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Methylphenidate Blocks Effort-Induced Depletion of Regulatory Control in Healthy Volunteers

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Abstract

A recent wave of studies—more than 100 conducted over the last decade—has shown that exerting effort at controlling impulses or behavioral tendencies leaves a person depleted and less able to engage in subsequent rounds of regulation. Regulatory depletion is thought to play an important role in everyday problems (e.g., excessive spending, overeating) as well as psychiatric conditions, but its neurophysiological basis is poorly understood. Using a placebo-controlled, double-blind design, we demonstrated that the psychostimulant methylphenidate (commonly known as Ritalin), a catecholamine reuptake blocker that increases dopamine and norepinephrine at the synaptic cleft, fully blocks effort-induced depletion of regulatory control. Spectral analysis of trial-by-trial reaction times revealed specificity of methylphenidate effects on regulatory depletion in the slow-4 frequency band. This band is associated with the operation of resting-state brain networks that produce mind wandering, which raises potential connections between our results and recent brain-network-based models of control over attention.

Keywords

methylphenidate, cognitive control, regulation, ego depletion, reaction time variability, spectral analysis, slow 4, attention, self-control, brain, psychopathology

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Humans have a remarkable ability to regulate their thoughts, motives, and behavioral tendencies. Yet it is an all-too-familiar fact that attempts at self-control often end in failure. Indeed, frequent regulation failures are the hallmark of a number of everyday problems (e.g., excessive spending, overeating) as well as psychiatric conditions (e.g., attention-deficit/hyperactivity disorder, or ADHD). Why is it that humans have unparalleled abilities for self-regulation, and yet attempts at regulation are so often unsuccessful?

A potentially important contributing factor is suggested in the recently proposed *strength* model of self-control (Baumeister, Bratslavsky, Muraven, & Tice, 1998). This model holds that the capacity to exercise sustained regulatory control is—akin to the tiring of a muscle—limited and depletable. Over the last decade, more than 100 studies have provided support for this model using the *dual-task paradigm* (Hagger, Wood, Stiff, & Chatzisarantis, 2010). During Phase 1 of this paradigm, each participant performs one of two versions of a task that are matched in all respects, except that one version requires the sustained use of regulatory control, and the other does not. In the immediately following second phase, all participants are given another task (differing from the Phase 1 task) that also demands the use of regulatory processing. These studies have reliably found that engaging in effortful regulation during Phase 1 tasks diminishes regulatory control during Phase 2.

Independently, neurobiological investigations have revealed important roles for the catecholamine neurotransmitters dopamine and norepinephrine in regulatory processing (Arnsten & Pliszka, 2011; Robbins, 2005). This view is based on multiple lines of evidence. For example, ADHD, a serious psychiatric disorder that

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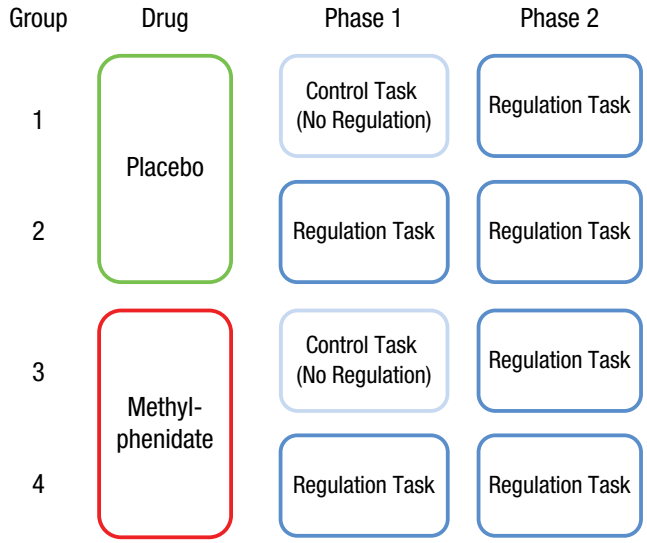

Fig. 1. Schematic illustrating the experiment's 2 × 2 design. In the dual-task paradigm, half the participants performed a task requiring effortful regulation in Phase 1, and all participants performed a regulatory task in Phase 2. This paradigm was crossed with a pharmacological manipulation in which a placebo or methylphenidate was administered 60 min prior to the start of Phase 1.

involves prominent deficits in regulatory control, is associated with distributed disturbances in the brain's catecholamine system (Bellgrove, Hawi, Kirley, Gill, & Robertson, 2005). Additionally, catecholamine-boosting psychotropic drugs reliably enhance regulatory processing (Pietrzak, Mollica, Maruff, & Snyder, 2006; Robbins, 2005). It is not currently known, however, whether acute pharmacological manipulation of brain catecholamine levels specifically affects the aforementioned phenomenon of regulatory depletion (i.e., the impairment of regulatory control resulting from prior effortful regulation).

In the study reported here, we investigated whether the depletion of regulatory control is affected by pretreatment with methylphenidate, a psychostimulant medication that reliably increases brain dopamine and norepinephrine levels (Volkow, Fowler, Wang, Ding, & Gatley, 2002). We also wanted to determine whether the effects of regulatory depletion and methylphenidate are associated with specific *spectral profiles*, that is, with specific patterns of oscillatory variation in task performance over time. We were interested in this issue because of recent findings regarding the default network, a brain network that is implicated in mind wandering and task-irrelevant thought (Mason et al., 2007; Weissman, Roberts, Visscher, & Woldorff, 2006).

The default network exhibits spontaneous oscillations at a very low frequency. It is hypothesized that the oscillatory activity of the network is manifested as variability in trial-to-trial reaction times in the so-called slow-4 frequency band (Castellanos et al., 2005)—a band that represents oscillations in the 13- to 37-s range. According to recent network-regulation models of attentional control (Castellanos & Proal, 2012; Sonuga-Barke & Castellanos, 2007), in individuals or in states associated with poor attentional control, there is insufficient regulation of the default network, which leads to elevated variability in the slow-4 band. We reasoned that if these network models are correct, then depletion of regulatory control as a result of prior effortful regulation would be associated with elevated variability, specifically in the slow-4 band (because of insufficient regulation of the default network). Further, we hypothesized that methylphenidate's effects on the depletion of regulatory control would specifically modulate this band.

Method

The experiment had a 2 × 2 design in which the dual-task paradigm was crossed with pharmacological manipulation using methylphenidate or a placebo (see Fig. 1). Guidelines suggest that for analysis-of-variance (ANOVA) designs in which factors have moderate effects, a sample size in the range of 30 participants per cell yields roughly 80% power (Van Voorhis & Morgan, 2007). We expected a large effect size for the effect of depletion with the Phase 1 task (Cohen's d = 0.8–1.0) on the basis of prior studies (Hagger et al., 2010) and pilot testing (without drug pretreatment), and we recruited 108 participants, 27 per group (mean age = 22.5 years, SD = 4.8; 68 female, 40 male; 81 Caucasian, 12 Asian, 5 African American, 7 who marked "more than one race/other," and 3 who gave no answer). Participants were recruited using a University of Michigan–sponsored online recruitment Web site. They were eligible if they were between the ages of 18 and 35 years, were not actively using psychotropic medications, and did not have any medical disorders or health symptoms (assessed through two health questionnaires) that might raise concerns for adverse effects with methylphenidate. Eighty-nine of the participants were university students or faculty.

Using a double-blind procedure, placebo or methylphenidate capsules identical in appearance were administered 60 min prior to the experiment to coincide with the window in which it was expected that methylphenidate would exert its cognitive effects (Swanson & Volkow, 2003). Participants next completed a brief practice session for the tasks while waiting for the 60-min period to elapse. Phase 1 effortful regulation was manipulated with a modified version of the letter-*e* task (Baumeister et al., 1998) lasting 7 min 30 s and consisting of 150 trials. In this task, words are shown one at a time. In the regulation version of the task, participants press a button when a word with the letter *e* is shown, but they must withhold the response if the *e* is next to or one extra letter away

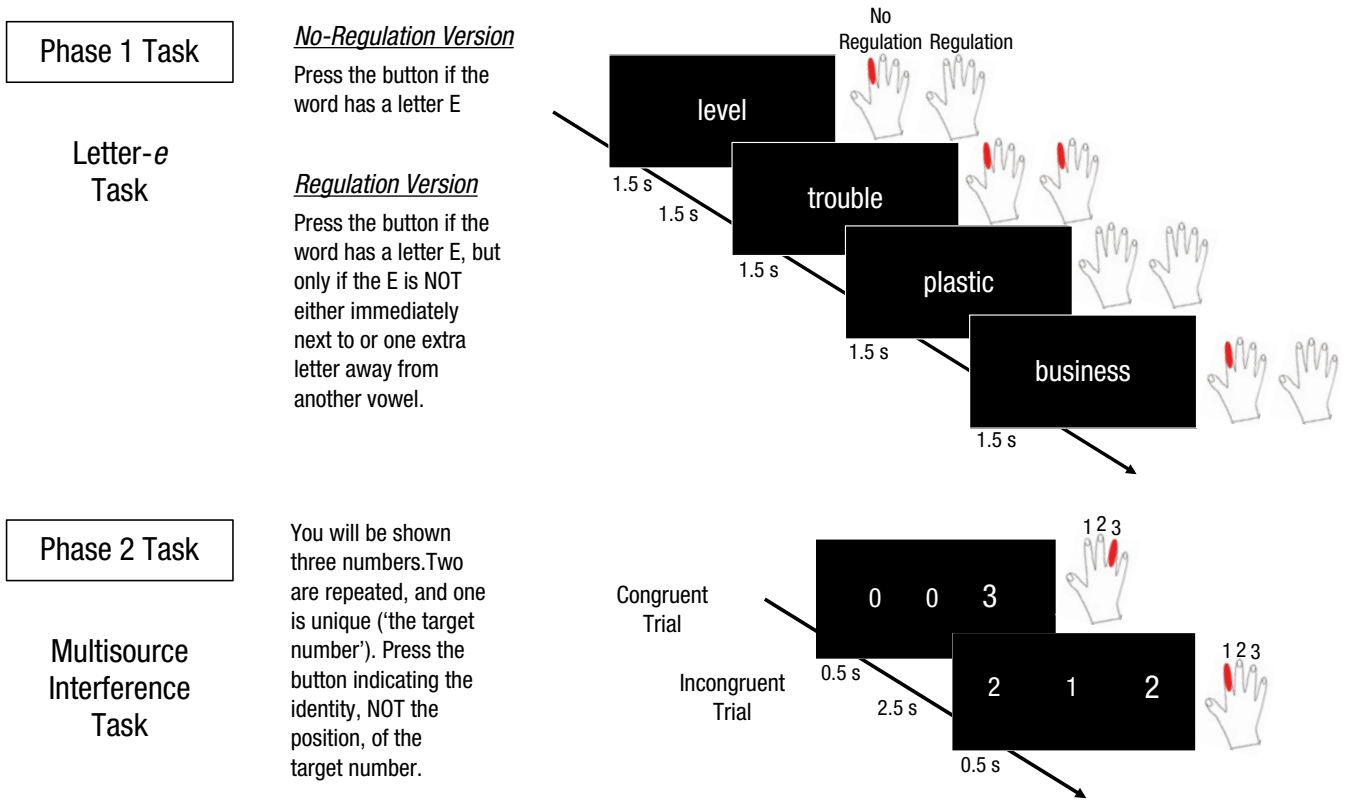

Fig. 2. Illustration of the tasks in Phases 1 and 2. In Phase 1, each participant performed either the no-regulation version or the regulation version of the letter-*e* task. In Phase 2, all participants performed the multisource interference task. During congruent trials, the position of the unique target number matched its identity (e.g., "3" in the third position), and the target appeared in a larger font than the other two numbers (both of which were always 0). During incongruent trials, position, size, and stimulus-related cues were irrelevant and thus had to be suppressed. Instructions given to participants at the start of each task are shown. For the letter-*e* task, trials consisted of a stimulus presented for 1.5 s followed by a 1.5-s response period, and for the multisource interference task, the stimulus was presented for 0.5 s and followed by a 2.5-s response period. For Phase 1, shaded fingers indicate that the correct response was a button press; for Phase 2, shaded fingers indicate which button press was the correct response.

from another vowel (Fig. 2). The no-regulation version is matched in all respects, except that participants press a button whenever a word with the letter *e* is shown, regardless of where in the word the *e* appears (i.e., no suppression of prepotent tendencies is required). The letter-*e* task was selected because it was previously shown (Hagger et al., 2010) to have among the highest effect sizes in inducing regulatory depletion.

Regulatory control was then tested at Phase 2 with a modified multisource interference task (Bush, Shin, Holmes, Rosen, & Vogt, 2003; Fig. 2, bottom) lasting 10 min and consisting of 200 trials, 100 congruent and 100 incongruent, presented in an interspersed, pseudo-random order. In this task, three single-digit numbers between 0 and 3 are presented horizontally on screen. Two numbers are the same, and one (the target) is unique. Participants have to press one of three response buttons that corresponds to the identity of the target number (rather than to the target's position on screen). On congruent trials, the target number's identity corresponds to its position (e.g., the target number is 3, and it appears in the third position to the far right). This number appears in a larger size than the other two numbers, and the other numbers are always 0. During incongruent trials, the target number's identity does not correspond to its position (e.g., the target number is 1, and it appears in the second position). On these trials, the digit that is larger than the other two need not be the target number (any one the three digits might be of larger size), and the other two numbers are not 0. Thus irrelevant position, size, and stimulus-related cues must be suppressed. For the letter-*e* task, trials began with a stimulus presented for 1.5 s, followed by a 1.5-s response period, after which the next trial immediately began. For the multisource interference task, the stimulus was presented for 0.5 s, followed by a 2.5-s response period prior to the start of the next trial.

The primary dependent measure was reaction time variability (RTV) during the Phase 2 task. RTV has been hypothesized to reflect levels of attentional control (Castellanos et al., 2005; Douglas, 1999). Control over attention is required to maintain task-directed focus and

prevent the emergence of task-irrelevant spontaneous thoughts (Weissman et al., 2006). When attentional control is reduced, this leads to more frequent waning or lapsing of attention, which in turn produces variability in trial-to-trial reaction times (Douglas, 1999).

Variability in reaction times was calculated using ex-Gaussian modeling (Dawson, 1988). In a pure Gaussian model, the distribution of reaction times can be characterized in terms of two parameters representing the mean (μ) and standard deviation (σ), where σ represents the variability of reaction times across trials. Observation of actual reaction time distributions, however, shows that they are significantly positively skewed. They have sharp left boundaries, due to the smaller number of very short reaction times, and long right tails, due to the larger number of very long reaction times. This skewed shape has been found to be well fit with ex-Gaussian models (Dawson, 1988), derived from summing a Gaussian and an exponential distribution. This model is parameterized with Gaussian mean μ and standard deviation σ. In addition, there is a second variability parameter, τ, that is associated with the exponential distribution and that captures the longer right tail. Because we were interested in how fatigue and methylphenidate affected the overall variability of reaction times across trials, RTV was calculated as the sum of the two variability parameters, σ and τ.

Participants were included in the analysis according to accuracy and outlier criteria commonly applied to studies employing RTV as the dependent measure (Karalunas, Huang-Pollock, & Nigg, 2013): greater than 80% accuracy (on both Phase 1 and Phase 2 tasks) and reaction time and RTV within 2 standard deviations for the Phase 2 task. Applying these criteria left 94 participants for the analysis (23 in each of two cells and 24 in each of two cells). RTV was calculated for trials with accurate responses only; reaction times for inaccurate responses and nonresponses were not included.

Fast Fourier transform (FFT), a spectral-decomposition method, was used to identify patterns of temporal variation in the sequence of trial-by-trial reaction times. In intuitive terms, if a participant has spikes in his or her reaction times every 20 s during the task, then FFT will correspondingly register elevated power at the 20-s frequency. We were particularly interested in power at neurophysiologically validated frequency bands (Penttonen & Buzsaki, 2003), which have been investigated in prior research on reaction times (Karalunas et al., 2013). We had an a priori hypothesis about the slow-4 band, which represents oscillations in the 13- to 37-s range (see the Introduction). The slow-3 band, which represents oscillations in the 5- to 13-s range, was chosen as a comparison band because this band plausibly contains variation related to cognitive processing of tasks, because it has been used as the comparison band in most previous studies (see Karalunas et al., 2013, for a review), and because other frequency bands (e.g., slow-2) contain oscillations too rapid to detect with standard tasks.

FFT was performed with subsequent trapezoidal integration of the power spectrum within slow-4 and slow-3 bands. In accordance with the procedures described elsewhere (Karalunas et al., 2013), prior to FFT analysis, reaction times were log-transformed, and the standard deviation of the reaction times was normalized to 1 for every participant to account for inequality of variances across conditions and to provide centering for tests of interaction.

Results

For incongruent trials, an ANOVA with drug (placebo vs. methylphenidate) and prior regulation (control task vs. regulation task) as factors revealed a significant Drug × Prior Regulation interaction, $F(1, 90) = 4.64$, $p = .03$, $\eta^2 = .045$, which indicates that prior regulation (i.e., during Phase 1) produced statistically different effects during Phase 2 in participants who received the placebo than in participants who received methylphenidate (Fig. 3a). In participants who received the placebo, performing the regulation task in Phase 1 produced significantly greater RTV at Phase 2 than did performing the control task, $t(45) = 2.38$, $p = .02$, $d = 0.69$, with no change in mean reaction time ($p = .65$). In participants who received methylphenidate, however, prior regulation did not affect Phase 2 RTV ($p = .57$). Similar patterns of results were observed for the σ and τ variability parameters taken individually (Fig. 3b). The distributions of reaction times for participants who completed a regulation task during Phase 1 are shown in Figure 3c. The distribution was wider (consistent with greater σ) and had a longer tail (consistent with greater τ) for participants who received the placebo than for those who received methylphenidate.

For congruent trials, neither methylphenidate, prior regulation, nor their interaction significantly affected RTV in Phase 2 (all ps $> .23$). A test of the three-way Trial Type (congruent vs. incongruent) × Drug × Prior Regulation interaction revealed a significant effect (trend level) for the σ variability parameter, $F(1, 90) = 3.00$, $p = .09$, $\eta^2 = .009$. This interaction was driven by the fact that in participants who received the placebo, prior effortful regulation elevated both σ and τ for incongruent trials in Phase 2, whereas it elevated only τ for congruent trials in Phase 2; for both kinds of trials, methylphenidate blocked these effects of regulation. This three-way interaction was not significant for τ ($p = .47$). Crucially, reduced RTV during Phase 2 incongruent trials was not achieved by compromising accuracy, as there was a small but statistically

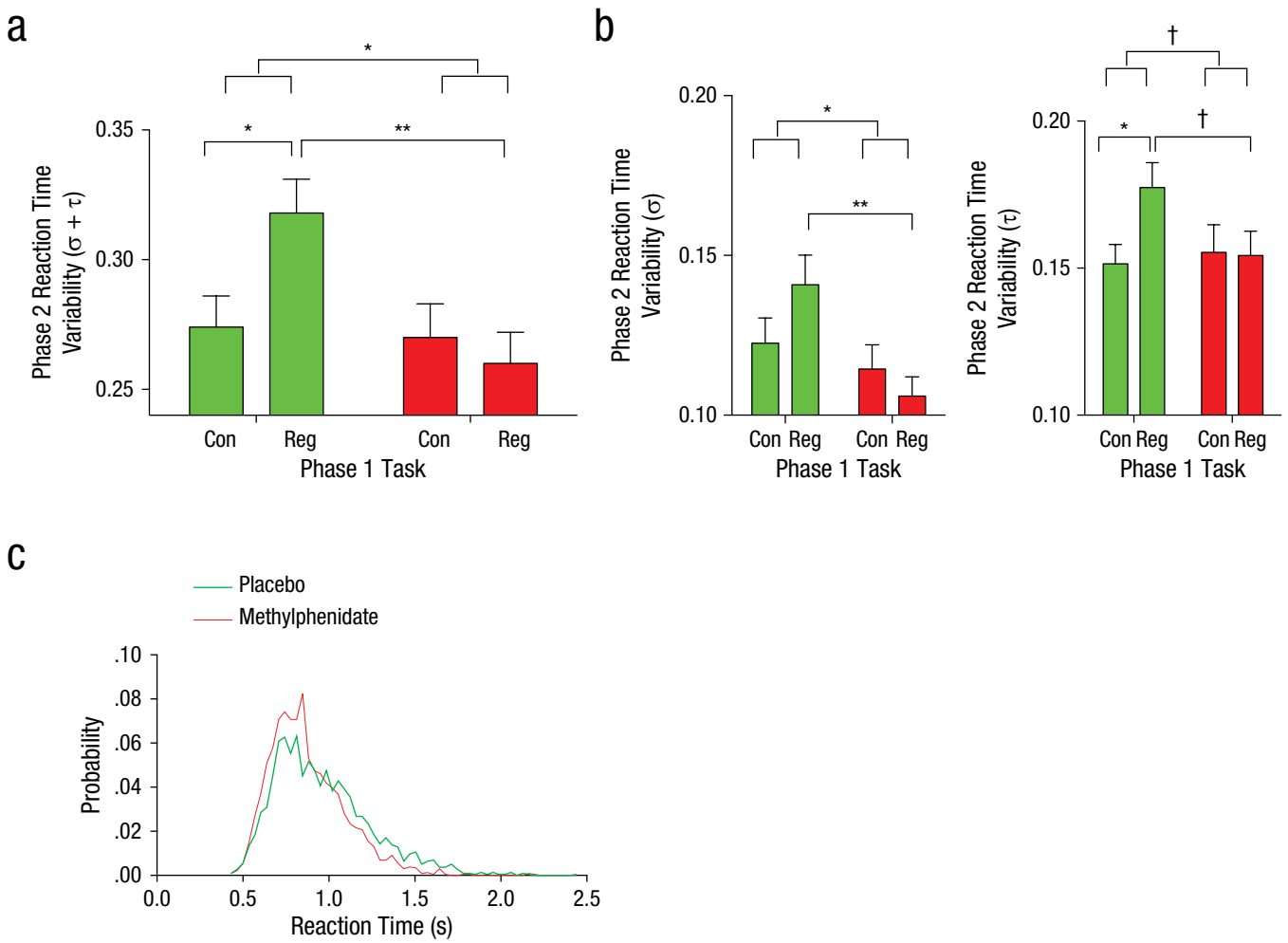

Fig. 3. Effects of methylphenidate and prior regulation on reaction time variability in incongruent trials. Mean reaction time variability during Phase 2 is shown in (a) as a function of task completed in Phase 1 (Con = control task, Reg = regulation task) and type of drug administered. Combined results are shown for the σ (Gaussian variability) and τ (exponential variability) parameters. In (b), results from (a) are broken down separately for σ and τ. In (a) and (b), asterisks indicate significant differences between groups (*$p < .05$, **$p < .01$), the dagger indicates a marginally significant difference between groups ($^{\dagger}p < .1$), and error bars represent standard errors of the mean. The graph in (c) represents the reaction time distributions during Phase 2 for participants who had completed the regulation task in Phase 1. Results are shown separately for participants who received the placebo and those who received methylphenidate.

significant improvement in mean accuracy among participants receiving methylphenidate—main effect of drug: $F(1, 90) = 7.07$, $p < .01$, $\eta^2 = .045$—and this improvement did not differ as a function of prior regulation (Drug × Prior Regulation interaction: $p = .87$).

Using spectral analysis (Fig. 4), we observed a three-way Band (slow 4 vs. slow 3) × Drug × Prior Regulation interaction, $F(1, 90) = 4.20$, $p = .04$, $\eta^2 = .012$. In the slow-4 band, there was a significant Drug × Prior Regulation interaction, $F(1, 90) = 5.43$, $p = .02$, $\eta^2 = .057$. With placebo, prior regulation increased slow-4 power, $t(45) = 1.69$, $p = .10$, $d = 0.49$, but with methylphenidate, slow-4 power decreased, $t(45) = -2.38$, $p = .11$, $d = 0.47$. In the slow-3 band, these effects were not observed (main effect of drug, main effect of prior regulation, and Drug × Prior Regulation interaction: all ps > .29). Means and standard deviations for main dependent variables, as well as additional simple effects and interactions, are presented in Tables S1 and S2, respectively, in the Supplemental Material available online.

Discussion

This study advances the field's knowledge of the neurobiological basis of the depletion of regulatory control in two ways. First, we provided evidence for the first time that regulatory depletion that arises in the widely used dual-task paradigm can be pharmacologically manipulated: Pretreatment with the catecholamine-boosting agent methylphenidate blocks the depletion of regulatory

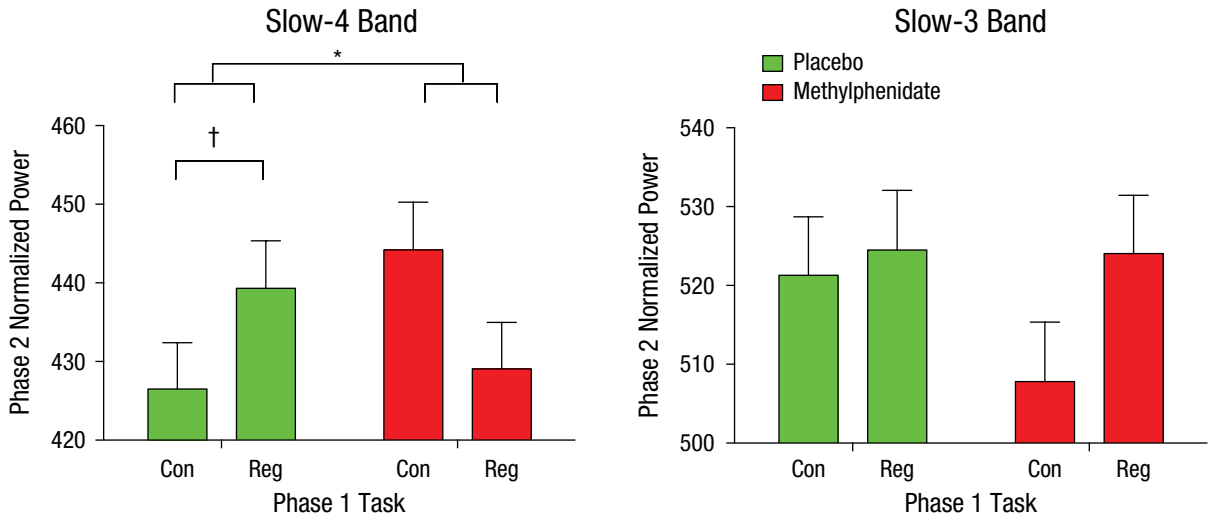

Fig. 4. Normalized power during Phase 2 as a function of task completed in Phase 1 (Con = control task, Reg = regulation task) and type of drug administered. Results are shown separately for the slow-4 and slow-3 power bands. The asterisk indicates a significant difference between groups ($p < .05$), the dagger indicates a marginally significant difference between groups ($p < .1$), and error bars represent standard errors of the mean.

control. Second, we utilized spectral-decomposition methods, which have not previously been applied to regulatory depletion. We found that prior regulation in Phase 1 increased spectral power in Phase 2 specifically in the slow-4 frequency band, a band associated with oscillations of the brain's default network, whereas methylphenidate reversed this effect. This finding is intriguing because it draws novel connections between regulatory depletion and emerging brain-network models of attentional control.

One hypothesis for explaining why methylphenidate blocks the depletion of regulatory control appeals to the drug's more general effects in increasing alertness and arousal. There is a large literature in psychology and ergonomic research examining the effects of methylphenidate and related compounds, such as amphetamine, on "vigilant" attention (Langner & Eickhoff, 2013). These studies nearly always used protracted, simple, monotonous tasks and showed that participants receiving these drugs were more alert and made fewer errors than participants who received a placebo (see Koelega, 1993, for a review). We believe, however, that this general-arousal hypothesis is unlikely to explain the results of the current study. First, in contrast to extended tasks used in prior research, we used relatively brief tasks that do not normally produce significant decrements in alertness and arousal due to time on task. Second, in contrast to prior studies that used task accuracy as the primary dependent measure, we used variability of reaction time, which is hypothesized to reflect one's level of attentional control specifically rather than arousal generally (Douglas, 1999).

Third, and perhaps most important, we manipulated the exertion of regulatory control during Phase 1 of the dual-task paradigm and found that methylphenidate's effects on variability of reaction times during Phase 2 were observed only in participants who had previously engaged in effortful regulation. Fourth, there was evidence for specificity of methylphenidate effects in incongruent trials, which require cognitive regulation, compared with congruent trials, which do not. In particular, after prior regulation, methylphenidate reduced both variability parameters of the ex-Gaussian distribution, σ and τ. However, it decreased σ in incongruent trials more than in congruent trials (whereas it decreased τ on both types of trials). These observations taken together suggest that methylphenidate had at least some specific effects on the depletion of regulatory control due to prior regulatory exertion and are hard to explain with the hypothesis that methylphenidate has only general, nonspecific effects on alertness and arousal.

Of note, it is often claimed that the individual parameters of the ex-Gaussian distribution have specific psychological interpretations. For example, it has been proposed that the Gaussian parameters (μ and σ) represent the perceptual phase of task processing, whereas τ represents the decision and action phase (Gordon & Carson, 1990). This interpretation, however, is based on limited evidence and remains controversial (Matzke & Wagenmakers, 2009). Further investigation into the psychological factors that drive σ versus τ variability will help to further clarify methylphenidate's apparent protective effects against regulatory depletion.

In contrast to the general-arousal hypothesis, an alternative hypothesis proposes relatively specific effects of methylphenidate on top-down regulatory processing. Effortful regulation of automatic responses is thought to be implemented by a set of relatively discrete prefrontal circuits, including regions in the dorsal and lateral prefrontal cortex and anterior cingulate (Heatherton & Wagner, 2011). In previous studies, regulatory depletion has been found to reduce activation of these prefrontal circuits (Richeson et al., 2003; Wagner & Heatherton, 2013). Catecholamine-boosting agents, in contrast, are known to enhance the effectiveness of these control circuits (Solanto, 2002), consistent with a large body of neuroimaging evidence (Costa et al., 2013; Rubia et al., 2011). If this picture is correct, then augmentation of brain catecholamines by methylphenidate might block the depletion of regulatory control by delivering a performance boost to the specific prefrontal control circuits whose functioning is normally compromised by prior regulatory exertion.

This study also draws links between regulatory depletion and recent network models of attentional control (Castellanos & Proal, 2012; Sonuga-Barke & Castellanos, 2007). According to these models, an important job for regulatory control circuits is to suppress the default network (Anticevic et al., 2012), a brain network involved in task-irrelevant spontaneous thoughts and that exhibits spontaneous oscillations in activity in the slow-4 frequency band. Our finding that depletion of regulatory control increases power in the slow-4 band might thus be interpreted in terms of enhanced default-network oscillatory activity, which in turn might be explained by diminished regulatory control over this network. Moreover, our finding that methylphenidate reverses this effect suggests a potential role for brain catecholamines in enhancing regulatory control over the default network. These hypotheses linking regulatory depletion with increased default network activity warrant direct investigation in future pharmaco-imaging studies.

In sum, effort-induced depletion of regulatory control is potentially important in explaining self-control failures in day-to-day life and in psychiatric disorders. Our results provide new insight into the physiological mechanisms by which effort-induced depletion occurs and could serve as a starting point for new lines of brain-based investigations into regulatory control failures.

Author Contributions

C. Sripada developed the study concept, tasks, and design. D. Kessler assisted with programming the tasks. Testing and data collection were performed by C. Sripada and D. Kessler. All authors analyzed and interpreted the data. C. Sripada drafted the manuscript, and D. Kessler and J. Jonides provided critical revisions. All authors approved the final version of the manuscript for submission.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at http://pss.sagepub.com/content/by/supplemental-data

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