Placebo Effects of Caffeine on Cycling Performance

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ABSTRACT

BEEDIE, C. J., E. M. STUART, D. A. COLEMAN, and A. J. FOAD. Placebo Effects of Caffeine on Cycling Performance. *Med. Sci. Sports Exerc.*, Vol. 38, No. 12, pp. 2159–2164, 2006. **Purpose:** The placebo effect—a change attributable only to an individual's belief in the efficacy of a treatment—might provide a worthwhile improvement in physical performance. Although sports scientists account for placebo effects by blinding subjects to treatments, little research has sought to quantify and explain the effect itself. The present study explored the placebo effect in laboratory cycling performance using quantitative and qualitative methods. **Method:** Six well-trained male cyclists undertook two baseline and three experimental 10-km time trials. Subjects were informed that in the experimental trials they would each receive a placebo, 4.5 $mg \cdot kg^{-1}$ caffeine, and 9.0 $mg \cdot kg^{-1}$ caffeine, randomly assigned. However, placebos were administered in all experimental conditions. Semistructured interviews were also conducted to explore subjects' experience of the effects of the capsules before and after revealing the deception. **Results:** A likely trivial increase in mean power of 1.0% over baseline was associated with experimental trials (95% confidence limits, −1.4 to 3.6%), rising to a likely beneficial 2.2% increase in power associated with experimental trials in which subjects believed they had ingested caffeine (−0.8 to 5.4%). A dose–response relationship was evident in experimental trials, with subjects producing 1.4% less power than at baseline when they believed they had ingested a placebo (−4.6 to 1.9%), 1.3% more power than at baseline when they believed they had ingested 4.5 $mg \cdot kg^{-1}$ caffeine (−1.4 to 4.1%), and 3.1% more power than at baseline when they believed they had ingested 9.0 $mg \cdot kg^{-1}$ caffeine (−0.4 to 6.7%). All subjects reported caffeine-related symptoms. **Conclusions:** Quantitative and qualitative data suggest that placebo effects are associated with the administration of caffeine and that these effects may directly or indirectly enhance performance in well-trained cyclists. **Key Words:** EXPERIMENTAL DESIGNS, DECEPTIVE ADMINISTRATION, ERGOGENIC AIDS, BELIEF EFFECTS

The placebo effect is a favorable outcome arising purely from the belief that one has received a beneficial treatment (4). It could be argued that in relation to sports performance and research, the placebo effect is widely acknowledged but little understood. Certainly, in common with practice in disciplines such as medicine and clinical psychology, sports scientists account for the possibility of a placebo effect in intervention studies by using a placebo control condition. However, despite evidence elsewhere that the placebo effect impacts a wide range of physiological, psychological, and behavioral variables (6), the placebo effect *per se* has received scant attention in sports science research. The few studies that have specifically addressed the placebo effect in sport (2,4,8,13), despite collectively providing little systematic information relating to its magnitude or mechanisms, do suggest that placebo effects might be associated with several nutritional and pharmacological interventions. For example, Clark et al. (4) reported placebo effects associated with carbohydrate supplementation in cycling performance. Subjects were allocated to three groups and were advised that the carbohydrate group would probably show the most improvement in performance. However, half of the carbohydrate group was randomized to receive the placebo, and half of the placebo group was randomized to receive the carbohydrate. Those in the third group were informed, correctly, that there was a 50:50 chance that their drink would contain carbohydrate. Results indicated a difference in mean power between the told-carbohydrate and told-placebo groups of 3.8% (95% confidence/likely limits/range = 0.2 to 7.9%).

Clark and colleagues made several recommendations for future placebo-effect research, including the use of crossover designs and the exploration of factors that might account for individual differences in placebo responsiveness. The aim of the present study was twofold: first, to use a crossover design to investigate whether athletes given a placebo under the impression it was a performance-enhancing substance would perform at a higher level than in control conditions, and

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secondly, to ascertain how the athletes themselves attributed any perceived or observed changes in performance.

METHOD

Subjects. Institutional ethics approval and written informed consent from all subjects were obtained. Subjects were well-trained competitive male cyclists (N = 7, age = 30 ±11 yr, height = 180 ± 6.3 cm, weight = 75 ± 5.1 kg) recruited from local cycling teams. Before the performance trials, and with the aim of catalyzing or reinforcing beliefs about caffeine, subjects were provided with literature reviewing the findings of published research into caffeine and cycling performance and detailing anecdotal evidence regarding the use of caffeine among elite cyclists. The efficacy of this manipulation was assessed in poststudy interviews. Initial analyses of experimental performance trials indicated that the power output of subject 4 varied by up to 20% between adjacent trials. His data were removed from further statistical analysis, but his interview responses are of interest and are reported below.

Procedure. Subjects each performed two 10-km habituation trials and one $\dot{V}O_{2max}$ test on the SRM cycle ergometer (Ingenieurburo Schoberer, Julich, Germany). The SRM was set up to exactly replicate the subjects' habitual riding position. Subjects performed five maximal-effort 10-km time trials (each preceded by a standardized, progressive 20-min warm-up), in the order of one prebaseline (control), three experimental, and one postbaseline (control). Subjects were informed that they would perform one experimental trial in each of three conditions: placebo, 4.5 $mg \cdot kg^{-1}$ caffeine (moderate dose), and 9.0 $mg \cdot kg^{-1}$ caffeine (high dose), on a randomly assigned double-blind basis. However, a deceptive administration protocol (14) was employed: an identical placebo capsule was administered in each experimental trial. No caffeine was administered during the study. Measures were power, oxygen uptake, heart rate, and blood lactate concentration, taken every 2 km at the thumb.

Each of the trials was separated by a 3- to 10-d gap, and the subjects were asked to maintain their usual training and diet during the study but to refrain from heavy training for 24 h before each trial. They were also asked not to consume any caffeine after 6:00 p.m. the night before testing to control for the effects of caffeine already consumed (14).

Subjects in caffeine research might engage in an active search for symptoms to identify to which experimental condition they have been allocated (15). Recent research has suggested that physical activity masks several of the expected cognitive effects of caffeine (7). Thus, to ensure that the integrity of the experimental deception was maintained, capsules were not administered until the subject was seated on the ergometer and pedaling.

To limit the potential for subjects to employ any pacing strategies based on performance in previous trials, the only performance-related feedback available to them during trials was the distance they had covered. Similarly, to preclude the possibility that knowledge of performance data would contaminate *post hoc* attributions (e.g., the attribution of a random increase in power to caffeine irrespective of any real perceptions of caffeine effects), feedback of all performance data was withheld until the completion of the study.

***Post hoc* measures.** Questionnaires were administered after each experimental trial. Items included "Which condition—placebo, low-dose caffeine, or high-dose caffeine—do you think you completed today?" "To what extent did the capsule effect your performance" and "Did you experience any side effects?" Subjects were reminded at this stage that they would complete only one trial per condition. Although subjects were given the opportunity to revise allocation of trial to condition at the end of the experimental phase of the study, none chose to do so.

Analyses. Performances in baseline trials were averaged to estimate changes in treatment trials. Changes in log-transformed mean power, oxygen uptake, lactate, and heart rate between trials were analyzed using one-way repeated-measures ANOVA. Recently, Batterham and Hopkins (3) proposed the use of magnitude-based inference, whereby the smallest worthwhile effect is identified and justified, confidence limits are interpreted in relation to this effect, and probabilities that the true effect is beneficial, trivial, and/or harmful are derived. Data below are presented in accordance with these suggestions. Paton and Hopkins (16) have suggested that the smallest practically beneficial improvement in performance for a road cyclist is that equivalent to an approximately 1.5% increase in power output; consequently, this value was adopted as the threshold level for the interpretation of confidence intervals.

The experimental design relied on a deceptive administration protocol. Ethical guidelines of the American Psychological Association (1) and our institutional ethics committee required that subjects be thoroughly debriefed at the conclusion of the data collection. The debrief process was incorporated into poststudy interviews carried out in the week after the final performance tests. Two semi-structured interview schedules were prepared. Schedule 1, delivered before revealing the results and the deception, included questions such as "Did you expect caffeine to effect your performance?" "What symptoms did you experience?" and "Do you think that the caffeine affected your performance?" Schedule 2, delivered after the results and the deception had been revealed, investigated subjects' previous responses in light of their knowledge that they had received no caffeine. Interviews were conducted by the first and second authors with one subject at a time in a private office. Each interview lasted between 60 and 100 min, and each was tape recorded with the subject's permission. All interviews were transcribed, and data were analyzed using inductive content analysis as demonstrated by Jackson (9). However, the resulting analysis seemed overly complex and raised several themes, for example "trust in experimenters" and "future use of caffeine," which went beyond the scope

TABLE 1. Means and standard deviations for power output, blood lactate, oxygen uptake, and heart rate by condition (N = 6).

	Power (W)	Lactate (mM)	$\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	Heart Rate (bpm)
Pre baseline	276.6 (39.1)	10.7 (1.5)	57.8 (9.0)	178.1 (10.1)
Placebo	274.3 (46.3)	10.5 (3.3)	60.2 (8.2)	169.2 (14.4)
4.5 $mg \cdot kg^{-1}$	280.6 (37.1)	10.8 (2.5)	56.8 (11.0)	172.4 (13.9)
9.0 $mg \cdot kg^{-1}$	285.9 (38.7)	11.3 (2.7)	57.8 (10.8)	171.4 (14.2)
Post baseline	278.9 (41.0)	10.7 (3.1)	58.0 (8.8)	172.4 (14.7)
Mean baseline	277.7 (42.3)	10.7 (2.3)	57.9 (8.8)	172.2 (12.2)
Mean experimental	280.3 (40.5)	10.9 (2.7)	58.3 (9.7)	171.0 (13.9)
Mean caffeine	283.2 (37.8)	11.0 (2.7)	58.8 (9.7)	171.94 (14.0)

of the present study. Subsequently, we adopted a less analytical approach by summarizing responses relevant to placebo effects.

RESULTS

Mean values by condition for all measured variables are presented in Table 1. Mean and standard deviations for percentage differences in power over baseline, confidence intervals, and likelihood of worthwhile effects are presented in Table 2. Interpretation of confidence intervals revealed a likely trivial difference in power between pre- and postbaseline trials, suggesting no systematic learning or training effects. Overall, there was no practically beneficial difference in power between mean baseline and mean experimental conditions, although a dose–response relationship was evident, with the placebo condition being associated with a mean *decrease* in power compared with baseline, whereas possibly beneficial and likely beneficial increases in power were associated with the moderate- and high-dose caffeine conditions, respectively. The within-subject coefficient of variation (CV) for log-transformed power was calculated at 2.7%. No substantial difference was evident between the CV for baseline and for experimental conditions.

Interpretation of 95% confidence intervals indicated no substantial differences between mean baseline and either mean experimental or mean caffeine conditions for heart rate, oxygen uptake, and blood lactate.

Interview data (N = 7) indicated that five subjects (subjects 1, 2, 3, 4, and 6) attributed direct performance effects to the capsules, subject 5 was unsure whether to attribute performance effects to the capsules, and subject 7 reported no performance effects (note that interview data for subject 4 is included despite his experimental data being removed from statistical analyses above). Performance data were consistent with the interview data of five subjects—for example, subjects 2 and 6, whose experimental and interview data both suggest that they experienced a placebo effect, subject 4, whose interview and performance data arguably suggested a negative placebo or "nocebo" effect, and subject 7, whose experimental and interview data both suggest that he did not experience a placebo effect. Interview responses suggested three specific areas of interest; expectation of caffeine effects, perceived effects of caffeine on performance, and potential mechanisms.

Expectation of caffeine effects. Four subjects (subjects 1, 2, 3, and 4) indicated that they expected the capsules to have a positive effect on their performance. However, no clear relationship between belief in caffeine and performance emerged. For example, subject 6, whose performance data suggested that he experienced a significant placebo response and who subsequently indicated that he believed this to be the case, reported very low *a priori* expectation of caffeine effects. Subject 4, however, indicated that he was expecting caffeine "to have a mega effect" and went on to describe how, on the basis of symptoms experienced during what he believed was the high-dose caffeine trial, he was unable to complete it, stating "I felt terrible, that must have been the big dose of caffeine." It is possible that this poor performance may have resulted from illness.

Effects on performance. Five subjects (subjects 1, 2, 3, 4, and 6) reported direct effects of caffeine on performance. Subject 1 reported that during certain tests, "it got to the point at which on the previous test you really [feel] the pain to the legs and you start to go down a bit, on another test I got to that stage but then I lifted again," and "you get a bit more aggressive you sort of pick up the rpm again and you think to yourself, 'this must be the caffeine.'" Subject 2 suggested, "when I thought I was on the 9 mg of caffeine I went faster, I felt more on top of it whereas all the other times I felt like I was having to dig in just to keep the pedals turning over. I think I was pushing a bigger gear than normal, I was able to push harder with less pain." Subject 6 suggested, "the first time I had the tablet was definitely an improvement on the [trial] before. I was surprised actually how different it felt, whether that was [the tablets] or not I still obviously don't know, but certainly that first tablet I took I thought, 'well, this is a damn sight easier than it was last time.'" He suggested that during experimental trials, "it was easier to put the effort in, there wasn't any tiredness creeping in, I was actually expecting to start feeling tired at a particular point normally after about 10 min on the bike and it didn't so you think 'oh great, well I'll press a little bit harder and I'll go a little bit faster.'" Subject 7 suggested, "one particular day when I turned up and did it I felt really zippy, you pedal and you pedal hard and you're out of breath but you feel you can ride at that threshold and a little bit higher," but this subject also added, "whether it was because of the caffeine, I don't know."

Placebo mechanisms. Six subjects (all except subject 7) suggested potential placebo-effect mechanisms.

TABLE 2. Mean and SD percentage differences in power over baseline, confidence intervals, and practical significance of effects (N = 6).

	Percent Change over Mean Baseline (%) (Mean [SD])	95% CI (Lower to Upper)	Percent Chance That Effect is Beneficial (Trivial/Harmful)
Mean experimental	1.0 (2.4)	−1.4 to 3.6	34 (64/2)
Placebo	−1.4 (3.1)	−4.6 to 1.9	4 (51/46)
4.5 $mg \cdot kg^{-1}$	1.3 (2.7)	−1.4 to 4.1	45 (53/2)
9.0 $mg \cdot kg^{-1}$	3.1 (3.4)	−0.4 to 6.7	86 (13/1)
Mean caffeine	2.2 (3.0)	−0.8 to 5.4	72 (26/1)

These explanations fell into four broad categories: a) pain reduction (six subjects)—for example, "the pain went away," "I don't think there was so much pain," "It's not that you feel it more or less you can just tolerate [pain] a bit more," "I was able to push harder with less pain," and "[you can ride] without it hurting and that's the difference"; b) belief–behavior relationships (four subjects)—for example, "because you think that you've taken caffeine, there must be something in the brain that might tell you've taken something that's gonna make you go better so it does," "there is this great big tablet and you think 'there must be a huge dose in there therefore this is gonna do something really good' and perhaps just that pure belief or hope that it was gonna do something did do something," and "you just believe that it's gonna make you stronger and you believe in it enough to actually make you stronger, so you try to bring yourself up to the level of the difference that it's supposed to make, you actually raise your game to try and match the tablet"; c) attentional changes (two subjects)—for example, "you're focusing on something else that's helping you so it actually takes your attention away from hurting so much'"; and d) arousal changes (two subjects)—for example, "it calms you because you know you are getting something to help you... I tend to ride better if I'm more relaxed. In my job—I'm a fireman—you've only got this air on the back and that's all you've got. When you go into a lighted [building], if you keep calm as you can you use less air, so you're more efficient if you're more calm."

DISCUSSION

When subjects were administered a placebo capsule they believed to be caffeine, they produced, on average, substantially greater power than at baseline. Furthermore, effects were stronger when subjects believed they had ingested higher doses of caffeine. The coefficient of variation for power was comparable with previous research on elite cyclists (15) and lower than for several current lab-based cycle performance tests (5), suggesting that the observed effects are unlikely to be the results of random biological or mechanical variations. Using recently published criteria (16), we are able to state that the effects observed are likely to be of practical benefit to a road cyclist in competition. Furthermore, some of these effects are similar in magnitude to those attributed to caffeine in several published performance studies (19).

Subjects in the present study had a 67% expectation of caffeine ingestion. Had we adopted a design that more closely replicated real-life drug-administration protocols, that is, a deceptive no-blind design in which subjects had a 100% expectation of caffeine administration, we might have expected performance effects of greater magnitude. Use of such a design might also have permitted us a greater degree of confidence in stating that observed effects resulted directly from the intervention (i.e., they were placebo effects) and not from a process whereby a subject simply felt good on one or more days and attributed those feelings to the effects of ingested caffeine. We acknowledge that the potential for subjects to attribute random changes in performance to the ingestion of caffeine was a limitation of the experimental design. However, a secondary aim of the present study was to investigate the mechanisms underlying subjects' attribution of trial to condition, and therefore we aimed to leave subjects in some doubt as to whether they had ingested caffeine in any one trial. It was anticipated that subsequent interview data might elucidate the mechanisms underlying subjects' allocation of trial to condition. To a certain extent, this approach was fruitful. For example, two subjects indicated that because they believed they had already received caffeine in the first and second experimental trials, they had low or zero expectation of caffeine administration in the third (subject 2 suggested, "maybe going into the final day having had a really fast day and one relatively fast makes you think 'hang on a second you can't be given caffeine again'"). This finding suggests that subjects' assumptions about what has or has not been administered in previous trials—placebo or drug—might influence performance in subsequent trials.

Potential placebo mechanisms—for example, whether the placebo effect is manifest as a direct effect on performance or whether a subject's awareness of caffeine symptoms leads to a revised pacing strategy and, thereby, enhanced performance—are of some significance to sports performance research. In this respect, it is interesting that ANOVA revealed no differences between baseline and experimental conditions in any measured physiological variables, which suggests that changes in performance may not have been the result of deliberate changes in pace (this finding, however, might also be a statistical anomaly resulting from the small sample and the fact that the CV for these indices are usually somewhat higher than those for power (5)). It is certainly logical to argue that an athlete performing at volitional maximal power output would not be able to revise his or her pacing strategy and produce still greater power on becoming aware of the subjective symptoms of caffeine ingestion. Conversely, an athlete performing below maximal volitional power output may, on becoming aware of such symptoms, use these as a cue to revise a pacing strategy and produce greater power. Certainly, subjects in the present study reported both perceived caffeine symptoms as well as perceived direct effects on performance. Each subject volunteered at least one caffeine symptom, and somewhat surprisingly, even after being informed of the deception, none of the subjects reappraised these perceptions. This pattern of responses may, as Kienle and Kiene (10) suggest, simply demonstrate a desire to please the researchers. However, we argue that, on the basis of previous research in psychology and medicine using substances such as painkillers, alcohol, and caffeine, the most parsimonious explanation is that individuals tend to experience symptoms consistent with those of the substance they believe they have ingested.

Interestingly, subjects 5 and 7, who reported the fewest caffeine-related symptoms and the least confidence in having experienced a placebo effect, also produced the highest mean power overall. Subject 6, who produced lowest mean power overall, reported arguably the largest and least ambiguous placebo effect. These findings hint at a relationship between training status and placebo responsiveness, as suggested in previous sports performance research (4).

Five subjects attributed direct performance effects to the capsules, one was unsure whether to attribute performance effects to the capsules, and one reported no performance effects. As stated above, performance data are consistent with the interview data of some subjects and less so with others. This is not necessarily surprising, because we can never be sure, even if one subject's mean baseline and mean experimental speeds are similar, that a placebo effect did not bring up to par one or more experimental performances that would otherwise have been below par, or vice versa. Similarly, it is possible that a subject, recognizing symptoms of caffeine ingestion, may have revised his pacing strategy accordingly and increased his power output, but to such a degree that he fatigued prematurely, resulting in a below-par performance overall. It should also be remembered that all interview responses are based on two somewhat unreliable processes, human perception and human recall; no amount of triangulation will unravel that particular problem.

All subjects proposed at least one possible mechanism that might explain observed placebo effects, and each of these, at one level or another, involved belief. Proposals varied from the vague (e.g., "something in the brain") to the specific, such as endorphin-driven pain reduction. (The latter proposal, placebo analgesia, a potential mechanism that was suggested by all subjects in the present study, is currently attracting considerable attention in contemporary medical research and practice (6).) An interesting mechanism was proposed by subject 5, a firefighter, who described how the placebo effect might operate by enabling him to feel less anxious and thus enable his cardiorespiratory and musculoskeletal systems to function more efficiently, producing greater work at a given metabolic cost. Such a mechanism might theoretically not be associated with any changes in physiological parameters such as oxygen uptake or blood lactate and could explain the lack of any observed changes in such variables in the present study.

In summary, once a subject is informed that he or she is to be given a substance that will enhance performance, several subject- or environment-specific psychological processes, such as belief, pain sensation, expectancy, and arousal may be modified. Each of these processes might have an impact on performance depending on its respective direction, intensity, and valence. It is reasonable to suggest that these processes might also be modified on the basis of new information or feedback once performance is under way. Thus, the search for the mechanisms underlying the placebo effect will likely be a complex process. Experimental designs that seek not only to demonstrate the effect but also to provide some explanation are required. Kirsch and Weixel (11), having demonstrated empirically that a double-blind protocol reduced the magnitude of placebo effects in relation to deceptive no-blind conditions, suggested: "If double-blind administration produces psychological effects that in some instances are opposite to those produced by clinical administration of drugs... then double-blind procedures may not be appropriate methods by which to evaluate drug effects" (p. 323). Despite the requirement for either more subjects or more trials per subject, the use of no-placebo controls alongside placebo and experimental conditions, or of deceptive no-blind conditions (e.g., the balanced placebo design (14)) would give sports scientists more insight into the mechanisms underlying many interventions. Such approaches might also be used to investigate the intuitively appealing proposal that placebo and pharmacological/nutritional effects do not act in isolation but, rather, combine in either an additive or interactive way. It is reasonable to suggest that researchers should also examine the impact of belief on performance, either controlling for, or treating as independent variables, the beliefs of subjects in intervention studies. Such strategies might provide a clearer picture of the mechanisms underlying the ergogenic effects of many commercially available products and, in doing so, might allow athletes and sports science professionals alike to make more well-informed decisions in relation to the use of such products.

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