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Short communication

Morning stimulant administration reduces sleep and overnight working memory improvement

Tenzin Tselha[a,*,1], Lauren N. Whitehurst[b,1], Benjamin D. Yetton[a], Tina T. Vo[c], Sara C. Mednick[a]

[a] Department of Cognitive Science, University of California, Irvine, USA
[b] Department of Psychology, University of California, San Francisco, CA, USA
[c] Department of Psychology, University of California, Riverside, CA, USA

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ABSTRACT

The goal of cognitive enhancement is to improve mental functions using interventions including cognitive training, brain stimulation and pharmacology. Indeed, psychostimulants, commonly used for cognitive enhancement purposes, while preventing sleep, have been shown to increase working memory (WM) and attention. Sleep, however, is also important for cognitive function; thus, understanding the interaction between stimulants, sleep and cognition may inform current approaches to cognitive enhancement. We used a double-blind, placebo controlled, repeated measure design to investigate the effect of morning administration of a commonly used stimulant, dextroamphetamine (DEX, 20 mg), on repeated, within-day and overnight WM performance, as well as on sleep in healthy young adults. Compared with placebo (PBO), we found no within-day benefit of DEX on WM. After sleep, DEX performed worse than PBO and the overnight improvement in performance in the PBO condition was absent in the DEX condition. Moreover, sleep quality was negatively affected by DEX administration. In summary, we found no cognitive boost from psychostimulants across a day of wake and a blockade of overnight WM increases with the stimulant, compared to PBO.

1. Introduction

As human society has gradually evolved to value mental capabilities over physical ones, the desire to enhance mental aptitudes seems a befitting response to the demands of the modern world. This compulsion to compete and outpace others is a motivation behind the pursuit of cognitive enhancement, in which individuals seek to 'amplify and extend core mental abilities' to improve performance on a range of cognitive domains, including working memory, attention, and control processes [1]. Many are turning to pharmacology, including readily available stimulant drugs like caffeine and nicotine, that have been shown to improve alertness, vigilance, and attention [2,3]. Another growing trend in students and young professionals is the off-label use of prescription psychostimulants to promote wakefulness and boost cognitive performance. These drugs, such as methylphenidate (MPH), dextroamphetamine (DEX), and mixed-salt amphetamine, prescribed for the treatment of Attention-Deficit-Hyperactivity-Disorder (ADHD) are currently being diverted into college campuses and work-places for their perceived cognitive enhancing effects. Though, compared with PBO, psychostimulants enhance performance in the context of sleep deprivation [4,5], studies in healthy non-sleep deprived adults show conflicting findings [6], with positive [7–10], negative [11,12], and null effects [13,12,14].

Working memory (WM) is widely believed play a core role in cognitive ability, and has been shown to correlate with broad measure of cognitive ability and fluid intelligence [15,16]. Studies of psychostimulant effects on WM in healthy, well-rested adults report a mix of findings. Among the positive outcomes, a within-subject study compared the impact of 10 mg and 20 mg of DEX to PBO on a WM digit span task in healthy young adults. Compared with PBO, DEX showed a dose-dependent improvement in performance [13]. Additionally, Mattay et al. [17] investigated the effect of D-amphetamine (0.25 mg/kg body weight) on an N-back task performance. They found that D-amphetamine benefitted the more demanding 3-back vs 2-back condition [17]. On the other hand, Ilieva et al. [12] administered 10 mg mixed salt amphetamine in healthy young subjects to study the objective and subjective effects of the drugs on a range of cognitive tasks, including WM (digit span and object-*N*-back) and found no stimulant-related benefit for WM. Accordingly, a meta-analysis found that the overall effect of psychostimulants on cognitive enhancement is inconclusive

* Corresponding author.
E-mail address: ttselha@uci.edu (T. Tselha).
[1] Contributed equally and share co-first authorship.

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[6], and that the literature is plagued by several issues that make comparison across studies difficult, including different subject demographics, drug compounds, and dosages.

Sleep is another unconsidered factor that might help explain the discrepant findings across studies. Sleep is usually categorized into Non-Rapid Eye Movement (NREM) sleep and Rapid Eye Movement (REM) sleep. Within NREM sleep, the stages of sleep (1–3) progress into lower frequency, higher amplitude waves on the electroencephalographic (EEG) recordings. REM sleep is characterized by high frequency, mostly desynchronized waves that show a similar pattern to wake. A large body of research has demonstrated that sleep, and specifically individual sleep stages, support a wide range of cognitive processes [18]. NREM Stage 2 and Stage 3 supports long-term memory formation [19], whereas REM sleep has been linked with the processing of emotions [20].

Sleep also supports WM. [21] Kuriyama et al., showed that sleep, compared with wake, accelerates improvement in WM performance [21]. They trained participants on an N-back task with either 10 h of wake or nighttime sleep between retesting. Significantly greater WM improvement was seen in the sleep group compared with the wake group. Similarly, a recent study compared a period of wake to a period of nocturnal sleep between WM test sessions, and showed an improvement in performance across the sleep session, compared to wake in both children and adults [22]. Along the same lines, sleep deprivation negatively affects WM performance. In one study, healthy young subjects were tested on an N-back task during an extended period of over-night wakefulness. Their task accuracy and reaction time deteriorated in conjunction with an increase in both subjective and objective measures of sleepiness; including brain activity in the delta (0–4 Hz) and theta (4–8 Hz) frequency bands [23].

Importantly, amphetamines promote wakefulness by reducing total sleep time, sleep efficiency (total sleep time/minutes in bed), minutes in REM and Stage 3, and increasing Stage 2 [24,25]. However, the impact of psychostimulant sleep disruption on cognitive processes has not been thoroughly investigated. One unexamined question is whether the deleterious impact of stimulants on sleep may play a role in the drug's impact on cognition. Most studies examining the effect of these drugs on cognition do not measure sleep. Given the growing trend in use/abuse of these drugs and recent understanding of the importance of sleep for health and cognition, the goal of the present study was to measure the impact of psychostimulants on WM and sleep. Using a double-blind, placebo-controlled, repeated measures design, we examined the effect of dextroamphetamine (DEX, 20 mg) on repeated WM testing and overnight sleep. We administered DEX in the morning on Day 1 and tested WM several times across the day, subjects then slept in the lab while monitored with polysomnography and were tested on WM in the morning. We hypothesized that DEX would promote a temporary boost to WM compared with PBO. In addition, we predicted a significant deterioration in nighttime sleep in the DEX group, compared with PBO, followed by significant decreases in WM performance the next morning.

2. Methods

A total of 46 healthy (22 female), non-smoking participants between the ages of 18–39, with no personal history of psychological, neurological, or chronic illness participated in the study. To control for prior sleep, subjects were required to keep a specific sleep schedule. Specifically, subjects went to sleep and woke up within a two-hour bedtime and wake time window—Bedtime: 10:00PM-12:00AM; Wake time: 6:00 − 8:00AM. Subjects were asked to maintain this regular sleep schedule for 7 days prior to each experimental visit to ensure approximately 7 h of sleep each night. For the night before the study day, the subjects had to ensure that they get at least 7 h of sleep and adjust their sleep schedule to report to the lab the next day by 8:00am. This schedule was confirmed via daily sleep diaries and a wrist-based activity monitor (Philips Respironics, USA). Participants gave informed consent to participate in the experiment, which was approved by the Western Institutional Review Board and the University of California, Riverside Human Research Review Board. Participants received monetary compensation for their participation in the study.

We used a double-blind, placebo-controlled design in which all subjects experienced both drug conditions. Each visit occurred a week apart to allow for drug washout. Each visit corresponded to one of the drug conditions, DEX or PBO, and drug conditions were counterbalanced across participants. Participants were extensively screened for their eligibility to participate in this study and were excluded if they did not follow a regular sleep schedule or if they reported: personal history or familial history of a mental illness, substance abuse, personal history of head injury with a loss of consciousness greater than two minutes or seizures, irregular sleep/wake cycles, history of parasomnias, and any cardiac or respiratory illness that may affect cerebral metabolism. Eligibility was determined during an in-person assessment in which research personnel conducted a structured clinical interview for DSM-IV psychological disorders as well as reviewed a series of self-report health and wellness questions as approved by the study physician. In addition, we administered the Assessment of Hyperactivity and Attention [26] to screen for symptoms of ADHD. After the in-person eligibility interview, participants underwent a standard health and physical exam conducted by the study physician to certify their health and eligibility. Participants were then required to submit to a urine toxicology test to ensure they had not used any substances not permitted by the study prior to their participation. All subjects were naïve to or had limited contact with (< 2 lifetime uses and no use in last year) the active medication in the study.

On each experimental day, subjects arrived to the lab at 8:00am (Fig. 1). After confirming that the subjects followed the required sleep schedule and adequately slept the night before, they were given breakfast. Their subjective sleepiness of the initial morning (AM1) was assessed with the Karolinska Sleepiness Scale (KSS) questionnaire [27]. The baseline performance for the WM task (details discussed below) was assessed at 8:30am. At 9:00am, participants received their first drug administration, which was either DEX or PBO. Seventy-five minutes later, another WM assessment was taken (Test 1), followed by a break during which participants could watch television, eat lunch, or work on their computer.

After drug administration, subjects' vital signs were monitored every hour. Subjects were allowed to leave the lab after 4 h of monitoring if their: 1) systolic blood pressure was below 140 and diastolic blood pressure was below 90, 2) resting heart rate was below 100 beats per minute, 3) gait measurements were sufficient, and subjects did not report experiencing a racing heart, dizziness, headache, or nausea. Upon their departure, subjects were told to refrain from caffeine, naps, and exercise during their time out of lab and were asked to confirm abstention upon arrival back to the lab. Subjects returned to the lab for another WM testing session at 9:00pm (Test 2). After completion of the task, subjects were then attached with 32-channel electroencephalogram (EEG) cap to monitor their sleep throughout the night (see below). Lights out occurred at 11:00PM and subjects were provided 10 h of time in bed. This was to ensure that the subjects had enough sleep opportunity. Subjects were awoken the next morning at 9:00am. After taking a KSS questionnaire (AM2), the subjects were tested on the WM task at 10:30am (Test 3) before being permitted to leave the lab at 11:00am. Before leaving, subjects were provided a final blood pressure reading, pulse reading, and gait assessment to ensure subjects' safety upon leaving the lab. For all subjects, the on-call doctor was regularly consulted throughout the study and for any concerns about subjects' ability to leave the lab.

We administered 20 mg of DEX, a stimulant drug that inhibits the reuptake of catecholamines, dopamine, and noradrenaline, prepared by MDMX Corona Pharmacy. DEX is an FDA approved drug to treat ADHD [28]. We chose 20 mg dosage as previous works by De Wit et al. [13]

Baseline Performance	Drug: PBO or DEX	Test 1	Break	Test 2	Sleep with PSG	Test 3
8:30AM	09:00AM	10:15AM		9:00PM	11:00PM-9:00Am	10:30AM

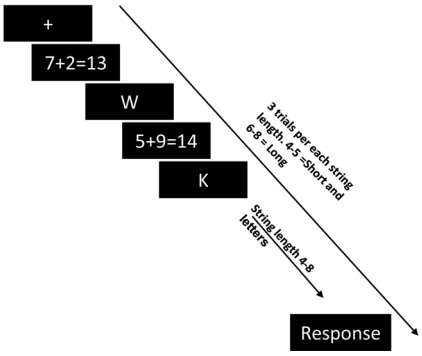

Fig. 1. Protocol and Task Figure.

showed an improved performance in WM digit span task at this dosage. The PBO as made of microcrystalline cellulose and contained no active medications. DEX powder was encapsulated and visually indistinguishable from the PBO capsules.

We utilized an operation span task (Fig. 1) that measured a subject's capacity to maintain and actively manipulate information in WM prior to a response time. We chose the operation span task as it engages and captures both the memory retention and online processing capacity of WM. Participants were shown a string of letters (4–8) on a computer screen and were asked to remember each letter in the exact order they were presented. Between the letters, subjects were shown simple mathematical equations (e.g. $4 + 2 = 6$ and $6 + 3 = 5$) and were prompted to use keyboard response to determine if the presented equations were correct or incorrect. Participants were provided three seconds to respond to each equation. The mathematical task was utilized as a distractor task to discourage online rehearsal of the letters. After each trial, subjects were provided a short break and moved to the next trial after a keyboard press. Subjects were required to maintain at least 70% accuracy on the mathematical distractor for the trial to be included in the analyses. Subjects were given practice trials at the start of each test session. Test trials were grouped into "short" versus "long" conditions in which short trials were defined as 4–5 to-be-remembered letters and long trials were defined as 6–8 to-be-remembered letters.

We employed the Karolinska Sleepiness Scale (KSS), a 9-point scale () to measure the sleepiness of the participants throughout the study day visits. KSS was tested at the onset of the initial morning visit (AM1) and the morning after the experimental sleep night (AM2).

2.1. Data Reduction

12 subjects did not complete both visits due to scheduling conflicts. For these subjects, we used their behavioral and sleep data, when applicable, and degrees of freedom are reported with each analysis for clarity.

2.2. Statistical Analysis

For prior sleep data analysis, we used paired *t*-tests to compare total sleep time (TST), WASO (Wake After Sleep Onset), SE (Sleep Efficiency) of the 7 days of sleep prior to each visit (DEX vs PBO) and the night before each visit (DEX vs PBO). Average bed/wake times and delay between wake time and test time are also reported. We ran 2 (DEX vs PBO visits) X 2 (AM1 vs AM2) RM ANOVA to investigate if the KSS score between PBO vs DEX visits and AM1 vs AM2 are comparable. To investigate the effects of the two drug conditions on the sleep quality, we used paired sample *t*-test. We examined the impact of DEX or PBO on nighttime sleep variables via a *t*-test on variables of: SE, TST, WASO, minutes in S1, S2, SWS and REM. To compare WM performance in the two conditions (DEX vs PBO), we first examined raw performance across each test and employed 2 (string length) X 2 (drug condition) X 3 (Performance Test) RM ANOVAs to compare the performance change between the DEX and PBO conditions across each Testing instance (Test 1: 75 min. post drug; Test 2: 12 h post drug; Test 3: 24 h post drug). To examine performance change, we calculated difference scores between baseline and each test and utilized the same 2 (string length) × 2 (drug condition) × 3 (Performance Test) RM ANOVAs. To control for differential drug absorption rates across our subjects due to weight, we entered weight (mean centered) as a covariate in each of these analyses. We also considered sex as a covariate in our analysis but performance nor sleep outcomes varied as a function of sex, so it is not included in the presented analyses. Lastly, we employed Pearson's correlations to examine the relationships between sleep features and WM performance. We consider $p < 0.05$ as significant and report effect sizes wherever applicable. For paired *t*-tests, we used repeated measure design effect size(d_{RM}) calculation which takes correlation coefficient into account [29] and for ANOVA we report partial eta squared. IBM SPSS Version 25 software was used for all statistical calculations.

3. Results

3.1. Prior sleep

We first confirmed that there were no significant differences in actigraphy for the seven days prior to the in-lab visits. Sleep features were similar for both the week prior and the night before for PBO vs DEX visits: TST ($t_{28} = 0.114$, $p = .91$, $d_{RM} = 0.022$), SE ($t_{28} = 2.0$, $p = .055$, $d_{RM} = 0.44$), WASO ($t_{28} = -2.139$, $p = 0.041$, $d_{RM} = 0.43$) and eve of the experimental day: TST ($t_{28} = -0.62$, $p = .53$, $d_{RM} = 0.118$), SE ($t_{28} = 0.8$, $p = 0.42$, $d_{RM} = 0.146$), WASO ($t_{28} = -1.63$ $p = 0.1$, $d_{RM} = 0.33$). During the seven days prior to each experimental visit, subjects slept an average of 7 h 53 min of sleep, and for the night before each experimental visit, subjects slept an average of 7 h 6 min of sleep. For the week before each experimental visit, the average bed and wake times were 11:57 pm and 7:47 am for PBO visit, and 11:52pm and 7:40 am for DEX visits. The average bedtimes and wake-up times for the eve of the experimental night's sleep were 11:30pm and 6:48am for PBO visit and 11:22pm and 6.53 am for DEX visit. These times indicate that subjects may have been sleepier on the

Table 1
Sleep Variables Analysis.

	Placebo	Dextroamphetamine	
TST (min)	537 ± 44	488 ± 69	*
SE(%)	92 ± 5	84 ± 9	*
WASO(min)	30 ± 26	52 ± 34	*
Stage 1 (min)	13 ± 8	20 ± 10	*
Stage 2 (min)	285 ± 50	267 ± 53	*
SWS (min)	108 ± 38	97 ± 33	*
REM (min)	130 ± 32	102 ± 36	*
Stage 2 Onset(min)	10.37 ± 1.41	22.43 ± 3.57	*
REM Onset(min)	56.03 ± 9.755	65.79 ± 11.45	*
Stage 3 Onset(min)	20.63 ± 1.69	33.96 ± 3.90	*
Stage 1 (%)	2.59 ± 1.60	4.357 ± 2.3	*
Stage 2 (%)	52.99 ± 8.21	55.48 ± 8.34	*
SWS (%)	20.27 ± 7.26	22.44 ± 14.7	
REM (%)	24.129 ± 5.192	20.739 ± 5.89	*

experimental morning, but that this sleepiness was similar across DEX and PBO conditions. On the study day visits, the mean delay between wake-up time and test-time was 102 min for PBO and 97 min for DEX. Morning sleepiness assessed by the KSS in the AM on Day 1(AM1) and Day 2(AM2) was not significantly different across drug conditions or sessions (AM1 vs AM2). With 2 × 2 RM ANOVA on KSS score (Drug condition visit (PBO vs DEX)) X Session (AM1 vs AM2), we did not find a significant main effect $F_(136) = 0.133$, $p = 0.717$ partial eta square = 0.004 or interaction effect, Drug X Session : ($F_(136) = 0.456$, $p = 0.504$ partial eta square = 0.013). The mean KSS score were DEX: AM1 = 3.43, AM2 = 3.6 and PBO: AM1 = 3.37 and AM2 = 3.86. In sum, we confirmed that the prior sleep was similar for the two drug conditions, and sleepiness did not differ between drug conditions.

3.2. Stimulant vs placebo and nighttime sleep

To examine the impact of stimulants on subsequent nighttime sleep, we measured the effect of DEX vs PBO on seven polysomnographically-measured variables using paired samples t-tests: TST, Stage 1, Stage 2, SWS and REM mins, SE, and WASO using a paired samples T-tests (Table 1). Compared with PBO, the DEX condition showed lower SE ($t_{33} = 5.47p < 0.001$), lower TST ($t_{33} = 4.68$ $p < 0.001$), higher WASO ($t_{33} = -3.71$, $p = 0.001$), decreased minutes in REM sleep ($t_{33} = 4.54$, $p < 0.001$), S2 ($t_{33} = -2.06p = 0.047$), and SWS ($t_{33} = 2.41$, $p = 0.022$), whereas S1 duration was significantly longer ($t_{33} = -3.48$, $p = 0.001$). Also, onset to REM, S2 and S3 duration was longer for DEX ($t_{33} = 4.71$, $p < 0.001$, $t_{33} = -2.06$, $p = 0.001$ and $t_{33} = 3.95$, $p < 0.001$, respectively).

3.3. Stimulant vs placebo and WM

Next, we examined the impact of DEX vs PBO on WM performance both pre- and post-sleep. First, we confirmed that there were no differences in baseline performance across the two experimental days with a paired *t*-test. For both short trials ($t_{33} = -318$ $p = 0.752$ $d_{RM} = 0.07$) and long trials ($t_{33} = -1.77$ $p = 0.084$ $d_{RM} = 0.29$), no significant differences were found. Using a 2 × 2 X 3 RM ANOVA (string length (short vs long) X drug condition (DEX vs PBO) X Test Performance (Test 1, Test 2, Test 3), we found a main effect of string length ($F_(1,32) = 190.437$, $p < 0.0001$, partial eta square = 0.856), with short strings having better performance, but no main effect of drug ($F_(1,32) = 0.203$, $p = 0.655$, partial eta square = 0.006), or session ($F_(2,64) =$) $= 0.922$, $p = 0.403$, partial eta square = 0.028). We did find a significant string length X drug condition X test performance interaction ($F_(2,64) = 4.04$, $p = 0.022$, partial eta square = .112). Post hoc analysis revealed a significant difference between Test 1 and 3 for PBO ($d_{RM} = 0.45$, $p = 0.009$,) but no such difference for DEX ($d_{RM} = 0.08$, $p = 0.72$) (Fig. 2A).

Next, we calculated difference scores to examine the change in performance from baseline to Test 1 (Day 1 AM), Test 2 (Day 1 PM) and Test 3 (Day 2 AM). Again using a 2 × 2 × 3 RM ANOVA (string length (short vs long) X drug condition (DEX vs PBO) X Test performance difference from baseline (at Test 1, at Test 2, at Test 3), we discovered no main effects of string length ($F_(1,32) = 1.124$, $p = 0.297$, partial eta square = 0.034), drug ($F_(1,32) = 1.945$, $p = 0.173$, partial eta square = 0.057), or session ($F_(2,64) = 0.922$, $p = 0.403$, partial eta square = 0.028). However, a significant string length X drug condition X test performance interaction emerged ($F_(2,64) = 4.04$, $p = 0.022$, partial eta square = 0.112) (Fig. 2B). Post hoc analysis revealed for long trials, individuals performed better after PBO compared to DEX at Test 3 only (post-sleep) ($\Delta = 7.2\%$; $p = 0.012$). Additionally, for long trials, participants showed a significant 5.1% increase in performance from Test 1 to Test 3 in the PBO condition ($p = 0.009$), however no difference from Test 1 to Test 3 was present in the DEX condition ($\Delta = 0.07\%$; $p = 0.72$). Lastly, individuals showed more improvement for long trials at Test 3 compared to short trials ($\Delta = 7.0\%$; $p = 0.013$). No other significant differences across conditions or sessions were detected for short trials (Δ's < 2%; p's > 0.86). Taken together, these results suggest WM training may benefit from a night of sleep and that DEX may block overnight WM performance enhancements.

3.4. Sleep and WM correlations

Lastly, to determine if WM improvement was correlated with nighttime sleep variables, we correlated sleep features with performance change (Test 3 - baseline WM performance) and at Test 3. We did not find significant correlations amongst any of the variables (all p values > 0.5) for both short and long trials.

4. Discussion

The present study examined the immediate and delayed impact of a psychostimulant on WM and sleep in well-rested, healthy adults. We found that stimulants administered in the morning significantly disrupted nighttime sleep. Importantly, contrary to our hypothesis, no significant difference in WM performance between DEX and PBO was present at either the 75-min or 12-hr post-drug delay. However, after a night of sleep (24 + hrs post-drug administration), the DEX condition performed significantly worse than the PBO condition. Even more, in the PBO condition, performance after sleep showed significant WM improvement compared to Test 1, but no such improvement was present in the DEX condition. These results suggest good sleep may be important to WM training and that sleep impairment, in this case induced by stimulant administration, may block WM performance gains.

One caveat to our findings is that subjects' night time sleep before the experiment was curtailed due to the early experimental start time (8AM), and this may have elevated levels of sleepiness and sleep inertia at the start of the study. However, this restriction pertained for both PBO and DEX conditions, and no differences in total sleep time was found for the week prior or the eve of the experimental day, suggesting that poor prior sleep could not completely explain the drug differences on performance. Also, with a mean delay time between wake-up and test time of more than 1.5 h for the both visit days, our participant would have typically recovered from sleep inertia [30–32]. This is also supported by average low score on the KSS at AM1, which did not differ across drug conditions.

Few studies have investigated the cost of off-label prescription psychostimulant use for sleep and cognitive performance in healthy, well-rested adults. This is surprising given that sleep is important for proper cognitive function, and the primary outcome of stimulants is increased wakefulness. In the present study, we noted that DEX impaired sleep quality, with lower sleep efficiency, increased WASO, and decreased SWS, as well as dampening sleep-dependent enhancement of WM performance. Thus, even a morning administration of DEX

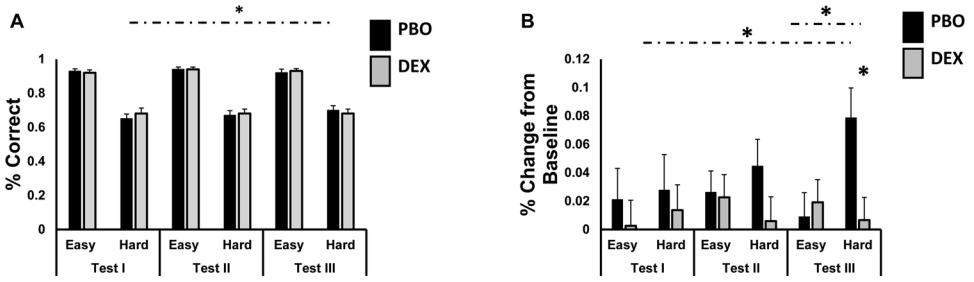

Fig. 2. Stimulants disrupted normal task improvement. A. Working memory performance at various time points (after 75 min, 12 h and 24 h). *Signifies over-all interaction effect. **B.** Working memory performance improvement at various time points (after 75 min, 12 h and 24 h) from the baseline. *Differences between PBO and DEX at various Test session and improvement.

deteriorated nighttime sleep and post-sleep WM performance.

Contrary to our expectations, we did not find increased WM ability after stimulants at either the 75-min or 12-hr delay. Previous studies have also failed to find an acute impact of stimulants on cognitive performance. For example, Ilieva et al. [12] found no evidence of cognitive enhancement 75 min. after 20 mg dose of mixed-amphetamine salt in an N-back WM task. However, other studies reported a significant benefit of stimulants for WM performance [13,17]. One potential reason for our null results relates to the inverted U-shape theory of baseline WM capacity [33] and optimal arousal theory [34], which posits that beneficial effects of stimulants would be maximum for individuals with low baseline performance, and minimal benefits would occur for individuals at moderately high baseline performance [35]. Our subject pool of healthy, young adults are likely at the peak of their working memory capacity [36], and in the context of this argument, positioned at the peak of inverted U. As such, they may not have been able to reap as much benefit from stimulants as individuals at lower performance capacities. Consistent with this idea, a study using methylphenidate showed that baseline performance was negatively correlated with errors made on a spatial working memory (SWM) task. The investigators noted that the lower the baseline performance, the more the stimulant group showed improvement in error reduction [17]. Future studies might examine this by comparing samples with lower and higher performance capacities.

Given the growing trend in off-label stimulant drug usage in healthy, well-rested adults [6], these findings have implications for public health, with a specific impact on the debate of stimulant use for cognitive enhancement. Along with the known adverse side effects from these drugs including addiction, psychosis, cardiovascular disease and sudden death [37], disruption of sleep and impairment of sleep-dependent cognition should be taken into consideration. An effective alternative approach of sleep hygiene education and napping interventions may better support a wide range of cognitive and health functions.

Conflicts of interest

None

Grants

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