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Misuse of Prescription Stimulants Among College Students: A Review of the Literature and Implications for Morphological and Cognitive Effects on Brain Functioning

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Prescription stimulant medication, the most frequently recommended treatment for college students with attention deficit/hyperactivity disorder (ADHD), has become increasingly available on college campuses. Research investigating prescription stimulant misuse among college students indicates that significant numbers of students without ADHD are taking prescription stimulants to enhance their cognitive performance. This article systematically reviews studies concerning misuse of prescription stimulants among college students with and without ADHD as well as the cognitive and morphological brain changes associated with prescription stimulants in humans and other animals. Whether these morphological changes are accompanied by improved cognitive performance remains equivocal. Implications of this body of literature are discussed and suggestions for future research are advanced.

Keywords: prescription stimulant misuse, morphological changes, brain functioning, ADHD

Attention-deficit/hyperactivity disorder (ADHD) is a chronic and pervasive disorder that typically persists across the life span (Wilens, Faraone, & Biederman, 2004). The disorder is characterized by clinically significant difficulties with inattention, impulsivity, and/or hyperactivity and is estimated to affect 3–7% of the school-aged population (American Psychiatric Association, 2013). Longitudinal studies suggest that the majority of children and adolescents diagnosed with ADHD display symptoms into adulthood, and the manifestation of these symptoms changes over time (Biederman, Mick, & Faraone, 2000). Recently there has been an increase in research investigating ADHD in the adult population (e.g., Barkley, Murphy, & Fischer, 2008), however relatively little research has been conducted with college students with the disorder, despite the fact that increasing numbers of students with ADHD are pursuing higher education (DuPaul & Weyandt, 2009). Although the precise number of college students with ADHD is unknown, preliminary studies suggest that ADHD symptoms affect approximately 2–4% of the college student population (DuPaul et al., 2001; Heiligenstein, Conyers, Berns, & Smith, 1998; Weyandt, Linterman, & Rice, 1995). According to Guthrie (2002) two of five college students with disabilities have ADHD or a learning disability, and the majority of these students are males.

A variety of treatment options are available for ADHD, including academic, behavioral, and pharmacological approaches, and treatment typically requires a multimodal approach, tailored to meet the needs of the individual (Weyandt, 2007). Stimulant medications are among the most frequently prescribed treatments for college students with ADHD (Baverstock & Finlay, 2003) and were described by Spencer, Biederman, and Wilens (1998) as the "first-line drug of choice for uncomplicated ADHD at any age" (p. 559). More recently Adler and colleagues (2009) described prescription stimulants as a safe and long-term effective treatment for ADHD in adults, and Staufer and Greydanus (2005) recommended stimulants specifically for the management of ADHD in college students.

Given that increasing numbers of students with ADHD are attending college and that stimulant medication is often prescribed for these students it is reasonable to conclude that stimulant medication is increasingly available on college campuses. Indeed, when Weyandt et al. (2009) explored the prevalence of stimulant misuse on a college campus located in the northeast region of the United States and questioned students whether prescription stimulants were "easy to get on campus," 50% agreed or strongly agreed that they were. The nonmedical use of prescription stimulants for purposes other than prescribed, also referred to as diversion, recreational use, illicit use, misuse, or abuse, has become a growing problem on college campuses. Babcock and Byrne (2000) reported that 16.4% of college students at a northeastern university reported taking methylphenidate (MPH) recreationally. More recent studies exploring illicit use of stimulant medications have reported prevalence rates among college students that range from 5.3% (Dupont, Coleman, Bucher, & Wilford, 2008) to 34% (DeSantis, Webb, & Noar, 2008).

The main motivating reasons students report misusing prescription stimulants include to (a) improve academic performance, (b) help with concentration and focusing, (c) improve test perfor-

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mance, and (d) for recreational purposes (e.g., Arria & Wish, 2006; Weyandt et al., 2009; Dussault & Weyandt, 2013). Given research that supports increasing numbers of college students are misusing stimulants and are using them primarily for cognitive enhancement, the question arises whether prescription stimulants have a similar effect on the brain functioning of students with and without ADHD. The extant literature suggests that stimulants may have a similar neurochemical effect regardless of disorder status, in children and adults. For example, two seminal studies in the 1970s by Sprague and Sleator (1977) and Rapoport et al. (1978) demonstrated that *both* children with and without the disorder showed positive cognitive and behavioral responses to stimulant medications, and since that time, several studies with healthy adults have also demonstrated positive cognitive effects (see Smith & Farah, 2011 for a review).

Although the specific effects of prescription stimulants on brain functioning are not fully understood, the stimulant medications mixed-salt amphetamines (e.g., Adderall XR) and methylphenidate (MPH), the two most commonly prescribed medications for ADHD, are thought to primarily impact neurotransmitter systems involving dopamine and to a lesser extent norepinephrine (Berridge et al., 2006). Recent research contrasting the effects of stimulant medication with nonstimulant medication on ADHD symptoms, however, suggests that the resulting improvements from stimulants may involve more than the transporter and receptor actions of dopamine and norepinephrine such as activation of second messenger systems and morphological changes such as cellular adaptations (Schulz et al., 2012).

Given that misuse of prescription stimulants is occurring among college students and the main motivation reported by students for misusing stimulants is to enhance academic, that is, cognitive performance, and preliminary studies that suggest stimulants alter brain functioning and morphology, the purpose of the present study is to systematically review (a) prescription stimulant misuse among college students with and without ADHD, (b) cognitive effects of prescription stimulants among individuals with and without ADHD and (c) morphological brain changes associated with prescription stimulants in humans with and without ADHD and other animals.

Method

Search and Retrieval

A systematic literature review was conducted according to Okoli and Schabram's (2010) eight-step guide. Accordingly, all researchers were trained in the protocol for searching and identifying relevant articles. We attempted to identify and retrieve all empirical studies published after 2003 that examined nonmedical stimulant misuse and diversion among college students, cognitive effects of prescription stimulants, and morphological effects on the brain of prescription stimulants. The search and retrieval process included a comprehensive search of the following bibliographic databases: PsycINFO, PsycARTICLES, MEDLINE, and ScienceDirect. Keywords and eligibility criteria were established separately for each subject. To identify and retrieve empirical studies that examined stimulant misuse among college students the following keywords were used: *Prescription Stimulants + Misuse, Prescription Stimulants + Illicit, Methylphenidate + Misuse, Methylphenidate + Illicit, Prescription Stimulants + Nonmedical, Methylphenidate + Nonmedical*. Keywords used to identify research examining the cognitive effects of prescription stimulants included the following: *ADHD + Prescription Stimulants + Cognitive Effect, ADHD + Medication + Cognitive Effect, Prescription Stimulants + Neuropsychological Effects, Methylphenidate + Neuropsychological Effects, Prescription Stimulants + Executive Functions*. Finally, keywords used to identify research examining morphological effects on the brain of prescription stimulants included the following: *Prescription Stimulants + Brain Effects, Methylphenidate + Physiological Effects, Stimulants + Physiological Effects, Methylphenidate + Brain Effects*.

Eligibility Criteria

Studies for all search subjects were selected for review based on the following criteria:

1) The study was published in English.
2) The study was published no earlier than 2003.
3) The study included a minimum sample size of 20 subjects.
4) The study used an original dataset (meta-analyses were excluded).

Eligibility criteria established specifically for research examining stimulant misuse and diversion among college students included:

1) The study was relational, experimental, or quasi-experimental.
2) The study was conducted in North America.
3) The sample included only college students.
4) The sample included students with and without ADHD (samples including only students with ADHD were excluded).
5) The study explored at least one of the following variables related to stimulant misuse: Lifetime or annual prevalence related to the entire population or established separately for sex, Greek affiliation, or ADHD status, the top three reasons for stimulant misuse, and/or the way of obtaining the drug.

Eligibility criteria established specifically for research examining the cognitive effects of prescription stimulants included:

1) The study was experimental or quasi-experimental.
2) The sample included human subjects only and included no special groups except for persons with ADHD and Learning Disabilities (LD).
3) The study addressed variables related to cognitive effects of prescription stimulants.

Eligibility criteria established specifically for research the morphological effects on the brain of prescription stimulants included:

1) The study was relational, experimental, or quasi-experimental.
2) The sample included no special groups except for persons with ADHD.
3) The study addressed variables related to morphological effects on the brain of prescription stimulants.

Results

Nonmedical Stimulant Misuse Among College Students

A total of 22 studies were identified that met eligibility criteria related to stimulant misuse within college student samples and

were included in this study (see Table 1). Three studies were published across multiple articles, one of which included longitudinal findings therefore only the most recent article was included (Garnier-Dykstra, Caldeira, Vincent, O'Grady, & Arria, 2012). The identified studies reported lifetime prevalence rates for the population overall (n = 19) and prevalence rates by sex (n = 7), Greek affiliation (n = 5), and ADHD or prescription status (n = 9). For prevalence rates, one study (Rozenbroek & Rothstein, 2011) reported students engaging in nonmedical stimulant use separately from students engaging in both medical and nonmedical stimulant use; these estimates were combined. As shown in Table 2, studies that were identified also reported reasons for misuse (n = 18), ways of obtaining drugs (n = 9), and administration methods (n = 8).

Prevalence rates. Estimates of lifetime prevalence rates ranged from 5.3% to 35%. Eight studies reported lifetime prevalence rates that were less than 10%. Weyandt and colleagues (2009) reported that 7.5% of the surveyed college students in their study reported using non-prescribed stimulants within the past 30 days and 9.3% reported using outside of the past year.

Lifetime prevalence rates in similar ranges were reported by Dupont et al. (5.3%; 2008), Lord et al. (6.7%; 2009), Teter, McCabe, LaGrange, Cranford, & Boyd (8.3%; 2006), Rozenbroek and Rothstein (2011; 9.7%), and Weyandt et al. (9.3%; 2009). Rabiner et al. (2009) surveyed more than 3400 college students within two universities and reported that 8.9% of the sample used nonmedical ADHD medication since beginning college and 5.4% used nonmedical use of ADHD medications within the past month. Finally, a study conducted in the Midwest with over 3,000 students reported a lifetime prevalence rate of 8.5% in three different articles (McCabe, 2008; McCabe, Boyd & Teter, 2009; McCabe & Teter, 2007).

Another nine studies reported lifetime prevalence rates that ranged between 11% and 20%. In 2005, Hall et al. surveyed 381 college students and found that 13.7% endorsed the illicit use of prescribed stimulant medications. White, Becker-Blease, and Grace-Bishop (2006) surveyed 1,000 college students, 16.2% of whom reported abusing or misusing stimulant medication; half of these students reported misusing stimulants once or twice over the past year, a third reported misusing them one to two times per month, and 15.5% reported misusing them two to three times per week. Reporting slightly higher rates, Judson and Langdon (2009) found that 20% of 333 students sampled across two colleges in the northeast endorsed using illicit prescription stimulants. Reporting comparable rates, Dussault and Weyandt (2013) conducted a study including five universities in five regions of the United States and found that 19.8% of the sample endorsed using prescription stimulants for nonmedical use. Most recently, Garnier-Dykstra and colleagues (2012) reported on the results of a longitudinal study assessing nonmedical use of prescription stimulants and found that among a sample of more than 1200 students, 13.3% (weighted) had used prescription stimulants nonmedically by their first year of college, and 31% (weighted) used prescription stimulants nonmedically by their fourth year.

Finally, two other studies, both conducted on the eastern coast of the United States, reported notably high prevalence rates (between 30% and 34%). DeSantis et al. (2008) found that 34% of more than 1800 students in their study reported using ADHD medications illegally. Janusis and Weyandt (2010) reported prevalence rates related to students with and without disabilities specifically; when combined, 30% of both groups reported using prescription stimulants for nonmedical purposes between rarely and frequently.

Prevalence rates by gender. Studies assessing gender differences reported mixed findings. White and colleagues (2006) found no difference in stimulant misuse between males and females, yet other studies have reported that males are more likely to misuse stimulants than females (Garnier-Dykstra et al., 2012; Rabiner et al., 2009; Teter et al., 2005). Of the seven studies included in the present study that reported lifetime prevalence rates by sex, five studies reported higher prevalence rates for males than females; the other two studies, in which females demonstrated higher prevalence rates than males, were conducted with special populations (pharmacy and dental students). Although effect sizes for sex differences were not reported within *any* of the included studies, we calculated Cohen's d based on estimates of male and female sample sizes of the general population studies to be very large, including .88 for first-years and 3.47 for fourth-years (Garnier-Dykstra et al., 2012), 4.80 (Dussault & Weyandt, 2013), 2.40 (Hall et al., 2005), 5.38 (DeSantis et al., 2008), and 4.74 (Rabiner et al., 2009). Effect size estimates were based on Cohen's estimates of small, medium, and large (Cohen, 1992). Based on these findings, it appears that male students misuse prescription stimulants at higher rates than female students.

Prevalence rates by Greek affiliation. It also appears that specific characteristics of the undergraduate institution may impact the likelihood of illicit stimulant use. Specifically, a few studies found that students who participate in Greek organizations are more likely to misuse stimulant medication than those who do not participate in these organizations (Dussault & Weyandt, 2013; Lord et al., 2009; Rabiner et al., 2009), and the five studies that reported separate prevalence rates for students affiliated and unaffiliated with Greek organizations found a higher prevalence rate overall for students affiliated with Greek organizations (Clegg-Kraynok et al., 2011; DeSantis et al., 2008; Dussault & Weyandt, 2013; Lord et al., 2009; Rabiner et al., 2009). Although exact effect sizes were not included within any of the included studies addressing prevalence rate differences between students affiliated with Greek organizations and students who were not affiliated with Greek organizations, we calculated effect sizes of Cohen's d to be very large, including 6.93 (Clegg-Kraynok et al., 2011), 15.88 (DeSantis et al., 2008), and 9.83 (Rabiner et al., 2009).

ADHD and prescription status. Although some studies excluded participants with an ADHD diagnosis or prescription stimulant from their analyses (e.g., DeSantis et al., 2008; Weyandt et al., 2009), others reported findings separately for each group. Although one of the eligibility criteria for this current review involved comparisons of prevalence rates between students with and without ADHD, the results were reported differently across studies and were therefore excluded from the tables. Specifically, three articles reported on misusers and illicit users of stimulant medication broken down by ADHD and/or prescription status and five articles reported the prevalence rates of misuse or illicit use within groups of students reporting an ADHD diagnosis and/or prescription for a stimulant medication.

White and colleagues (2006) reported that among more than 1000 college students, 16.2% reported misusing or abusing MPH; 90% of these students had never been diagnosed with ADHD or

Table 1
Illicit Stimulant Use and Misuse Prevalence Rates

Reference	Region, school type (number)	Stimulant description	Sample size	Special population	Prevalence (lifetime if not otherwise indicated)				
					Overall	Sex		Greek affiliation	
						M	F	Y	N
Barrett, Darredeau, Bordy, & Pihl (2005)	Canada, University (1)	Methylphenidate	100	No	—	—		—	
Clegg-Kraynok, McBean, & Montgomery-Downs (2011)	Mid-atlantic/southeast, University (1)	Prescription stimulants (methylphenidate, etc.)	492	No	14.4%			31.8%	13.9
DeSantis, Webb, & Noar (2008)	Southeast, University (1)	ADHD stimulants	1733	No	34%	39%	30%	48%	23%
Dupont, Coleman, Bucher, & Wilford (2008)	NR, Universities (2)	Methylphenidate	2087	No	5.3%	—		—	
Dussault & Weyandt (2013)	Northeast/Southeast/Northwest/Southwest/Midwest. Universities (5)	Prescription stimulant	1033	No	19.8%[a]	26.0%[a]	17.3%[a]	25%[a]	15.9%[a]
Garnier-Dykstra, Caldeira, Vincent, O'Grady, & Arria (2012)	Midatlantic, University (1)	Prescription stimulants for ADHD	1253	Longitudinal	Year 1 13.3%$_{wt}$ Year 4 31%$_{wt}$	Year 1 13.9%$_{wt}$ Year 4 34.2%$_{wt}$	Year 1 12.7%$_{wt}$ Year 4 27.8%$_{wt}$	—	
Hall, Irwin, Bowman, Frankenberger, & Jewett (2005)	Midwest, University (1)	Stimulants	381	No	13.7%[a]	17%[a]	11%[a]	—	
Janusis & Weyandt (2010)	Northeast, University (1)	Prescription stimulant	165	No	30.3%[a]	—		—	
Judson & Langdon (2009)	Northeast, Colleges (2)	Prescription stimulant	333	No	20%	—		—	
Lookatch, Dunne, & Katz (2012)	Mid-Atlantic, university (1)	Prescription stimulants	206	No					
Lord et al. (2009)	Northeast, College (1)	Prescription stimulant	950	Pharmacy students only	6.7%	6.5%	6.8%	20%	5.3%
McCabe (2008); McCabe & Teter (2007); McCabe, Boyd & Teter (2009)	Midwest, University (1)	Stimulant medication	3639	No	8.5%	—		—	
McCabe, Teter, & Boyd (2006a); McCabe, Teter, & Boyd (2006b); McCabe & Boyd (2005); Teter, McCabe, Cranford, Boyd, & Guthrie (2005)	Midwest, University (1)	Stimulant medication	9161	No	8.1%	—		—	
McNiel et al. (2011)	Southcentral, Dental schools (multiple)	Prescription ADD Stimulant medication	243	4th year dental students only	12.4%[a]	10%[a]	13%[a]	—	
Rabiner et al. (2009)	Southeast, Universities (2)	ADHD medication	3407	No	8.9%[b]	7.2%[b]	4.5%[b]	10.5%[b]	4.2%[b]
Rozenbroek, & Rothstein (2011)	Mid-Atlantic University	Prescription stimulants to treat ADHD	413	No	9.7%	—		—	
Scotter & Meaux (2008)	NR, College	Prescription stimulants	404	No	10.4%[a]	—		—	
Stone & Merlo (2011)	Southeast, University (1)	Prescribed stimulant	383	No	13.2%	—		—	
Teter, McCabe, Boyd, & Guthrie (2003)	Midwest, University (1)	Methylphenidate (included weight-loss pill)	2250	No	—	—		—	
Teter, McCabe, LaGrange, Cranford, & Boyd (2006)	Midwest, University (1)	Prescription stimulants (methylphenidate & amphetamine)	4580	No	8.3%	—		—	
Weyandt et al. (2009)	Northeast, University (1)	Prescription stimulants (e.g., Ritalin, Adderall, Concerta)	363	No	9.3%				
White, Becker-Blease, & Grace-Bishop (2006)	Northeast, University (1)	Stimulant medications (Ritalin, Adderall, Cylert, Dexedrine, Concerta)	1025	No	16.2%	—		—	

[a] Did not specify time period. [b] Since beginning college.

Table 2
Top Three Reasons for Misuse, Ways of Obtaining, and Administration Methods

Reference	Reasons for misuse	Way of obtaining drug	Administration method
Barrett, Darredeau, Bordy, & Pihl (2005)	Recreational (70%)	Peers with prescription (77.8%)	Oral (88%)
	Study purposes (30%)	Own prescription (11.1%)	Intranasal (50%)
		Theft (4%)	Smoking (4%)
Clegg-Kraynok, McBean, & Montgomery-Downs (2011)	Study/work (41.4%)	Given by friend with prescription (59.2%)	Oral (89.7%)
	Concentrate (28.6%)	Acquaintance with prescription (39.7%)	Intranasal (29.4%)
	Get high (10%)	Purchased from friend with prescription (17.6%)	
DeSantis, Webb, & Noar (2008)	Stay awake/study longer (72%)	Friends with prescription (87%)	—
	Concentrate (66%)	Significant others (4%)	
	Help memorize (36%)	Strangers (8%)	
Dupont, Coleman, Bucher, & Wilford (2008)	Work/study (35.8%)	Friends, family members or acquaintances (free; 90%)	Oral (50.1%)
	Party (35.8%)	Purchased (5.5%)	Intranasal (47.2%)
	Work/study & party (18.3%)	Other (1.8%)	
Dussault & Weyandt (2013)	Perform better in my schoolwork (70.4%)	—	—
	Perform better on tests (70.0%)		
	Focus better in class (58.6%)		
Garnier-Dykstra, Caldeira, Vincent, O'Grady, & Arria (2012)	Year 1	Year 1	Year 1
	Study (73.8%)	Friend with prescription (76%)	Swallowed whole (89.3%)
	Curiosity (18.7%)	Friend without prescription (14.9%)	Snorted (13.7%)
	Stay awake to party (12.9%)	Other (10%)	Swallowed crushed (2.5%)
	Year 2	Year 2	Year 2
	Study (91.3%%)	Friend with prescription (78.3%)	Swallowed whole (92.7%)
	Stay awake to party (9.8%)	Friend without prescription (21.3%)	Snorted (14.3%)
	Get high (6.2%)	Other (12.6%)	Swallowed crushed (2.2%)
	Year 3	Year 3	Year 3
	Study (89%)	Friend with prescription (77.4%)	Swallowed whole (92.4%)
	Stay awake to party (12.3%)	Friend without prescription (17.6%)	Snorted (16.6%)
	Other (6.6%)	Other (8.3%)	Swallowed crushed (2.3%)
	Year 4	Year 4	Year 4
	Study (91.5%)	Friend with prescription (73.9%)	Swallowed whole (91.9%)
	Stay awake to party (14.5%)	Friend without prescription (16.7%)	Snorted (12.8%)
	Get high (6.4%)	Other (8.1%)	Swallowed crushed (3.4%)
Hall, Irwin, Bowman, Frankenberger, & Jewett (2005)	Finals week (27%)		Oral 63%
	Before tests (15.4%)		Intranasal 11.5%
Janusis & Weyandt (2010)	—	—	—
Judson & Langdon (2009)	Improve concentration (28.8%)	—	—
	Increase alertness or stay awake (23.4%)		
	Become high (6.3%)		
Lookatch, Dunne, & Katz (2012)	Help with studying or with completing a paper	—	—
	To increase focus and concentration		
	To pull an "all-nighter."		
Lord et al. (2009)	Help with concentration (80%)	Friends (75%)	—
	Improve school performance (59%)	Acquaintances (28%)	
	Increase energy (39%)	Parents (9%)	
McCabe (2008); McCabe & Teter (2007); McCabe, Boyd, & Teter (2009)	Self-treatment only (26.4%)	—	Oral (58.5%)
	Recreational only (19.3%)		Intranasal & oral (6.6%)
	Mixed (54.2%)		Other (injecting, smoking, inhaling; 6.6%)

(*table continues*)

Table 2 (*continued*)

Reference	Reasons for misuse	Way of obtaining drug	Administration method
McCabe, Teter, & Boyd (2006a); McCabe, Teter, & Boyd (2006b); McCabe & Boyd (2005); Teter, McCabe, Cranford, Boyd, & Guthrie (2005)	Help with concentration (58%) Increase alertness (43%) Provide a high (43%)	Friends & peers (67.6%) Family (3.1%) Did not specify (28.6%)	—
McNiel et al. (2011)	Improve attention or concentration (70%) Recreation (17%) Higher grades (13%)	Friends (87%) Parents/family members (7%) Physicians (7%)	—
Rabiner et al. (2009)	Concentrate better while studying Be able to study longer Feel less restless while studying	Given by student with prescription Purchased from student with prescription Given by student without prescription	—
Rozenbroek & Rothstein (2011)	Help me study/perform better at school (53.9%) Just to try it/curiosity (20.0%) Makes me feel good (15.1%)	—	—
Scotter & Meaux (2008)	—	—	—
Stone & Merlo (2011)	Help study (57.1%) Improve focus (20.4%) Experiment (8.2%)	—	
Teter, McCabe, Boyd, & Guthrie (2003)	—	—	—
Teter, McCabe, LaGrange, Cranford, & Boyd (2006)	Help with concentration (65.2%) Help study (59.8%) Increase alertness (47.5%)	—	Oral (95.3%) Intranasal (38.1%) Smoking (5.6%)
Weyandt et al. (2009)	Perform better on schoolwork Perform better on tests Focus better in class	—	—
White, Becker-Blease, & Grace-Bishop (2006)	—	—	Oral (55%) Intranasal (40.3%) Other (4.4%)

prescribed a stimulant and 9.8% reported having ADHD and a prescription for stimulants. Similarly, Scotter and Meaux (2008) reported that while 14.7% of a sample of 404 college students reported misusing prescription stimulants, 12.5% of these students also reported having a diagnosis of ADHD. In 2005, Teter and colleagues reported that 14% of the students who reported illicitly using prescription stimulants also reported having a prescription for stimulant medication at some point in their lifetime. No differences in motivations for illicit use between students with and without a prescription in their lifetime were found.

Arria et al. (2008) reported on data from the College Life Study including a sample of more than 1200 first-year students and found that among the 45 students with ADHD, one third reported either overusing their prescription stimulant or using someone else's prescription stimulant. When broken down by type of misuse, 26.7% of students with ADHD had overused their prescribed stimulant medication at least once in their life; however, only 15.6% had ever used someone else's prescription stimulants. Compared with the latter type of misuse by students with ADHD, students without a prescription for an ADHD stimulant endorsed nonmedical use of prescription stimulants at a higher rate (18.1%). Reporting on the College Life Study three years later, Arria and colleagues (2013) found that by their fourth year in college, 55.8% of the 86 students who reported having an ADHD diagnosis endorsed the nonmedical use of prescription stimulants for studying; however, only 38% of the entire sample endorsed this. McNiel and colleagues (2011) reported that of the dental and dental hygiene students reporting a diagnosis of ADHD in their sample, all had received a prescription for a stimulant at some time in their life and only 10% ever misused their stimulant by taking more than the prescribed dose. Finally, although Judson and Langdon (2009) did not report exclusively on students with ADHD, they did offer separate lifetime prevalence rates of stimulant misuse for students with a prescription (47.6%) and students without a prescription (18.3%).

Reasons for illicit stimulant use and misuse. Students have reported a number of reasons for illicit use of stimulant medications encompassing both academic and recreational reasons. Of the 18 studies included, 15 identified academic reasons as college students' primary motivation for illicit use of stimulant medication. For example, Dussault & Weyandt reported that students most commonly misused prescription stimulants to perform better in schoolwork (70.4%) and tests (70%) and to focus better in class (58.6%). Teter and colleagues (2005) found that the most common reason students took illicit stimulant medications was to help with concentration (58%) and increase alertness (43%), although 43% did report using stimulant medications to provide a high. Other studies included in the review reported similar findings. It is critical to note that although students report academic motivations as their main reason for misusing stimulants, there is a dearth of studies that have investigated

whether stimulants *actually* lead to enhanced academic performance among college students.

Obtaining prescription stimulants and administration method. Regarding access to prescription stimulants, *all* the studies included in this review reported that students procured medication from friends and peers. Other sources from which students obtain stimulant medication included family (Dupont et al., 2008 Lord et al., 2009; McCabe et al., 2006b; McNiel et al., 2011), prescriptions (Garnier-Dystra et al., 2012; Barrett et al., 2005), theft (Barrett et al., 2005), worksite (Lord et al., 2009), and physicians (McNiel et al., 2011). Once students begin to partake in illicit stimulant medication, they are most likely to take it orally or intranasally (Clegg-Kraynok et al., 2011; Garnier-Dystra et al., 2012; Barrett et al., 2005; Dupont et al., 2008; Hall et al., 2005; McCabe & Teter, 2007; Teter et al., 2006; White et al., 2006); a much smaller number of students have reported taking MPH by smoking (4%) or injections (2%) (Barrett et al., 2005; McCabe & Teter, 2007; Teter et al., 2006).

Summary of prescription stimulant misuse. Based on the studies included in the present review, a number of findings are relatively clear. First, although prevalence rates seem to vary across studies, all of the studies found that a substantial percent (at least 5% and in many cases, higher) of college students report misusing prescription stimulants. Further, male students, compared with female students, and students affiliated with Greek organizations, compared with students who are unaffiliated, have endorsed higher usage rates of prescription stimulants. Although the literature comparing students with and without ADHD and stimulant misuse is scant, the present findings offer some evidence that students with ADHD may also be vulnerable to stimulant misuse and illicit stimulant use. Additionally, although students have reported using prescription stimulants for recreational purposes, the majority of studies included in the present review reported academic (i.e., cognitive enhancement) reasons as the primary motive students take nonprescribed stimulants. It is also clear that students are most likely to procure stimulant medication from peers, and once they have obtained the medication the majority of students may take it orally.

Cognitive Effects of Prescription Stimulants

Of 33 studies reviewed (see Table 3), only 10 reported effect sizes (i.e., Cohen's *d*), which ranged from *small* to *large* (d = .01–1.2). The most common tasks used in the studies included executive functioning tasks (n = 20), followed by memory (n = 9) and cognitive tasks (n = 6). Twenty-nine of the 33 studies reviewed reported specific types of medication used by participants in the study and dosages of medication (n = 23), 14 of that included a placebo. Eleven of the 33 studies included a control group.

Results revealed that stimulants were associated with improved performance on neuropsychological and cognitive tasks, particularly with respect to response inhibition and working memory for both children and adults with ADHD (Brackenridge, McKenzie, Murray, & Quigley, 2011; Coghill, Rhodes, & Matthews, 2007; Klimkeit, Mattingley, Sheppard, Lee, & Bradshaw, 2005; Murray et al., 2011).

Several studies examined the effects of stimulants on cognitive performance over time. For example, Gimpel et al. (2005) compared the Verbal, Performance and Full Scale IQ scores, as measured by the Wechsler Intelligence Scale for Children, Third Edition, with children with ADHD who were taking stimulant medication compared to controls and found at a one-year follow-up that those taking stimulant medication had better test performance. Similar results have been found in cross-cultural samples (Jung et al., 2011; Zhang, Jin, & Zhang, 2011).

Tucha et al. (2011) evaluated the problem-solving abilities of adults with ADHD on and off MPH and found those treated with MPH showed marked improvement in convergent thinking (i.e., algorithmic approaches to arrive at a solution) as measured by a Tower of London task, however no effect was observed on participants' divergent thinking (i.e., heuristic approaches to generate various solutions) while taking MPH. Studies investigating the effects of MPH on response inhibition have also produced significant findings (e.g., Nandam et al., 2011; Allman et al., 2010).

Summary of Cognitive Effects

It was only possible to include a sample of the studies available concerning stimulants and cognitive performance, however, of those included in the review, findings supported that stimulants were associated with improved performance on tasks related to attention, impulsivity, memory and response inhibition in both children and adults with ADHD. Although not the focus of this review, other reviews are available concerning the effects of stimulants on cognitive performance of healthy adults (e.g., Smith & Farah, 2011). There is a dearth of studies, however, concerning college students, and replication of previous studies in college samples is sorely needed.

Morphological Effects on the Brain of Prescription Stimulants

Of the 1007 studies that the search yielded, 19 studies were identified as meeting inclusion criteria (see Table 4). Of the 19 identified studies, 10 examined humans, 8 studied rats, and one study used mice. Primary independent variables included administration of prescription stimulants, either experimentally (n = 15) or as indicated by stimulant medication history (n = 4). Dependent variables included measurements of regional cerebral blood flow, cerebrum, cerebellum, basal ganglia, striatum, cortical regions, hippocampus, locus coeruleus, neurons, and frontoparietal regions. None of the studies reported relevant effect sizes.

Studies examining morphological changes. Sobel and colleagues (2010) analyzed the morphological aspects of the basal ganglia nuclei (caudate, putamen, and globus pallidus) in children with ADHD, compared with their non-ADHD peers and found the ADHD group had reduced putamen volume relative to the comparison group. Notably, symptom severity was positively correlated with local inward deformations of the surface of the putamen. Additionally, surface analyses revealed significant *outward* deformations of all three basal ganglia nuclei among ADHD participants who had been treated with stimulants, compared with untreated ADHD participants. Interestingly, the outward deformations were observed in similar locations as the inward deformations in the ADHD group, compared with control participants.

Regarding the cerebellum, Bledsoe, Semrud-Clikeman, and Pliszka (2009) found that the cerebellar vermis was significantly

Table 3
Cognitive Effects of Prescription Stimulants

Reference	Area of cognition tested	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Implications of significance	Participant demographics
Allman et al. (2010)	Executive function; working memory; oculomotor activity	0.3 mg/kg	D-amphetamine	24	Placebo, D-amphetamine	$p = .04$	D-amphetamine performance better than placebo.	Adults, age 18–34
Allman, Ettinger, Joober, & O'Driscoll. (2012)	Oculomotor activity	20 mg	MPH[a]	29	Placebo then drug; reverse order	$p < .004$; $p < .001$; $p < .001$; $p < .004$	MPH decreased visually-guided saccade latency; MPH increased smooth pursuit gain; number of saccades during pursuit decreased with MPH; proportion of predictive saccades increased on MPH.	Male adults
Barnett, Maruff, & Vance (2005)	Visuospatial memory	Varied	Stimulant medication	159	Control, ADHD medicated, ADHD non-medicated	$p = .00$; $p = .01$	Memory impairment in non-medicated and medicated ADHD children compared to controls.	Children age 6–12
Biederman et al. (2011)	Executive function; intelligence; reading;	Medication was titrated to optimal response (a maximum daily dose of 1.3 mg/kg; initial dose of 36 mg).	OROS MPH[b]	223	Placebo, MPH	$p = .048$	Improvement in functioning from OROS MPH compared to placebo.	Adults, 19–60
Biederman et al. (2008)	Executive function; neuropsychological variables	Varied	Stimulant medication	253	ADHD no meds; ADHD medicated; control	$p = .04$ & $p = .03$; $p = .002$, $p < .001$, $p = .003$; $p < .001$	Medicated ADHD performed higher on sustained attention & verbal learning than non-medicated; medicated ADHD and non-medicated performed poorer than controls on various measures.	Adolescents, young adults, age 15–25 years.
Boonstra, Kooij, Oosterlaan, Sergeant, & Buitelaar (2005)	Executive function	Maximum dose 1 mg/kg/day	MPH[a]	43	Placebo, MPH[a]	$p = .001$;	Overall significant effect of treatment on performance.	Adults age 20–55
Brackenridge, McKenzie, Murray, & Quigley (2011)	Executive function (response inhibition)	Unspecified	MPH[a]	46	Control, ADHD medicated, ADHD non-medicated	$p = .02$	Different reaction time between medicated/unmedicated condition (unmedicated had faster reaction time); no difference compared to control.	Children age 6–14

Table 3 (*continued*)

Reference	Area of cognition tested	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Implications of significance	Participant demographics
Broyd et al. (2005)	Executive function (response inhibition)	Varied, $M = 28$ mg/day	MPH[a]	36	Control, ADHD	$p < .05$	MPH may not necessarily improve response execution, but rather enhances inhibitory processing.	Children age 8–14
Coghill, Rhodes, & Matthews (2007)	Executive function (response inhibition)	0.3 mg/kg, 0.6 mg/kg	MPH[a]	73	Placebo, low, high dose	$p < .001$; $p < .02$	MPH improved Go/No Go performance; improved recognition memory tasks.	Children, adolescents age 7–15
DeVito et al. (2008)	Executive function (gambling task)	.5 mg/kg	MPH[a]	21	Control; ADHD non-medicated & ADHD medicated; placebo	$p < .01$	Medicated ADHD group bet more conservatively than placebo group.	Children
Gimpel et al. (2005)	Intelligence			31	ADHD non-medicated & ADHD medicated	$p < .001$	Improvement in IQ 1 year later in medicated group, no significant improvement in non-medicated group.	Children, 6–13
Hellwig-Brida, Daseking, Keller, Petermann, & Goldbeck (2011)	Intelligence; attention	Individually tailored doses.	MPH[a]	67	ADHD non-medicated & ADHD medicated	NS ($p > .05$)	Non-significant effects for intelligence and attention.	Male children with ADHD diagnosis, age 6–13.
Jung et al. (2007)	Intelligence; executive function	18 mg or 36 mg	MPH[a]	83	ADHD	$p < .01$	MPH improved intelligence test performance.	Children age 6–12
Klimkeit, Mattingley, Sheppard, Lee, & Bradshaw (2005)	Executive function	Not specified	Stimulant medication	25	Control, ADHD medicated, ADHD non-medicated	$p < .01$; $p < .01$	Significant difference in inattentive errors between non-medicated and controls. Non-significant effects for medicated and controls; significant difference in impulsive errors for both ADHD groups compared to controls.	Children
Kubas, Backenson, Wilcox, Piercy, & Hale (2012)	"Cold" executive working memory, "hot" self-regulation, neuropsychological impairments		MPH[a]	56	Double-blind, placebo-controlled, within-subjects (baseline, placebo, low MPH, high MPH); ADHD (all three subtypes)	$p < 0.001$; $p < 0.001$; $p < 0.001$; $p < 0.001$; $p < 0.001$; $p < 0.001$; $p < 0.001$	Significant effects of MPH on various cognitive/neurospsychological measures. MPH response generally poorer for those with no or low impairment at baseline, MPH response better among those with moderate or high impairment at baseline.	Children age 6–16

(*table continues*)

Table 3 (*continued*)

Reference	Area of cognition tested	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Implications of significance	Participant demographics
Langleben et al. (2006)	Executive function	10 to 30 mg	MPH[a]	24	Control; ADHD on-MPH; ADHD off-MPH	$p = .028$	MPH improved Stroop test response interference scores in ADHD children (not in healthy controls).	Children age 8–12
Mikami et al. (2009)	Response inhibition; visual memory (executive function)	72 mg (OROS); 30 mg	OROS® MPH[b]; se-AMPH ER	35	Males & females, med 1 then med 2; reverse order	$p < .01$	Improved neuropsychological performance for medicated ADHD (no effect for sex).	Adolescents
Murray et al. (2011)	Achievement; reading; attention, executive functioning	18 mg–54 mg	OROS MPH[b]	68	OROS MPH & placebo	$p < .0001$; $p < .0001$; $p < .0001$; $p < .0001$	Significant differences between placebo & OROS MPH on various measures.	Children age 9–12
Nandam et al. (2011)	Executive functioning	MPH 30 mg, ATM 60 mg, CIT 30 mg	MPH[a], ATM, & CIT	24	MPH 30 mg, ATM 60 mg, CIT 30 mg, or PLAC	$p < .01$	MPH resulted enhanced response inhibition compared to other drugs/ placebo.	Males age 18–35
O'Driscoll et al. (2005)	Oculomotor planning; executive functioning	.5 mg/kg to a maximum of 30 mg	MPH[a]	32	Control; on/off medication	$p = .013$, $p = .007$, $p = .008$, $p = .026$, $p = .013$	MPH associated with improved motor planning and response inhibition performance in participants with ADHD-Inattentive and ADHD-Combined.	Children, adolescents age 7–15
Pasini et al. (2013)	Working memory; inhibition; planning	0.5 mg/kg	MPH[a]	108	10/10 allele, 9/10 allele, and 9/9 allele	$p < .001$	Response inhibition improvement at 4 weeks for all genotypes, improvements at 8 and 24 weeks for 9/10 & 10/10 genotype. Planning: improvement at 24 weeks for all genotypes. Working memory: improvement at 4 weeks for all genotypes, improvement at 8 weeks for 10/10, and 24 weeks (but $p = .002$ for 24).	Children, adolescents age 7–15
Rhodes, Coghill, & Matthews (2006)	Neuropsychological performance	0.3 mg/kg, 0.6 mg/kg	MPH[a]	73	Placebo, low, high dose	$p < .05$	Significance at .05 level or greater for MPH: latency correct, latency incorrect, delayed matching to sample (0s, 4s, 12s), reaction time latency 5-choice. No significance on primary measures.	Children, adolescents age 7–15

Table 3 (*continued*)

Reference	Area of cognition tested	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Implications of significance	Participant demographics
Schweitzer, Lee, Hanford, Zink, Ely, Tagamets,. . . Kilts (2004)	Executive function	.5 mg/kg for the first week, .75 mg/kg for the second week, and up to 1.0 mg/kg for the third week	MPH[a]	21	Control, ADHD	$p = .02$	MPH improved PASAT performance; significant difference between controls/ADHD pretest but not posttest.	Adults
Semrud-Clikeman, Pliszka, & Liotti (2008)	Achievement; executive function; attention	Caried	Amphetamine or MPH[a]	94	Control, non-medicated ADHD, medicated ADHD, LD	$p = .028$; $p = .05$; $p = .02$	Non-medicated group performed significantly poorer than controls on various measures.	Children, adolescents age 9–15
Snyder, Maruff, Pietrzak, Cromer, & Snyder (2008)	Executive function	18–90 mg qAM	Adderall XR; Concerta	67	ADHD (on-medication then off-medication), ADHD (off-medication then on-medication), non-ADHD control	$p < .05$	Improvement from unmedicated trial to medicated on several measures.	Children
Tucha, Tucha, Sontag, Stasik, Laufkotter, & Lange (2011)	Problem-solving abilities	10 to 45 mg	MPH[a]	44	ADHD on medication; ADHD unmedicated; control	$p < .05$; $p < .05$	Differences in divergent thinking between ADHD and controls and in convergent thinking between medicated and non-medicated participants with ADHD.	Adults
Turner, Blackwell, Dowson, McLean, & Sahakian (2005)	Spatial working memory; visual memory; spatial span/sustained attention	30 mg	MPH[a]	24	Placebo; drug	$p = .026$; $p = .006$; $p = .026$	MPH improved performance on several measures.	Adults
Turner, Clark, Dowson, Robbins, Sahakian (2004)	Short-term memory; visual memory; executive function	200 mg	Modafinil	20	Placebo, drug	$p < .001$; $p = .017$; $p = .016$; $p = .007$; $p = .009$; $p = .004$; $p = .028$	Modafinil related to improved performance on various measures.	Adults
Turner et al. (2003)	Memory; attention; executive function	100 mg or 200 mg	Modafinil	60	Placebo, low dose (100mg), high dose (200mg)	$p < .05$	Modafinil related to improved performance on digit span compared to placebo; medicated group obtained solution in fewer moves compared to placebo; Increased deliberation time on gamble task.	Adults
Wigal et al. (2011)	Academic performance; intelligence; reading	18–54 mg/day	MPH[a]	71	Placebo first, then MPH and reversed	$p < .0001$	MPH associated with better scores compared to placebo and significant improvements on a number of measures.	Children with ADHD, age 9–12

(*table continues*)

Table 3 (*continued*)

Reference	Area of cognition tested	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Implications of significance	Participant demographics
Wilson, Cox, Merkel, Moore, & Coghill (2006)	Visual memory; attention span; response inhibition	72 mg Concerta/30 mg Adderall XR	Concerta & Adderall XR	35	Concerta, Adderall XR and placebo	$p < .001$; $p = .042$	Concerta and Adderall XR both related to decreased distractibility and increased visual short term memory; no significant difference between drugs.	Adolescents age 16–19
Yildiz Oc et al., (2007)	Oculomotor	.5–1.5 mg/kg	MPH[a]	21	ADHD pre-treatment/ post-treatment	$p = .001$	BGT scores before MPH treatment compared with after MPH were significantly decreased.	Children
Zhang, Jin, & Zhang (2011)	Intelligence/ Achievement	Varied	MPH[a]	237	Untreated, MPH	$p < .05$; $p < .05$; $p < .01$; $p < .01$	PIQ and FSIQ scores higher than the control group at post-test; Intervention group had significant increase in Verbal IQ; PIQ; FSIQ.	Children

[a] Methylphenidate. [b] Oral osmotic methylphenidate.

Table 4
Morphological Effects on the Brain of Prescription Stimulants

Reference	Tasks	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Brain region
Claussen, & Dafny (2012)	Investigated the neurophysiological properties of the caudate nucleus neurons in response to acute and chronic administration of MPH in freely behaving animals, previously implanted with permanent semi microelectrodes.	2.5 mg/kg	MPH[a]	26	Rats; within-subjects	$p < 0.05$; $p < 0.05$; $p < 0.05$	Basal ganglia (caudate nucleus)
Costa, Riedel, et al. (2013)	Explored effects of MPH administration on brain activity using fMRI and 2 response inhibition tasks among 52 healthy males.	40 mg	MPH[a]	52	Humans, double-blind, placebo-controlled, within subjects	$p = 0.05$	Basal ganglia (putamen)
Sobel et al. (2010)	Individuals with and without ADHD examined using anatomical MRI. Conventional volumes and surface morphology for the basal ganglia measured to investigate effects of childhood medication history.	N/A	N/A	104	Humans; cross-sectional, case-control design, correlational	$p = 0.005$; $p < 0.0001$;	Basal ganglia
Bledsoe, Semrud-Clikeman, & Pliszka (2009)	Determine whether cerebellar morphology is different in treatment-naïve versus chronically treated children with ADHD.	N/A	N/A	47	Humans; between groups, correlational design (ADHD no meds; ADHD meds, non-ADHD no meds	$p = 0.004$; $p = 0.001$	Cerebellar vermis
Martins et al. (2006)	Evaluated oxidative damage in the rat brain and the differential age-dependent response to MPH after acute and chronic exposure in young and adult male rats.	1, 2 & 10 mg	MPH[a]	20	Rats; experimental and control	$p < 0.05$	Cerebellum, prefrontal cortex, hippocampus, striatum, cerebral cortex
Gomes et al. (2009)	Superoxide level in submitochondrial particles evaluated in response to treatment with MPH in the age-dependent manner in rats. MPH was administrated acutely or chronically.	1, 2 or 10 mg/kg (acute or chronic)	MPH[a]	40	Rats; within-subjects, placebo controlled	$p < 0.05$	Cerebellum, hippocampus, striatum
Lee et al. (2005)	Investigated how long-term oral medication of MPH affects resting regional cerebral blood flow in ADHD children, using single photon emission computerized tomography (SPECT).	0.3–1.0 mg 2x a day	MPH[a]	40	Humans, quasi-experimental design	$p < 0.01$; $p < 0.05$	Cerebral blood flow

(*table continues*)

Table 4 (*continued*)

Reference	Tasks	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Brain region
Szobot et al. (2003)	Studied effects of MPH on brain activity in male children and adolescents with ADHD, using the blood flow radiotracer technetium-99m ethyl cysteinate dimer (99mTc-ECD) and single-photon emission tomography (SPET).	0.70 mg/kg	MPH[a]	36	Humans: experimental, randomized, double blind, and placebo controlled	$p = 0.019$; $p = 0.015$	Cerebral blood flow
Shaw et al. (2009)	Determined prospectively whether psychostimulant treatment for ADHD was associated with differences in the development of the cerebral cortex during adolescence.	N/A	N/A	67	Humans; correlational study	$p = 0.02$; $p = 0.05$	Cerebral cortex
Scherer et al. (2009)	Evaluated effect of acute and chronic administration of MPH on Na+, K+-ATPase activity in cerebrum of young and adult rats.	1.0, 2.0, or 10.0 mg/kg (for both acute and chronic admin.)	MPH[a]	40	Rats; quasi-experimental design	$p < 0.05$	Cerebrum
Tomasi et al. (2011)	fMRI used with working memory and visual attention tasks to explore whether 20 mg oral MPH increased activation in the dorsal attention network and deactivation in the default mode network and improved performance during cognitive tasks in healthy men.	20 mg	MPH[a]	32	Humans; experimental, placebo-controlled, randomized	$p < 0.05$; $p < 0.05$	Dorsal attention network (DAN); lingual gyrus, cerebellum
Bush et al. (2008)	Investigate whether MPH hydrochloride osmotic-release oral system would increase fMRI activation, compared with placebo, in the dorsal anterior midcingulate cortex (daMCC) and other frontoparietal regions subserving attention during the Multi-Source Interference Task (MSIT).	36 mg (maximum dose 1.3 mg/kg)	MPH[a]	21	Humans, randomized, placebo controlled	$p < 0.0001$; $p < 0.000001$	Frontoparietal regions
Lee et al. (2008)	Investigated effects of MPH and atomoxetine (ATX) on cell proliferation and neuronal differentiation in the dentate gyrus (DG) of the adolescent mouse by 5-bromo-2 -deoxyuridine (BrdU) and doublecortin (DCX) immunohistochemistry.	2.5, 5 & 10 mg MPH, 4, 8 &16 mg/kg ATX	MPH[a] & ATX	84	Mice; between subjects design	$p < 0.05$	Hippocampus

Table 4 (*continued*)

Reference	Tasks	Doses of medication	Types of medication	Sample size	Groups compared	Significance	Brain region
Devilbiss & Berridge (2006)	Examined effects of low-dose MPH on LC tonic and phasic discharge in halothane anesthetized rats.	0.25, 0.5, 1.0, 2.0 and 4.0 mg/kg	MPH[a]	88	Rats; within-subjects design	$p < 0.05$	Locus coeruleus discharge (brainstem)
Urban, Waterhouse, & Gao, (2012)	Juvenile and adult rats treated with MPH or saline. Neuronal excitability and synaptic transmission in pyramidal neurons of prefrontal cortex explored. Recovery from MPH treatment also examined at 1, 5, & 10 weeks following drug cessation.	1 mg/kg	MPH[a]	41	Rats; experimental, placebo-controlled, randomized	$p < 0.05$	Pyramidal neurons of prefrontal cortex
Stoy et al. (2011)	Examined differences in reward processing in adulthood (independent of actual ADHD) depending on MPH treatment during childhood.	N/A	N/A	35	Humans; correlational	$p < 0.001$; $p < 0.001$	Reward processing (VTA)
Boikess, & Marshall (2008)	Western blotting and radioimmunocytochemistry used to examine spinophilin (dendritic spine protein). Spinophilin determinations conducted in striatum and other subcortical regions implicated in psychostimulant-induced neuroplasticity.	1–8 mg/kg	D-Amphetamine	48	Rats; experimental and control	$p < 0.05$ $p < 0.001$	Striatum (and other subcortical regions)
Volkow et al. (2012)	Assessed whether dopamine increases elicited by MPH were associated with long-term clinical response, using PET [11C]raclopride. 20 adults evaluated before and after 12 months of clinical treatment with a titrated regimen of oral MPH.	1 mg/kg	MPH[a]	20	Humans; within-subjects	$p < 0.001$	Striatum, prefrontal and temporal cortices
Prieto-Gomez et al. (2004)	Behavioral and electrophysiological experiments using male rats were performed before and after MPH treatment. Intracellular recording of neuronal activity before and after electrical stimulation identified ventral tegmental area-dopamine (VTA-DA) neurons. Whole-cell patch clamping to study post-synaptic currents on VTA-DA neurons.	0.6, 2.5, and 10.0 mg/kg (10 mg dose chosen as most effective)	MPH[a]	48	Rats; 4 groups (1 control and 3 w/ diff. doses of MPH). 16 horizontal rat brain slices also used	$p < 0.05$	VTA-DA neurons

[a] Methylphenidate.

smaller in children with ADHD who had received treatment with stimulants, compared with treatment-naïve children with ADHD, as well as typically developing children. In addition to the basal ganglia and cerebellum volume, Shaw et al. (2009), found greater cortical thinning in a group of adolescents with ADHD children not treated with stimulants compared to the group taking stimulants, and this effect *remained* after controlling for IQ and gender.

Studies exploring brain activation patterns. In addition to volumetric studies, Bush et al. (2008) conducted a randomized, placebo-controlled fMRI study that explored whether MPH resulted in changes in activation in the dorsal anterior midcingulate cortex (daMCC) and other frontoparietal regions associated with attention, compared with placebo, among adults diagnosed with ADHD. At baseline, no differences were found between the experimental and control groups on measures of daMCC activation; however, after 6 weeks of MPH treatment, the experimental group had significantly higher daMCC activation than the control group. An interaction was found between the type of treatment (placebo vs. MPH) and response to MPH treatment, wherein those who responded well to MPH treatment had significantly *higher* daMCC activation than those who responded poorly to treatment. In addition to increased daMCC activation, the MPH group displayed significantly higher activation in the dorsolateral prefrontal cortex and the parietal cortex, as well as the caudate, premotor cortex, thalamus and cerebellum.

In a correlational study, Stoy et al. (2011) examined differences in reward processing among adults with a history of childhood ADHD, both with and without a history of MPH treatment in childhood, compared to an age, handedness and smoking behavior matched group, using fMRI. BOLD-responses were measured in the ventral striatum during anticipation of reward, and in the orbitofrontal cortex during task outcome. During the anticipation of reward, no differences were found for activation across groups; in contrast, whole-brain analyses indicated group differences between ADHD and non-ADHD participants, as well as between childhood-drug-naïve and childhood-MPH-treated participants during both anticipation and outcome stages. Decreased activity in the left inferior frontal cortex (BA 45) was found among childhood-drug-naïve participants, whereas participants with a history of stimulant treatment childhood displayed decreased activity in the right inferior frontal cortex (BA 46), compared with the non-ADHD group. This difference did not remain significant, however, after controlling for IQ.

Lee and colleagues (2005) also explored activation patterns in those with and without ADHD and investigated whether regional cerebral blood flow (rCBF) during resting state was affected by MPH administration among children with ADHD, using single photon emission computerized tomography (SPECT). Results revealed diminished rCBF in the ventral part of the middle prefrontal and the orbitofrontal cortices in the right hemisphere before treatment among the ADHD participants as compared to a non-ADHD control group. After 4–5 weeks of MPH treatment, however, this decreased rCBF normalized, and measured to be nearly equal to that of the control group. Further, rCBF decreased in the right striatum after treatment, whereas the superior region of the prefrontal cortices demonstrated increased rCBF. In addition, bilateral occipitotemporal regions displayed reduced rCBF after treatment for the ADHD participants. In contrast, decreased rCBF in the posterior cerebellar cortex in the ADHD group relative to the control group were unaffected by treatment.

In a double-blind, randomized, placebo-controlled study conducted with healthy males (non-ADHD), Costa et al. (2013) demonstrated that MPH administration resulted in increases in the BOLD signal in the putamen, compared with placebo, during unsuccessful go/no-go response inhibition trials. In contrast, no increases in the BOLD signal were found for successful trials. Costa and colleagues (2013) noted that converging evidence suggests that MPH has a more pronounced effect on "striatal mediated performance monitoring than on inhibitory networks" (p. 1185). Further, the authors speculated that MPH exerts its neural effects by up-regulating basal ganglia in people both with *and* without ADHD. Other studies, such as Szobot et al. (2003), reported acute MPH administration in adult males with ADHD results in reduced rCBF in the left posterior parietal region compared to a placebo condition suggesting a "posterior attentional system" (p. 425) may be involved in mediating the effects of MPH on ADHD symptoms. Interestingly, MPH in healthy adult males was found to increase activity in the dorsal attention network (DAN) and the default mode network (DMN) and in the cerebellum during working memory and visual attention tasks (Tomasi et al., 2011). These findings were supported by Volkow et al. (2008) who found that MPH (compared with placebo) administration during a cognitive task resulted in significantly reduced metabolic activity in the parietal cortex, cingulate gyrus, and thalamus, all of which are involved in exerting attentional control. In contrast, significant metabolic activity differences were not observed between the MPH and placebo conditions on a neutral nontask. In a later study, Volkow et al. (2012) explored how MPH affected dopaminergic activity using PET, and found significant increases in dopamine in the striatum, as well as the prefrontal and temporal cortices. After 12 months of MPH treatment, dopamine in the striatum had *decreased* and these changes were associated with significant reductions in participant-reported symptoms of ADHD.

Studies on rats and mice. Based on the articles in this review, research with other animals corroborates morphological changes found in human samples as a result of MPH treatment. For example, Prieto-Gomez et al. (2004) investigated the physiological properties of MPH on the ventral tegmental area's dopaminergic neurons (VTA-DA) in preadolescent male rats. Using behavioral experiments, MPH resulted in adaptive changes in the VTA-DA neurons' and modulated the glutaminergic receptors via NMDA and non-NMDA receptors. The results suggested that the effect of MPH and the input from the prefrontal cortex to the VTA-DA neurons are mediated by NMDA and kainate/AMPA receptors and may participate to induce behavioral sensitization to stimulants.

Claussen and Dafny (2012) investigated the role of acute and chronic MPH treatment on caudate nucleus (CN) single unit neuronal activity in rats. Semi microelectrodes permanently implanted in the rats indicated the majority of CN units showed an increase in neuronal firing rate after acute MPH treatment and further increase after repetitive MPH administration. These findings suggest that the same repetitive dose of MPH can elicit neurophysiological sensitization, that is, the neurons respond more rapidly after MPH administration. Boikess and Marshall (2008) also found evidence of neuronal changes after MPH administration twice daily for 5 weeks via an increase of the protein spinophilin in the septum, hippocampus, amygdale, and the cingulate cortex. Simi-

larly, Lee et al. (2008) used immunohistochemistry to study the effects of MPH and atomoxetine (ATX) on hippocampal neurogenesis in the dentate gyrus of 7 groups of adolescent mice and found that a single dose of 10 mg/kg MPH increased cell proliferation and neuronal differentiation in the subgranular zone(SGZ) and increased brain derived neurotrophic factor (BDNF) level in the dentate gyrus.

Investigating potential age-related effects of MPH on brain morphology, Scherer et al. (2009) studied differences between acute and chronic exposure to MPH in young and adult rats. Specifically, activity of Na^+, K^+-ATPase, a membrane-bound enzyme involved in cellular excitability and cell energy metabolism, was measured in the hippocampus, prefrontal cortex and striatum. Results revealed in both young and adult rats, acute MPH exposure increased Na^+, K^+-ATPase activity in all three regions evaluated. Urban, Waterhouse, & Gao (2012) also explored age and dose dependent effects of MPH administration on neuronal excitability and synaptic transmission but did so in layer 5 pyramidal neurons of the prefrontal cortex (PFC) of rats. Findings revealed distinct age-dependent actions of MPH on prefrontal neurons as juvenile PFC neurons were supersensitive to very low doses of MPH, as measured by whole-cell patch clamp recordings. Both single-dose and chronic treatment regimens of MPH resulted in significant *decreases* of neuronal excitability and synaptic transmission in PFC layer 5 pyramidal neurons in juvenile neurons and furthermore, higher doses of MPH induced long-lasting depressant effects on juvenile PFC neurons.

With regard to potential negative effects of stimulants on cellular function, Martins et al. (2006) studied oxidative damage in rat brain and differential age-dependent response to MPH after acute and chronic exposure in the cerebellum, prefrontal cortex, hippocampus, striatum, and cerebral cortex of young (25 days old) and adult (60 days old) male rats. Findings revealed significant differences in oxidative stress parameters between acute and chronic MPH administration, dependent on dose and age of exposure. In young rats chronically exposed to MPH there was a dose-dependent increase in lipid peroxidation in the cerebellum, prefrontal cortex, hippocampus, and striatum as well as protein carbonilation in the cerebral cortex. On the other hand, oxidative damage was not found in any region of the brains of adult rats chronically exposed to MPH. These findings suggest that in young rats but not adult rats, MPH induces oxidative damage to the brain. This study did not, however, measure behavioral effects that may or may not be associated with oxidative damage.

Gomes et al. (2009) suggested that MPH treatment, depending on age and treatment regimen, can influence the production of superoxide radical anions which in some conditions may be toxic in the brain. The researchers evaluated the superoxide levels in submitochondrial particles in the striatum, cerebellum, and hippocampus of young and adult rats in response to acute or chronic MPH treatment. The study showed that in *young rats*, the acute MPH administration in all doses (1, 2, or 10 mg/kg) increased the production of superoxide in the cerebellum and, in high doses (10 mg/kg), in the hippocampus. Chronic treatment, however, did not have any effect. In *adult rats*, acute treatment had no effect but chronic treatment resulted in decreased production of superoxide in the cerebellum at lower doses.

Summary of morphological effects. Collectively, these studies provide preliminary evidence that for individuals with ADHD, prescription stimulants are associated with normalized brain activity patterns, similar to that of individuals without ADHD. In contrast, among individuals with ADHD not taking stimulants, brain activity patterns appear to be different in several regions of the brain, particularly the basal ganglia and frontal-striatal regions. Additionally, preliminary studies suggest that individuals with ADHD who do not take stimulant medication may experience more rapid cortical thinning (e.g., Shaw et al., 2009), compared with those with ADHD who take prescription stimulants. Further, while taking prescription stimulants, use of attentional resources appears to be more efficient for both individuals with and without ADHD. Similarly, other-animal studies suggest that brain activity may be enhanced by both acute and chronic stimulant medication, such as more rapid neural responses, upregulation of dendritic proteins (spinophilin), neurogenesis, and increased Na^+,K^+-ATPase activity in cell membranes. It is important to note, however, that stimulant administration has also been associated with oxidative damage in the rat brain, and that the young rat brain relative to the adult rat brain may be more vulnerable to potential detrimental effects of stimulants on brain development.

Discussion

Stimulant Misuse Among College Students

This review indicates that a substantial percentage of college students are engaging in nonmedical stimulant use, and these findings are consistent with previous research (Kaye & Darke, 2012). Overall rates vary across studies ranging from 5.3% (Dupont et al., 2008) to 34% (DeSantis et al., 2008). Rates also vary based on lifetime prevalence versus more recent use, with lifetime rates significantly higher than more recent use estimates. For example, a large scale survey conducted by McCabe and colleagues nearly 9 years ago, involving nearly 11,000 college students within 119 different colleges, revealed a lifetime prevalence of 6.9%, whereas 4.1% of students reported illicitly using stimulant medication in the past year, and 2.1% reported doing so in the past month (McCabe, Knight, Teter, Wechsler, 2005). Other studies, including the National Survey on Drug Use and Health (NSDUH) (Kroutil et al., 2006), found approximately 34% of the national sample reported misusing ADHD medications in their lifetimes, with the majority (80%) falling between the ages of 12 and 25. Based on the current review it appears as though prescription stimulant misuse is continuing to rise, and rates vary by age, year in college, and region of the United States.

The present review provides support for higher rates of stimulant misuse among male students compared with females. Interestingly, several studies reported that although prevalence rate differences emerged between males and females in their college sample, there were no gender differences for motives to use prescription stimulant medications (Low & Gendaszek, 2002; Teter et al., 2005). It is important to note, however, that moderating variables (e.g., academic differences, choice of major) as well as the various methods for estimating prevalence rates across studies may have contributed to the mixed findings.

The present review also affirmed that a paucity of studies have examined differences in prescription stimulant misuse between students with and without ADHD. Of those studies that have been conducted, the findings are inconsistent, which may reflect a

difference between having a diagnosis of ADHD and misusing a valid prescription or obtaining stimulants illicitly. To date the relationship between prescription stimulant misuse and disability status is virtually unexplored. A recent study by Janusis and Weyandt (2010) involving more than 165 college students with and without disabilities (ADHD, learning disability, mental health disability, vision disability, hearing disability, physical and chronic disability, executive functioning disorder, and Asperger's syndrome) as determined by the university's Disability Services for Students as well as self-identification on a demographic questionnaire, found 46.8% of students without disabilities and 20.9% of students with disabilities endorsed having used prescription stimulants for purposes other than medical between "rarely" and "frequently." Although students with and without disabilities did not differ on overall ratings generated by the Stimulant Survey Questionnaire, that encompassed questions on different types of and reasons for stimulant misuse, both groups were using at a similar high level. Further, the prevalence estimates reported between students with (including ADHD) and without disabilities generated a large effect size (Cohen's $d = 4.76$), suggesting there may be important differences between rates of endorsement.

Results regarding Greek affiliation offer an unclouded picture. Specifically, studies included in this review consistently found that students affiliated with Greek organizations engaged in nonmedical use of stimulants at higher rates than students who are not Greek.

In terms of access to stimulants, the most common source of prescription stimulant appears to be peers and friends. Other sources include family and the medical community. Additionally, although some studies (e.g., McCabe & Teter, 2007) have reported that students inject or smoke prescription stimulants, based on the present review, most students use prescription stimulants orally.

Cognitive and Morphological Findings

Studies included in the review revealed the primary reason for misusing prescription stimulants was for academic purposes, that is, cognitive enhancement. Whether the cognitive enhancement actually occurs in college students without ADHD remains an empirical question; however, studies included in the current review consistently found that prescription stimulants were associated with improved performance on neuropsychological and cognitive tasks in children and adults with ADHD. The most robust findings were in the area of executive functions including response inhibition, working memory, and visual-spatial working memory in both children and adults with attention-deficit/hyperactivity disorder (ADHD; e.g., Brackenridge et al., 2011; Coghill et al., 2007; DuPaul et al., 2012). These findings, in conjunction with studies that have explored effects of stimulants in healthy adults (see review by Smith & Farah, 2011), suggest that prescription stimulants have the potential to serve as cognitive enhancers. Ideally a double-blind placebo-controlled study would be conducted with college students without ADHD, similar to the landmark studies conducted with children by Sprague and Slater (1977) and Rapoport et al. (1978) and more recently DuPaul et al. (2012) to explore whether college students with and without the disorder show positive effects to stimulant medications. Because of ethical issues, however, it may be difficult to conduct such a study with college students in the United States. Recently, however, Zeeuws and Soetens (2007) and Zeeuws, Deroost, & Soetens (2010a, 2010b) conducted studies with volunteers from a university in Belgium and found acute administration of D-amphetamine resulted in improvements in long-term but not short-term memory (STM).

Numerous empirical questions remain regarding the effects of prescription stimulants on cognition. Specifically, are stimulants truly cognitive neuroenhancers, and if so, what aspects of cognition are improved? Conversely, are there aspects of cognition that are deleteriously affected by stimulants? If neuroenhancement is supported, what is the mechanism by which this improvement occurs? As discussed in this review, research with humans and other animals suggests that stimulants may alter brain morphology by increasing gray and white matter (Nakao et al., 2011; Shaw et al., 2009), induce cellular changes, and may decrease bilateral ventral striatum volume in individuals with ADHD (Hoekzema et al., 2012). Human studies have found that while taking prescription stimulants, use of attentional resources appears to be more efficient for both individuals with and without ADHD. Additionally, studies have provided preliminary evidence that for individuals with ADHD, prescription stimulant intake is associated with normalized brain activity patterns, similar or equal to that of individuals without ADHD. In contrast, among individuals with ADHD not taking stimulants, brain activity patterns appear to be different in several regions of the brain, particularly the basal ganglia and frontal-striatal regions (Bush et al., 2008; Lee et al., 2005). These results are consistent with other-animal studies that have found a high density of dopamine transporter protein and extracellular dopamine in the caudate and thalamus regions. Other-animal studies also suggest that both acute and chronic stimulant medication results in more rapid neural responses, upregulation of dendritic proteins (spinophilin), neurogenesis, and increased Na^+,K^+-ATPase activity in cell membranes which, although speculative, may account for some of the cognitive improvements found in human studies. To help clarify the underlying physiological mechanisms involved in stimulant effects, it is critical that studies demonstrate that stimulants are associated with physiological changes (e.g., increased rCBF or glucose metabolism, neurgenesis, increased ion activity) *and* these changes are accompanied by cognitive and/or behavioral changes (e.g., increased attention, decreased impulsivity, improved memory, improved productivity).

It is plausible, however, that stimulants may have deleterious effects on brain morphology and cognition, and this area warrants further investigation. For example, other-animal studies have found that stimulant administration has been associated with oxidative damage in the rat brain, and that the young rat brain relative to the adult rat brain may be more vulnerable to potential detrimental effects of stimulants on brain development (Urban et al., 2012). Recently Goitia and colleagues (2013) reported that MPH resulted in synaptic alterations of thalamic nuclei and GABA transmission in mice. It is unknown, however, whether these changes were long-lasting or whether they were associated with cognitive–behavioral changes. Additionally, preliminary studies suggest that for individuals with ADHD, withholding stimulant medication may result in more rapid cortical thinning compared with brain development among those with ADHD taking prescription stimulants (e.g., Shaw et al., 2009), although replication of these studies is needed. When taken as prescribed, stimulants are regarded as safe, although not without risks. For example, adverse

side effects, although rare, can include psychosis, seizures, and cardiac events such as tachycardia, hypertension, myocardial infarction, or sudden death (Westover & Halm, 2012).

Implications for College Students

Recently Hanson et al. (2013) analyzed the discussion of prescription stimulants on Twitter posts and found tweets peaked during college and exam periods, and the nature of these tweets confirmed the use of prescription stimulants as a study aid among college students. The findings from this social media outlet corroborate the extant literature and the results of the current review, indicating that significant numbers of college students without ADHD are taking prescription medication primarily to enhance their academic functioning, that is, cognitive enhancement. This practice raises again the question whether stimulants enhance cognitive and academic functioning or whether students simply believe they do so. A recent study by Looby and Earleywine (2011) found that students who thought they were receiving MPH but received a placebo reported enhanced mood compared with the control group, however no improvements were found on cognitive tasks. Furthermore, studies suggest that college students report that their use of nonprescribed stimulants is justified, or at the very least no cause for concern, and should be made available over the counter (White & colleagues (2006; Dodge, Williams, Marzell, & Turrisi, 2012). These findings suggest that prescription stimulant misuse may be becoming acceptable (i.e., part of a cultural norm on college campuses). It is also plausible that a percentage of students who misuse stimulants are self-medicating because of higher levels of internal restlessness and other symptoms associated with ADHD, as a recent study found that 71.11% of Northern Virginian college students who misused stimulants also screened positive for ADHD symptoms and were nearly seven times as likely to demonstrate ADHD symptoms than those who did not misuse stimulants (Peterkin, Crone, Sheridan, & Wise, 2010).

Recent studies suggest that prescription stimulant misuse is not limited to college students but may extend to other settings such medical schools (Webb, Valasek & North, 2013) and even vocational and grammar schools (Franke et al., 2011). Given these findings, as well as research that suggests prescription stimulant misuse is often viewed as justified, misuse is likely to continue and as Katsurra and McGrogan (2013) noted, "it is possible that neuroenhancement may become commonplace in the future" (p. 77).

Conclusion

In conclusion, the present review substantiates that significant numbers of college students without ADHD are taking prescription stimulants to enhance their cognitive performance. Students who are most likely to misuse prescription stimulants include Greek-affiliated and male students. Based on the current review, students are most likely to obtain prescription stimulants from their peers and to take them orally. Studies support that stimulants enhance cognitive performance of individuals with ADHD, particularly attention, memory, self-regulation, and executive functions. Whether prescription stimulants improve these abilities *and* academic performance in college students without ADHD who take prescription stimulants for the purpose of cognitive enhancement remains equivocal. Physiological research with other animals and preliminary studies with humans supports that stimulants alter brain chemistry, primarily by targeting the dopaminergic system, and that use of stimulants results in morphological changes in the brain. It is remains unknown whether morphological changes in humans are short or long term, and whether these changes are actually accompanied by cognitive enhancement. These questions are important to address as misuse of prescription stimulants is a prominent issue on college campuses and is likely to remain as students continue to seek methods to increase academic success. Additional research is needed to better understand the physiological, morphological, and cognitive effects of prescription stimulants, and to help develop appropriate intervention and prevention programs to address the misuse of prescription stimulants among the college student population.

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