

# Psychotropic placebos reduce the misinformation effect by increasing monitoring at test

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A psychotropic placebo can help people resist the misinformation effect, an effect thought to be caused by a shift to more stringent source monitoring. When this shift occurs has been unclear. To address this issue we gave some people—but not others—a phoney cognitive-enhancing drug we called R273. Shortly afterwards, everyone took part in a misinformation effect experiment. To gather evidence about source monitoring we surreptitiously recorded time to read the misleading postevent narrative, and response time at test. Our findings suggest that people shifted to more stringent source monitoring at test. Moreover, people with higher working memory capacity (WMC) performed better than people with lower WMC—but only when they were told they had received R273, a finding that fits with research showing that WMC can confer advantages in situations demanding effortful control, but not when automatic heuristics suffice.

One of the puzzles of human behaviour is how taking a substance that does nothing can cause something. Phoney painkillers can lessen our pain or make it worse; phoney alcohol can lead us to do things we might otherwise resist, and phoney feedback can even cause us to shed body fat (Cheong & Negoshi, 1999; Colloca & Bendetti, 2006; Crum & Langer, 2007). Perhaps Kirsch (2004, p. 341) said it best: “Placebos are amazing.”

Recent research shows that placebos can also improve memory performance. Clifasefi, Garry,

Harper, Sharman, and Sutherland (2007) gave people R273, a phoney “cognitive enhancing drug”. Later, when they participated in a misinformation effect experiment (Loftus, Miller, & Burns, 1978; Tousignant, Hall, & Loftus, 1986), people who took R273 were more resistant to the effects of misleading postevent information. These results were both interesting and puzzling: since R273 is really nothing more than lime-flavoured baking soda, how is it that people who took it were better able to ward off misleading

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suggestions? That is the question we address in the experiment reported here.

People have pondered the workings of placebos for centuries, but only recently have we begun to understand them. Today, the prevailing account of the placebo effect is that when people expect a substance to produce a certain outcome, they automatically set in motion a chain of behaviours to produce that outcome—but misattribute the outcome to the placebo, instead of to themselves (Kirsch, 1997, 2004; Kirsch & Lynn, 1999; see also Stewart-Williams & Podd, 2004, for a review). Kirsch and colleagues would say that R273 produces response expectancies, and response expectancies produce effects reflected in people's physiological or mental experiences (see Kirsch, 1997). A closer look at the misinformation effect literature gives us clues about those effects and people's resulting experiences.

## THE MISINFORMATION EFFECT

The typical misinformation effect experiment comprises three stages. People watch an event, then read a summary of it—a summary riddled with misleading postevent information (PEI)—and then take a memory test to report only what they saw, usually choosing between the actual and the suggested details. In hundreds of published studies the pattern is the same: people are worse at correctly remembering event details that were the targets of misleading information (“misled items”) than details that were not (“control items”; Bonto & Payne, 1991; Sutherland & Hayne, 2001; Takarangi, Parker & Garry, 2006; Tousignant et al., 1986).

Both cognitive and social factors influence the misinformation effect. On the cognitive side, people's ability to keep track of—and then detect discrepancies between—the event and PEI affects their susceptibility to the misinformation effect. Several factors influence this ability, such as people's age, where they focus their attention during the event, and their neural activity during the event and PEI phases (Loftus, Loftus, & Messo, 1987; Okado & Stark, 2005; Tousignant et al., 1986). On the social side, the misinformation effect rises and falls with the credibility, power, and likeability of the “misinformation messenger”, the other person who prepared the misleading summary and to whom people must capitulate if they are misled (Assefi & Garry, 2003; Dodd & Bradshaw, 1980; Echterhoff, Hirst,

& Hussy, 2005; French, Garry, & Mori, in press; Vornik, Sharman, & Garry, 2003).

These findings are consistent with the notion of source monitoring, the process by which people are thought to evaluate the quality of information—their memories, knowledge, and beliefs—on myriad cognitive and social dimensions, ultimately, classifying the evaluated information as true or false (Johnson, 2007; Johnson, Hashtroudi & Lindsay, 1993; Lindsay, in press). These dimensions include spatial, temporal, and emotional details, the accordance with other information, and what other people have to say. As source monitoring improves, people become less misled. In the misinformation effect literature, “improved” source monitoring usually means that people shift from quicker, heuristic source monitoring to slower, more effortful, stringent source monitoring (see, for example, Chambers & Zaragoza, 2001; Hekkanen & McEvoy, 2002; see also Johnson et al., 1993 for a detailed account of the mechanism).

## Placebos and the misinformation effect

On the basis of this research, Clifasefi et al. (2007) reasoned that a placebo could improve people's resistance to misleading PEI if it led them to do more stringent source monitoring, misattributing that behaviour to the placebo, instead of to themselves. To examine their hypothesis, Clifasefi et al. recruited people for a bogus drug trial to examine the effects of a phoney drug, R273, touted as a “close cousin” (p. 114) of a drug long used by US military radar operators to produce “significant and sustained improvement in their ability to detect changes in their visual field, and to quickly and accurately distinguish enemy target signatures from simple environmental noise” (p. 114). In reality, R273 was nothing more than a mixture of lime flavouring and baking soda. Clifasefi et al. gave everyone a dose of the mixture, telling half of them they were receiving the active version of the drug, and telling the other half that they were receiving the inactive version. After a short delay, everyone took part in a misinformation effect experiment.

There were two important findings. First, although “Told Inactive” people showed the typical misinformation effect, “Told Drug” people did not. Second, Told Drug people were more likely to agree that the drug improved their cognitive abilities. Taken together, a Kirsch

(2004) account of the results would be this: people's response expectancies about R273 were reflected as more careful and deliberate source monitoring, which in turn produced resistance to the misinformation effect. The question remains: By what mechanism?

There are at least two ways in which more stringent source monitoring could reduce susceptibility to the misinformation effect (Chambers & Zaragoza, 2001; Hekkanen & McEvoy, 2002; Lindsay & Johnson, 1989; Loftus, 2005; Mitchell, Johnson, & Mather, 2003; Zaragoza & Lane, 1994). The first is at the misleading summary. We know that people who read the misleading summary more slowly—whether because of their own preference or an experimenter's instruction to do so—perform better on the later memory test (Greene, Flynn, & Loftus, 1982; Tousignant et al., 1986). The mechanism here is that when people read more slowly, they can scrutinise the summary more carefully, and spot the discrepancy between what they saw and what they read (Greene et al., 1982; Loftus, 1992; Tousignant et al., 1986). If R273 produces response expectancies that lead Told Drug people to scrutinise the summary, we might find that they take longer than Told Inactive people to read the misleading passages.

The second point at which more stringent source monitoring could operate is at test. We know that people who take longer to respond to test items tend to be more accurate (Echterhoff et al., 2005; Hekkanen & McEvoy, 2002; Loftus, Donders, Hoffman, & Schooler, 1989). The logic here was first laid out by Johnson et al. (1993): the standard memory test—a choice between event and suggested information—encourages people to respond on the basis of familiarity (rather than the source of that familiarity). The problem is that the standard method probably makes the suggestion more familiar, because people saw the misleading detail more recently and because the test itself encourages a response bias based on familiarity. As a result, people take the test by relying on efficient, heuristic source monitoring, answering misled items quickly and inaccurately. In fact, Loftus et al. (1989) found that people responded fastest when they incorrectly chose a suggested detail. By contrast, stringent source monitoring is effortful and slower, requiring people to override their tendency to judge accuracy on the basis of familiarity. If R273 produces response expectancies that lead Told Drug people to shift to more effortful source

monitoring at test, we might find that they take longer than Told Inactive people to answer misled test items—particularly for items that were answered correctly

In the experiment we present here, our primary interest was to determine if we could find empirical support for the notion that Told Drug people would engage in stringent source monitoring, and if so, when would they do so: at the postevent summary, at the test, or both? To address this question, we used the same procedure as Clifasefi et al. (2007) but surreptitiously recorded the time it took for people to read the postevent summary, and to respond to questions at test.

### IS THE PLACEBO EFFECT RELATED TO WORKING MEMORY CAPACITY?

We had a secondary interest in identifying whether certain people—namely, people with higher working memory capacity (WMC)—might respond differently to our placebo. A growing body of research suggests that WMC is the ability to draw on cognitive resources to focus on important information while shutting out distracting information (Engle, 2002; Engle & Kane, 2004; Heitz & Engle, 2007; Kane, Bleckley, Conway, & Engle, 2001; Marcizo, Bajo, & Soriano, 2006; Unsworth, Schrock, & Engle, 2004). This ability is the essence of successful source monitoring; perhaps not surprisingly, people with higher WMC—as measured by the OSPAN (Unsworth, Heitz, Schrock, & Engle, 2005)—tend to be more resistant to some false memories, such as those produced by the Deese, Roediger, and McDermott (DRM; Deese, 1959; Roediger & McDermott, 1995) paradigm (McCabe & Smith, 2002; Watson, Bunting, Poole, & Conway, 2005).

In a DRM task, people are shown a list of thematically related words (e.g., *bed, rest, awake, pillow*) that are all highly related to a particular non-presented target word (e.g., *sleep*). Later, on a recognition memory task, people must identify which words were actually presented on the original list, and often people make the mistake of falsely identifying the critical target word. In the DRM people are thought to rely on relatively automatic criteria, such as familiarity, to determine if they heard the lure word earlier. By its very nature, an automatic mechanism does not demand controlled attention, and so it makes sense that WMC differences do not emerge in the

typical DRM experiment. But high-span people should be better able to control their attention—that is, to do deliberate, strategic source monitoring—and reduce their DRM errors accordingly, if something alerts them to override their reliance on familiarity. That something is a warning: in two different studies McCabe and Smith (2002) and Watson et al. (2005) found that higher-span people were less likely to recall hearing lure words—but only if they had been warned about the DRM illusion beforehand. Without the warning, WMC did not matter. These results suggest that people with higher spans could capitalise on a warning in a way that those with lower spans could not, using their superior cognitive control to block the automatic tendency to otherwise report the lure word.

We used a similar logic to hypothesise that if Told Drug people act in line with R273's response expectancies, they could adopt the deliberate, strategic source-monitoring strategies similar to those that should be produced by a warning about the DRM. If so, then higher-span people should be better able to identify discrepancies between what they saw and what they read, and use their cognitive control to block their tendency to otherwise report the suggested detail. Thus, we should see a relationship between span and resistance to misleading suggestion among Told Drug people, but not Told Inactive people, for whom R273 should not produce response expectancies.

## METHOD

### Participants

A total of 96 introductory psychology students took part for course credit. They were run in groups of three or fewer, and did not interact with one another during the session.

### Design

We used a  $2 \times 2$  mixed design with drug instruction (Told Drug or Told Inactive) as the between-participants factor and PEI (control or misled) as the within-participants factor. To control for potential time of day effects (Hasher, Goldstein, & May, 2005) we ran two experimental sessions in the morning and two in the afternoon.

### Procedure

We based our procedure on that developed by Clifasefi et al. (2007), with some minor modifications. There were five stages.

*Stage 1: OSPAN.* In a departmental mass testing session, we obtained scores from 759 first-year psychology students on the automated Operation Span task (OSPAN; Unsworth et al., 2005). The OSPAN task consisted of 75 trials in which people solved mathematical equations while attempting to remember a sequence of random letters. This sequence continued until the end of each trial, at which point people had to choose, from a grid of 12 letters, which letters had been presented. There were three to seven letters per trial. To control for the possibility that people would concentrate only on remembering the letters and not solve the maths equations, maths accuracy had to remain above 85%; the computer issued a warning if accuracy dropped below this point.

For each person we calculated an OSPAN score as the total number of letters correctly recalled on successfully completed trials. After excluding 66 people because they did not complete all 75 trials or because their overall maths accuracy fell below 85%, we were left with scores from 693 people. These scores ranged from 0 to 75, with a median of 40 ( $M = 38.92$ ,  $SD = 18.40$ ) and were comparable to those reported in other published studies (see Unsworth et al., 2005).

A week later we advertised a new and ostensibly unrelated experiment restricted to those who had taken part in the mass testing session. This experiment was advertised as a clinical trial testing the efficacy of a new cognitive enhancing drug, R273, on visual and verbal modes of learning.

*Stage 2: Cultivating response expectancies.* In this second phase we worked to cultivate response expectancies about R273. To that end we set up a small laboratory on campus that was ostensibly run as a joint partnership between a fictitious drug company, Jinal Placard, and our department. We took pains to give the laboratory a high-security, corporate ambience: we fitted a fake swipe card to the door, installed a dummy security camera, created high-quality promotional Jinal Placard posters with a corporate logo, mounted the posters, and hung them on the walls of the laboratory. The experimenter (SP) told

people that she was a PhD student working in collaboration with the pharmaceutical company. To further enhance her credibility she appeared in the laboratory toting a Jinal Placard mug, using a desk littered with Jinal Placard pens, and sitting nearby a Jinal Placard first aid kit.

We ran sessions of no more than three people. They came into the laboratory and sat in individual compartments so they could see the experimenter and the front of the room, but they could not see each other. Next we showed people a fictitious promotional movie about R273, purportedly produced by its (also fictitious) pharmaceutical company. In reality, we created the movie using Final Cut Pro 3 and iDVD 4 software from Apple, Inc.®; it lasted for approximately 6 minutes. The movie informed people that previous trials had shown R273 to be safe and effective in increasing mental alertness and cognitive functioning. The movie also informed people that a “close cousin” of R273 had been tested on US military radar operators and had significantly increased their ability to detect and distinguish changes in their visual fields. A scientist explained the mechanism of action. At no stage did the movie, or the experimenter, mention the word “memory” or a related concept. To further increase response expectancies we warned people that R273 produced some mild physiological side effects, such as an increase in heart rate, a slight head rush, and mild tingling in the fingertips.

*Stage 3: Drug administration.* After the response expectancy phase, the drug administration phase began. The experimenter recorded each person’s weight and told them we would use the information to determine the exact amount of substance each would receive. Next the experimenter recorded weight data into a computer and waited while the computer software “randomly assigned them to their drug condition”. These steps, too, were completely bogus. We told people whether they were to receive the active drug or inactive compound, and then they watched as the experimenter weighed the dose of R273 or inactive compound, transferring it from clearly labelled bottles onto a scientific balance. This substance was mixed with a little water and then distributed to each person, and they drank their portion. They were informed that R273 needed several minutes to take effect, and in the meantime the experimenter played a 10-minute clip from an action movie that was instead designed to

stop people from introspecting about their physiological and psychological state.

*Stage 4. Misinformation effect.* When the action clip ended, the misinformation effect phase began. People watched one of two versions of a slide series depicting a man shoplifting various items from a bookstore (see Loftus, 1991). There were 62 slides; each slide appeared for 2.5 seconds, and the entire event lasted 2 minutes and 57 seconds. Eight of the slides contained a critical item—that is, an item that would later be described with generic or misleading information in the misleading postevent summary. After a 12-minute filler task, people read the misleading summary. There were four versions, each counterbalanced so that every critical item from the slides appeared equally often as a control and misled item. Every combination of drug and PEI summary also appeared equally often across the four time of day sessions.

We divided the 541-word summary into 16 passages, each with a mean of 33.69 words ( $SD = 9.53$ , Range = 14–54) and used Superlab 4.0 to present them on a 14-inch iBook, which each person used throughout the session. Eight of these passages referred to a critical event item, and contained a mean of 34.88 words ( $SD = 9.33$  Range = 14–41). Four of the eight passages were *control* items, those described only generically, while the remaining four were *misled* items, those described inaccurately. People read the passages at their own pace and pressed the space bar to move from one passage to the next. While they did so, we covertly measured reading speed.

*Stage 5: Memory test.* After a 3-minute distractor task, we tested people’s memory for the original event. Using the iBooks, everyone took a 20-item, two-alternative forced-choice memory test. Eight of the questions referred to the critical items from the slide sequence; the four questions about misled items forced people to choose between the item that appeared in the event and an item that was only suggested in the summary. The remaining questions were fillers. For every question, people also rated their confidence on a scale of 1 (*Not at all Confident*) to 5 (*Very Confident*). Again, we covertly recorded response time for each question.

*Manipulation check.* Finally, we asked people to complete a short questionnaire that we used as a manipulation check. On a 1 (Not at all) to 5 (Very much so) scale, they rated the degree to

which they had experienced specific cognitive effects associated with R273.

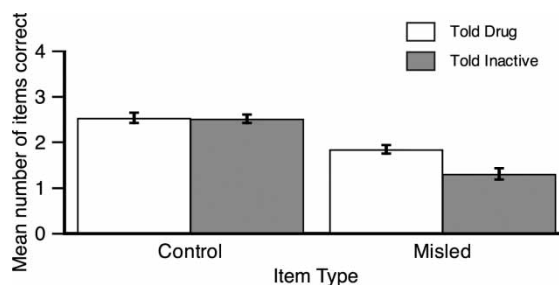
## RESULTS AND DISCUSSION

### Manipulation check

Before turning to our primary analyses, we first determined whether Told Drug people would report that they had observed cognitive effects in line with some of R273's response expectancies. We classified our six cognitive effects measures according to whether people were told they had received the active or inactive version of the drug. As Figure 1 shows, people believed the manipulation: Told Drug people reported better cognitive abilities than Told Inactive people,  $F(1, 93) = 79.15, p < .01, \text{partial } \eta^2 = .80$ . But Told Drug people did not report uniformly better cognitive abilities, as the interaction between drug instruction and cognitive ability ratings shows,  $F(5, 89) = 5.38; p < .01, \text{partial } \eta^2 = .23$ . Follow-up pairwise comparisons revealed that Told Drug people rated every ability significantly higher than their Told Inactive counterparts (all  $p < .01$ ) except for attention,  $t(94) = 1.69, p = .09$ .

### Replication

To make sure we had replicated Clifasefi et al.'s (2007) basic pattern of memory performance, we first calculated each person's mean correct responses to the four control questions and the four misled questions. We classified those scores according to whether we told people we had given them the active or inactive version of R273, and display them in Figure 2.

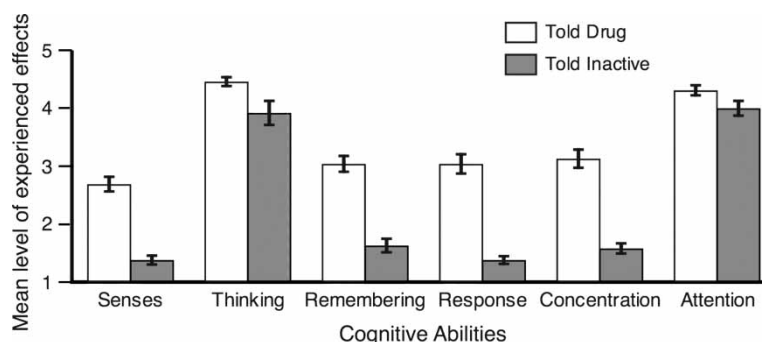


**Figure 2.** Mean Memory Performance (+SE of cell means) by group. Told Drug ( $n = 48$ ); Told Inactive ( $n = 48$ ).

Figure 2 shows that, in line with Clifasefi et al.'s findings, Told Drug people were more resistant to the misinformation effect than Told Inactive people. Although both groups fell sway to misleading suggestion, Told Drug people were less susceptible. More importantly, Told Drug people's resistance was not merely a consequence of better memory for the event, because they were as accurate as Told Inactive people on control items. Put another way, there was an interaction between drug instruction and PEI  $F(1, 94) = 4.43, p = .04, \text{partial } \eta^2 = .05$ . In addition, both groups were similarly accurate on control items,  $t(94) = .11, p = .91$ , but Told Drug people were better able to resist misleading suggestions,  $t(94) = 2.47, p = .02, \text{Cohen's } d = .49$ .

### Confidence

We might expect Told Drug people would be more confident about the accuracy of their memories. Instead we found that they were less confident, although Told Drug and Told Inactive people tended to be similarly confident about control items and less confident about misled items. Regardless of what version of the drug we told them they received, people were more confident



**Figure 1.** Mean score for manipulation check (+SE of cell means) by group. Told Drug ( $n = 48$ ); Told Inactive ( $n = 48$ ).

about their responses to misleading items than control items (see also Assefi & Garry, 2003; Loftus et al., 1989). In short, a 2 (Drug instruction)  $\times$  2 (PEI) mixed ANOVA found a main effect for drug instruction  $F(1,94) = 4.23$ ,  $p = .04$ , partial  $\eta^2 = .04$ , an effect for PEI  $F(1, 94) = 66.85$ ,  $p < .01$ , partial  $\eta^2 = .42$  and a marginally significant interaction,  $F(1, 94) = 3.56$ ,  $p = .06$ .

### Evidence for shift to more stringent source monitoring

We now turn to our primary research questions: Was there empirical support for the notion that Told Drug people would engage in stringent source monitoring? If so, did they do so at the postevent summary, at the test, or both? To answer these questions, we had two directional predictions.

First, if R273 leads people to engage in more stringent source monitoring, we might expect Told Drug people to read the misleading portions of the summary more slowly than Told Inactive people. To address this issue we first calculated each person's mean reading time of the filler passages—text about neither control nor misled items—and then did the same for passages containing misled items. These results appear in Table 1. We then performed an M-estimator robust regression, using both drug instruction and reading speed of filler passages to predict reading speed of misled passages. In M-estimator robust regression, the impact of outliers is minimized by dampening the contribution that outliers make to the model. The resulting model is a good fit to the majority of the observations without any loss of data. M-estimator robust regression, then, is preferable to more popular analyses, such as ordinary least squares regression, and has the added virtue of being supported

**TABLE 1**  
Means (ms) and Standard Deviations for Reading Speed of PEI

	<i>Told Drug</i>		<i>Told Inactive</i>	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
Filler Passages	7794.24	2424.43	8012.32	2118.25
Misled Passages	8642.87	3755.73	9242.15	3532.55

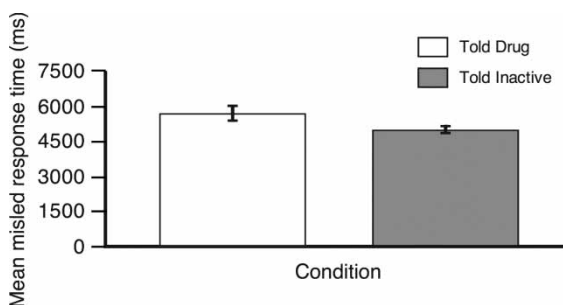
Note: reading speed for filler passages did not differ between Told Drug and Told Inactive conditions,  $t(94) < 1$ .

by the APA task force on statistics (Wilkinson & Task Force, 1999; Wright & London, in press). The robust regression showed that once we controlled for baseline reading speed of the filler portions, drug condition did not predict how long people took to read the misleading parts of the narrative,  $t(93) < 1$ . That is, we found no evidence that Told Drug people used more effortful source monitoring during the narrative than their Told Inactive counterparts: reading speed for the misleading portions of the narrative was the same.

Second, we had a similar prediction for response time at test: more stringent source monitoring might lead Told Drug people to respond to misleading test items more slowly than Told Inactive people. To address this prediction we calculated baseline response time by determining the mean time each person took to answer filler items. We then did the same for misled items. Again, we used robust regression, with both drug instruction and filler response time to predict misled response time. After controlling for filler response time, we found that drug instruction was a significant predictor of misled response time,  $t(93) = -2.16$ ,  $p = .03$ . Figure 3 shows that Told Drug people took longer to respond to test items. These findings lend support to our hypothesis that Told Drug people used more effortful and deliberate source-monitoring strategies during the test than their Told Inactive counterparts.

### Working memory capacity

Recall that we had a secondary interest in the relationship between WMC and our placebo effect, and expected to see a relationship between span and resistance to misleading suggestion among Told Drug people but not among Told



**Figure 3.** Mean Response Time to Filler and Misled Items on Test (+SE of cell means) by group. Told Drug ( $n = 48$ ); Told Inactive ( $n = 48$ ).

Inactive people. To examine this issue we obtained OSPAN scores from the 693 people who took part in mass testing, and found 76 of them who had later volunteered for our session. Of these 76, 36 had been randomly assigned to the Told Drug condition, and 40 to the Told Inactive condition. Mean OSPAN score did not differ between conditions: Told Drug = 45.44 ( $SD = 17.00$ ), and Told Inactive = 40.28 ( $SD = 21.12$ ),  $t(74) = 1.17$ ,  $p = .12$ .

We calculated partial correlations between OSPAN and misled performance, and found that OSPAN was significantly associated with resistance to misleading suggestion among Told Drug people, but not among Told Inactive people,  $pr(33) = .38$ ,  $p = .02$ ;  $pr(37) = .06$ ,  $p = .72$  respectively. One possible counter-explanation for our results is that higher-span participants who received R273 were more gullible, more likely to report having felt the drug's effects. In this line of thinking, higher-span participants performed better because they were more susceptible to the response expectancy. To investigate this possibility we calculated the partial correlation between OSPAN and a score summing responses across all six drug effects. In fact, the partial correlation was  $pr(33) = -.55$ ,  $p < .01$ —and  $pr(37) = -.11$ ,  $p = .49$  in the Told Inactive condition—suggesting that Told Drug higher-span participants tended to report fewer of R273's effects than Told Drug lower-span participants. Thus, we do not find this counter explanation a compelling account of the relationship between WMC and our placebo effect.

Considered as a whole, our findings suggest that R273 “works” because people use more stringent source monitoring at test. Specifically, Told Drug people answered misled test items more slowly than their Told Inactive counterparts. Our data also suggest that R273's effectiveness is related to WMC. OSPAN was associated with better resistance to misleading suggestion, but only when people were told they would receive R273. This finding fits with other research showing that higher-span people are not necessarily less susceptible to false memories such as the DRM illusion; instead, susceptibility depends on whether the task demands effortful control. In the DRM, for example, higher-span people are better able to respond to a warning about the illusion and avoid it (McCabe & Smith, 2002; Watson et al., 2005).

The conditional advantage that higher-span people possess is not limited to false memory research. For example, like the DRM, good performance on the Stroop also demands effortful

control. Kane and Engle (2003) varied the mix of congruent vs incongruent trials to encourage a habit of ignoring the word—and then found that higher-span participants were better able to break the habit to avoid errors on incongruent trials. However, when the mix did not encourage the unhelpful habit, span did not affect errors. Put another way, higher-span participants possessed an advantage in a context demanding effortful control, but no advantage in when automatic heuristics were good enough. Taken together, this research suggests that R273 “works” because it produces response expectancies that demand attentional control—and higher-span people are better able to reflect that response expectancy. When the response expectancy is absent, as with Told Inactive people, there is less demand for attentional control. In this sense, the R273 response expectancies may be similar to warnings in DRM studies.

Our results also extend the literature examining the effects of warnings on the misinformation effect. Research shows that both explicit and implicit warnings can reduce the effects of the misleading PEI (Echterhoff et al., 2005; Greene et al., 1982). Although both warnings targeted the reliability of the narrative, Greene et al.'s came just before people read it, while Echterhoff et al.'s came after. In both studies, timing data suggested that people who were warned shifted to more deliberate source monitoring at the next opportunity—Greene et al.'s participants read the narrative more slowly, and Echterhoff et al.'s responded to misleading test items more slowly.

Of course, in our experiment we had neither an explicit nor implicit warning, nor did we implicitly or explicitly tie the effects of R273 to any particular part of our method. Instead, if R273 creates a response expectancy, then its effects might vary with task demand, manifesting at those points in the method where people can act in line with the expectancy. Because R273's response expectancies are oriented around cognitive abilities, it seems plausible that the demands of the test would most likely be reflected in behaviour at test than behaviour at any other point in the session. In this way, R273's effects on behaviour may be like the relationship between WMC and mind wandering. Kane et al. (2007) found that people with high WM were no better at focusing on a task than low-WM people, unless the task was hard, which is when high-span people were better.

It is important to emphasise that although we did not find any evidence for a shift to more



strategic source monitoring at the PEI, it does not mean that R273 had no effect on source monitoring at the PEI. Source monitoring involves many attributes, and we measured but one. Moreover, we cannot assume Told Drug people's experiences at the narrative are independent of their experiences when taking the test later.

Why should we care that a placebo can induce resistance to the misinformation effect? After all, it is not as if people could presciently take R273 before witnessing a crime. But this paradigm should be useful in examining mechanisms that the SMF predicts. One prediction is that, if R273 leads people to engage in more strategic monitoring at test rather than at PEI, then giving it to people just before test should produce the same resistance to suggestion as giving it to them at the very start of the session. Another prediction comes from the close parallel between relatively automatic vs strategic monitoring processes in the SMF and similar processes thought to be involved in prospective memory. Literature shows that for some kinds of prospective memory tasks people can just rely on cues of the situation to remind them about what they need to do, whereas in other tasks they must actively monitor the situation and ultimately remind themselves (Einstein et al., 2005; Kliegel, Martin, McDaniel, & Einstein, 2004). Therefore examining the effect that R273 has on improving memory for our "to do" lists could be a fruitful avenue for both theoretical and applied research. From a more practical perspective, our research implicates the role of cognitive control in some memory failures, and suggests they can be corrected with warnings, training, or a combination. These are just some of the many important questions that are open to further research. For now, Kirsch's assessment of placebos is borne out by the small but growing literature studying placebo effects on memory: placebos truly are amazing.

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