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Symptom Self-Reports Are Susceptible to Misinformation

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We examined whether self-reported symptoms are affected by explicit and implicit misinformation. In Experiment 1, undergraduates ($N = 60$) rated how often they experienced somatic and psychological symptoms. During a subsequent interview, they were exposed to misinformation about 2 of their ratings: One was inflated (upgraded misinformation), whereas another was deflated (downgraded misinformation). Close to 82% of the participants accepted the upward symptom misinformation, whereas 67% accepted the downward manipulation. Also, 27% confabulated reasons for upgraded symptom ratings, whereas 8% confabulated reasons for downgraded ratings. At a follow-up test, some days later, participants ($n = 55$) tended to escalate their symptom ratings in accordance with the upgraded misinformation. Such internalization was less clear for downgraded misinformation. There was no statistically significant relation between dissociativity and acceptance or internalization of symptom misinformation. In Experiment 2, a more subtle and implicit form of misinformation was employed. Undergraduates ($N = 50$) completed a checklist of symptoms and were provided with feedback for some symptoms (targets), misleadingly suggesting that a slight majority of their peers experienced these targets on a regular basis. Next, participants rated the checklist again. Overall, symptom ratings went down for control but not for target symptoms. Taken together, our results demonstrate that symptom reports are susceptible to misinformation. The systematic study of symptom misinformation may help to understand iatrogenic effects in psychotherapy.

Keywords: symptoms, medically unexplained symptoms, misinformation, iatrogenic effects, dissociation

Symptoms do not necessarily indicate the presence of an underlying illness (e.g., Constantinou, Bogaerts, Van Diest, & Van den Bergh, 2013). A telling example is a condition referred to as medically unexplained symptoms (MUSs) in which patients have debilitating complaints such as ex-

treme fatigue, pain, and concentration difficulties in the absence of biomedical dysfunctions. As many as 25%–50% of primary care patients report to have MUSs, rendering it one of the most prevalent problems encountered in general medical practice (Bogaerts et al., 2010). Some authors (e.g., Page & Wessely, 2003) have argued that exposing individuals to extensive history taking and medical tests may escalate innocuous symptoms to such degree that they develop into MUSs. Kouyanou, Pither, Rabe-Hesketh, and Wessely (1998) compared MUS patients attending a pain clinic with patients whose pain symptoms were related to organic pathology (e.g., vascular disease) and observed that possible iatrogenic factors such as hospital admissions, computed tomography and MRI

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scans, and medication prescriptions were more often present in the first group.

More recent studies have accumulated evidence that iatrogenic side effects may also occur in individuals who undergo psychotherapy for their psychological problems. For example, in a survey by Crawford and colleagues (2016) among patients ($N = 14.587$) who had been given psychotherapy, 5% reported that they experienced lasting bad effects from their treatment. Patihis and Pendergrast (2018) conducted a an age-representative survey among adults in the United States and found that those who had consulted with psychotherapists who discussed the possibility of repressed abuse memories were 20 times more likely to recover abuse memories than those whose therapists did not.

Little experimental attention has been devoted to iatrogenic factors that may lead to escalated symptom reporting. One candidate mechanism is misinformation. Because symptoms are often ambiguous and subjective in nature, symptom reports may be susceptible to misinformation—akin to the misinformation effect that is well documented in the memory literature (Loftus, 2005; see also Merckelbach, Jelicic, & Jonker, 2012). For instance, Baumann, Cameron, Zimmerman, and Leventhal (1989, Study 1) misinformed some undergraduates with false feedback suggesting they had raised blood pressures, whereas others were informed correctly that they had normal blood pressure readings. The false feedback group subsequently more often reported symptoms that laypeople associate with high blood pressure (e.g., flushed face, headaches) than the comparison group.

Merckelbach, Jelicic, and Pieters (2011) had undergraduates ($N = 78$) rate to what extent they experienced common symptoms such as fatigue, concentration difficulties, and low mood on a 5-point scale ranging from *never* to *all the time.* After a short interval, participants were interviewed about why they had evaluated the symptoms the way they did, but unbeknownst to them, two symptom ratings were manipulated by upgrading the ratings with two full-scale points. In total, 49 participants (63%) failed to notice both manipulations, as indicated by the fact that they provided confabulated reasons for symptom ratings they had never given. At a 1-week follow-up session, participants who

had accepted the misinformation rated the symptoms that had previously been the targets of misinformation higher than nonmanipulated control symptoms. This pattern of symptom escalation is suggestive of misinformation internalization, and it was absent in those who had rejected the misinformation.

In the two experiments described below, we further explored symptom misinformation and subsequent changes in symptom reports. Specifically, we addressed the following questions. First, can we replicate the phenomenon of symptom escalation due to upward manipulations and does the reverse (i.e., symptom de-escalation) occur with misinformation that downgrades the severity of symptoms (Experiment 1)? Second, are people who display changes in their symptom reports after exposure to misinformation higher on dissociativity compared with those who do not exhibit such changes (Experiments 1 and 2)? Dissociativity refers to traitlike alterations in consciousness, memory, and perception, and it encompasses experiences such as daydreaming, derealization, depersonalization, and amnesia (e.g., Condon & Lynn, 2015). The failure to integrate thoughts, feelings, and experiences into consciousness, as seen in dissociative individuals, results in binding disruptions that may explain why some studies found highly dissociative individuals to relatively easily accept and adopt misinformation (e.g., Eisen & Lynn, 2001; but see Patihis, in press). Third, does a more subtle form of misinformation (i.e., just providing people with misleading information about symptom endorsement rates in their peers) produce symptom escalation, and if so, is this a matter of social compliance or private acceptance (Experiment 2; Zaki, Schirmer, & Mitchell, 2011)?

Experiment 1

This experiment aimed at replicating the phenomenon described by Merckelbach et al. (2011), specifically that symptom escalation occurs when people accept upgraded versions of their symptom scores. The experiment also examined to what extent misinformation that takes the form of symptom downgrading may lead to symptom de-escalation. This is relevant, because trivializing symptoms may explain why people may come to mistrust their symptoms (e.g., Pavelko & Myrick, 2016). Furthermore,

we investigated whether dissociativity levels are related to changes in symptom reports after exposure to misinformation. We predicted that internalizing misinformation as indicated by symptom escalation—or symptom de-escalation for that matter—would be connected to raised dissociativity levels.

Method

Participants. Sixty students (M_{age} = 22.1 years, SD = 2.9, range = 18–32, 47 women) from Maastricht University participated in the study in return for course credits and/or a financial compensation (15 euros; see below). The sample size was based on a power analysis that assumed (as per Merckelbach et al., 2011) an effect size (d) of 0.70 for the difference between target and control symptoms at retest in symptom misinformation-accepting participants. With alpha set at 0.05 and a power of 0.8, this implies an n of 20, which must be multiplied by a factor of 3 when the misinformation acceptance rate and the attrition rate at Time 2 (T2) are conservatively set at 50% and 30%, respectively. Ethics approval was obtained from the standing human subjects committee of the Faculty of Psychology and Neuroscience, Maastricht University, the Netherlands (ERCPN-173–08-03–2016).

Materials.

Checklist for Symptoms in Daily Life. The Checklist for Symptoms in Daily Life (CSDL; Wientjes & Grossman, 1994) consists of 39 common somatic and psychological symptoms (e.g., tension, sleepiness). Respondents rate the degree to which they experienced the symptoms over the past year on a 5-point scale, ranging from *never* (1) to *very often* (5). Scores are averaged to obtain a total CSDL score (range = 1–5), with higher scores indicating higher symptom reports. The CSDL has been found to have acceptable reliability (Cronbach's alpha = .92; Wientjes & Grossman, 1994) and is widely used in psychosomatic research (Sütterlin et al., 2013). In the current study, Cronbach's alphas at Time 1 (T1) and T2 were .92 and .94, respectively.

Dissociative Experiences Scale. The Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986) is a self-report scale that measures trait dissociation. Participants indicate on 100-mm visual analog scales (anchors: 0 = *never;* 100 = *always*) to what extent they experience 28 dissociative experiences in daily life. Examples include feelings of depersonalization and derealization and memory difficulties (i.e., dissociative amnesia). We calculated total DES scores by summing across items (range: 0–100). van IJzendoorn and Schuengel (1996) provide meta-analytic evidence for the sound psychometric properties of the DES (Cronbach's alphas > .90; test–retest rs = .78–.93). Cronbach's alpha for the total DES Scale (T2) in the current experiment was .93.

Design and procedure. Experiment 1 was a within-subjects design, in which participants rated the same CSDL symptoms at two different time points (T1, T2). To mask its true purpose, the experiment was embedded within an unrelated study on memory. In that study, participants viewed a series of neutral pictures (e.g., animals), after which they were given a filler task (i.e., playing Tetris). Following this, participants were given a retrieval task in which they were instructed to recall the pictures they had seen previously. Next, participants carried out another filler task (i.e., playing Bejeweld) but also completed the CSDL (T1), after which they again were instructed to recall the pictures. Fifteen minutes after they had completed the CSDL, participants were interviewed about four CSDL symptoms. During the interview, participants were asked to explain why they had rated these symptoms the way they did. However, the ratings of two symptoms ("targets") were manipulated so as to create misinformation: One rating was upgraded 2 points and one downgraded 2 points, whereas two unaltered symptoms served as "controls." For instance, if the participant originally indicated being "seldom" confused, he or she would be asked to tell more about why he or she "often" felt confused, or if the participant had indicated that he or she "often" felt headaches, the item would be downgraded to "seldom" feeling headaches. A random-number generator was used to select target and control symptoms from the CSDL. When participants did not notice the down- or upgrading of symptoms during the interview, we categorized them as "accepters." Following Johansson, Hall, Sikström, and Olsson (2005), participants' explanations for their symptom ratings during the interview were coded into three distinct categories: (1) "do not know"/ short and uncertain answers (e.g., "maybe due

to exercise"), (2) explanations in accordance with the original symptom ratings (e.g., "yes, but I sometimes do feel dizzy" when asked why they never feel dizzy), and (3) confabulations/ extensive explanations (e.g., "Yes, I often feel very cold. As you can see, that is why I am wearing a warm sweater now"). Thus, when participants provided reasons for the manipulated score, they would be categorized as "confabulators." Confabulators were a subgroup of misinformation-accepting participants.

After the interview, participants were given the CSDL (T2) and the DES to take home and fill out. Participants were compensated with vouchers or course points for the main study, but they were awarded an extra voucher of 15 euros when they delivered the questionnaires back within 7 days (they could deliver it back between 5 hr and 7 days after the testing). On average, participants returned their questionnaire after 5 days. At T2, 55 participants returned a complete CSDL, whereas 54 returned a complete DES. The different sample sizes at T1 and T2 explain the fluctuating degrees of freedom in the Results section. There was no difference in total CSDL scores (T1) between those who returned the T2 questionnaires and those who did not: $t(57^{1}) = 1.16$, $p = .25$. Participants who increased their symptom scores at T2 for the upward manipulated targets are referred to as "escalators." Upon return of the take-home questionnaires, participants were debriefed about the purpose of the study.

Results

The data file can be found at https:// dataverse.nl/. During the interview right after T1, participants could reject both manipulations, accept one and reject the other, or accept two manipulated symptoms. Six participants (10%) rejected both the upward and downward manipulations, 19 (32%) accepted one of the manipulations, and 35 (58%) accepted both types of manipulations. Close to 82% ($n = 49$) of the participants accepted the upward manipulation, whereas 67% ($n = 40$) accepted the downward manipulation, a difference that was not statistically significant, McNemar's exact $p = .064$. However, participants more often reacted with confabulatory responses to upward than downward misinformation: 16 (27%) versus 5 participants (8%), McNemar's exact $p = .013$.[2]

To test whether participants had adopted the upward misinformation at follow-up, we performed a 2 (Groups: accepters vs. rejecters) $\times$ 2 (Symptoms: target vs. control) $\times$ 2 (Time: T1 vs. T2) repeated-measures analysis of variance (ANOVA) on relevant CSDL ratings. There was no statistically significant interaction effect between groups and symptoms, $F(1, 52) < 1.0$, or between groups and time, $F(1, 52) < 1.0$. However, the critical three-way interaction between groups, symptoms, and time attained significance: $F(1, 52) = 9.77$, $p < .01$, $\eta_p^2 = .16$. Follow-up simple effect analyses using paired t tests indicated that the rejecters did not increase symptom scores from T1 to T2 for upgraded targets, $t(10) = 1.93$, $p = .08$, or for control symptoms, $t(10) < 1.0$ (see Figure 1). In contrast, at T2, accepters had increased their scores for the upgraded symptoms ($M_{Time\ 1} = 1.75$, $SD = .78$, $M_{Time\ 2} = 1.95$, $SD = .80$), $t(43) = 2.03$, $p = .04$, Cohen's $d = 0.30$, but had decreased scores for control symptoms ($M_{Time\ 1} = 2.21$, $SD = 1.18$, $M_{Time\ 2} = 1.95$, $SD = 1.09$), $t(42) = 2.54$, $p = .01$, Cohen's $d = 0.41$.

To test whether participants internalized the downward misinformation at follow-up, we performed a 2 (Groups: accepters vs. rejecters) $\times$ 2 (Symptoms: target vs. control) $\times$ 2 (Time: T1 vs. T2) repeated-measures ANOVA on symptom ratings. This yielded a significant interaction of symptoms and time, $F(1, 53) = 9.85$, $p < .01$, $\eta_p^2 = .157$. There was no significant interaction between groups and symptoms, $F(1, 53) = 3.53$, $p = .06$, $\eta_p^2 = .063$, or between groups and time, $F(1, 53) < 1.0$. More important, there was a significant three-way interaction between groups, symptoms, and time, $F(1, 53) = 5.85$, $p = .02$, $\eta_p^2 = .09$.

Follow-up t tests showed that rejecters did not significantly change their target and control ratings from T1 to T2, $t(19) = 1.71$, $p = .10$ and

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[1] Degrees of freedom fluctuate due to missing values at T1 and T2. Thus, of the 60 participants at T1, one had missing values on the CSDL and was excluded from the analyses.

[2] Because we were primarily interested in confabulations, we combined rejecters, those who gave brief explanations, and those who gave explanations that were in accordance with their original ratings into one category and tested the frequencies in this lump category against confabulations for upward and downward misinformation.
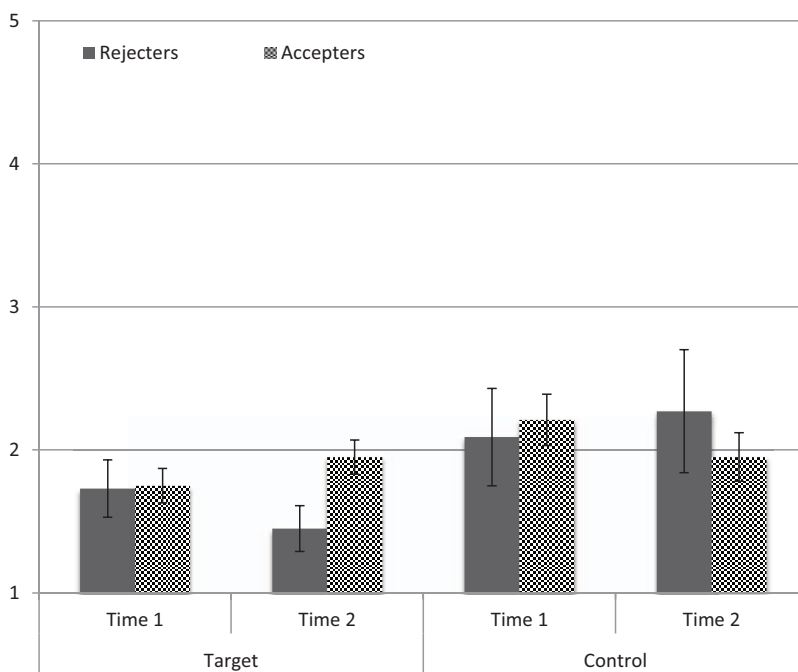
Figure 1. Mean symptom scores (1–5) for target (upgraded) and control items at Time 1 (T1) and Time 2 (T2) of participants who accepted ($n = 44$) and those who rejected ($n = 11$) symptom misinformation. Error bars represent standard errors of the means.

$t(19) = 1.00$, $p = .33$, respectively (see Figure 2). Likewise, accepters did not change their scores of control symptoms, $t(34) < 1.0$. However, there was a significant decrease from T1 to T2 for target symptom ratings; $M_{\text{Time 1}} = 3.46$ ($SD = 0.61$) and $M_{\text{Time 2}} = 2.69$ ($SD = 1.02$), $t(34) = 5.41$, $p < .01$, Cohen's $d = 1.02$. Despite randomization, accepters scored target symptoms higher than control symptoms at T1: $M_{\text{target}} = 3.46$ ($SD = 0.61$) and $M_{\text{control}} = 1.91$ ($SD = 1.11$), $t(39) = 7.53$, $p < .01$. Thus, the significant decline of target ratings over time in this subgroup may reflect regression to the mean rather than internalization of misinformation.

We looked at DES scores of those who accepted or rejected misinformation. Independent samples t tests indicated that for upward manipulations, accepters ($n = 43$) and rejecters ($n = 11$) did not significantly differ in dissociativity, $t(52)^{[3]} < 1.0$. Neither were there differences in DES scores between accepters and rejecters for downgraded targets, $t(52) = 1.07$, $p = .28$. We also examined DES scores of those who confabulated reasons for upward manipulations ($n =$ 16) versus those who did not ($n = 38$). The groups did not differ in DES scores, $t(52) < 1.0.^{[4]}$

Because the long-term impact (T2) of misinformation was most straightforward for symptom upgrading, we next focused on participants who had increased their symptom scores for the upward manipulated target from T1 to T2 (escalators; $n = 13$) and compared them with those who had equal or lower target scores at T2 (nonescalators; $n = 41$). Escalators and nonescalators did not differ in their DES scores, $t(52) < 1.0$, with means being 18.89 ($SD = 14.11$) and 16.28 ($SD = 11.29$).$^{[5]}$

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[3] Only 54 participants returned a complete DES at T2.

[4] For downward manipulations, only five participants confabulated reason, and of those, only four handed in a complete DES at T2. Thus, this group was too small to run a meaningful statistical test.

[5] Neither did we find differences between escalators and nonescalators when we restricted the comparison to socalled DES-T items (see Experiment 2). Means here were 12.23 ($SD = 15.50$) and 9.55 ($SD = 8.15$), respectively, $t(52) < 1.0$.
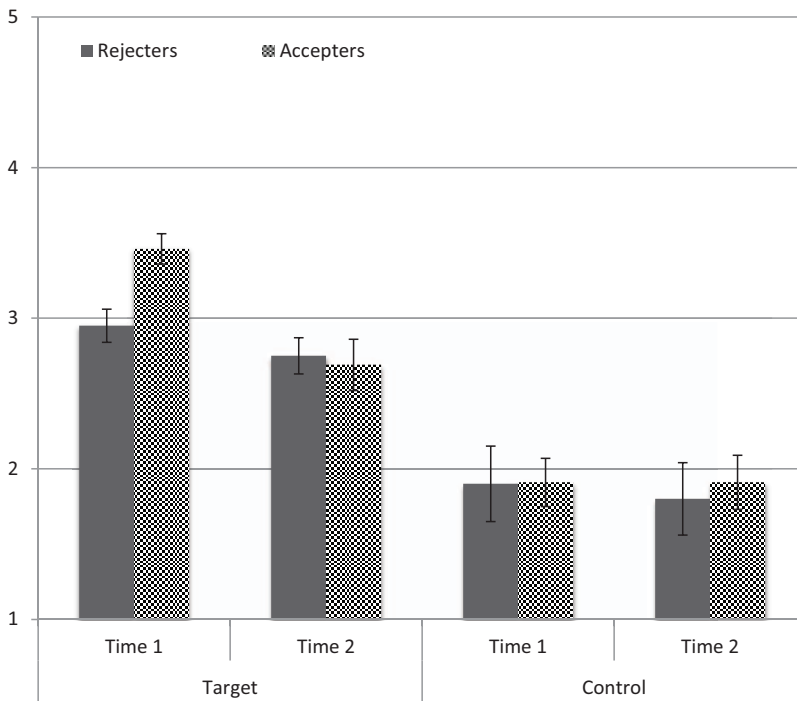
Figure 2.   Mean symptom (1–5) scores for target (downgraded) and control items at Time 1 (T1) and Time 2 (T2) of participants who accepted ($n = 35$) and those who rejected ($n = 20$) symptom misinformation. Error bars represent standard errors of the means.

Discussion

Our results can be catalogued as follows. First, we replicated the phenomenon observed by Merckelbach et al. (2011)—namely, that people often accept misinformation that pertains to their own symptom reports. Thus, close to 82% of the participants accepted the upward manipulation of their symptom scores during the interview. Second, earlier studies (Baumann et al., 1989; Castro et al., 2001; Merckelbach et al., 2011) only looked at upward manipulations and related this to iatrogenic effects of symptom misinformation (for a clinical example, see Merckelbach et al., 2012). Our study demonstrates that acceptance of downward misinformation also occurs (i.e., close to 67% accepted the downward manipulation). Still, the impact of downward misinformation was less pronounced than that of upward manipulations. Specifically, people more often engaged in confabulating reasons for upgraded than for downgraded manipulations (27% vs. 8%). The reason for this asymmetry is probably that upward ma-

nipulated ratings ("sometimes, often, very often") more readily invite attributions than downward manipulated ratings ("never, seldom, sometimes"): If you believe that you have a symptom, there is something to explain; if you do not think you have a symptom, there is nothing to explain. This is reminiscent of the effort-after-meaning phenomenon, that is, the tendency to make sense of ambiguous information (Bartlett, 1932). Third, we replicated Merckelbach et al.'s (2011) observation that those who accept upgraded manipulations during the interview subsequently tend to adopt upgraded versions of their symptom ratings. Such a long-term effect was less straightforward in the case of downgraded symptom intensity manipulations. Although we did find that accepters exhibited a statistically significant decline in target symptom ratings over time, the interpretation of this finding was complicated by baseline differences in target and control symptoms. Clearly, this issue warrants further study.

Fourth, we did not find any statistical differences in dissociative symptom levels between those who accepted and those who rejected symptom misinformation. Such differences were also absent when we compared confabulators with nonconfabulators or escalators with nonescalators. In hindsight, our speculation that changes of symptom ratings after misinformation might be related to heightened dissociativity is naive given the high acceptance rates. It may well be the case that acceptance of straightforward symptom manipulation is a highly situational phenomenon that does not or minimally depends on trait factors. Germane to this is the dual encoding interference (DEI) hypothesis that was recently advanced by Patihis (in press). By this view, traitlike characteristics such as dissociativity operate during both encoding of the original information (e.g., original symptoms ratings) and encoding of the subsequent misinformation (e.g., manipulated symptom ratings). Thus, a traitlike characteristic that undermines encoding does so when the original information is stored—making the original memory trace weak—but also when misinformation is processed, which would make the representation of misinformation and therefore its retroactive interference weak. This way, the DEI hypothesis explains why researchers have not been very successful in identifying traits that are powerful predictors of misinformation acceptance. However, the DEI leaves open the possibility that dissociative individuals are relatively more sensitive to types of misinformation (e.g., "social" misinformation; see below) that do not capitalize on interference with encoding of the original events.

Experiment 2

In Experiment 2, we explored a more subtle form of misinformation that was inspired by Zaki et al. (2011). These authors had participants rate the attractiveness of faces, but on some trials, they were given false feedback about how their peers had evaluated the faces. When participants rated the faces for a second time, their evaluations were affected by peer feedback, such that participants increased attractiveness ratings when their peers ostensibly

had evaluated a face as more attractive than they had.

In the current study, we asked participants to rate symptoms, and for some symptoms, we provided them with misleading information about how their peers had rated these symptoms. We wanted to test whether participants would modify symptom ratings in accordance with group norms. And if so, does this merely reflect public compliance? Or do changes in symptom ratings also occur when participants are explicitly told to disregard feedback information and to rate symptoms in accordance with their original ratings ("consistency instructions")? Furthermore, we investigated whether the tendency to adopt social misinformation about symptoms is particularly pronounced in people who score high on dissociative symptoms, as work on misinformation and interrogative suggestibility seems to indicate (e.g., Merckelbach, Muris, Rassin, & Horselenberg, 2000). We were also interested in two traits that are related to dissociativity: alexithymia (Bagby, Parker, & Taylor, 1994) and fantasy-proneness (Rauschenberger & Lynn, 1995). Alexithymia (Bagby et al., 1994) refers to habitual difficulties in describing internal experiences. Due to their poor interoceptive awareness, people high on alexithymia are more open to external sources of information. Likewise, people high on fantasy-proneness may overvalue information provided by others. For example, researchers have determined that medical students high on fantasy-proneness more often develop the medical student syndrome compared with their low fantasy-prone counterparts (Candel & Merckelbach, 2003). Thus, we anticipated that those who magnify target symptoms ratings after social feedback will score higher on dissociation, alexithymia, and fantasy-proneness than those who do not magnify their symptoms ratings.

Method

Participants. The sample consisted of 50 students who received course credits for their participation in a single-session online experiment (M_{age} = 21.9 years, SD = 2.0, range = 18–26, 36 women). The study was approved by the standing ethical committee of the Faculty of Psychology

and Neuroscience at Maastricht University, the Netherlands (ERCP-173_08_03_2010_V1).

Materials.

CSDL. As in Experiment 1, we used the CSDL (Wientjes & Grossman, 1994) consisting of 39 symptoms that are rated on a 1 (*never*) to 5 (*very often*) scale. Cronbach's alphas in the current study were .94 at T1 and .95 at T2.

Dissociative Experiences Scale–Taxon. To reduce the length of our test battery, we administered only a subset of DES items—namely, the eight items that measure pathological dissociation, including derealization, depersonalization, psychogenic amnesia, and identity alteration. Together, these eight items constitute the so-called Dissociative Experiences Scale–Taxon (DES-T; Waller, Putnam, & Carlson, 1996) index. DES-T scores correlate at $r > .85$ with total DES scores (e.g., Maaranen et al., 2005), although some authors have questioned its temporal stability (Maaranen et al., 2008). Participants indicated on 100-mm visual analog scales (anchors: $0 = never;$ $100 = always$) to what extent they experienced these dissociative experiences in daily life. We calculated total DES-T scores by summing across items (range: 0–100). Cronbach's alpha was .82.

Toronto Alexithymia Scale−20. The Toronto Alexithymia Scale−20 (TAS-20; Bagby et al., 1994) is a self-report scale that measures difficulties in identifying and describing emotions. Its 20 items are rated on a 5-point-Likert scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). Illustrative items are "I am often confused about what emotion I am feeling" and "It is difficult for me to find the right words for my feelings." Scores are summed to obtain a total TAS-20 score that ranges from 20 to 100, with higher scores indicating higher levels of alexithymia. Kooiman, Spinhoven, and Trijsburg (2002) found for TAS-20 scores in their heterogeneous samples of students and patients Cronbach's alphas of .79–.82 and a test–retest stability of $r = .74$. Cronbach's alpha in the current experiment was .74.

Creative Experiences Questionnaire. The Creative Experiences Questionnaire (CEQ; Merckelbach, Horselenberg, & Muris, 2001) is a yes/no self-report measure of fantasy-proneness that consists of 25 items such as, "My fantasies are so vivid that they are like a good movie" and "When I was a child, I had an imaginary friend." Merckelbach et al. (2001) reported a Cronbach's alpha of .72 and a test–retest stability of $r = .95$ for CEQ scores in a student sample. The total CEQ score is calculated by summing the number of yes answers. Cronbach's alpha in the current experiment was .83.

Design and procedure. Experiment 2 was a within-subjects design, in which participants rated the same CSDL symptoms at the start (T1) and at the end (T2) of the session. Using the online survey environment Qualtrics, participants were informed that we were interested in symptoms and their daily frequency among students. Participants were told that they would be asked to estimate how often they themselves experienced common symptoms and that whenever their pattern of symptomatology would deviate from their peers, they would be informed accordingly. Next, participants completed the CSDL for a first time (T1), and while doing so, certain symptoms (targets) were flagged and followed by misinformation. The target items were selected on the basis of the prevalence data collected by Wientjes and Grossman (1994). More specifically, we selected symptoms with the lowest base rate from each of the seven symptom categories represented in the CSDL (see Appendix). For example, one target was "tingling in face," which had a prevalence of 7.2% in the Wientjes and Grossman (1994) sample. Right after participants had rated a target symptom, they were exposed to misinformation that appeared on the center of the computer screen (e.g., "56% of students *often* have this symptom"). The misinformation was subtle, suggesting that a slight majority of peers experienced at least regularly the pertinent target symptom. Symptoms were presented one by one on the screen, and only by clicking on a "next" button would participants be presented with a new symptom. Participants could not go back to previous symptoms. The misinformation message stayed on the screen until participants clicked to see the next symptom. Target symptoms were evenly distributed over the CSDL and were in positions 1, 15, 17, 20, 25, 32, and 38. All other symptoms served as controls.

Following the CSDL items, participants completed the TAS-20, DES-T, and CEQ. In the final part of the experiment (T2), participants once more filled out the CSDL but under different instructions. One group (the control

group) was instructed that rating symptoms is a difficult job and that people sometimes may have second thoughts about how they rated certain symptoms on a checklist. Next they were told, "That's why we want you to complete the CSDL for a second time. There are no good or wrong answers; what counts is your opinion right now." A second group (the consistency group) was instructed that rating symptoms is a difficult job and that people might be misled by all kinds of irrelevant information such as information about how other people rate symptoms. They were next instructed as follows: "That's why we want you to complete the CSDL for a second time. Ignore what you have seen about other students; try to complete the CSDL in the same way as you did the first time you had it. What counts is your consistency." Participants were randomly assigned to the control ($n = 26$) or the consistency group ($n = 24$). CSDL items were administered in a different order than during T1 (they were in positions 1, 8, 10, 14, 15, 19, and 30).

Results

The data file can be found at https:// dataverse.nl/. To test whether participants had adopted the peer misinformation at T2, we performed a 2 (Groups: control vs. consistency instructions) $\times$ 2 (Symptoms: target vs. control) $\times$ 2 (Time: T1 vs. T2) repeated-measures ANOVA on CSDL symptoms. The main effect of group and all interaction effects involving group fell short of significance, $Fs\ (1, 48) \leq 1.41$, $ps \geq .24$, and so we will disregard this factor in what follows. Control items were rated as more frequently experienced than target symptoms. Thus, the main effect of symptoms was statistically significant, $F(1, 48) = 179.29$, $p < .01$, but this is, of course, an artifact of item selection (i.e., we selected low base rate symptoms as targets). The main effect of time was statistically significant, $F(1, 48) = 12.44$, $p < .01$, $\eta_p^2 = .21$, and this was qualified by a Symptom $\times$ Time interaction, $F(1, 48) = 39.02$, $p < .01$, $\eta_p^2 = .45$. As Figure 3 shows,
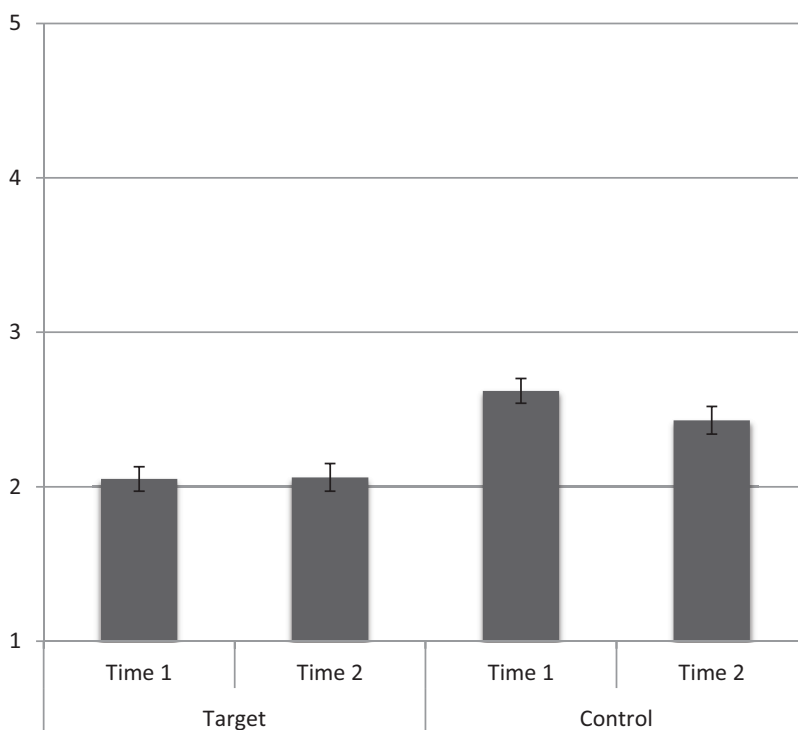

Figure 3. Mean symptom scores (1–5) for target and control symptoms at Time 1 (T1) and Time 2 (T2) ($N = 50$). Error bars represent standard errors of the means.

severity ratings of control symptoms declined from T1 to T2, $M_{\text{Time 1}} = 2.62$ ($SD = 0.53$) and $M_{\text{Time 2}} = 2.43$ ($SD = 0.63$), $t(49) = 7.76$, $p < .01$, whereas they were stable for target symptoms, $M_{\text{Time 1}} = 2.05$ ($SD = 0.58$) and $M_{\text{Time 2}} = 2.06$ ($SD = 0.66$), $t(49) < 1.0$. To take this pattern— decline for control symptoms, stable scores for targets—as evidence for the impact of peer misinformation would be an over-interpretation because it may just reflect regression to the mean for control items (that had an initially higher rating due to item selection). Overall, there was no escalation of target symptom ratings after exposure to misinformation.

The control and consistency group did not differ with regard to dissociativity as measured with the DES-T items, alexithymia (TAS-20), or fantasy-proneness (CEQ), all ts (48) ≤ 1.33, all ps > .19. Looking at the Pearson product–moment correlations between these trait measures in the full sample, we found, as was to be expected on the basis of previous studies (Merckelbach, Boskovic, Pesy, Dalsklev, & Lynn, 2017), that DES-T correlated significantly and positively with TAS-20, $r(50) = .53$, $p < .01$. Similarly, as has been documented in previous studies (e.g., Rauschenberger & Lynn, 1995), dissociativity (DES-T) correlated significantly and positively with fantasy-proneness (CEQ), $r(50) = .49$, $p < .01$. However, the correlation between TAS-20 and CEQ was not statistically significant, $r(50) = .19$, $p = .18$.

We next compared escalators ($n = 19$), that is, those who increased their scores for target symptoms from T1 to T2, with nonescalators ($n = 31$) with regard to their DES-T, TAS-20, and CEQ scores. Although escalators had somewhat higher dissociation, alexithymia, and fantasy-proneness scores than nonescalators, these differences did not attain statistical significance, all ts (48) ≤ 1.98, all ps > .05 (see Table 1).

General Discussion

Research on misinformation usually pertains to memory (e.g., Loftus, 2005), but the experiments described here illustrate that it might be worthwhile to pursue such effects in the context of symptom reports. Many studies have demonstrated that people have limited access to their bodily reactions (e.g., Pennebaker, 2000; Rietveld & van Beest, 2007). It is this limited access

Table 1

Mean and Standard Deviations (in Parentheses) for Escalators and Nonescalators on Dissociativity (DES-T), Alexithymia (TAS-20), and Fantasy-Proneness (CEQ)

Variable	Escalators ($n = 19$)	Nonescalators ($n = 31$)	$t(48)$
DES-T	20.98 (18.63)	12.42 (12.08)	1.98
TAS-20	61.32 (10.19)	58.32 (8.35)	1.13
CEQ	9.16 (4.56)	7.03 (4.76)	1.56

Note. DES-T = Dissociative Experiences Scale–Taxon; TAS-20 = Toronto Alexithymia Scale−20; CEQ = Creative Experiences Questionnaire.

that may make symptoms ambiguous and symptom reports sensitive to misinformation. The results of Experiment 1 show that misleading information about symptom severity might easily go undetected and may even affect how people later on report about their symptoms. This was more obvious for upward than for downward manipulated symptoms, an asymmetry that is reminiscent of studies that found it easier to elicit nocebo than placebo effects in nonsymptomatic participants who are at the lower end of symptom intensities (e.g., Colloca, Petrovic, Wager, Ingvar, & Benedetti, 2010). A more systematic analysis of upward and downward manipulations and their iatrogenic effects may help to understand how certain medical interventions may intensify or potentially trivialize symptom reporting. Thus, the escalation effect of upward misinformation that we found in Experiment 1 and that replicates a similar phenomenon described by Merckelbach et al. (2011; see also Baumann et al., 1989; Castro et al., 2001) may provide a model for how extensive history taking during which a wide array of potential symptoms are explored may intensify symptom reports to such degree that MUSs develop (Kouyanou et al., 1998; Page & Wessely, 2003). Germane to this is a study by Villemure, Nolin, and Le Sage (2011), who used two methods to interview mild traumatic head injury patients about their symptoms: spontaneous free recall of symptoms and an extensive checklist on which patients had to identify their symptoms. The second method yielded consistently more symptoms than the first one. One explanation for this pattern is that checklists may unwillingly convey the misinformation to patients that they are expected to experience

certain symptoms (see also Andreasson et al., 2017; Edmed, Sullivan, Allan, & Smith, 2015).

Merckelbach et al. (2011) found that participants who accepted symptom manipulations did not score higher on social desirability than participants who rejected symptom manipulations. These authors conceptualized acceptance of symptom misinformation as an instance of choice blindness, that is, people's poor monitoring of the choices they made earlier, which makes them susceptible to misinformation. Note that the high misinformation acceptance rates found in Merckelbach et al. (2011) and in Experiment 1 are not unusual in the extant literature on choice blindness. For example, researchers who manipulated preferences for pictures of faces reported blindness rates of 70–80% (Johansson et al., 2005). Ready acceptance of misinformation has also been well documented in memory research (e.g., Loftus, 2005; Loftus & Pickrell, 1995; see also Stille, Norin, & Sikström, 2017). Here, misinformation is incorporated in such way that it affects, for example, food preferences and behavior even after long intervals (e.g., Bernstein, Laney, Morris, & Loftus, 2005). However, just how acceptance of symptom misinformation, blindness, and memory misinformation are related to each other is currently not clear and deserves systematic study (see, for a conceptual analysis, Stille et al., 2017).

Overall, escalating effects of symptom misinformation were absent in Experiment 2, where we provided undergraduates with *subtle* misinformation about how their peers had rated certain symptoms. Neither were there differences in symptom scores between participants who were instructed to be consistent in their ratings while disregarding feedback about peers and participants who were not given such consistency instructions. A subgroup of participants did exhibit symptom escalation from T1 to T2, but they did not score significantly higher on pathological dissociation, alexithymia, or fantasy-proneness than nonescalators. Like Experiment 1, the absence of straightforward differences between escalators and nonescalators in Experiment 2 with regard to these traitlike characteristics might be explained by the DEI hypothesis (Patihis, in press). According to this hypothesis, characteristics that undermine the encoding of original information will also interfere with the encoding of misinformation, such

that the impact of misinformation becomes less pronounced. However, both Experiments 1 and 2 relied on undergraduate samples in which variability in dissociation, alexithymia, and fantasy-proneness is relatively restricted. Furthermore, looking at Table 1, the pattern of group differences is in the expected direction, and one comparison (i.e., DES-T) approached significance ($p = .054$). Of course, this may represent a false positive. Still, whether certain groups of people are more vulnerable to symptom escalation due to misinformation is an issue worthy of further investigation, preferably with a study that directly compares subtle and less subtle forms of symptom misinformation in heterogeneous samples.

Two limitations of our experiments deserve comment. To begin with, Experiment 1 used a random selection of symptoms, resulting in a broad variety of manipulated target symptoms and nonmanipulated control symptoms. However, some symptoms (e.g., fatigue) have a higher a priori base rate and might be more easily internalized than other symptoms (e.g., fainting). Thus, a random selection procedure creates strong baseline fluctuations and therefore statistical noise that may obscure effects. On the other hand, in Experiment 2, we capitalized on symptoms with a low base rate and made these symptoms the targets for misinformation. Yet, this potentially introduces another complication, notably stronger regression-to-the-mean tendencies for control than for target symptoms, which makes effects difficult to interpret. A second limitation is that Experiments 1 and 2 leave open the possibility that social demand characteristics affected our results. Indeed, what is now needed is a paradigm that allows for disentangling the contributions of social demand and poor internal monitoring to people's acceptance of misinformation targeted at plausible symptoms. Providing people with positive incentives for accurate symptom reports and conducting rigorous exit interviews may clarify the role of both factors.

To conclude, few studies have looked into the effects of symptom misinformation, although there are good reasons to assume that this type of misinformation plays a role in the etiology of MUSs (Page & Wessely, 2003) and in recovered memory therapy (Patihis & Pendergrast, 2018). Our results indicate that (a) symptom misinformation is easily accepted by partici-

pants, and (b) misinformation may affect subsequent symptom ratings in such way that symptom escalation occurs. Both findings are relevant to our understanding of how iatrogenic interventions of clinicians may produce worsening of illness in their patients. Although some older studies provided anecdotal descriptions of such effects in the context of psychotherapy (e.g., Fetkewicz, Sharma, & Merskey, 2000), research on iatrogenesis is still in its infancy (Moritz et al., 2015). The main contribution of the current work is that it suggests a laboratory model for the systematic study of this underresearched phenomenon.

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Appendix

List of Target Symptoms

List of target items in Experiment 2 (category, item number, prevalence of "sometimes" in Wientjes & Grossman sample,[6] symptom misinformation):

Gastrointestinal symptoms, 13, shivering, 32.5%, "51% of students regularly have this symptom."

Tingling sensations, 18, tingling in face, 7.2%, "56% of students often have this symptom."

Respiratory symptoms, 5, need for air, 14.5%, "53% of students sometimes have this symptom."

Cardiac symptoms, 31, irregular heart rate, 20.5%, "57% of students regularly have this symptom."

Sensations of warmth, dizziness, and fainting, 32, fainting, 4.8%, "51% of students often have this symptom."

Psychological symptoms, 9, feeling anxious, 34.9%, "59% of students sometimes have this symptom."

Unclassified symptoms, 38, fits of crying, 14.6%, "52% of students regularly have this symptom."

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[6] Wientjes and Grossman (1994), Table 1.