The creative brain
Some insights into the neural dynamics of flexible and persistent creative processes

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CHAPTER 6

Methylphenidate Does not Affect Convergent and Divergent Creative Processes in Healthy Adults

Abstract

An increasing number of healthy people use methylphenidate, a stimulant that increases dopamine and noradrenaline levels in the brain, to help them focus over extended periods of time. While methylphenidate may indeed facilitate cognitive functions related to cognitive stability, such as working memory and persistence, it may impair processes characterized by cognitive flexibility, including the flexible processes that contribute to creativity. In this study, we investigated whether methylphenidate affected convergent and divergent creative processes in a sample of healthy participants. Also, we assessed whether such effects depended on individual differences in ADHD symptoms and working memory capacity. Contrary to our expectations, methylphenidate did not affect participants' creative performance on any of the tasks. Also, methylphenidate effects did not depend on individual differences in hyperactivity–impulsivity and baseline working memory capacity. Thus, although methylphenidate effects on creativity may be underestimated in our study due to several methodological factors, our findings do not suggest that methylphenidate impairs people's ability to be creative.

In recent years, the number of people using methylphenidate (a psychostimulant that increases levels of dopamine and noradrenaline in the brain) has strongly increased (De Jongh, Bolt, Schermer, & Olivier, 2008; Maher, 2008; Smith & Farah, 2011). While some of these people use this drug as prescribed medication to treat symptoms of attention-deficit/hyperactivity disorder (ADHD), an increasing number of healthy people use methylphenidate and similar stimulants as cognitive enhancers to boost their ability to concentrate over extended periods of time (Cakic, 2009; Greely et al., 2008; Maher, 2008). While the term *cognitive enhancer* (also referred to as *smart pills*, *cosmetic neurology*, or *botox for the brain*) implies that the effects of such enhancers are unquestionably beneficial, this assumption may be overly optimistic. Indeed, these substances may facilitate cognitive processes that support cognitive stability, such as working memory, vigilance, and response inhibition (e.g., Linssen, Sambeth, Vuurman, & Riedel, 2014; Marquand et al., 2011; Minzenberg & Carter, 2008), but they may simultaneously impair flexible cognitive processes, such as those that contribute to creativity (Mohamed, 2016; Müller et al., 2013).

Creativity, the ability to come up with outcomes (e.g., ideas, poems, problem solutions) that are both original and useful, benefits from cognitive flexibility (e.g., Chermahini & Hommel, 2012; Nijstad et al., 2010). Flexible creative processes include seeing associations between concepts that are only remotely related, switching between different task approaches, and divergent thinking (generating multiple ideas in response to open-ended questions; Guilford, 1967). Alternatively, creative outcomes may result from more analytical, persistent processes that require sustained attention and perseverance, such as thinking along a certain line and convergent thinking (the recombination of familiar and closely related information into novel ideas according to certain rules) (Cropley, 2006; Lucas & Nordgren, 2015; Nijstad et al., 2010; Roskes et al., 2012). In real life, creativity probably results from a balance between flexible and persistent creative processes rather than one or the other – a balance that seems to be strongly modulated by dopamine (Boot et al., 2017b; Hommel & Colzato, 2017).

The neurotransmitter dopamine plays a major role in the cognitive control of goal-directed behavior by regulating the balance between cognitive flexibility and stability (Cools & D'Esposito, 2011; Cools et al., 2007; Frank et al., 2001; McNab & Klingberg, 2008). While dopamine in the prefrontal cortex seems to facilitate cognitive stability (e.g., the manipulation of information in working memory, suppression of task-irrelevant information), dopamine in the subcortical striatum supports cognitive flexibility (e.g., task switching, updating of representations in working memory). Similarly, dopamine seems to modulate flexible and persistent processes in creativity, as indicated by an increasing number of studies (e.g., Chermahini & Hommel, 2012; Mayseless et al., 2013; Zabelina et al., 2016a; Zhang et al., 2014b), although direct evidence for dopaminergic modulation of creativity is lacking at present (Boot et al., 2017b). For example, genetic variations in prefrontal and striatal dopamine availability are associated with differences in flexible and persistent creative performance (Mayseless et al., 2013; Zhang et al., 2014a, 2014b). By increasing dopaminergic (and noradrenergic) activity in fronto-striatal brain areas (Arnsten & Dudley, 2005; Bymaster et al., 2002; Kuczenski & Segal, 1997), methylphenidate may affect flexible and persistent creative processes in opposing ways. By improving the ability to focus, methylphenidate may facilitate persistent creative processes, but, at the same time, this may be detrimental to the cognitive flexibility that other creative processes require. Thus, the effects of methylphenidate on creativity may depend on the specific creative processes required for the task at hand.

Moreover, the effects of dopamine on cognition seem to follow an inverted-U-shaped function (Cools & D'Esposito, 2011; Durstewitz & Seamans, 2008; Vijayraghavan et al., 2007). While both low and high levels of dopamine are associated with impaired cognitive performance, medium dopamine levels are associated with optimal performance. This implies that the effects of methylphenidate and similar substances may depend on individuals' baseline dopamine levels (Clatworthy et al., 2009; Cools et al., 2007; Frank & O'Reilly, 2006; Kimberg, D'Esposito, & Farah, 1997; Mehta et al., 2000; Van der Schaaf, Fallon, Huurne, Buitelaar, & Cools, 2013). For example, Cools and colleagues (2007) showed that the effects of bromocriptine (a dopamine agonist) on

working memory performance depended on people's impulsivity, a personality trait associated with both dopaminergic functioning and enhanced creativity (Boot et al., 2017d; Feist, 1998). In this study, bromocriptine enhanced the updating of information in working memory in highly impulsive participants with low baseline working memory capacity, while it did not affect working memory performance in low-impulsive participants with high baseline working memory capacity. Similarly, moderate levels of dopamine seem to be associated with optimal creative performance, while both low and high dopamine levels are associated with suboptimal performance (Boot et al., 2017b; Chermahini & Hommel, 2010, 2012). Therefore, methylphenidate effects on creativity may also depend on individual differences in baseline dopaminergic functioning, as indicated by their working memory capacity or hyperactivity–impulsivity, one of the core symptoms of ADHD that has been associated with enhanced creativity (Boot et al., 2017d).

In the present study, we set out to investigate methylphenidate effects on convergent and divergent processes in creativity in a sample of healthy participants and we tested whether such effects depend on individual differences in hyperactivity–impulsivity symptoms of ADHD and working memory capacity. Along the same lines, a recently published study showed that the effects of methylphenidate on divergent thinking in a sample of healthy participants depended on participants' baseline levels of novelty seeking, an aspect of impulsivity that is also associated with dopaminergic functioning (Gvirts et al., 2016). In this study, methylphenidate enhanced divergent thinking in participants with lower baseline novelty seeking scores (and presumably reduced baseline levels of striatal dopamine; Zald et al., 2009), but decreased divergent thinking in participants with higher baseline novelty seeking scores (and elevated levels of striatal dopamine at baseline). Similarly, we expected that methylphenidate would increase divergent thinking in people with low hyperactivity–impulsivity symptoms and high baseline working memory capacity, but that it would decrease divergent thinking in people with high hyperactivity–impulsivity scores and low baseline working memory capacity. However, we expected that methylphenidate would increase convergent thinking in highly hyperactive–impulsive people and low baseline working memory

capacity, but that it would decrease convergent thinking in people with low hyperactivity–impulsivity scores and high baseline working memory capacity.

Method

Participants, Design, and Procedure

We recruited 48 right-handed Dutch native speakers (28 females) with a mean age of 22.12 years (SD = 2.39) to take part in this double-blind placebo-controlled randomized within-subjects study. All but one participant had completed or was currently attending some form of higher education (i.e., university or comparable). Sample size was determined based on a power analysis in G*Power (Faul et al., 2007) with an α of 0.05 and a power of 0.90, indicating that a sample of at least 44 participants would be required to detect pharmacological effects of moderate size (η^2_p = 0.05 – 0.22; De Dreu et al., 2014) on the variables of interest. Participants were extensively screened prior to participating in the study to exclude any (history of) physical or mental illness that could affect their response to methylphenidate. A complete list of the exclusion criteria is shown in Appendix II. Participants received €100 for their participation.

Participants took part in two sessions during which they received a capsule with 20 mg of methylphenidate or a visually identical placebo. The two sessions were scheduled at least one week apart to ensure drug washout. Methylphenidate has a half-life of 2 to 3 hours (Kimko, Cross, & Abernethy, 1999). The two sessions for each subject started at the same time of day and all sessions ended before 5 pm, because dopamine levels naturally rise in the evening (Barbato, Ficca, & Muscettola, 2000). The order of the drug and placebo sessions was randomized across participants. Participants were asked to refrain from drinking alcohol and smoking cigarettes within the 24 hours prior to the sessions. Moreover, they were not allowed to drink caffeinated drinks on the testing days. Light snacks were provided during the sessions.

The present experiment was part of a larger study that included language and working memory tasks (including EEG recordings). The data of these tasks are not reported here, because they are not relevant for the present research

questions. Session 1 lasted for approximately 5 hours. At the start of this session, participants were subjected to a medical screening, consisting of a medical history checklist and blood pressure measurement. Subsequently, participants completed baseline measures of working memory capacity, current ADHD symptoms, (self-reported) creative abilities, and alertness. Approximately 1 hour after administration of the capsule containing either 20 mg methylphenidate or a placebo, participants started a series of tasks including an alertness measure, a language task, and several working memory tasks. Methylphenidate levels peak around 2 hours after oral administration (Kimko et al., 1999). About 2.5 hours after capsule administration, participants completed three creativity tasks that took about 20 minutes to complete. Over the course of the session, participants' blood pressure and heart rate were assessed four times: 1) during the medical screening (approximately 1 hour before capsule administration), 2) directly before capsule administration, 3) approximately 1 hour after capsule administration and 4) at the end of the session (approximately 3 hours after capsule administration). At Time 1, 3 and 4, participants' subjective mood was also assessed. Session 2 did not include a medical screening and baseline measures, but was otherwise identical to Session 1 and took approximately 4 hours.[1]

Creativity Tasks

In both sessions, participants were presented with three creativity tasks, the order of which was counterbalanced across participants.

Alternate Uses Task

We measured divergent thinking using the Alternate Uses Task (AUT; Guilford, 1967). In four separate two-minute trials per session, we asked participants to generate as many new, original ways to use a common object as they could think of. The two sets of four objects (Set 1: brick, chord, towel, book; Set 2: newspaper, can, cardboard box, belt) were matched in terms of the (variance in) flexibility and originality of ideas that were generated in a previous study (data not presented here). Within each session, the objects were presented in randomized order. The order of the sets across sessions was counterbalanced across participants. Subsequently, two trained coders, blind to the conditions

that the ideas belonged to, scored participants' ideas in terms of fluency (the number of non-redundant ideas), flexibility (the number of conceptual categories the ideas belonged to), and originality (the extent to which an idea is uncommon). To obtain a measure of flexibility, ideas were categorized into different conceptual categories. For example, for the newspaper, the idea "to hit somebody" is coded in the category 'as a weapon', whereas the idea "use to light up the fireplace" is coded in the category 'as fuel'. To obtain a measure of originality, each idea was scored for the extent to which it was novel and uncommon on a 5-point scale (1 = *not original at all*, 5 = *very original*). Originality ratings were averaged across all ideas for each individual to correct for differences in fluency. Interrater reliability for both flexibility (Cohen's κs > .96, ps < .001) and originality (*ICC*s > .86, ps < .001) was good. For each session, fluency, flexibility and originality scores were averaged across the four objects into reliable indices of divergent thinking ($\alpha_{fluency}$ > .84, $\alpha_{flexibility}$ > .71, $\alpha_{originality}$ > .76). Across sessions, participants generated an average of 7.93 ideas (*SD* = 2.63) in 5.68 categories (*SD* = 1.53) with an average originality of 1.83 (*SD* = 0.26), which is comparable to the ideas generated in other studies (e.g., Boot et al., 2017d).

Remote Associates Test

We assessed convergent thinking using the Remote Associates Test (RAT: Mednick, 1962). This task required participants to identify associations among words that are not obviously connected. In each session, participants received a set of 10 items in which they were given three words (e.g., *jar*, *stain*, *blue*) and had to generate a word that associated with all of them (i.e., *ink*). The two sets of items were matched in terms of difficulty based on the solution rate in previous datasets and the order of sets was counterbalanced across participants. This task was self-paced and participants could skip an item if they were unable to retrieve its correct solution. The number of correctly solved items was our measure of convergent thinking. On average, participants solved 5.40 items (*SD* = 1.53) across sessions.

Alternate Names Task

We measured rule divergent and rule convergent processes in creativity using the recently developed Alternate Names Task (Boot et al., 2017a), an adaptation of the Pasta task (De Dreu et al., 2014; Dijksterhuis & Meurs, 2006; Marsh et al., 1999). During this task, participants were asked to generate as many new names as possible for items in a specific category within one minute, given three examples of non-existing names, all ending with the same letter(s). For example, examples in the category 'martial arts' were 'nikato', 'kai do', and 'sadamo'. From their responses, indices for rule convergent thinking (the number of items ending with the cue in the instructions, i.e., 'o') and rule divergent thinking (the number of items not ending with an 'o') could be created. Moreover, we created indices for repetitions (the number of times in which participants consecutively generated names with the same ending), switches (number of times in which participants switched from one ending, e.g., 'e', to another ending, e.g., 'a'), and the number of unique name endings (Boot et al., 2017a; De Dreu et al., 2014). During each session, participants generated new names for two sets of five categories. The sets were matched in terms of rule divergence and convergence based on previous datasets. The order of sets across sessions was counterbalanced across participants. We removed existing names and duplicates from the data prior to data analysis. Two additional participants were excluded from this particular analysis, because they generated too few valid ideas ($M < 1.5$) during at least one of the sessions.

Because the resulting variables were strongly skewed, we log-transformed them to approach a normal distribution. Convergent names associated positively with category repetitions ($r = .98$, $p < 0.001$), and both were z-transformed and aggregated as a measure of convergent ideation. Similarly, divergent thinking, category switches, and the number of non-redundant endings were z-transformed and formed a reliable index of divergent ideation ($\alpha = 0.84$).

ADHD Symptoms

We assessed hyperactivity–impulsivity and inattention symptoms of ADHD using the 23-item ADHD rating scale for adults (Kooij et al., 2005). For each ADHD symptom, participants rated its frequency in the past 6 months using a 4-point scale, from 1 (never or rarely) to 4 (very often). Sample items of the

hyperactivity–impulsivity subscale are "difficulty awaiting turn" and "interrupt or intrude on others". Sample items of the inattention subscale are "easily distracted" and "difficulty organizing tasks and activities". Reliability of the total scale (α = .82) and subscales ($\alpha_{hyperactivity\text{-}impulsivity}$ = .76; $\alpha_{inattention}$ = .73) was good. On average, participants reported a total ADHD score of 1.73 (SD = 0.30, range = 1.26-2.48), a hyperactivity–impulsivity score of 1.82 (SD = 0.39, range = 1.17-2.92), and an inattention score of 1.65 (SD = 0.32, range = 1.09-2.27).

Working Memory Capacity

Baseline working memory capacity was assessed using a Dutch version of the automated reading span (Daneman & Carpenter, 1980; Unsworth, Heitz, Schrock, & Engle, 2005). In this task, participants were required to judge whether presented sentences made sense or not by clicking a button saying 'True' or 'False'. Following each sentence, the participant was asked to memorize a letter that appeared on the screen. After sets of 3 to 7 sentence–letter combinations, participants were asked to report back the to-be-remembered letters in the current set in the correct order. In total, the task contained 75 sentence judgements and letters to be remembered. The order of the set sizes (each appearing three times) was randomized across participants. Performance on this task was scored as the total number of correctly recalled letters during this task (M = 60.09, SD = 9.70, range = 32 − 75).

Alertness

To assess potential drug effects on general alertness and vigilance, participants completed two control tasks in both sessions. Prior to capsule administration, participants completed the box completion task (Salthouse, 1996). During this paper–and–pencil task, participants were presented with ten rows of ten square boxes that were still open on one of the sides. Participants were required to close all squares as quickly as possible by drawing a line. Performance on this task was scored in terms of total completion time. In addition, participants completed the number cancellation task (Lewis & Kupke, 1977) approximately one hour after capsule administration. This paper–and–pencil task required participants to scan 28 rows of 35 digits and to cross out all numbers 6 and 9 while ignoring all other digits. Performance on the number

cancellation task was scored in terms of total completion time and the number of missed targets.

Subjective Mood and Physical Symptoms

Subjective mood and current physical symptoms were assessed using visual analogue scales (Bond & Lader, 1974). Participants rated their subjective mood on 16 dimensions (e.g., 'tense – relaxed, 'muzzy – clear-headed') by moving a slider between opposite ends of the dimensions. We calculated factors for *alertness*, *contentedness*, and *calmness* as suggested by Bond and Lader (1974). Similarly, participants rated to what extent they were currently experiencing each of 10 physical complaints (e.g., headache, dry mouth, dizziness).

Data Analysis

We tested our hypotheses using mixed linear models with within-subjects factors Drug (methylphenidate *vs.* placebo) and Order of treatment (methylphenidate in Session 1/placebo in Session 2 vs. the reverse). We included random intercepts to account for these within-subjects factors. To assess whether effects depended on participants baseline working memory capacity or hyperactivity–impulsivity symptoms, we included these (centered) scores and their interactions with Drug and Order as covariates in separate analyses.[2] We applied a Bonferroni correction for multiple comparisons based on the number of dependent variables in our study (α = .05/6 = .008). Drug effects on general alertness, mood, and blood pressure were assessed using repeated measures analyses of variance (ANOVAs) with within-subjects factors Drug (methylphenidate vs. placebo) and Time (pre vs. post capsule administration).

Results

Methylphenidate Effects on Alertness, Mood, and Physiology

Participants' performance on the box completion task indicated that there were no differences in general alertness between sessions prior to capsule intake ($t(45)$ = 0.71, p = .485). Moreover, performance on the number cancellation task indicated that methylphenidate did not affect participants' alertness, either in terms of completion time ($t(45)$ = −0.16, p = .875) or number of omission errors

($t(45)$ = −1.58, p = .121). Methylphenidate did not affect participants' subjective alertness ($F(1,44)$ = 1.68, p = .202, η^2_p = .04), contentedness ($F(1,44)$ = 0.15, p = .705, η^2_p < .01), calmness ($F(1,44)$ = 1.01, p = .321, η^2_p = .02), or the degree of physical complaints ($F(1,44)$ < .01, p = .979, η^2_p < .01) reported on the visual analogue scales.

As expected, methylphenidate did affect participants' systolic ($F(1,44)$ = 9.44, p = .004, η^2_p = .17) and diastolic ($F(1,44)$ = 5.25, p = .027, η^2_p = .10) blood pressure. While systolic blood pressure decreased over the course of the placebo session (M_{t1} = 116.11, SD_{t1} = 1.51; M_{t2} = 112.83, SD_{t2} = 1.53; $F(1,44)$ = 19.46, p < .001, η^2_p = .30), it did not change after methylphenidate administration (M_{t1} = 115.32, SD_{t1} = 1.62, M_{t2} = 115.66, SD_{t2} = 1.52; $F(1,44)$ = 0.12, p = .726, η^2_p < .01). Diastolic blood pressure increased after methylphenidate administration (M_{t1} = 71.11, SD_{t1} = 1.13, M_{t2} = 74.11, SD_{t2} = 0.95; $F(1,44)$ = 15.86, p < .001, η^2_p = .26), whereas it did not after placebo (M_{t1} = 70.72, SD_{t1} = 1.07; M_{t2} = 71.60, $SD_{t2\ time2}$ = 0.90; $F(1,44)$ = 1.89, p = .176, η^2_p = .04). Also, methylphenidate affected participants' heart rate ($F(1,44)$ = 7.12, p = .011, η^2_p = .14). Heart rate decreased over time in both the methylphenidate and placebo session, but it decreased less strongly after methylphenidate (M_{t1}= 68.77, SD_{t1} = 1.66, M_{t2} = 66.04, SD_{t2} = 1.67; $F(1,44)$ = 7.30, p = .010, η^2_p = .14) than after placebo administration (M_{t1} = 67.87, SD_{t1} = 1.94; M_{t2} = 61.32, SD_{t2} = 1.57; $F(1,44)$ = 52.73, p < .001, η^2_p = .54).

Methylphenidate Effects on Creative Performance

Effects depending on individual differences in hyperactivity–impulsivity symptoms

AUT. For AUT fluency, there were no main effects of drug ($F(1,42)$ = 1.50, p = .228), order of treatment ($F(1,42)$ = 0.10, p = .760), or hyperactivity–impulsivity scores ($F(1,42)$ < 0.01, p = .927). After correction for multiple comparisons, there were no significant two-way interactions between drug and order ($F(1,42)$ = 4.85, p = .033), drug and hyperactivity–impulsivity ($F(1,42)$ < 0.01, p = .954), or order and hyperactivity–impulsivity ($F(1,42)$ = 0.15, p = .703), and no three-way interaction between drug, order, and hyperactivity–impulsivity ($F(1,42)$ = 0.41, p = .523). We did not find any main effects of drug ($F(1,42)$ = 0.79, p = .379), order ($F(1,42)$ = 0.09, p = .766), or hyperactivity–impulsivity ($F(1,42)$ = 0.97, p = .331)

on the flexibility of ideas during the AUT. Also, none of the two-way and three-way interactions were significant [drug × order: $F(1,42) = 2.50$, $p = .121$; drug × hyperactivity–impulsivity: $F(1,42) = 0.17$, $p = .679$; order × hyperactivity–impulsivity: $F(1,42) = 0.04$, $p = .839$; drug × order × hyperactivity–impulsivity: $F(1,42) = 0.35$, $p = .556$]. Similarly, we did not find any main effects [drug: $F(1,42) = 1.95$, $p = .170$; order: $F(1,42) = 0.92$, $p = .342$); hyperactivity–impulsivity: $F(1,42) = 1.55$, $p = .221$] or interactions [drug ×order: $F(1,42) = 0.02$, $p = .899$; drug × hyperactivity–impulsivity: $F(1,42) = 3.03$, $p = .089$; order × hyperactivity–impulsivity: $F(1,42) = 0.24$, $p = .624$; drug × order × hyperactivity–impulsivity: $F(1,42) < 0.01$, $p = .958$] on the originality of ideas during the AUT.

RAT. None of the main effects on the number of correctly solved RAT items was significant [drug: $F(1,42) = 0.04$, $p = .841$; order: $F(1,42) = 0.08$, $p = .785$; hyperactivity–impulsivity: $F(1,44) = 0.13$, $p = .721$]. Also, we did not find any two-way interactions between drug and order (F(1,42) = 1.09, p = .302), drug and hyperactivity–impulsivity ($F(1,42) = 0.36$, $p = .550$), and order and hyperactivity–impulsivity ($F(1,44) = 0.23$, $p = .637$). There was no three-way drug × order × hyperactivity–impulsivity interaction ($F(1,42) = 0.05$, $p = .832$) on the number of RAT solutions.

Alternate Names Task. For rule convergent thinking during the Alternate Names Task, there were no main effect of drug ($F(1,40) < 0.01$, $p = .959$), order ($F(1,40) = 0.03$, $p = .859$), or hyperactivity–impulsivity ($F(1,40) = 0. 06$, $p = .806$). We found a significant interaction between drug and order ($F(1,40) = 21.58$, $p < .001$), indicating that, regardless of the order of methylphenidate/placebo administration, participants generated more convergent names in the second session compared to the first session (i.e., equivalent to a session effect; Figure 6.1). Follow-up analyses for the Order groups separately (i.e., methylphenidate in Session 1/placebo in Session 2 vs. the reverse) confirmed that participants who received methylphenidate during the first session generated fewer ideas after methylphenidate compared to placebo ($b = -0.50$, $t(20) = -3.32$, $p = .003$), whereas participants who received methylphenidate during the second session generated more ideas after methylphenidate compared to placebo ($b = 0.51$, $t(20) = 3.25$, $p = .004$). There were no other significant interactions [drug × hyperactivity–impulsivity: $F(1,40) = 0.18$, $p = .674$; order × hyperactivity–

impulsivity: $F(1,40) = 0.19$, $p = .670$; drug × order × hyperactivity–impulsivity: $F(1,40) = 4.06$, $p = .051$]. For rule divergent thinking during this task, none of the main effects [drug: $F(1,40) = 0.83$, $p = .368$; order: $F(1,40) = 0.02$, $p = .890$; hyperactivity–impulsivity: $F(1,40) = 3.86$, $p = .056$] or interactions [drug × order: $F(1,40) = 0.41$, $p = .528$; drug × hyperactivity–impulsivity: $F(1,40) = 0.01$, $p = .913$, order × hyperactivity–impulsivity: $F(1,40) < 0.01$, $p = .967$; drug × order × hyperactivity–impulsivity: $F(1,40) = 1.93$, $p = .173$] was significant.[3]
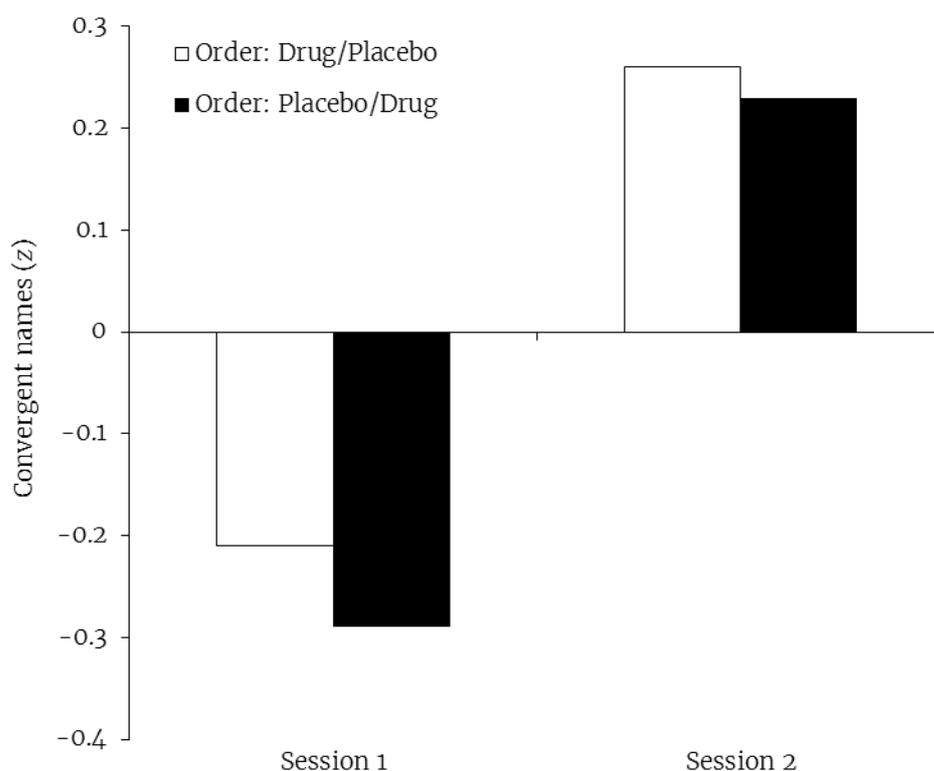

Figure 6.1. The number of convergent names that participants generated during the Alternate Names Task in both sessions. Regardless of whether participants received methylphenidate or placebo, they generated more convergent names during the second session compared to the first session.

Effects depending on individual differences in baseline working memory capacity

AUT. We did not find any main effects of drug ($F(1,42) = 0.73$, $p = .397$), order ($F(1,42) = 0.04$, $p = .851$), or working memory capacity ($F(1,42) = 0.93$, $p = .340$) on fluency during the AUT. There were no significant two-way interactions

between drug and order ($F(1,42) = 4.32$, $p = .044$), drug and working memory ($F(1,42) = 2.34$, $p = .134$), or order and working memory ($F(1,42) = 0.51$, $p = .481$). The three-way interaction between drug, order, and working memory capacity ($F(1,42) = 5.58$, $p = .023$) was not significant after correcting for multiple comparisons. For AUT flexibility, none of the main the effects [drug: $F(1,42) = 0.30$, $p = .587$; order: $F(1,42) = 0.13$, $p = .723$; working memory: $F(1,42) = 1.41$, $p = .241$] or interactions [drug × order: $F(1,42) = 1.85$, $p = .181$; drug × working memory: $F(1,42) = 2.16$, $p = .150$; order × working memory: $F(1,42) = 0.38$, $p = .540$; drug × order × working memory: $F(1,42) = 3.99$, $p = .052$] was significant. For originality of ideas during the AUT, there were no significant main effects [drug: $F(1,42) = 1.65$, $p = .206$; order: $F(1,42) = 0.52$, $p = .474$; working memory: $F(1,42) = 1.06$, $p = .310$] or interactions [drug × order: $F(1,42) = 0.13$, $p = .721$; drug × working memory: $F(1,42) = 0.11$, $p = .740$; order × working memory: $F(1,42) = 3.19$, $p = .081$; drug × order × working memory: $F(1,42) = 0.29$, $p = .591$].

RAT. For the number of correct solutions during the RAT, we did not find any significant main effects [drug: $F(1,42) = 0.07$, $p = .792$; order: $F(1,42) = 0.24$, $p = .625$; working memory capacity: $F(1,44) = 3.03$, $p = .089$]. There were no significant two-way or three-way interactions [drug × order: $F(1,42) = 0.66$, $p = .423$; drug × working memory: $F(1,42) = 1.63$, $p = .208$; order × working memory: $F(1,42) = 0.07$, $p = .791$; drug × order × working memory: $F(1,42) = 0.12$, $p = .732$].

Alternate Names Task. For rule convergent thinking during the Alternate Names Task, there were no significant main effects [drug: $F(1,40) = 0.09$, $p = .765$; order: $F(1,40) = 0.09$, $p = .762$; working memory capacity: $F(1,40) = 0.32$, $p = .577$]. We found a significant interaction between drug and order ($F(1,40) = 19.30$, $p < .001$), again showing that participants who received methylphenidate during the first session generated fewer ideas after methylphenidate compared to placebo ($b = -0.46$, $t(20) = -2.97$, $p = .008$), whereas participants who received methylphenidate during the second session generated more ideas after methylphenidate compared to placebo ($b = 0.52$, $t(20) = 3.24$, $p = .004$). None of the other interactions was significant [drug × working memory: $F(1,40) = 0.81$, $p = .374$, order × working memory: $F(1,40) = 1.89$, $p = .177$; drug × order × working memory: $F(1,40) = 0.34$, $p = .566$]. For rule divergent thinking, we did not find any significant main effects [drug: $F(1,40) = 0.58$, $p = .453$; order: $F(1,40) = 0.11$, p

= .741; working memory: $F(1,40) = 0.01$, $p = .916$] or interactions [drug × order: $F(1,40) = 0.35$, $p = .558$; drug × working memory: $F(1,40) = 0.10$, $p = .752$; order × working memory: $F(1,40) = 0.13$, $p = .723$; drug × order × working memory: $F(1,40) = 0.04$, $p = .846$].

Discussion

In the present study, we aimed to investigate whether methylphenidate affects convergent and divergent creative processes in healthy participants and, if so, whether such effects depend on individual differences in hyperactivity–impulsivity and baseline working memory capacity. We did not find evidence indicating that methylphenidate affected participants' performance on any of the creative tasks in this study. Also, methylphenidate effects did not depend on individual differences in hyperactivity–impulsivity and baseline working memory capacity. Although participants generated more convergent ideas during one of the creativity tasks in the second session of the study, methylphenidate did not affect this increase in productivity. Methylphenidate significantly increased blood pressure and heart rate compared to placebo, suggesting that our manipulation was successful, but it did not affect participants' (subjective or objective) alertness or self-reported mood.

These results are not in line with recent findings by Gvirts and colleagues (2016), who showed that methylphenidate effects on divergent thinking depended on individual differences in novelty seeking, an aspect of impulsivity associated with dopaminergic functioning (Depue & Collins, 1999). Our failure to replicate these findings may be due to insufficient power to detect methylphenidate effects on creativity in the present study. Generally, effect sizes are small in creativity research, particularly for divergent thinking tasks due to subjective scoring of performance (Farah et al., 2009; Silvia et al., 2008). In line with our findings, several studies focusing on the effects of other drugs that influence dopamine and other neurotransmitters, such as Modafinil and amphetamines, did not find straightforward effects on creative processes (Farah et al., 2009; Mohamed, 2016; Müller et al., 2013). The (often marginally) significant effects observed in these studies were usually characterized by complex interactions between individual differences, baseline task performance,

or the order in which participants performed tasks. Thus, demonstrating drug effects on creative processes may be complicated and may require studies using larger samples.

In addition, the 20 mg dose of methylphenidate that we administered to participants in our study may have been insufficient to influence creative performance. Previous studies found that methylphenidate increased vigilance and (subjective) alertness (Linssen et al., 2014; Swart et al., 2017; Van der Schaaf et al., 2013), whereas we did not observe such increased subjective or objective alertness, suggesting that our modulation may not have been sufficiently strong. However, other studies have demonstrated methylphenidate effects on cognitive (and creative) performance using a similar dose (Campbell-Meiklejohn et al., 2012; Gvirts et al., 2016; Swart et al., 2017; Van der Schaaf et al., 2013) and showed that the effects of 20 mg of methylphenidate on response inhibition and memory consolidation were comparable to effects of a higher dose of 40 mg (Linssen, Vuurman, Sambeth, & Riedel, 2012). Although participants in the present study completed the creativity tasks towards the end of a 4- to 5-hour session and approximately 2.5 hours after capsule administration, we think it is unlikely that the interval between drug administration and task performance can explain the present null findings. Plasma levels of methylphenidate peak after approximately 2 hours and methylphenidate has a half-life of 2 to 3 hours (Kimko et al., 1999). However, we cannot exclude the possibility that participants' fatigue levels at the end of the session influenced their creative performance in both the drug and placebo session. Thus, methylphenidate effects on creativity may be underestimated in the present study due to several methodological factors.

Alternatively, it is possible that methylphenidate simply does not affect creative performance. These null findings do not provide support for a recently proposed model of dopaminergic modulation of creativity (Boot et al., 2017b). Based on converging but indirect evidence (e.g., Chermahini & Hommel, 2012; Zabelina et al., 2016a; Zhang et al., 2014b), this model proposes that flexible and persistent processes in creativity are modulated by dopamine in fronto-striatal brain areas. Moreover, it suggests that the relationship between dopamine in these areas and creative performance follows an inverted-U-shaped function,

similar to the relationship between dopamine and other cognitive processes (Clatworthy et al., 2009; Van der Schaaf et al., 2013). This model predicts that increasing dopamine levels in the striatum would affect flexible creative processes (such as divergent thinking), whereas increasing prefrontal dopamine levels would influence persistent processes (including convergent thinking), and that the direction of effects would depend on people's baseline dopamine levels. Our findings here do not indicate that increasing dopamine levels through methylphenidate administration affects either flexible or persistent creative processes. However, several studies have observed methylphenidate effects on neural activity in the absence of behavioral effects (Dodds et al., 2008; Volkow et al., 2004). Thus, future studies should investigate whether methylphenidate modulates activity in fronto-striatal brain areas during creative tasks.

Although several studies showed that effects of methylphenidate and similar dopaminergic agonists depended on baseline working memory capacity and impulsivity (Cools et al., 2007; Frank & O'Reilly, 2006; Kimberg et al., 1997), these individual differences did not influence methylphenidate effects in the present study. Possibly, the range of working memory and impulsivity scores in our highly educated, healthy sample was too limited to demonstrate such effects. Also, we assessed individual differences in impulsivity together with hyperactive symptoms of ADHD, using a questionnaire developed for clinical diagnosis of ADHD, whereas previous studies demonstrated impulsivity-dependent effects of methylphenidate and similar substances using the more extensive Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995) that focuses on trait impulsivity (e.g., Cools et al., 2007; Swart et al., 2017). The ADHD questionnaire that we used to assess individual differences in impulsivity may have been less sensitive to such differences in healthy participants. Alternatively, it is possible that methylphenidate effects on creativity depend on other individual differences than those under investigation here, such as baseline creative performance (Farah et al., 2009).

Finally, the present null findings are in line with studies in adults with ADHD that showed that creative performance did not differ between people who used medication to treat their ADHD and people who did not (Boot, Nevicka, & Baas, 2017c; White & Shah, 2011). Also, studies on the effects of methylphenidate

on creativity in children with ADHD have obtained mixed results. While some found divergent thinking to be impaired when children were taking methylphenidate (González-Carpio Hernández & Serrano Selva, 2016), others found no effects (Funk, Chessare, Weaver, & Exbey, 1993) or even enhanced performance after methylphenidate administration (Douglas et al., 1995). Thus, evidence for an effect of medication on creativity in participants with ADHD is inconsistent at present and may be further investigated in future studies.

Conclusion

In sum, although methylphenidate effects on creativity may be underestimated in the present study, our findings indicate that the use of methylphenidate as a cognitive enhancer is not unequivocally detrimental to people's ability to generate original ideas and solve creative problems. Although methylphenidate may negatively affect other aspects of performance (in some people) and ethical objections to the use of such substances remain (Greely et al., 2008; Van der Schaaf et al., 2013), our results do not indicate that methylphenidate also affects convergent or divergent creative processes, regardless of individual differences in working memory capacity or impulsivity

Footnotes

[1] For the second half of participants, we slightly changed this procedure. These participants took part in the medical screening several days prior to the first session. Moreover, these participants completed half of the baseline measures during Session 1 and the other half during Session 2, so that both sessions took approximately 4 hours. (Methylphenidate effects on) creative performance did not differ between the first and second half of participants (all Fs < .130, all ps > .260).

[2] To explicitly test the possibility that hyperactivity–impulsivity and baseline working memory capacity would be related to drug effects in a curvilinear rather than a linear way, we repeated these analysis with participants' squared hyperactivity–impulsivity and working memory scores as predictors. However, these quadratic variables did not significantly predict (drug effects on) creative performance.

[3] In addition, we explored possible interactions between the drug and inattention symptoms of ADHD. None of these interactions was significant after correcting for multiple comparisons (all Fs < 5.21, all ps > .028).