On the feeling of doing: Dysphoria and the implicit modulation of authorship ascription

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Abstract

The experience of authorship arises when we feel that observed effects (e.g., the onset of a light) are caused by our own actions (e.g., pushing a switch). This study tested whether dysphoric persons' authorship ascription can be modulated implicitly in a situation in which the exclusivity of the cause of effects is ambiguous. In line with the idea that depressed individuals' self-schemata include general views of uncontrollability, in a subliminal priming task we observed that dysphoric (compared with nondysphoric) participants experienced lower authorship of action effects when the self-concept was primed. Priming the potential effects of an action just prior to their occurrence, however, increased experiences of authorship in all participants and eliminated the effect of self-concept priming on dysphoric participants' authorship experiences. These findings suggest that the human mental system seizes on a match between primed and actual action effect to establish a sense of authorship, even in a state of depression when persons have weak self-views of causing behavioral outcomes.

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Introduction

The experience of personal authorship arises when we feel that we are doing things. This feeling of causation emerges when the perception of an effect (e.g., onset of a light) corresponds with the effect that is expected to result from performing an action (e.g., pushing a switch). However, such causal analyses suffer from a fundamental uncertainty, as there is always the possibility that action-effects result from some other source. Furthermore, one cannot directly observe causal connections between one's own actions and effects. Thus, conscious causation is always an inference and never something directly observable. Recent work suggests that experienced authorship of events is biased by the mere thoughts we have about events just prior to their occurrence (Wegner, 2002; Wegner & Wheatley, 1999). This study explored the implicit modulation of...
Spurred by Beck's (1967) depressive schema model and the reformulated learned helplessness model (Abramson, Seligman, & Teasdale, 1978), much social-cognitive research on depression has been guided by the idea that perceptions of personal control and causation differ between depressed and nondepressed individuals. These differences result from cognitive schemata that are triggered by self-referential processes (e.g., Bargh & Tota, 1988; Kuiper & MacDonald, 1982; Pyszczynski, Holt, & Greenberg, 1987). Thus, depressed and dysphoric people’s schemata automatically reveal their nature when the self-concept is activated, thereby expressing less perceived control and causation over action–effects. There are some data that speak to this suggestion. In a study by Alloy and Abramson (1979), dysphoric and nondepressed students engaged in a series of action/effect contingency trials in which their action (e.g., pressing a key or not) produces an effect (e.g., the onset of a light) by chance. After the task they were asked to estimate the overall degree of causing the outcome. Because participants were the sole cause of the events and the aggregated post-test measure of causation renders the judgment more self-referential (e.g., Fu, Koutstaal, Fu, Poon, & Cleare, 2005), the self-concept was likely to have been activated in all participants. Alloy and Abramson found that dysphoric individuals’ estimates of causation were lower than those of nondysphoric people (for a discussion of this effect see e.g., Ackermann & DeRubeis, 1991; Dobson & Franche, 1989).

The present work aimed to extend this research by testing whether dysphoric individuals experience less authorship when the self-concept is subliminally primed before the occurrence of an action effect in a situation in which the exclusivity of the cause of effects is ambiguous. Specifically, we experimentally varied the mere accessibility of the self-concept and, rather than an overall post-test measure of causation, asked participants on a trial-by-trial basis to indicate whether they themselves or another agent (i.e., the computer) caused an observed effect following their action. Recent research shows that subliminal exposure to first-person singular pronouns (e.g., “‘I’) primes self-schemata (Dijksterhuis, 2004; Mussweiler & Bodenhausen, 2002; Schubert & Häfner, 2003). In line with this research, we reasoned that self-priming trigger self-schemata of uncontrollability in dysphoric people, and that these general thoughts about uncontrollability decrease their experience of causing action effects.

Depressed and dysphoric individuals are likely to experience decreased authorship when there is no salient information about alternative causes for effects (cf. Weary & Gannon, 1996). Thus, if depressive self-schemata will decrease feelings of doing under conditions where causation is ambiguous, less ambiguity may attenuate this effect. Less ambiguity can be brought about by relevant information. One such salient piece of information is prior knowledge about action effects. A match between primed and observed effect information is a key source for grasping a sense of authorship (Aarts, Custers, & Wegner, 2005; Frith, Blakemore, & Wolpert, 2000; Pronin, Wegner, McCarthy, & Rodriguez, in press; Wegner & Wheatley, 1999). This basic role of effect information renders people prone to rely on it to establish personal feelings of causation. According to Wegner (2002, 2003), in such cases the mind can produce apparent mental causation: the experience of personally causating events that arises whenever our thoughts are inferred to cause these events—whether we truly caused them or not. Thus, the mere priming of the representation of action effects enhances the feeling that one causes the effect when it actually occurs.

Aarts et al. (2005) tested this idea. They designed a task in which the participant and the computer each move a single gray square. The two squares independently traverse in opposite directions on a rectangular path consisting of eight white tiles on a display. Participants’ task was to press a key to stop the movement of the squares. This action turned one of the eight white tiles black. In reality, the computer determined which of the tiles would turn black. However, from a participants’ perspective this black tile could represent the location of either the participants’ square or the computer’s square at the time they pressed stop. Thus, the task was devised in such a way that either the participant or the computer could have caused the square to stop on the observed position, rendering the exclusivity of the cause of effects ambiguous (cf. Wegner & Wheatley, 1999). Accordingly, the position of the black square could be conceived of as the possible effect resulting from participants’ action of pressing the stop key. The position of the black square was primed or not, just before participants stopped the movement. Because the position of the black square was determined by the computer, actual control over causing the stops was absent. Results showed that subliminal priming of the position substantially enhanced feeling of personally causing the square to stop. Further experimentation showed that
the effect priming results were independent of participants’ goal of, or potential real control over causing the effects, suggesting that one can experience authorship of effects merely because one thinks about them just before they occurred.

Given that the mental system heavily relies on a match between primed and observed effect information to establish a sense of personal authorship, we tested whether effect priming also affects dysphoric people’s authorship ascription by employing the subliminal priming task described above (Aarts et al., 2005). We expected effect priming to increase feelings of authorship. However, dysphoric (in comparison with nondysphoric) participants were expected to show decreased feelings of causation when the self-concept is primed, but only in the absence of effect primes.

Method

Participants and design

One hundred and twelve undergraduates participated in the study receiving 2 Euros in return. They were randomly assigned to experimental conditions.

Assessment of dysphoria

Participants filled out the BDI (Beck, 1967, 1972), which was administered during the study. Following the cut-points suggested by Beck, participants with a score of 9 or below were categorized as nondysphoric (BDI-scores 0–9; \(M = 5.92\)) and those with a score higher than 10 as dysphoric (BDI-scores 10–18; \(M = 12.92\)).

Task and procedure

Participants worked in separate cubicles on the task. They learned that the study was designed to examine people’s feelings of causation and how these feelings come and go. For this purpose, they had to move a gray square rapidly traversing a rectangular path in a counterclockwise direction by pressing and holding the S-key. This path consisted of eight white tiles. They also were told that the computer independently would move another gray square along the path at the same speed, but in opposite direction (clockwise). At a certain point in time, participants had to stop the movement immediately by pressing the Enter key in response to the message “stop.” This action turned one of the eight white tiles black, representing the location of either their square or the computer’s at the time they pressed stop. Thus, the black square either did or did not represent the effect of their action. Participants did not have actual control, however, as the computer determined which of the tiles would turn black. Cues for responding were displayed in the middle of the rectangular path. Participants were instructed to keep focused on it during the task. After each trial, they indicated whether they had caused the square to land on that position or the computer had caused it. This authorship judgment was measured on a 10-point answer scale from not at all me (1) to absolutely me (10). The position of the black square was presented twice on each of the eight tiles of the path. The experimental task thus consisted of 16 trials. Trials were randomly presented.

Events in a trial

Each trial started with a warning signal. Next, the message “start” was presented until participants pressed the S-key. One second after participants pressed (and held) the S-key, the participant’s and computer’s square started to move along the path in alternating motion. Squares were displayed for 60 ms on each position. Thus, the speed of one lap was 960 ms [60 ms * 8 positions * 2 (participant’s and computer’s square)]. The number of laps in a trial that were completed before the message “stop” appeared could vary between 8 and 10, and was randomly determined by the computer. From the moment that the message “stop” was presented, only the eight empty white tiles were visible until the participant pressed “Enter.” On that response, a black square was presented after 100 ms, for 1 s. The placement of this square was always 4 positions farther than the last position of the participants’ square before the message “stop” had appeared. So, for example, the black
square was presented in the right lower corner position after the participant’s last square was presented in the left upper corner position; the black square was presented in the right middle position after the participant’s last square was presented in the left middle position, etc.

**Effect-priming**

In eight trials, the position of the black square was flashed before the message “stop.” Thus, the primed location always corresponded with the presented location of the black square. The effect prime (e.g., black square on lower corner right) occurred 40 ms after the last presentation of the participant’s square (e.g., upper corner left). Effect primes were presented for 34 ms, and were 46 ms later followed by the message “stop” (the total time for the priming event thus is 120 ms). In the no-effect-priming condition the position of the black square was not flashed (the position was presented in white for 34 ms). The priming event was employed for every possible location, resulting in eight replications of the effect-priming condition and the no-effect-priming condition.

**Self-priming**

In each trial, for half of the participants the word “ik” (“I” in Dutch) was presented at the moment they had to press the stop key. Specifically, the word “stop” was first presented for 100 ms after which “ik” was presented for 23 ms, followed by the word “stop”. In the no-self-prime condition the prime “de” (“the” in Dutch) was used. The word “stop” remained on the screen until participants pressed the ENTER key.

**Measurement of potential control**

To assess participants’ potential control over producing the effects, the computer measured participants’ time (in ms) to push the Enter key in response to the message “stop.” Because the location of the black square was always four positions farther than the last presentation of the participant’s square, the time from the onset of the last position of the participants’ square to the onset of the position of the black square was 960 ms/2 = 480 ms. Accordingly, the time between the message “stop” and the onset of the black square was 300 ms (480 ms minus the 60 ms from the last presentation of the participant’s square, and minus 120 ms for the effect-priming event). Furthermore, the primary response time required for the participant’s square to land exactly on the position indicated by the black square at half of its presentation time was 330 ms (300 + 30 ms). For each trial we calculated the absolute difference between the response time after the message to stop and the initial time required to land exactly on the position of the black square at half of its presentation time (i.e., 330 ms). This way, for each trial we have a measure of potential control by estimating how close participants had landed to the position of the black square. The smaller the absolute difference, the more likely they actually could have caused the square to land on the position (see Aarts et al., 2005).

**Debriefing**

As in our previous work (Aarts et al., 2005), debriefing showed that no participants had seen the position-primes. They also had not seen the self-primes. Furthermore, none of the participants realized the true nature of the study. Two participants were omitted because they indicated that they misunderstood the task instructions.

**Results**

**Effects on experienced authorship**

The average ratings of experienced authorship across the eight non-prime trials and eight prime trials were subjected to a 2 (dysphoria: nondysphoric vs. dysphoric) × 2 (self-prime: no vs. yes) between-participants × 2 (effect-prime: no vs. yes) within-participants ANOVA. The main effect of effect prime was significant,
participants’ experienced authorship was higher after effect priming than after no effect priming. The analysis also yielded a self-prime × dysphoria interaction effect, but this effect was not significant, $F(1, 106) = 1.88, p = .17, \eta^2 = .02$. Of importance, these effects were qualified by a significant three-way interaction effect, $F(1, 106) = 5.20, p = .02, \eta^2 = .05$. No other effects were reliable, $F’s < 1.60$. Fig. 1 presents the means of each cell in the design.

To gain further insight into the three-way interaction effect and to test our specific predictions, we conducted follow-up tests to analyze the effects of dysphoria and self-prime on experienced authorship within the no-effect-prime and effect-prime condition separately. These tests revealed a significant Self-prime × Dysphoria interaction effect within the no-effect-prime condition, $F(1, 106) = 5.85, p = .02$, but not in the effect-prime condition, $F < 1$. Subsequent analyses within the no-effect-prime condition showed that dysphoric participants’ authorship ratings were lower in the self-prime condition than in the no-self-prime condition, $F(1, 106) = 8.00, p = .006$, whereas nondysphoric participants’ authorship ratings were unaffected by the self-primes, $F < 1$. Furthermore, within the no-effect-prime condition nondysphoric participants did not differ from dysphoric participants in the no-self-prime group, $F < 1$, whereas they did differ in the self-prime group, $F(1, 106) = 5.69, p = .02$.

**Potential control**

The averaged absolute difference scores across the eight non-prime trials and eight prime trials were subjected to the ANOVA described above. The analysis showed no significant main and interaction effects, $F’s < 1.24$. The mean absolute difference score was 70 ms (SD = 43). These findings indicate that the priming events did not affect participants’ potential control over causing the effects.

**Discussion**

The present results disclosed two intriguing findings about the role of implicitly activated thoughts in biasing the experience of authorship of observed action effects. Dysphoric participants experienced decreased authorship when their self-schema was primed, supporting the suggestion that their feeling of doing was affected by general thoughts about uncontrollability. However, these effects on authorship ascription were alleviated when the specific effect of the action was primed just before the effect occurred. These findings are in line with Wegner’s (2002, 2003) theory of apparent mental causation, according to which experienced authorship arises whenever our thoughts are consistent with, and inferred to cause behavioral events.

![Fig. 1. Mean authorship ratings as a function of depression, self-primes and action–effect primes.](image-url)
However, our data extend this general notion showing that priming of action–effects implicitly modulates the experience of authorship even in depressed persons having weak self-views of causing behavioral outcomes. Depression is also related to underestimation of performance. Hence, it may be argued that the decreased feelings of authorship were not caused by perceptions of uncontrollability, but by perceptions of failure. That is, the task might be perceived as a race between oneself and the computer, and the self-primes activated thoughts about failure that caused dysphoric participants to conclude that they lost the contest. It should be noted, though, that the task did not involve a strong performance element. Participants simply pushed a key to stop the movement of their square, and were asked whether they or the computer caused the square to stop on the presented location. However, since we did not directly test the “perception of uncontrollability” account in this study, it still awaits further empirical scrutiny. It might be the case, for example, that the production of action effects that are unwanted or unpleasant might be perceived as consistent with a dysphoric person’s self-concept, and that the experience of authorship for such negative effects could be enhanced by self-primes (cf., Wegner, 2002).

The present study may have implications for interventions designed to encourage dysphoric and depressed people to experience more self-causation in a situation in which the exclusivity of the cause of outcomes is ambiguous. First, research (Fu et al., 2005) suggests that depressive self-schemata are more likely to affect perceptions of confidence if people retrospectively judge their overall level of confidence after a series of trials than on a trial-by-trial basis. In line with this notion, our data showed that the self-concept was not active (and influential) when authorship was assessed after each action effect event. Thus, a first implication may be that eliciting feelings of authorship on a trial-by-trial basis could render the influence of dysphoric and depressed persons’ self-concepts less likely. However, when self-concepts are primed for whatever reason and dysphoric and depressed people therefore might disavow authorship, one could prime effects of their actions just before they occur in order to help them to achieve a sense of authorship. Such training may cause them to experience stronger authorship by relying more readily on a match between expected and observed outcomes as a sign of successful control, even though their self-concept is accessible.

The present study recruited participants whose BDI-scores felt within the mild depression or dysphoric range, and treated dysphoria as an individual-difference variable to examine the potential consequences of self-schema and action-effect priming for authorship processing among depressed people (see for a similar approach, e.g., Weary & Gannon, 1996). In previous research, relations between depression, self-schemata and perception of control have often been observed by comparing nondysphoric and dysphoric samples. As such, our findings may have implications for research on depression. However, although the effects established in dysphoric individuals are often considered to simulate the processes operating in more clinically depressed individuals, we believe that the present effects should be tested in clinical populations as well. Only then can firm conclusions be drawn about the idea that priming of action–effects and the self-concept affects authorship processing among more severely depressed individuals as it does among normal individuals with dysphoria.

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