Caffeine and placebo effects improve 1000-m running performance and pacing strategy: a balanced placebo design study

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Keywords:	belief, deception, ergogenic aids, nutrition, sport supplements

Abstract

Purpose: To investigate the placebo effect of caffeine on pacing strategy and performance over 1000-m running time-trials using a balanced placebo design. **Methods:** Eleven well-trained male middle-distance athletes performed seven 1000-m time-trials (one familiarisation, two baseline and four experimental). Experimental trials consisted of the administration of four treatments: informed caffeine/received caffeine (CC), informed caffeine/received placebo (CP), informed placebo/received caffeine (PC), and informed placebo/received placebo (PP). Treatments were randomized. Split times were recorded at 200-, 400-, 600-, 800- and 1000-m and peak heart rate (HR_{peak}) and rating of perceived exertion (RPE) were recorded at the completion of the trial. **Results:** Relative to baseline, participants ran faster during CC ($d = 0.42$) and CP ($d = 0.43$). These changes were associated with an increased pace during the first half of the trial. No differences were shown in pacing or performance between baseline and the PC ($d = 0.21$) and open administration of placebo ($d = 0.10$). No differences were reported between treatments for HR_{peak} ($\eta^2 = 0.084$) and RPE ($\eta^2 = 0.009$). **Conclusions:** Our results indicate that the effect of believing to have ingested caffeine improved performance to the same magnitude as actually receiving caffeine. These improvements were associated with an increase in pace during the first half of the time-trial.

Key words: belief, deception, ergogenic aids, nutrition, sport supplements

Introduction

The placebo effect is a desirable outcome resulting from a person's belief and/or learned response to a treatment or situation.[1] Although there is considerable evidence for the effect placebos can have on sports performance,[2] empirical evidence within sport and exercise science has remained largely static in regards to the degree to which placebo effects interact with the verum components of a treatment. Attempts to quantify the placebo effect in sport and exercise science often rely exclusively on randomized control trials in which participants' belief about the treatment they have been administered is held constant by blinding. Using this type of design nevertheless does not provide sufficient information about whether there are any interactions between a treatment and the belief that the treatment will influence performance.[3] Authors in placebo effect research[4,5] have therefore advocated the use of the four-treatment, balanced placebo design,[6] which allows an assessment of each possible combination of what the participant believes they have taken and what they have actually taken.

To our knowledge, seven studies have used the balanced-placebo design to examine the placebo effect on sport performance.[4,5,7-11] While most studies using this design have reported significant placebo effects on time-trial performance,[5,7,9] few studies have investigated the potential mechanisms related to its response. Since the mid-1990s, there has been an exponential increase in the number of studies investigating the placebo effect and the neurobiological pathways underlying this phenomenon.[2] Qualitative data suggest that placebo effects may be associated with a reduction in pain sensation, arousal regulation and increases in motivation,[12] which may be mediated and moderated by various neurobiological pathways, such as the endogenous opioid and neurotransmitter pathways.[13] However, while there is mounting evidence of the mechanisms underpinning this phenomenon, it is unclear how placebo effects affect sport performance during the actual measure itself. It reasonable to suggest that after ingesting caffeine, for example, athletes may anticipate an offset in fatigue and alter their exercise behaviour. Thus, athletes' pacing strategy may depend on their belief regarding the effect of a substance and their subsequent decisions during performance.

Pacing strategies are set according to an athlete's expectation of the task they are required to perform, based on previous experiences that were used to form a performance template.[14] Numerous studies have manipulated pacing strategies through deception about timing, the presence of a competitor and inaccurate feedback. [15] Konings and colleagues[16] reported that when riding against a virtual opponent, time to complete 4-km cycling times trials improved compared to no opponent due to a faster pace at the start of the time-trial. It has been suggested that this change in pacing behaviour is influenced through neurotransmitters, such as dopamine, which are affected by motivation, drive and perception of effort.[17] Based on this, if an athlete receives a treatment they believe to be performance enhancing, that athlete may be more likely to change their pacing strategy, thereby impacting on performance. However, to the authors' knowledge, no study has investigated the effects of a placebo treatment on pacing strategy.

In this study, we used a balanced placebo design to examine the placebo effects of caffeine on pacing strategy and performance over 1000-m running time-trials. By using a balanced placebo design, we specifically aimed to: 1) determine the influence both placebo and caffeine have on performance and 2) analyse participants' pacing strategies after administration of deceptive and open treatments of caffeine and placebo. We also aimed to

establish whether any changes in performance were associated with changes in peak heart rate and whether this was made possible by participants' propensity to knowingly exert more effort.

Method

Participants and statistical power

Eight participants were estimated to provide an *a* priori statistical power of 0.80. This estimation was based on a study design using repeated measures ANOVA, an *a-value of* 0.05 and an explained effect of 1.4 ± 1.6%.[18] In case of drop out, fifteen participants were initially recruited. Four withdrew (two due to injury and two because of a conflicting timetable), leaving eleven well-trained male middle-distance athletes (mean ± SD: age = 25.2 ± 5.6 yrs; height = 176.3 ± 8.1 cm; body mass = 66.8 ± 6.1 kg; daily caffeine consumption; 269 ± 43 $mg \cdot d^{-1}$). Eligibility criteria stipulated that participants must be nationally ranked in the United Kingdom for 800-, 1500-, 3000- or 5000-m, aged between 18 and 35 and have trained minimally five days per week for at least 3 months prior to the start of the study. Only light-moderate caffeine (200-350 $mg \cdot day^{-1}$) users were included in the study to control for individual differences and familiarity of the effects of caffeine.[19] The study was anticipated to last approximately four weeks. For this reason, only males were recruited to avoid confounding performance variation in the mid-luteal phase of the menstrual cycle.[20] Institutional ethics approval was granted, in agreement with the Declaration of Helsinki. Participants were informed that participation was voluntary and they had the right to withdraw at any time during the course of the study. Participants provided written informed consent after reading the study information sheet.

Design

We used a quasi-randomised, repeated measures, balanced placebo design to determine the effects of caffeine and placebo on 1000-m running time-trial performance. Participants performed seven trials: familiarisation, two baseline and four as part of the balanced placebo design. The four balanced placebo design trials were as follows:

1. Informed caffeine and given caffeine (CC) – participants were informed they received caffeine and did
2. Informed caffeine and given placebo (CP) – participants were informed they received caffeine but received placebo
3. Informed placebo and given caffeine (PC) – participants were informed they received placebo but received caffeine
4. Informed placebo and given placebo (PP) – participants were informed they received placebo and did

The balanced-placebo 1000-m trials were randomised using a computer generated programme (www.randomization.com) and participants were deceived about the treatment they received in CP and PC. Particiapts ran 1000-m and split times were recorded at 200-, 400-, 600-, 800- and 1000-m. Peak heart rate (HR_{peak}) and ratings of perceived exertion (RPE) were recorded immediately after the trial.

Performance measure and equipment

All trials were run on a 400-m, tartan track, in accordance with the International Association of Athletics Federation's standards (polymer synthetic tartan track, with a depth of three centimetres). Participants ran two and a half laps (1000-m) around the track as fast as possible, with no assistance (e.g. pacemakers or external feedback). Times and splits were measured using an automated, single-beam photocell, light gate system (Smartspeed Pro™, Fusion Sport Inc., Australia) and were mounted in lane 1 of the 200- and 400-m start/finish line. Single-beam light gate systems are the most common method for measuring running performance and have shown to have good reliability.[21] Weather measurements for wind speed (m/s), temperature (°C), relative humidity (%) and wind chill (°C) were recorded using the Pasco weather sensor (PS-2174, Pasco, Roseville CA, USA) attached to the Xplorer GLX graphing data-logger (PS-2002, Pasco, Roseville CA, USA). Minimal differences were reported for all time-trials (wind speed = 0.5 ± 0.2*m/s*; temperature = 18.5 ± 1.9°C; relative humidity = 53.5 ± 0.9%).

Caffeine and placebo treatments

Based on previous research in the deceptive administration of caffeine,[4] in the CC and CP treatments, participants ingested 200-mL of chilled saline with 3.0 mg·kg^{-1} of anhydrous caffeine (Myprotein; Norwich, England). The dosage of 3.0 mg·kg^{-1} caffeine was chosen as it has been suggested to be optimal for improving performance lasting ~3-minutes.[22] Given that peak plasma caffeine typically occurs 45-minutes post-ingestion,[23] participants were asked to consume the treatments 1-hour prior to the start of the time-trial. In the CP and PP treatments, participants consumed 200-mL of chilled saline only. In placebo effect research, the validity of the balanced-placebo design relies on the credibility of the deception in the CP and PC treatments. Extensive pilot testing was therefore conducted to ensure that no taste or palpability differences could be identified between placebo and caffeine treatments.

Belief manipulation

Before any data collection, participants attended a short presentation on the benefits of caffeine on middle-distance running performance delivered by the first author. Participants were provided with literature reviewing the findings of published research on caffeine and middle-distance running and were informed that caffeine was previously a banned performance enhancing substance. To further augment the belief that caffeine is performance enhancing, and in line with current recommendations for reporting fine details of participant contact and communication,[2] anecdotal evidence relating to the first authors' experience in the use of caffeine was explained. At the time of data collection, the first author competed as an international level athlete against notable Olympians and participants were informed that caffeine acted as potent ergogenic aid during competition. The efficacy of this manipulation of beliefs was supported by data collected in post-study interviews.

Procedure

Participants performed seven 1000-m running time-trials. All trials were performed on Monday and Friday evening at the same location. The time between trials allowed an adequate wash out period for caffeine supplementation[24] and is sufficient for middle-distance trained athletes to fully recover.[25]

For all trials, participants were instructed to arrive in 'race-shape' condition. High intensity exercise 48 hours preceding the trials was not permitted, as well as the consumption of alcohol or sport supplements. Participants were asked to adhere to their regular pre-race diet, rest and warm-up routines. Participants began all trials at the same time of day to minimise circadian variation in performance[26] and each trial was started by a green LED, which would flash up on the photocell. To limit the potential for participants to employ pacing strategies based on knowledge of previous trials and performance during trials, they did not to wear a watch and were given no encouragement. No information about split times was given and the results of the trials were given after all data had been collected. HR_{peak} was recorded using a Polar stopwatch (Heart Monitors, Polar Ltd, Finland) and RPE from 0 (nothing at all) to 10 (maximal) was measured using the Borg Category Ratio[27] immediately after participants completed the trial.

For familiarisation trials, participants were informed: "Today you are performing a familiarisation trial" and for baseline trials 1 and 2, participants were informed "Today you are performing a baseline trial". For balanced placebo design trials, participants were further reminded about which treatment they had received. For CC and CP treatments, participants were informed: "Today you will be performing the trial with caffeine" and for PC and PP treatments, participants were told: "Today you will be performing the trial with no caffeine." Upon completion of all data collection, participants were debriefed about the true nature of the study.

Data analysis

Times to complete the 1000-m time-trials for baseline 1 and baseline 2 and each split (200-, 400-, 600-, 800- and 1000-m) were inputted into an online reliability spreadsheet.[28] Data were log transformed to reduce nonuniform errors and the intraclass correlation (ICC) and Pearson correlation (*r*) provided estimates of reliability. The precision of ICC was interpreted as extremely high (0.99); very high (0.90), high (0.75) moderate (0.50) and low (0.20).[28] *r* was interpreted as trivial (<0.1), small (0.3), moderate (0.5), large (0.5), very large (0.7), nearly perfect (0.9) and perfect (1.0). In addition, paired samples *t*-tests were conducted to determine any systematic difference in performance between baseline 1 and baseline 2.

Data were entered into SPSS version 24.0 (IBM, Armonk, NY) and tested for homogeneity of variance, normal distribution and anomalies. Repeated measures ANOVA identified differences in time to complete 1000-m time-trials between each treatment (i.e. baseline, CC, CP, PC and PP) and split (i.e. 200-, 400-, 600-, 800- and 1000-m). Differences in HR_{peak}, RPE and mean time to complete the 1000-m trials between each treatment were also established using repeated measures ANOVA. Greenhouse-Geisser epsilon was reported when sphericity was violated and post-hoc LSD tests were used. Cohen's *d* was calculated to determine the effect size (*d*) of the mean differences. Differences between 0.2 and <0.5 were interpreted as a small effect, between 0.5 and <0.8 as moderate, and >0.8 as large.[29] Data are presented as mean ± standard error of the mean with statistical significance set at $p<0.05$

Results

Times were similar between baseline 1 and baseline 2 at 200- (mean differences = -0.48 ± 0.34 s, $P = .290$, $r = 0.897$, ICC = 0.90), 400- (0.04 ± 0.40 s, $p = 0.936$, $r = 0.776$, ICC = 0.77), 600- (-0.56 ± 0.30 s, $p = 0.217$, $r = 0.885$, ICC = 0.85), 800- (-0.13 ± 0.53 s, $p = 0.149$,

r = 0.584, ICC = 0.61) and 1000-m (0.60 ± 0.61 s, p = 0.189, r = 0.614, ICC = 0.67). The average of these two time-trials was thus used to measure baseline. Mean times to complete 1000-m trials in all treatments are shown in table 1.

Main analyses

Repeated measures ANOVA (treatment × split) reported differences between treatment ($F_{(4, 160)}$ = 6.162, p = 0.006; η^2 = 0.381) and split ($F_{(4, 160)}$ = 9.288, p < 0.001; η^2= 0.482). No difference in time was shown for treatment × split ($F_{(4, 160)}$ = 1.055, p = 0.266; η^2= 0.108).

Differences in time between treatments

Compared to baseline, participants ran faster in CC (mean differences = 0.64 ± 0.11 s, p <0.001, d = 0.42) and CP (0.66 ± 0.18 s, p = 0.004, d = 0.43) treatments. Compared to PP, participants ran faster in CC (0.80 ± 0.18 s, p = 0.001, d = 0.47) and CP (0.83 ± 0.21 s, p = 0.002, d = 0.48) treatments. All differences between mean times to complete the trials and treatments are shown in figure 1.

Differences in treatment between splits

At the 200-m split and compared to PP, participants ran faster in CC (mean differences = 0.94 ± 0.29 s, p = 0.009, d = 0.42) and CP (1.21 ± 0.38 s, p = 0.010, d = 0.57). At the 400-m split, participants ran faster in CC compared to baseline (-0.87 ± 0.25, p = 0.006, d = 0.55), PC (-0.91 ± 0.28 s, p = 0.009, d = 0.54) and PP (-1.69 ± 0.28 s, p = 0.001, d = 0.84). Similarly, participants ran faster at 400-m in CP compared to baseline (-0.68 ± 0.27 s, p = 0.031, d = 0.41), PC (-0.72 ± 0.31 s, p = 0.044, d = 0.41) and PP (-1.40 ± 0.28 s, p = 0.001, d = 0.72). At the 600-m split, participants ran faster in CP compared to baseline (-0.94 ± 0.27 s, p = 0.005, d = 0.64) and PP (-0.81 ± 0.33 s, p = 0.043, d = 0.47). Participants also ran faster at 600-m in PC compared to baseline (-1.01 ± 0.31 s, p = 0.008, d = 0.60), CC (-0.61 ± 0.23 s, p = 0.024, d = 0.36) and PP (-0.88 ± 0.33 s, p = 0.023, d = 0.46). No differences were shown between any treatments at the 800-m split (p > 0.05), but participants ran faster at 1000-m in CC compared to baseline (-1.08 ± 0.43 s, p = 0.030, d = 0.52) and PP (-0.98 ± 0.40 s, p = 0.035, d = 0.45). All differences between each treatment and split are shown in figure 2.

Differences in peak heart rate and RPE between treatments

Repeated measures ANOVA revealed no differences between treatments for HR_{peak} ($F_{(4, 40)}$ = 1.198, p = 0.327, η^2 = 0.084) and RPE ($F_{(4, 40)}$ = 0.892, p = 0.641, η^2 = 0.009). Across all treatments, mean HR_{peak} and RPE average scores ranged from 180 to 184 bpm (183.5 ± 2.3 bpm) and 9 to 10 (9.6 ± 0.4), respectively.

Discussion

We used a balanced placebo design to investigate the effect of a placebo and caffeine on pacing strategy during 1000-m running time-trials. Collectively, our results indicate that the belief of receipt of caffeine improved performance, which was associated with a significant increase in speed during the first 400-m of the time-trial. In contrast, the hidden and open administration of caffeine and placebo, respectively, did not improve performance compared

to baseline. Participants ran faster between 400- and 600-m during the hidden administration of caffeine, but time to complete the trial overall was similar to baseline.

In our study, the effect of believing to have ingested caffeine improved performance to the same magnitude as actually receiving caffeine. These findings complement previous findings in this area, in which participants were able to significantly improve their performance after being falsely informed they had received caffeine.[30,31] However, in addition to previous studies investigating the placebo effect of caffeine,[4,30-32] we also examined participants' pacing strategy during the trial, in order to establish if a change in pacing might help explain the performance improvements. Given that we informed participants that they had received caffeine in the CC and CP treatments, this information appears to have influenced their belief of how fast they could perform, influencing the goal-directed process of decision-making regarding how to distribute the available energy resources.[33] Results indicated that participants were significantly faster at 400-m than baseline and also faster at 200- and 400-m than when they were given a placebo and informed it was a placebo. This highlights that the belief of receipt of caffeine, influences the pacing strategy at the start of a 1000-m running time-trial, impacting on performance.

While both belief and actual receipt of caffeine improved performance at the start of the time-trial, only the actual receipt of caffeine improved performance in the latter stages. At 1000-m, participants ran significantly faster than baseline during the open administration of caffeine. This suggests that caffeine may offset fatigue during the final stages of a 1000-m time-trial. It has been reported that caffeine directly affects neuromuscular output,[34] which increases muscular endurance and subsequently offsets fatigue.[35] However, no improvements in performance at 1000-m were shown during the hidden administration of caffeine. Therefore, the belief of receipt of caffeine was primarily responsible for the ergogenic effect of caffeine. These results are similar to Atlas and colleagues, [36] who reported that the benefits of an opioid drug were augmented after open administration compared to hidden and to a placebo described as the drug. In the same study, follow up fMRI data revealed that drug and placebo effects activate different neurobiological pathways, suggesting that the benefits from the drug and placebo are additive. From the results reported in the present study, it could be suggested that caffeine and placebo use different neurobiological pathways that affect performance. Thus, when caffeine is administered openly, the verum and placebo components of caffeine may combine to provide a greater improvement in performance. However, while these data show additive effects for caffeine in the latter stages of the trial, it does not exclude the possibility that other treatments may show interactive effects (i.e. use the same mechanisms). A paucity of evidence in sport and exercise science is available in this area and future research needs to design studies that examines the additive or interactive effects of treatments and placebos.

Table 1 shows large variability between each treatment, which indicates that some participants may be more likely to respond to a placebo than others. It is recognised that a participant responding to a placebo can vary from study to study[1,2] and even those who do respond, may not do so consistently.[37] Researchers often focus on single-factor casual mechanisms such as expectation theory[4,31] or classical conditioning.[38,39] However, placebo effects are a manifestation of several factors, such as the context in which the treatment is administered, the person administering it, and the psychology of the athlete (e.g. personality, beliefs, and intentions). Beedie et al. [37] suggest that variability of the placebo effect can be a function of 1) an athlete's response to the verum component of a treatment (e.g. caffeine); 2)

an athletes response to the placebo component only; and 3) an athletes response to both the verum and placebo component. To increase knowledge and understanding of the placebo effect, research is needed that helps identify the mechanisms underlying the variation in placebo responsiveness.

Similar to previous research,[4,31,40] no differences in peak heart rate or perceived exertion was found between treatments. Given that the aim of a pacing strategy is to ensure physiological limits are not surpassed while performing at an optimal level,[15] a limitation of this study was that the growth curve of heart rate and perceived exertion during each trial was not measured. Future research should measure the differences in slopes of heart rate and RPE at each split to provide a better insight into the variability in intraindividual patterns of change over time between treatments.

Conclusion

In conclusion, this is the first study to show that the belief of receipt of caffeine improves 1000-m running time-trial performance on competitive level athletes. That is, believing to have ingested caffeine, improved performance to the same magnitude as actually receiving caffeine. These improvements were associated with an increase in speed during the first-part of the time-trial. While slight changes in pacing strategy were demonstrated during the mid-part of the time-trial with the hidden ingestion of caffeine, overall no changes compared to baseline were shown. Therefore, for practitioners aiming to maximise the benefits of caffeine on an athlete's performance, they should couple the administration of caffeine with a positive belief of its effectiveness to increase the likelihood of that athlete improving performance.

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Figure captions

Figure 1. Mean split time between each treatment. Note: Data are means ± 95% CI. * = $p < 0.01$ vs. CC and CP. ** = $p < 0.01$ vs. CC and CP

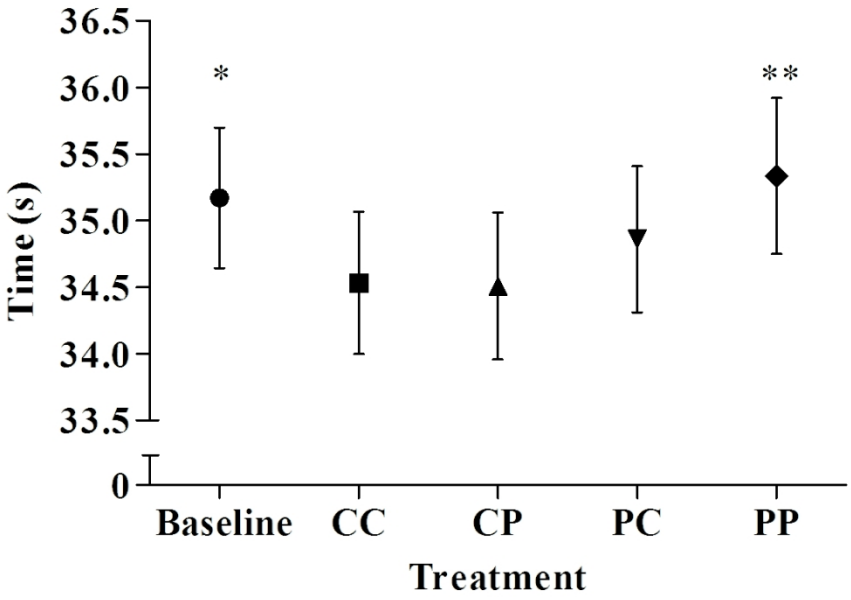
Figure 2. Differences in times between treatment and splits. Note: *PP vs. CC and CP ($p < 0.05$). **CC and CP vs. baseline, PC and PP ($p < 0.05$). #PC vs. baseline, CC and PP($p < 0.05$) and CP vs. baseline and PP ($p < 0.05$). †CC vs. baseline and PP ($p < 0.05$)

403 **Tables**

Table 1. Mean times (s) to complete 1000-m time-trials in each treatment

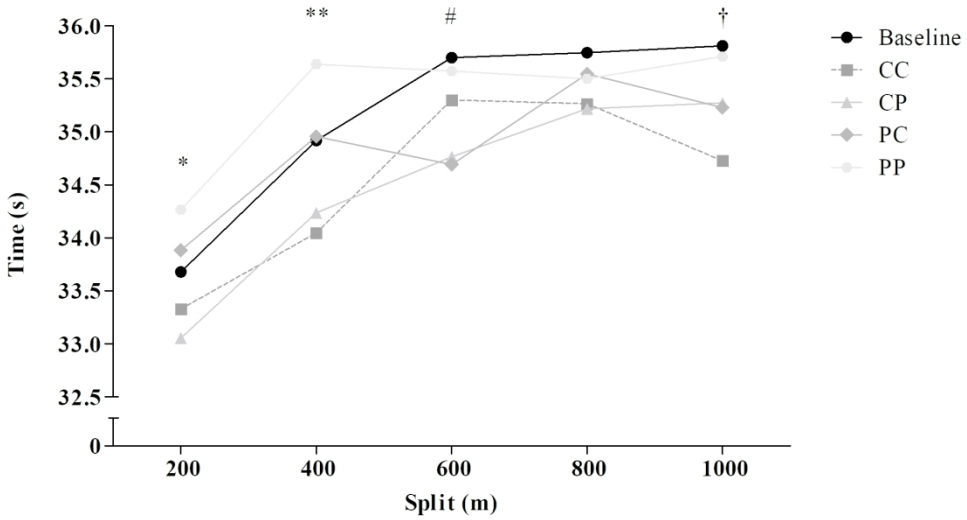
Participant	*Baseline*	*CC*	*CP*	*PC*	*PP*
1	166.9	164.3	165.4	172.1	165.1
2	187.3	182.9	180.9	187.1	193.1
3	179.4	174.9	175.4	174.7	178.2
4	176.4	171.1	170.5	173.7	175.3
5	168.4	164.1	160.3	163.0	164.8
6	180.4	178.4	178.7	177.8	184.5
7	169.3	164.6	165.9	165.9	169.8
8	166.3	162.5	163.7	164.3	168.1
9	183.3	180.4	179.2	182.4	181.6
10	175.2	173.3	173.6	173.9	179.1
11	181.8	182.8	184.4	182.4	183.9
Mean ± SEM	175.9 ± 0.55	172.7 ± 0.60	172.6 ± 0.60	174.3 ± 0.59	176.7 ± 0.68

Note: CC = Told caffeine/given caffeine; CP = Told caffeine/given placebo; PC = Told placebo/given caffeine; PP = Told placebo/given placebo



Mean split time between each treatment. Note: Data are means ± 95% CI. * = p < 0.01 vs. CC and CP. ** = p < 0.01 vs. CC and CP

109x74mm (300 x 300 DPI)



Mean split time between each treatment. Note: Data are means ± 95% CI. * = p <0.01 vs. CC and CP. ** = p < 0.01 vs. CC and CP

179x99mm (300 x 300 DPI)