to avoid detection of sudden gains as a result of imputed values. Our aim was to use the common practices in the field to examine whether sudden gains such as previously detected in the literature could be predicted by variability in symptoms.

Results

Dataset I: PE for PTSD in Children and Adolescents (Aderka et al., 2011)

Characteristics of intraindividual variability in symptoms. Before conducting our main analyses we examined the measure of intraindividual variability in symptoms, its descriptive statistics, and its relationship with overall change in treatment. Mean variability of symptoms in the sample was 3.86 ($SD = 1.60$) indicating that participants experienced an average change of approximately four points on the CPSS between consecutive sessions. Importantly, this average change score is nondirectional and does not necessarily indicate that the change was a reduction in symptoms, but rather refers to any change whether a reduction or an increase in symptoms. Variability of symptoms along the course of treatment ranged from a minimum of 1.44 to a maximum of 8.75 indicating that there are substantial individual differences in the variability scores. Skewness of variability scores was 0.86 and kurtosis was 0.09.[1] The correlation between variability and overall change during treatment (i.e., pretreatment scores minus posttreatment scores) was small in magnitude according to Cohen's (1988) classification and nonsignificant, $r = .25$, $p = .07$, *ns* Similarly, the correlation between variability and the size of sudden gains was very small and nonsignificant, $r = .08$, $p = .71$, *ns.* This suggests that variability on the one hand, and overall change or size of sudden gains on the other, measure independent aspects of the process of change in treatment.

Main analyses: The relationship between variability in symptoms and sudden gains. We compared individuals with and without sudden gains in their average variability along the course of treatment. Results indicated that individuals who experienced sudden gains ($M = 4.36$, $SD = 1.37$) had significantly more variability in symptoms compared with individuals who did not experience sudden gains ($M = 3.45$, $SD = 1.68$); $F(1, 58) = 5.08$, $p = .03$, partial $\eta^2 = 0.08$. This significant difference remained when controlling for overall change during treatment, $F(1, 55) = 4.33$, $p = .042$, partial $\eta^2 = 0.07$.[2] This indicates that the difference in variability between individuals with and without sudden gains cannot be accounted for by the greater overall change experienced by individuals with sudden gains.

To conduct an even more rigorous test for our hypothesis, we calculated the *pregain variability* in symptoms for all individuals with sudden gains. Thus, for all individuals with sudden gains, the calculation of variability was conducted only on sessions occurring before the sudden gain, excluding the sudden gain and its consequences (e.g., upward spiral of change). We then examined whether this pregain variability predicted sudden gains. Results indicated that individuals who experienced sudden gains ($M = 4.51$, $SD = 2.32$) had significantly more variability in symptoms before the gain compared with individuals who did not experience sudden gains ($M = 3.10$, $SD = 1.14$); $F(1, 40) = 7.06$, $p = .01$, partial $\eta^2 = 0.15$. In order to ensure that these results were not due to restricting the number of observations for individuals with sudden gains (but not for those without sudden gains), we conducted an additional analysis. We examined the average number of pregain observations for individuals with sudden gains and found this to be 5.69 ($SD = 1.89$). Then, we based our calculation of variability for individuals without sudden gains on their first six observations in order to equate the number (and timing) of observations between the groups. Similarly, results indicated that individuals who experienced sudden gains ($M = 4.51$, $SD = 2.32$) had significantly more variability in symptoms compared with individuals who did not experience sudden gains ($M = 3.12$, $SD = 1.28$); $F(1, 40) = 6.28$, $p = .016$, partial $\eta^2 = 0.14$. This suggests that even when examining only pregain variability in symptoms (excluding the gain itself and its resulting upward spiral), and variability in a similar number of sessions for individuals without sudden gains, those with sudden gains had greater intraindividual variability in symptoms compared to those without sudden gains.

Finally, we used logistic regression to predict sudden gain status (yes vs. no) using pregain variability. Results indicated that pregain variability significantly predicted sudden gain status ($B = 0.52$, $SE = 0.23$, Wald $Z = 5.25$, $df = 1$, $p = .02$) and that using pregain variability correctly assigned 81% of participants to either sudden gain or nonsudden gain status. The optimal cutoff score in variability to predict sudden gains was 2.8.

Dataset II: CBT for OCD in Adults (Aderka et al., 2011)

Characteristics of intraindividual variability in symptoms. We ran identical analyses to those conducted on the first dataset, and results appear in Table 1. Similar to Dataset I, intraindividual variability was found to be independent from change during treatment as well as from sudden gains magnitude.

Main analyses: The relationship between variability in symptoms and sudden gains. We ran identical analyses to those conducted on the first dataset, and results appear in Table 2. Similar to results from Dataset I, intraindividual variability was consistently found to be greater among individuals with sudden gains compared with individuals without sudden gains. Specifically, differences between individuals with and without sudden gains were found when examining variability along the entire course of treatment, when controlling for change during treatment, when examining pregain variability, and when examining variability in the first sessions (see Table 2). Finally, results from logistic regression indicated that pregain variability predicted sudden gains and correctly classified 69.2% of participants to sudden gain or nonsudden gain status. The optimal cutoff score in variability to predict sudden gains was 2.4.

[1] Skewness and kurtosis values of less than two in absolute value are acceptable and suggest that the distribution does not differ from normal (Gravetter & Wallnau, 2014).

[2] We could not control for size of sudden gains in the ANCOVA analysis as this variable existed only for individuals with sudden gains (a fact which removed all the individuals without sudden gains from the analysis and rendered the sudden gains variable meaningless). However, the nonsignificant correlation between variability and size of sudden gain among individuals with sudden gains suggests that size of sudden gains may be independent of variability.

Table 2
Intraindividual Variability as a Predictor of Sudden Gains

Predictor	Dataset I	Dataset II	Dataset III
Variability along the entire course of treatment			
M_{SG} (SD_{SG})	4.36 (1.37)	2.79 (1.07)	.28 (.12)
n_{SG}	27	23	24
M_{NSG} (SD_{NSG})	3.45 (1.68)	2.11 (1.04)	.23 (.12)
n_{NSG}	33	35	77
F	5.08	5.88	3.87
Df	1, 58	1, 56	1, 99
P	.03	.02	.05
Partial η^2	.08	.10	.04
Controlling for change during treatment			
F	4.33	4.08	8.19
D*f*	1, 55	1, 55	1, 98
P	.04	.048	<.01
Partial η^2	.07	.07	.08
Pregain variability			
M_{SG} ($SD_{SG)}$	4.51 (2.32)	2.96 (1.43)	.32 (.18)
n_{SG}	13	23	19
M_{NSG} (SD_{NSG})	3.10 (1.14)	2.04 (1.10)	.23 (.12)
n_{NSG}	29	35	77
F	7.06	7.62	7.17
D*f*	1, 40	1, 56	1, 94
P	.01	<.01	<.01
Partial η^2	.15	.12	.07
Number of pregain sessions *M* (*SD*)	5.69 (1.89)	6.63 (3.82)	6.56 (4.38)
Variability in the first sessions[a]			
M_{SG} ($SD_{SG)}$	4.51 (2.32)	2.96 (1.43)	.32 (.18)
n_{SG}	13	23	19
M_{NSG} (SD_{NSG})	3.12 (1.28)	2.20 (1.26)	.23 (.13)
n_{NSG}	29	35	77
F	6.28	4.43	5.19
D*f*	1, 40	1, 56	1, 94
P	.02	.04	.03
Partial η^2	.14	.07	.05
Logistic regression predicting sudden gains from variability			
B	.52	.51	4.39
SE	.23	.25	1.71
Wald *z*	5.25	4.13	6.55
Df	1	1	1
P	.02	.04	.01
% of individuals correctly classified	81.0	69.2	76.9
Cutoff score	2.8	2.4	.23

Note. SG = sudden gainers; NSG = nonsudden gains.
[a] First sessions constituted six sessions for Dataset I, and seven sessions in Datasets II and III based on the average number of pregain observations in each dataset.

Dataset III: Psychodynamic Treatment in Adults in a Naturalistic Setting (Atzil-Slonim et al., 2018)

As data on sudden gains from this sample have not been previously published, we include information on characteristics of sudden gains and their prediction of outcome prior to our main analyses in order to facilitate comparison with the sudden gains literature.

Characteristics of sudden gains. Of the 106 individuals receiving treatment in this sample, 25 (23.6%) experienced a sudden gain. Of these 25 individuals with sudden gains, 22 (88%) experienced a single sudden gain, and three (12%) experienced two gains during treatment. Thus, 28 gains were identified throughout the course of treatment. Sudden gains occurred throughout the length of treatment (see Table 3) with Sessions 5 and 9 as the modes (each with five sudden gains). The average magnitude of sudden gains in raw units was 0.79 (SD = 0.38) on the HSCL, and this represented a change of 1.46 standard deviations based on pretreatment standard deviation ($SD_{pretreatment}$ = 0.55).

Sudden gains and outcome. To examine whether sudden gains were related to outcome, we conducted a mixed ANOVA with treatment (pre vs. post) as a two-level within-subjects independent variable and sudden gains (present vs. absent) as a two-level between-subjects independent variable. The dependent variable was the BDI. We found a significant main effect of treatment such that posttreatment scores (M = 14.63, SD = 10.30) were significantly lower compared with pretreatment scores (M = 18.08, SD = 11.43); $F(1, 65) = 16.24$, $p < .001$, partial $\eta^2 = 0.20$. As expected, no main effect for sudden gains was found, $F(1, 65) = 2.66$, $p = .11$, partial $\eta^2 = 0.04$, but the Treatment × Sudden Gains interaction was significant, $F(1, 65) = 4.77$, $p =$

Table 3
Timing of Sudden Gains in Dataset III

Session	Number of gains	Percentage	Cumulative percentage
1	0	0%	0%
2	2	7.2%	7.2%
3	2	7.2%	14.4%
4	1	3.6%	18.0%
5	5	17.6%	35.6%
6	2	7.2%	42.8%
7	2	7.2%	50.0%
8	0	0%	50.0%
9	5	17.6%	67.6%
10	1	3.6%	71.2%
11	0	0%	71.2%
12	3	10.7%	81.9%
13	0	0%	81.9%
14	1	3.6%	85.5%
15	2	7.2%	92.7%
16	1	3.6%	96.3%
17	0	0%	96.3%
18	1	3.6%	99.9%
19	0	0%	99.9%

.03, partial $\eta^2 = 0.07$, indicating that individuals with sudden gains experienced a greater reduction in symptoms during treatment ($\Delta_{BDI} = 7.37$, $SD = 8.86$) compared with those without sudden gains ($\Delta_{BDI} = 2.09$, $SD = 8.02$). This is consistent with the sudden gains literature showing superior outcome for individuals experiencing sudden gains.

Characteristics of intraindividual variability in symptoms. We repeated the analyses conducted on previous datasets and found that similar to those datasets, intraindividual variability in symptoms was unrelated to change during treatment as well as to magnitude of sudden gains (see Table 1).

Main analyses: The relationship between variability in symptoms and sudden gains. Individuals with sudden gains had greater intraindividual variability in symptoms compared with individuals without sudden gains when examining variability throughout the course of treatment and controlling for change during treatment, when examining pregain variability, and when examining variability in the first sessions (see Table 2). When examining variability throughout the course of treatment without controlling for change, results were marginally significant (see Table 2). We also used logistic regression to predict sudden gain status (yes vs. no) using pregain variability. Results indicated that pregain variability significantly predicted sudden gain status and that using pregain variability correctly assigned 76.9% of participants to either sudden gain or nonsudden gain status.[3] The optimal cutoff score in pregain variability was 0.23.

Pretreatment Clinical Measures, Sudden Gains, and Variability

In all three datasets pretreatment measures were not found to be significant predictors of sudden gains (all $ps > 0.05$). In Datasets I and II, pretreatment measures (CPSS and Y-BOCS, respectively) were not significantly associated with variability in these symptoms ($r = .20$, $p = .12$, $n = 57$; $r = .02$, $p = .86$, $n = 60$; respectively). In Dataset III, pretreatment measures (OQ and BDI), were significantly associated with variability in the HSCL ($r = .29$, $p < .01$, $n = 98$; $r = .38$, $p < .01$, $n = 98$ for OQ and BDI, respectively). To examine whether pretreatment measures accounted for the association between variability and sudden gains in this dataset, we conducted two additional logistic regressions. The first predicted sudden gains using the variability measure and the OQ, and the second predicted sudden gains using the variability measure and the BDI. OQ did not emerge as a significant predictor ($B = -0.005$, $SE = 0.011$, Wald $Z = 0.23$, $df = 1$, $p = .63$) whereas variability significantly predicted sudden gains ($B = 3.97$, $SE = 1.69$, Wald $Z = 5.55$, $df = 1$, $p = .02$). Similarly, BDI did not emerge as a significant predictor ($B = 0.012$, $SE = 0.023$, Wald $Z = 0.26$, $df = 1$, $p = .61$) whereas variability significantly predicted sudden gains ($B = 3.46$, $SE = 1.71$, Wald $Z = 4.12$, $df = 1$, $p = .04$). These results suggest that the variability-sudden gains link cannot be accounted for by pretreatment symptoms.

Intraindividual Variability in Symptoms and Sudden Gain Reversals

In all three datasets, intraindividual variability in symptoms did not predict the reversal of sudden gains (defined as an erosion of 50% of the gain at any time during treatment following the gain; Tang & DeRubeis, 1999). Table 1 includes frequencies of sudden gain reversals as well as variability-reversals correlations for each dataset.

Discussion

Research on predictors of sudden gains has yielded mixed findings. Based on studies suggesting that variability in symptoms may play an important role in sudden gains, the present study examined intraindividual variability in symptoms during treatment as a potential transdiagnostic and transtherapeutic predictor of sudden gains. We examined three diverse datasets and found intraindividual variability in symptoms to be large, to be independent of improvement in treatment, and to consistently predict sudden gains, even when excluding the variability that stems from the gain itself and the ensuing upward spiral.

The measure of within-person variability in symptoms used in the present study has three important characteristics. First, the variance in this measure was large suggesting that some individuals have low symptom variability (i.e., fluctuate less) whereas others have high symptom variability (i.e., fluctuate more). This is in line with previous findings on symptom instability which has been found to differ between individuals (see Marwaha et al., 2014 for a review). Second, symptom variability was unrelated to improvement during treatment suggesting that it taps an orthogonal aspect of the process of change which is unaffected by the amount of improvement experienced. A potential criticism of the sudden gains literature is that it focuses on predicting outcome (i.e., symptom levels) using sudden gains (i.e., symptom improvements)

[3] We also juxtaposed variability in symptoms and early improvement in symptoms during the first four sessions of treatment as predictors of sudden gains based on studies highlighting the importance of early change during treatment (Gibbons et al., 1993; Gilboa-Schechtman & Shahar, 2006; Thomas & Persons, 2013). Results indicated that early improvement was not a significant predictor of sudden gains in any of the three datasets (all p's $> .05$).

and thus creates a circular/tautological argument (Stiles et al., 2003). Along these lines, some researchers have used different measures to tap sudden gains and treatment outcome in order to partly address this issue (Stiles et al., 2003; Vittengl et al., 2005). Accordingly, the second characteristic of the measure of intraindividual variability in symptoms used in the present study (i.e., its independence from symptom improvement) ensures that our predictor does not merely tap symptom improvement which is reflected in sudden gains. Third, our measure of within-person variability was based on a relatively small number of observations (approximately six) which is lower than the number of measurements used in recent studies examining similar constructs (e.g., self-esteem instability, 14 measurements; Farmer & Kashdan, 2014).

Whereas the idea that symptom fluctuations may predict sudden gains was suggested in the literature (Stiles et al., 2003; Vittengl et al., 2005), the present study is the first to examine intraindividual variability in symptoms as a predictor of sudden gains in treatment data. Specifically, findings from three independent treatment datasets with diverse characteristics suggest that it may be generalized across disorders, treatments, populations, and contexts. Moreover, this replication of analysis across three datasets reduces the risk that the findings are spurious and increases our confidence that the associations reported exist in the population of individuals with psychopathology receiving treatment. Demonstrating such replicability is important as researchers have recently failed to replicate published findings in psychology (e.g., Klein et al., 2014).

The use of intraindividual variability in symptoms has an advantage compared with some of the predictors described in the sudden gains literature in that it can be easily examined using existing data and does not require additional measurements (beyond those already used to identify sudden gains). Put differently, this predictor can be examined in any existing treatment dataset for which sudden gains have been identified. This is in contrast to most predictors which are based on additional self-report measures (e.g., measures of self-esteem; Kelly et al., 2005) or other study-specific measurements (e.g., coding of videotaped sessions; Norton et al., 2010). Thus, intraindividual variability in symptoms lends itself well to reexamination and replication and can be easily tested using reanalysis of existing data.

It is important to note that intraindividual variability in symptoms was not found to predict all sudden gains (81%, 69.2%, and 76.9% in our three datasets, respectively). Thus, it is certainly possible that cognitive changes (Tang & DeRubeis, 1999; Tang et al., 2005), therapist competence (Abel et al., 2016), or other factors may still predict some sudden gains that are not predicted by variability. Our findings cannot rule out the existence of additional predictors and in fact suggest the opposite—that additional predictors may exist and explain the residual variance unaccounted for by intraindividual variability. Nevertheless, intraindividual variability in symptoms predicted a large portion of the variance in all three datasets and assigned the majority of individuals to sudden gain versus no sudden gain status correctly.

A number of factors or processes can potentially lead to intraindividual variability in symptoms. First, intraindividual variability in symptoms may stem from random fluctuations. This explanation is consistent with Monte Carlo simulations in the field of sudden gains (Thomas & Persons, 2013; Vittengl et al., 2015). These studies found sudden gains in simulated datasets which included random fluctuations in addition to gradual change. Importantly, this explanation is not in contrast to the predictive role of symptom variability. It is possible for variables to occur at random and still have systematic predictive utility (e.g., winning the lottery). Thus, variability in symptoms may occur at random but may still systematically predict sudden gains. Relatedly, intraindividual variability in symptoms may stem from low reliability of measures administered repeatedly during treatment (Vittengl et al., 2015). Thus, measurement error can also contribute to intraindividual variability in symptoms. In addition, intraindividual variability in symptoms may be the result of contextual factors and changes in participants' daily lives (e.g., Spindler, Stopsack, Aldinger, Grabe, & Barnow, 2016).

Alternatively, intraindividual variability in symptoms may stem from systematic individual differences in symptom instability or volatility. Specifically, individuals have been found to have stable a priori differences in the extent to which their symptoms fluctuate (Marwaha et al., 2014) and these differences have been linked to mental health (Spindler et al., 2016) and response to treatment (Husen, Rafaeli, Rubel, Bar-Kalifa, & Lutz, 2016). Importantly, some fluctuations may be negatively associated with mental health (e.g., pretreatment fluctuations in negative affect; Husen et al., 2016) whereas others may be positively associated with mental health (e.g., fluctuations in positive affect; Spindler et al., 2016). Fluctuations which are positively associated with mental health can be conceptualized as a form of psychological flexibility representing a healthy ability to change in response to external contexts or events such as therapy (see Kashdan & Rottenberg, 2010 for a review). Both theoretical accounts and empirical findings have suggested that psychological flexibility is related to enhanced mental health and positive outcomes (Hayes, Strosahl, & Wilson, 2012; Kashdan & Rottenberg, 2010; Levin, Hildebrandt, Lillis, & Hayes, 2012). It is important to note however, that all the explanations of causes of intraindividual variability remain speculative and future studies are needed to shed light on the nature of intraindividual variability in symptoms and its causes.

Our findings have important clinical implications. First, administering weekly symptom measures can allow us to predict subsequent sudden gains based on variability in the first sessions. This is important as clinicians can be aware of potential sudden gains in advance and thus be ready to address gains in therapy following their occurrence to increase the probability of creating an upward spiral of improvement (Wucherpfennig, Rubel, Hofmann, & Lutz, 2017). Prediction of sudden gains based on data from the beginning of treatment may have advantages compared with many predictors which occur in the session prior to the gain and are thus more difficult to detect or utilize. For example, predicting sudden gains using cognitive changes would require observing videotapes of all treatment sessions and coding cognitive changes from all sessions during the week following the session (see Tang & DeRubeis, 1999 for the method of identifying cognitive changes). This process is much more resource- and time-intensive and would be very hard to implement in routine clinical conditions and in RCTs. Use of intraindividual variability during the first sessions may be a more cost-effective strategy as it only requires entering symptom scores for each session. Moreover, it is possible that intraindividual variability in symptoms may be measured before treatment using a number of weekly pretreatment measurements. While future research is needed to demonstrate a link between

pretreatment variability and sudden gains, this strategy may ultimately allow clinicians and clinical researchers to predict sudden gains prior to treatment and use this information to inform treatment planning. Future research can examine pretreatment variability and its relationship to sudden gains.

The present study has a number of limitations. First, our findings cannot be used to infer causality. Similar to all sudden gains studies to date, our findings suggest an association between pregain intraindividual variability in symptoms and sudden gains, and demonstrate predictive utility, but cannot be taken to indicate that variability *causes* sudden gains. Second, despite our efforts to choose diverse datasets to facilitate generalizability, it remains possible that results would not hold in other treatments or disorders. Future studies are needed to examine intraindividual variability in symptoms in additional contexts. Third, our formula for variability in symptoms represents one of many options to capture fluctuations in symptoms which are unrelated to improvement during treatment. It is certainly possible that other approaches to calculating variability would produce better results (e.g., calculating the number of positive or negative changes in symptoms). Future studies can examine different formulas for variability and their predictive utility in predicting sudden gains. Fourth, using intraindividual variability in symptoms requires repeated symptom measurements early in treatment and not all RCTs or naturalistic datasets have such repeated measurements.

These limitations notwithstanding, our findings point to a novel predictor of sudden gains, which is consistent, potentially generalizable, may cut across disorders, treatments, populations and contexts, and is easily derived without the need for additional measures. These characteristics make intraindividual variability in symptoms a promising predictor of sudden gains which has the potential to inform treatment planning and clinical decision making.

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Appendix

Reanalysis of Previously Reported Data

The article examines datasets from three RCTs and an open trial, and reanalyzes them with the purpose of predicting sudden gains. Thus, efficacy/outcome articles for all trials have been previously published, and are cited in the article. While efficacy and outcome have been the focus of these previous studies, the present study is focused on predicting sudden gains using intraindividual variability in symptoms. No overlap in independent or dependent variables exists between the present article and previous outcome studies.

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