1. **Article 1.** Depression. By: Oyama, Oliver, PhD, Piotrowski, Nancy A., PhD, Magill’s Medical Guide (Online Edition), 2013**HTML Full Text**

**Depression**

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**Anatomy or system affected:** Brain, heart, musculoskeletal system, psychic-emotional system

**Definition:** One of the most common psychiatric disorders to occur in most lifetimes, caused by biological, psychological, social, and/or environmental factors

**Causes and Symptoms**

The word “**depression**” is often used to describe many different things. For some, it defines a fleeting mood, for others an outward physical appearance of sadness, and for others a diagnosable clinical disorder. In any year, millions of adults suffer from a clinically diagnosed **depression**, a mood disorder that often affects personal, vocational, social, and health functioning. The fifth **edition** of the [*Diagnostic and Statistical Manual of Mental Disorders*](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293871889%22%0a%09%09%09%09&sl=ll) (DSM-5, 2013) of the American Psychiatric Association delineates a number of mood disorders that include clinical **depression**, known as major depressive disorder.

Neuroimaging can be a valuable tool in the diagnostic work-up of various psychiatric disorders including **depression**. By Helmut Januschka (Helmut Januschka) [GFDL (http://www.gnu.org/copyleft/fdl.html) or CC-BY-SA-3.0 (http://creativecommons.org/licenses/by-sa/3.0/)], via Wikimedia Commons

Major depressive disorder is characterized by a syndrome of symptoms, present during a two-week period and representing a clinically significant change from previous functioning. The symptoms include at least five of the following: depressed or irritable mood for most of the day, diminished interest in previously pleasurable activities, significant unintentional weight loss or weight gain, [insomnia](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293872050%22%0a%09%09%09%09&sl=ll) or hypersomnia, physical agitation or slowness, loss of energy or [fatigue](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2289093407%22%0a%09%09%09%09&sl=ll), feelings of worthlessness or excessive guilt, indecisiveness or a diminished ability to concentrate, and recurrent thoughts of death. The clinical **depression** cannot be initiated or maintained by another illness or condition.

Major depressive disorder is often first recognized in the patient’**s** twenties, while a major depressive episode can occur at any age. Women are twice as likely to suffer from the disorder than are men.

There are several potential causes of major depressive disorder. [Genetic factors](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2294416443%22%0a%09%09%09%09&sl=ll) may determine a person'**s** susceptibility to developing **depression** following stressful life events. Genetic studies suggest a familial link with higher rates of clinical **depression** in first-degree relatives. There also appears to be a relationship between clinical **depression** and levels of the brain’**s** neurochemicals, specifically decreased monoamines—the neurotransmitters dopamine, [norepinephrine](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2289143348%22%0a%09%09%09%09&sl=ll), and [serotonin](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%22100259358%22%0a%09%09%09%09&sl=ll). It is important to keep in mind, however, that anywhere from 15 to 20 percent of adults will experience major **depression** at some point in their lifetimes. Furthermore, not everyone has a biological cause for this **depression**. Common causes of clinical **depression** also include [psychosocial](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2289550636%22%0a%09%09%09%09&sl=ll) stressors such as the death of a loved one, financial stress, loss of a job and unemployment, interpersonal problems, or traumatic world events such as natural disasters and war. It is unclear, however, why some people respond to a specific psychosocial stressor with a clinical **depression** and others do not. Finally, certain prescription medications have been noted to cause or be related to clinical **depression**. These drugs include muscle relaxants, heart medications, hypertensive medications, ulcer medications, oral contraceptives, painkillers, narcotics, and steroids. Thus there are many causes of clinical **depression**, and no single cause is sufficient to explain all clinical depressions.

Other likely risk factors for **depression** include past alcohol dependence, insecure attachment to parents in early adolescence, and the experience of [childhood abuse or neglect](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2289185463%22%0a%09%09%09%09&sl=ll). Possible risk factors for **depression** that have been explored include cannabis use, low birth weight, high levels of television viewing and media exposure in adolescence, and head injury.

In the DSM-5, the existence of at least three manic symptoms (which is insufficient to satisfy the diagnostic criteria for a manic episode) within a major depressive episode is acknowledged by the specifier "major depressive disorder with mixed features." The presence of mixed features in an episode of major depressive disorder increases the likelihood that the illness exists in the bipolar spectrum, although separate criteria exist for the diagnosis of [bipolar disorder](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293871807%22%0a%09%09%09%09&sl=ll), which can share some symptoms with major **depression**.

Dysthymic disorder is another persistent depressive disorder characterized by chronic low-level **depression**. In the United States, the twelve-month prevalence of dysthymic disorder is estimated to be approximately 1.5 percent of the adult population. Dysthymic disorder is characterized by at least a two-year history of depressed mood and at least two of the following symptoms that cause clinically significant impairment in social, work, or other important areas of functioning: poor appetite or overeating, insomnia or hypersomnia, low energy or fatigue, low [self-esteem](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293872219%22%0a%09%09%09%09&sl=ll), poor concentration or decision making, or feelings of hopelessness. The individual cannot be without the symptoms for more than two months at a time, the disorder cannot be superimposed on another psychotic disorder, and it cannot be initiated or maintained by another illness or condition. Dysthymic disorder is more common in adult women, equally common in both sexes of children, and with a greater prevalence in families. The causes of dysthymic disorder are believed to be similar to those listed for major depressive disorder, but the disorder is less well understood than is **depression**.

In order to prevent the overdiagnosis of bipolar disorder in children, the DSM-5 added a new depressive disorder called disruptive mood dysregulation disorder (DMDD). This diagnosis is given to children up to the age of eighteen years who exhibit persistent irritability and frequent episodes of extreme emotional outbursts and behavioral dyscontrol. DMDD is characterized by severe and recurrent temper outbursts that are grossly out of proportion in intensity or duration to the situation at hand, occurring on average three or more times per week for one year or more. Diagnosis of DMDD requires the symptoms to be present in at least two settings (at school, at home, and/or in social settings), and the child cannot have gone three or more consecutive months without symptoms to be diagnosed with DMDD. Onset of DMDD must occur before the age of ten years, and diagnosis cannot be made for the first time before the age of six years or after eighteen years.

Also in the category of depressive disorders, the DSM-5 includes premenstrual dysphoric disorder (PMDD), which was previously categorized under Appendix B "Criteria Sets and Axes Provided for Further Study" in the DSM-IV, due to a strong body of evidence supporting its existence and the validity of the diagnostic criteria. PMDD is an extreme version of [premenstrual syndrome](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2287324455%22%0a%09%09%09%09&sl=ll) that affects approximately 2 to 5 percent of women of reproductive age. PMDD is characterized by the presence of symptoms for most of the time during the last week of the luteal phase of the [menstrual cycle](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2287690366%22%0a%09%09%09%09&sl=ll); these symptoms begin to remit within a few days of the onset of the follicular phase and are not present in weeks following menstruation. For the diagnosis of PMDD, a woman must have five or more of the following symptoms for most menstrual cycles during the past one year: markedly depressed mood or feelings of hopelessness, marked anxiety or tension, persistent anger or irritability or increased interpersonal conflicts, sense of difficulty in concentrating, lethargy or fatigue, marked changes in appetite, hypersomnia or insomnia, feelings of being overwhelmed or out of control, and/or physical symptoms such as headache, joint or muscle pain, and breast tenderness. These symptoms must also cause a clinically significant impact on functioning at work, school, and social settings or within personal relationships.

A final variant of clinical **depression** is known as [seasonal affective disorder](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2286196274%22%0a%09%09%09%09&sl=ll) (SAD). Patients with this illness demonstrate a pattern of clinical **depression** during the winter, when there is a reduction in the amount of daylight hours. For these patients, the reduction in available light is thought to be the cause of the **depression**. In the DSM-5, SAD is categorized as a mood disorder with a specifier called "with seasonal pattern."

**Treatment and Therapy**

Crucial to the choice of treatment for clinical **depression** is determining the variant of **depression** being experienced. Each of the diagnostic categories has associated treatment approaches that are more effective for a particular diagnosis. Multiple assessment techniques are available to the health care professional to determine the type of clinical **depression**. The most valid and reliable is the [clinical interview](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293871839%22%0a%09%09%09%09&sl=ll). The health care provider may conduct either an informal interview or a structured, formal clinical interview assessing the symptoms that would confirm the diagnosis of clinical **depression**. If the patient meets the diagnostic criteria set forth in the DSM-5, then the patient is considered for **depression** treatments. Patients who meet many but not all diagnostic criteria are sometimes diagnosed with a “subclinical” **depression**. These patients might also be considered appropriate for the treatment of **depression**, at the discretion of their health care providers.

Another assessment technique is the “paper-and-pencil” measure, or **depression** questionnaire. A variety of questionnaires have proven useful in confirming the diagnosis of clinical **depression**. Questionnaires such as the [Beck Depression Inventory](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293871795%22%0a%09%09%09%09&sl=ll), Hamilton **Depression** Rating Scale, Zung Self-Rating **Depression** Scale, and the Center for Epidemiologic Studies **Depression** Scale are used to identify persons with clinical **depression** and to document changes with treatment. This technique is often used as an adjunct to the clinical interview and rarely stands alone as the definitive assessment approach to diagnosing clinical **depression**.

Once a clinical **depression** (or a subclinical **depression**) is identified, several types of treatment options are available. These options are dependent on the subtype and severity of the **depression**. They include individual and group psychotherapy, light therapy, family therapy, [psychopharmacology](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293872181%22%0a%09%09%09%09&sl=ll) (drug therapy), electroconvulsive therapy (ECT), and other less traditional treatments. These treatment options can be provided to the patient as part of an outpatient program or, in certain severe cases of clinical **depression** in which the person is a danger to the self or others, as part of a hospitalization.

Clinical **depression** often affects the patient physically, emotionally, and socially. Therefore, prior to beginning any treatment with a clinically depressed individual, the health care provider will attempt to develop an open and communicative relationship with the patient. This relationship will allow the health care provider to provide patient education on the illness and to solicit the collaboration of the patient in treatment. Supportiveness, understanding, and collaboration are all necessary components of any treatment approach.

For the treatment of mild to moderate **depression** in adults, the American Psychiatric Association (APA) recommends [psychotherapy](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293872001%22%0a%09%09%09%09&sl=ll) as the initial treatment choice. The APA also recommends [antidepressant medications](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293871772%22%0a%09%09%09%09&sl=ll) as an initial treatment choice, whereas the National Institute for Clinical Excellence (NICE) recommends antidepressants only if the patient is unresponsive to initial psychosocial interventions. For moderate to severe **depression** in adults, the APA and the NICE recommend a combination of psychotherapy and antidepressants. The APA also recommends electroconvulsive therapy (ECT) for the treatment of severe unresponsive major **depression** in adults.

For the treatment of **depression** in children and adolescents, the recommended initial treatment choices include education, supportive treatment, and case management. If **depression** is complicated or chronic, psychotherapy may then be recommended. Interpersonal therapy and [cognitive-behavioral therapy](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2293871842%22%0a%09%09%09%09&sl=ll) have been shown to be among the best psychotherapeutic options for the treatment of **depression**. If the child or adolescent with **depression** is unresponsive to psychotherapy, he or she may benefit from some types of antidepressant medications; however, in most children with **depression**, antidepressants do not appear to be an effective treatment.

Psychotherapy refers to a number of different treatment techniques used to deal with the psychosocial contributors and consequences of clinical **depression**. In psychotherapy, the patients develop knowledge and insight into the causes of and treatment for their clinical **depression**. In cognitive psychotherapy, symptom relief comes from assisting patients in modifying maladaptive, irrational, or automatic beliefs that can lead to clinical **depression**. In behavioral psychotherapy, patients modify their environment such that social or personal rewards are more forthcoming. This process might involve being more assertive, reducing isolation by becoming more socially active, increasing physical activities or exercise, or learning relaxation techniques or other coping skills. Research upholds the effectiveness of these and other psychotherapy techniques for the treatment of **depression** and other mood disorders.

The primary types of medications used in the treatment of clinical **depression** in adults include [selective serotonin reuptake inhibitors](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2294416245%22%0a%09%09%09%09&sl=ll) (SSRIs), serotonin norepinephrine reuptake inhibitors (SNRIs), mirtazapine (Remeron), and bupropion (Wellbutrin). Monoamine oxidase inhibitors (MAOIs) should be restricted to patients who do not respond to other treatments. The health care professional will select an antidepressant based on [side effects](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2289093551%22%0a%09%09%09%09&sl=ll), dosing convenience (once daily versus three times a day), and cost.

Cyclic antidepressants represent one class of antidepressant medications. As the name implies, the chemical makeup of the medication contains chemical rings, or “cycles.” There are unicyclic (buproprion and fluoxetine, or [Prozac](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2289408076%22%0a%09%09%09%09&sl=ll)), bicyclic (sertraline and trazodone), tricyclic (amitriptyline, desipramine, and nortriptyline), and tetracyclic (maprotiline) antidepressants. These antidepressants function to either block the reuptake of neurotransmitters by the neurons, allowing more of the neurotransmitter to be available at a receptor site, or increase the amount of neurotransmitter produced. The side effects associated with the cyclic antidepressants—dry mouth, blurred vision, constipation, urinary difficulties, palpitations, and sleep disturbance—vary and can be quite problematic. Some of these antidepressants have deadly toxic effects at high levels, so they are not prescribed to patients who are at risk of [suicide](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2289312380%22%0a%09%09%09%09&sl=ll). Furthermore, in some patients, antidepressants such as SSRIs are associated with increased suicidal ideation, so patients should be carefully monitored as they begin an antidepressant treatment regimen.

Newer drugs are more specific in terms of the drug action. For instance, fluoxetine is a selective serotonin reuptake inhibitor (SSRI) and works specifically on the neurotransmitter serotonin. Similarly, buproprion is a norepinephrine and dopamine reuptake inhibitor (NDRI) and works specifically on the neurotransmitters norepinephrine and dopamine. More specific drugs generally create fewer side effects. Fewer side effects can be associated with greater medication compliance, making these drugs a more effective treatment for many individuals.

Monoamine oxidase inhibitors (isocarboxazid, phenelzine, and tranylcypromine) are another class of antidepressants. They function by slowing the production of the enzyme monoamine oxidase. This enzyme is responsible for breaking down the neurotransmitters norepinephrine and serotonin, which are believed to be responsible for **depression**. By slowing the decomposition of these transmitters, more of them are available to the receptors for a longer period of time. Restlessness, dizziness, weight gain, insomnia, and sexual dysfunction are common side effects of the MAOIs. MAOIs are most notable because of the dangerous adverse reaction (severely high blood pressure) that can occur if the patient consumes large quantities of foods high in tyramine (such as aged cheeses, fermented sausages, red wine, foods with a heavy yeast content, and pickled fish). Because of this potentially dangerous reaction, MAOIs are not usually the first choice of medication and are more commonly reserved for depressed patients who do not respond to other treatment options.

Electroconvulsive or [shock therapy](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2287690622%22%0a%09%09%09%09&sl=ll) is the single most effective treatment for severe and persistent **depression** that does not respond to other treatments. If the clinically depressed patient fails to respond to medications or psychotherapy and the **depression** is life-threatening, electroconvulsive therapy is considered. It is also considered if the patient cannot physically tolerate antidepressants, as with elderly patients who have other **medical** conditions. This therapy involves inducing a seizure in the patient by administering an electrical current to specific parts of the brain. The therapy has become quite sophisticated and much safer than when it was introduced in the mid-twentieth century, and it involves fewer risks to the patient. Patients undergo several treatments over a period of time. Some temporary memory impairment is a common side effect of this treatment.

A special treatment used for individuals with seasonal affective disorder is [light therapy](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2287690567%22%0a%09%09%09%09&sl=ll), or phototherapy. Light therapy involves exposing patients to bright light for a period of time each day during seasons of the year when there is decreased light. This may be done as a preventive measure and also during depressive episodes. The manner in which this treatment approach modifies the **depression** is unclear and awaits further research, but some believe it affects the internal clock of the body, or circadian rhythm. Studies of the effectiveness of light therapy have been mixed, but interest in this promising treatment is strong, as it may prove useful for working with nonseasonal mood disorders as well. It should be noted, however, that light therapy does have some risks associated with it. Caution must be used to protect the eyes and to use the light as directed. Additionally, the intensity of light must be correct so as to achieve therapeutic effects and not cause other problems. Finally, some individuals can experience manic episodes if they are exposed to too much light, so caution must be exercised in terms of the length of time for light exposure treatment sessions.

Surgery, the final treatment option for severe **depression**, is quite rare. Psychosurgery is used only after all treatment options have failed and the clinical **depression** is life-threatening. Vagus nerve stimulation (VNS) is a form of surgery that implants a stimulus generator on the [vagus nerve](http://eds.a.ebscohost.com.libdatab.strayer.edu/eds/detail/?sid=17fc5727-21de-4674-944c-9b6075944ee9@sessionmgr4007&vid=1&db=ers&ss=AN+%2287690419%22%0a%09%09%09%09&sl=ll); it is approved by the FDA for the treatment of severe unresponsive **depression**. Nonsurgical methods of creating similar stimuli have been explored as well.

**Perspective and Prospects**

**Depression**, or the more historical term “melancholy,” has had a history predating modern medicine. Writings from the time of the ancient Greek physician Hippocrates refer to patients with a symptom complex similar to the present-day definition of clinical **depression**.

The rates of clinical **depression** have increased since the early twentieth century, while the age of onset of clinical **depression** has decreased. Women appear to be at least twice as likely as men to suffer from clinical **depression**.

While most psychiatric disorders are nonfatal, clinical **depression** can lead to death. About 60 percent of individuals who commit suicide have a mood disorder such as **depression** at the time. In a lifetime, however, only about 7 percent of men and 1 percent of women with lifetime histories of **depression** will commit suicide. Though these numbers are high, what this means is that not everyone who is depressed will commit suicide. In fact, many receive help and recover from **depression**. There are, however, other costs of clinical **depression**. Billions of dollars are spent on clinical **depression**, divided among the following areas: treatment, suicide, and absenteeism (the largest). Clinical **depression** obviously has a significant economic impact on society, and major personal impacts on the lives of individuals suffering from **depression**.

Studies have shown a marked increase in **depression** among those in their twenties and thirties in the early twenty-first century. The American College Counseling Association reported in 2012 that there had been a 16 percent increase in visits to mental health counselors on college campuses since 2000. A reported 44 percent of college students experience **depression** symptoms. In a 2011 study, the CDC found overall that from the period between 1988–1994 to the period between 2005–2008, antidepressant use had increased by 400 percent.

The future of clinical **depression** lies in early identification and treatment. Identification will involve two areas. The first is improving the social awareness of mental health issues to include clinical **depression**. By eliminating the negative social stigma associated with mental health treatment, there will be an increased level of the reporting of **depression** symptoms and thereby an improved opportunity for early intervention, preventing the progression of the disorder. The second approach to identification involves the development of reliable assessment strategies for clinical **depression**. Data suggests that the majority of those who commit suicide see a physician within thirty days of the suicide. The field of psychology will continue to strive to identify biological markers and other methods to predict and identify clinical **depression** more accurately. Treatment advances will focus on the further development of nonpharmacological and pharmacological strategies to increase effectiveness.

1. **Article 2.** **Depression**, anxiety, and tobacco use: Overlapping impediments to sleep in a national sample of **college** **students**.

By: Boehm, Matthew A.; Lei, Quinmill M.; Lloyd, Robin M.; Prichard, J. Roxanne*. Journal of American* ***College*** *Health* , Oct2016, Vol. 64 Issue 7, p565-574, 10p, 3 Charts, 4 Graphs; DOI: 10.1080/07448481.2016.1205073, Database: [Education Source](http://libdatab.strayer.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=eue&bquery=XY+%26quot%3bimpediments%26quot%3b%5b100%5d+AND+(XY+%26quot%3bsleep%26quot%3b%5b93%5d+OR+XY+%26quot%3boverlapping%26quot%3b%5b92%5d+OR+XY+%26quot%3btobacco%26quot%3b%5b90%5d+OR+XY+%26quot%3bdepression%26quot%3b%5b68%5d+OR+XY+%26quot%3banxiety%26quot%3b%5b62%5d+OR+XY+%26quot%3bsample%26quot%3b%5b41%5d+OR+XY+%26quot%3bnational%26quot%3b%5b32%5d+OR+XY+%26quot%3bcollege%26quot%3b%5b27%5d+OR+XY+%26quot%3buse%26quot%3b%5b26%5d+OR+XY+%26quot%3bstudents%26quot%3b%5b18%5d)&cli0=FT&clv0=Y&type=0&site=ehost-live&scope=site&custID=strayer&groupID=main&profID=ehost#_blank)

ABSTRACT Objectives: To examine how tobacco use and depression/anxiety disorders are related to disturbed sleep in college students. Participants: 85,138 undergraduate respondents (66.3% female, 74.5% white, non-Hispanic, ages 18–25) from the Spring 2011 American College Health Association–National College Health Assessment II database. Methods: Multivariate analyses of tobacco use (none, intermediate, daily) and mental health (diagnosed and/or symptomatic depression or anxiety) were used to predict sleep disturbance. Results: Daily tobacco use was associated with more sleep problems than binge drinking, illegal drug use, obesity, gender, and working >20 hours/week. Students with depression or anxiety reported more sleep disturbances than individuals without either disorder, and tobacco use in this population was associated with the most sleep problems. Conclusions: Tobacco use and depression/anxiety disorders are both independently associated with more sleep problems in college students. Students with depression and/or anxiety are more likely to be daily tobacco users, which likely exacerbates their sleep problems.

Emerging adulthood is a formative time for overall health, a sensitive period for forming lifestyle patterns, and an age when mental illnesses often ﬁrst emerge.1 Colleges and universities in the United States are working to cope with record high levels of psychological distress among students, a trend that has been attributed to multiple factors, including increased ﬁnancial stress, the changing demographics of college students, increasing technology dependence, and the lifestyle changes young adults experience as they abruptly gain independence in decision making.2,3 Accordingly, colleges and universities are searching for ways to improve students’ health through early identiﬁcation and treatment of mental illness, identifying modiﬁable risk factors, and promoting healthier lifestyle choices. In April 2012, the American College Health Association (ACHA) introduced the Healthy Campus 2020 initiatives, which include goals to reduce the incidence of speciﬁc negative health behaviors, including nicotine use and sleep disturbance, by 10% by 2020.4 Data from the Spring 2015 ACHA–National College Health Assessment (NCHA) II show that within the last year, 15.8% of undergraduates have been diagnosed with or treated for an anxiety disorder, 13.1% have been diagnosed with or treated for depression, 20% have reported

that their academic performance has been adversely impacted by sleep problems, and that within the last month at least 10% have used tobacco.5 Recently, Ridner et al identiﬁed sleep quality and tobacco use as 2 of the most important predictors in students’ subjective sense of well-being.6 Depression, anxiety, and frequent tobacco use have all been independently linked to sleep problems, and those with depression and/or anxiety are more likely than others to use tobacco.7–9 To gain a better understanding of the relationships among these variables in college students, we used multivariate analysis on data from the Spring 2011 ACHA-NCHA II database to evaluate the relative impacts of anxiety, depression, and tobacco use on rates of sleep disturbance.

Depression, anxiety, and sleep disturbance The relationships between depression, anxiety, and sleep have been a topic of research for over 40 years. Among individuals with depression, sleep disturbances often include difﬁculty with sleep initiation, frequent early awakenings, and insomnia or hypersomnia.10 Depression is also associated with electrophysiological disturbances in sleep, including disinhibition of rapid eye movement (REM) sleep, REM sleep fragmentation, a reduction of slow-wave sleep, and circadian rhythm dysregulation.11 Sleep disturbances in anxiety disorders include difﬁculty in initiating and/or maintaining sleep, restless or unsatisfying sleep, nightmares, insomnia, and alterations in sleep architecture similar to those with depression.12 Depression and anxiety are often comorbid with insomnia, sleep problems exacerbate depressive and anxious symptoms, and insomnia is a risk factor in the development of depression and anxiety disorders.7,12–14 There is growing evidence from prospective population studies that disturbed sleep is in itself a predictor and modiﬁable risk factor for anxiety and depression. Data from a national survey of adults showed that frequent insufﬁcient sleep is associated with depressive and anxiety disorders, and the odds of having a sleep disorder are increased when both classes of psychiatric disorders are diagnosed.15 A population study of approximately 15,000 participants found that in those with depressive disorders, more than 40% experienced insomnia before the onset of the mood disorder symptoms and more than 20% experienced the symptoms concurrently.16 However, in individuals diagnosed with anxiety disorders, insomnia appeared concurrently (>38%) or after (>34%) the anxiety disorder.16 An adolescent population study (N > 1,000) found that among those with comorbid anxiety and depression, anxiety disorders preceded 73% of insomnia diagnoses, whereas insomnia preceded 69% of comorbid insomnia and depression cases. 17 Prospective studies have also shown that persistent sleep problems in childhood predict adult anxiety disorders.18 In adults, both anxiety and depression at baseline predict new cases of insomnia, and insomnia at baseline predicts new diagnoses of anxiety and depression 1 year later.19 Understanding the nature of these relationships should be of importance to college health professionals because sleep disturbances are clinically relevant for both evaluation and treatment of mental health. Individuals with anxiety disorders and poor sleep experience signiﬁcantly worse mental health related quality of life and increased disability, compared with those with anxiety disorders alone.20 Furthermore, insomnia severity is a predictor of suicidal ideation in individuals with depression, even after controlling for cofactors such as level of depressed mood and anhedonia.21 Treating insomnia improves depression and anxiety symptoms, and treating anxiety/depression improves insomnia.22–24 tobacco use, depression/anxiety, and sleep Tobacco use is more common among individuals with depression and/or anxiety than in the general population.9 In national surveys, people with a lifetime history of depression, anxiety, or comorbid anxiety and depression were more likely to be current smokers, smoke with higher intensity and dependence, and have lower success at quitting than those without a history of anxiety or depression.25 A 26-year longitudinal study demonstrated that smoking is associated with an increased risk of developing depression later in life; individuals who smoked more than 10 g of tobacco per day had signiﬁcantly higher depression rates than did nonsmokers.26 A 10-year longitudinal study of adolescents showed that both occasional and daily smokers with high levels of depression and anxiety symptoms had an approximately 2-fold increase in nicotine dependence in young adulthood, compared with those with low levels of adolescent depression and anxiety symptoms.27 Similarly, an earlier age of smoking onset predicts an earlier development of anxiety disorders, even after controlling for the effects of gender, education, and childhood trauma.28 Many people who are vulnerable to depression or anxiety use tobacco because they expect smoking to relieve negative affect.29 However, tobacco use might actually exacerbate symptoms of depression and anxiety through impairments to sleep. Polysomnography studies reveal that smokers have longer sleep latencies, less slow wave sleep, and less total sleep time compared with nonsmokers.8,30 Smokers tend to have higher REM densities, more leg movements throughout the night, and more breathing difﬁculties during sleep and report feeling less refreshed in the morning compared with nonsmokers.31 In a sample of US adults, those who were tobacco users had twice the odds of insufﬁcient sleep compared with non-tobacco users and secondhand smoke exposure was associated with insufﬁcient rest among nonsmokers.32 In addition, a 25-year prospective study showed that women who were chronic heavy smokers had a 2.76 times increased chance for reporting insomnia in late adulthood.33 In smokers, sleep deprivation increases the frequency of cigarette smoking, which can in turn negatively impact sleep.34 In contrast, a reduction in smoking is associated with improvements in sleep, and better sleep can help with successful reduction and cessation of smoking.9,35

Hypotheses We sought to examine how tobacco use and depression/ anxiety disorders are related to disturbed sleep in a national sample of college students. Speciﬁcally, we hypothesized that those with depression and anxiety, or probable undiagnosed anxiety and depression, will be more likely to have impaired sleep (H1) and more likely to use tobacco (H2) than those without these disorders. Furthermore, we hypothesized that tobacco use will be associated with increased reports of sleep disturbance (H3), particularly among those with anxiety/mood disorders (H4). This study is the ﬁrst to explore the interaction of these factors in a large national sample of college students.

Methods Survey We used data from the Spring 2011 American College Health Association–National College Health Assessment II (ACHA-NCHA II).36 The ACHA-NCHA II is a nationally recognized research survey that is used to collect precise data about students’ health habits, behaviors, and perceptions. It is systematically evaluated for reliability and validity by comparing common survey items with national studies such as the National College Health Risk Behavior Survey (Centers for Disease Control and Prevention [CDC]) and College Alcohol Study (Harvard School of Public Health). The survey consists of 294 questions regarding physical and mental health, health education, and alcohol, tobacco, and drug use, as well as questions about impediments to academic performance. The Spring 2011 survey was administered to a total of 105,781 students at 129 postsecondary institutions, including associate’s, baccalaureate, master’s, and research institutions. We limited our analysis to participants aged 18–25 (ND85,218) in order to eliminate age as a possible confounding variable. In addition, respondents whose data seemed purposely falsiﬁed (eg, students who indicated daily use of every type of drug [nD44] or a diagnosis of all 15 mental disorders [n D 36]) were excluded from the data analyses, reducing the number of student respondents in the sample to 85,138. The sample used for this analysis was 66.3% female, 73.7% white, 11.1% Asian/Paciﬁc Islander, 7.8% Hispanic/Latino, 5.7% black non-Hispanic, and 1.7% American Indian/ Alaskan Native/Hawaiian Native.

Classiﬁcation of tobacco use To quantify tobacco use, we coded responses to ACHANCHA Question 8, “Within the last 30 days, on how many days did you use: cigarettes, tobacco from a water pipe (hookah), cigars, little cigars, clove cigarettes, and smokeless tobacco,” into 3 categories. Participants in the None category reported “Never used” or “Have used, but not in last 30 days” (n D 64,369). Participants in the Intermediate category reported tobacco use “1–2 days, 3–5 days, 6–9 days, 10–19 days, or 20–29 days” in the last 30 days (n D 15,535), and Daily tobacco users responded “Used daily” (n D 3,890). We opted for this categorical grouping because the ACHA-NCHA II

does not include quantitative data regarding the number of times a day a person uses tobacco products.

Classiﬁcation of depression To get closer to capturing the true population, we included individuals who were undiagnosed but symptomatic for depression/anxiety in our analysis. Research indicates that at least 25% of individuals with depression are undiagnosed and less than half receive treatment.37,38 We combined individuals who responded that they had been formally diagnosed with depression (a positive response to ACHA-NCHA II Question 31, “Within the last 12 months have you been diagnosed or treated by a professional for depression (n D 7,924) and/or bipolar disorder (n D 955)”) with respondents who appeared to be symptomatic for depression. Participants who endorsed at least 3 of the following 4 statements were classiﬁed as having probable undiagnosed depression (n D 9,389): In the last 12 months, have you felt things were hopeless (Question 30A); felt very sad (30E); felt so depressed that it was difﬁcult to function (30F), and seriously considered suicide (30J). This estimation likely underestimates participants with undiagnosed depression because students who were formally diagnosed with depression reported an average of 2.7 of the 4 symptoms, whereas we set the cutoff for probable undiagnosed depression at 3 of 4 symptoms. The ACHA-NCHA II survey questions used for this classiﬁcation are similar to other criteria used for depression diagnosis (eg, DSM-V [Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition] diagnostic criteria for Depression, the Hamilton Rating Scale for Depression, and the Center for Epidemiologic Studies Depression Scale). Of the 17,205 individuals who were either diagnosed with or symptomatic for depression, 9,198 (53%) were also either diagnosed with or symptomatic for an anxiety disorder.

Classiﬁcation of anxiety It is estimated 20% of adults meet criteria for at least 1 anxiety disorder, but are not formally diagnosed.39 In order to get closer to capturing the true population, we combined individuals who responded that they had been formally diagnosed with an anxiety disorder (nD9,721), deﬁned as a positive response to Question 31, “Within the last 12 months have you been diagnosed or treated by a professional for panic attacks (nD4,231), obsessive compulsive disorder (n D 1,777), and/or anxiety (n D 8,898),” with respondents who appeared to by symptomatic for an anxiety disorder (n D 6,559). Of the 9,721 individuals diagnosed with an anxiety disorder, 5,185 had a comorbid anxiety disorder (eg, panic disorder and obsessive compulsive disorder). Individuals who endorsed at least 2 of the following 3 statements: experienced “tremendous stress” within the past 12 months (Question 37); “felt overwhelming anxiety within the last 2 weeks” (30G); and reported that anxiety has negatively affected academic performance within the last 12 months (45C) were classiﬁed as having probable undiagnosed anxiety. This estimation likely underestimates participants with undiagnosed anxiety because students who were formally diagnosed with anxiety reported an average of 1.2 of the 3 symptoms, whereas we set the cutoff for probable undiagnosed anxiety at 2 of 3 symptoms. The ACHA-NCHA II survey questions used for this classiﬁcation are similar to other criteria used for anxiety diagnosis (eg, DSM-V diagnostic criteria for Generalized Anxiety Disorder, Generalized Anxiety Disorder 7-item scale, Hamilton Anxiety Rating Scale).

Comorbid depression and anxiety In the sample population, 5,279 individuals were formally diagnosed with comorbid anxiety and depression. Within the sample, 498 individuals were formally diagnosed with depression and also reported symptoms of probable anxiety, 795 received a formal diagnosis of an anxiety disorder and reported symptoms of probable depression, and 2,626 individuals reported concurrent symptoms from both probable undiagnosed anxiety and undiagnosed depression groups as deﬁned above, yielding a total of 9,198 groupedin the comorbid classiﬁcation.

Reported sleep problems Self-reported sleep problems were deﬁned as the average score of responses to questions about sleep in the past 7 days: In the last week, how many days did you “get enoughsleep to feelwell rested in the morning” (Question 42,reverse scored); “awoketoo early and couldn’t get back asleep” (44A); “felt tired or sleepy during the day” (44B); “gone to bed because they could not stay awake any longer” (44C); and “had an extremely hard time falling asleep” (44D). The impact of poor sleep on daily life and academic performance was also assessed with the following questions: “Within last 12 months have sleep difﬁculties been traumatic or very difﬁcult to handle?” (33K) and “Within the last 12 months have sleep difﬁculties affected academicperformance?” (45CC).

Statistical analyses All statistical analyses were performed with IBM SPSS version 22 (Armonk, NY). We performed a 2 (depression: yes/no) £ 2 (anxiety: yes/no) £ 3 (tobacco:

none, intermediate, daily) multivariate analysis of variance (MANOVA) with least signiﬁcant difference (LSD) post hoc tests to calculate the various impacts of depression, anxiety, and tobacco on sleep. The alpha level was set at p < .01. Additionally, chisquare and odds ratio analyses were used to assess the impact of daily tobacco use, as compared with other factors, on responses to categorical questions regarding the impact of poor sleep on daily life and academic performance (questions 33K and 45CC).

Results Depression/anxiety Within the population sample, 9.9% were diagnosed with or symptomatic of depression (n D 8,007), 8.3% were diagnosed with or symptomatic of an anxiety disorder (n D 6,683), and 11.1% were diagnosed with and/or symptomatic of both anxiety and depression (n D 9,198). Of the 23,888 individuals diagnosed with or identiﬁed as having probable anxiety and/or depression, 45.4% received a formal diagnosis (n D 10,854) and 34.0% received some form of treatment (n D 8,124). Those with co-occurring anxiety and depression had the highest occurrence of a number of problematic factors, including traumatic sleep difﬁculties, insomnia diagnosis, suicide ideation/attempt, daily tobacco use, and marijuana use in the last month (Table 1). In support of hypothesis 1, there was a signiﬁcant association between depression/anxiety status (anxiety, depression, comorbid anxiety/depression, or no anxiety/depression symptoms or diagnoses) and the number of self-reported sleep problems (Figure 1). Those with diagnosed and/or symptomatic anxiety (F1,78908 D 793.29, p < .001) or depression (F1,78908 D 696.22, p < .001) had signiﬁcantly higher number of self-reported sleep problems in the last week than those without either disorder, but there was no signiﬁcant difference in the average number of sleep problems among those with anxiety compared with those with depression. There was a signiﬁcant interaction between depression and anxiety status; individuals with co-occurring depression and anxiety reported the highest average number of sleep problems per week (F1,78908 D 39.78, p < .001). Depression/anxiety status also signiﬁcantly predicted the proportion of students who reported that sleep problems negatively impacted their academic performance (x2(3, N D 80,597) D 7084.31, p < .001). Among those without anxiety or depression, 13.3% reported that sleep problems negatively impacted academic performance. In contrast, 31.2% of those with depression, 32.8% of those with anxiety, 47.6% of those with co-occurring depression and anxiety reported this occurrence.

Tobacco use Within the sample population, 76.8% reported not using tobacco in the last month (n D 64,369), 18.5% reported intermediate tobacco use (nD15,535) and 4.6% reported daily tobacco use (nD3,890). In support of hypothesis 2, those with depression and/or anxiety, or probable undiagnosed anxiety and/or depression, were more likely to use tobacco (x2(4, ND79,706)D1073.31, p < .001). In support of hypothesis 3, the frequency of tobacco use was associated with an increase in the number of selfreported sleep problems in the last week, regardless of depression/anxiety status (F2,78897 D 81.87, p < .001; Figure 2). Daily tobacco users reported a higher number of sleep complaints in the last week than both nonusers and intermediate users (p < .01). Tobacco use frequency also signiﬁcantly predicted the proportion of students who reported that sleep problems negatively impacted their academic performance (x2(2, N D 83,039) D 741.90, p < .001. Of students with no tobacco use in the last month, 18.7% reported that sleep problems negatively affected academic performance, whereas 25.7% of students with intermediate tobacco use and 32.8% of students with daily tobacco use reported this occurrence. Daily tobacco use increased the odds of sleep negatively affecting academic performance by 2.13 (95% conﬁdence interval [CI]: 1.99–2.28). Tobacco use and depression/anxiety Depression and anxiety status was signiﬁcantly associated with tobacco use frequency in support of hypothesis 1( x2(6, N D 79,706) D 1127.98, p < .001; Figure 3). Among nonusers, 27.1% were either diagnosed with or symptomatic for depression or anxiety, as compared with 36% of intermediate users and 44.9% of daily tobacco users. Anxiety categorization increased the odds of daily tobacco use by 2.15 (95% CI: 2.01–2.31) and depression categorization increased the odds of daily tobacco use by 2.31 (95% CI: 2.15–2.48). Fifty-six percent of daily tobacco users with cooccurring depression and anxiety reported that sleep problems negatively impacted their academic performance, whereas 50.5% of intermediate users and 45.3% of nonusers comorbid for these disorders reported negative academic impacts due to sleep problems. Regardless of disorder type, daily tobacco use was associated with both an increased reporting of academic problems caused by sleep difﬁculties, and of sleep being experienced as traumatic or difﬁcult to handle (Table 2).

To compare the impacts of tobacco on sleep problems relative to other risk factors, we provided a table of odds ratio comparisons for relevant factors as they relate to the likelihood of experiencing traumatic sleep difﬁculties in the last 12 months and reporting that sleep problems negatively impacted academic performance (Table 3). Depression/anxiety status was the strongest indicator for these sleep problems. Depression increased the odds of traumatic sleep difﬁculties by 4.11 (95% CI: 3.91–4.32), anxiety increased the odds by 3.23 (95% CI: 3.09–3.45), and comorbid depression/anxiety by 7.55 (95% CI: 7.20– 7.92). Tobacco use was the second strongest indicator for these sleep problems, as daily tobacco use increased the odds of traumatic sleep difﬁculties by 2.25 (95% CI: 2.11–2.41). Both depression/anxiety status and daily tobacco use had larger impacts on sleep than obesity, illegal drug use, binge drinking, gender, and working/volunteering 20 hours a week or more. Figure 4 compares the mean number of sleep problems across the 12 groups in the multivariate analysis. In support of hypothesis 4, the lowest number of sleep problems was observed in nonusers without depression or anxiety (2.18 § 0.01) and the highest number was observed in daily tobacco users with co-occurring depression and anxiety (3.63 § 0.04). Multivariate analysis revealed no signiﬁcant interactions between tobacco use and depression/anxiety status in relation to the average number of reported sleep problems (F2,78909 D .60, p > .05). Comment Our results provide strong evidence for the interconnected relationships between depression and anxiety, tobacco use, and sleep problems in young adults. Odds ratio analyses revealed that depression/anxiety status was the strongest predictor of sleep difﬁculties, and those diagnosed with or symptomatic for both disorders experienced the greatest frequency of self-reported sleep problems. Diagnosed/symptomatic individuals also showed a nearly 2-fold increase in the probability of daily tobacco use. In addition, daily tobacco use increased the risk for sleep problems more so than did obesity, illegal substance use, binge drinking, gender, and working/volunteering more than 20 hours a week.

Limitations As with any study of data from a large national sample, we are limited by a number of factors inherent in the survey design. First, all measures are self-reported. We do not have independent conﬁrmation of substance use, medical diagnoses, or sleep problems. Second, we are limited to the survey questions, so we do not have access to questions that are typically included in studies of sleep (eg, total sleep time, daily sleep diaries, circadian preference, and sleep schedule regularity), which limits direct comparisons with other studies of sleep and nicotine use. The ACHA-NCHA II also does not include questions about socioeconomic status (SES). A lower SES has been shown to increase the likelihood of insomnia, as well as smoking initiation and progression to regular use.40,41 Third, the survey respondents were disproportionately white and female and therefore not representative of the current demographics of US college students. Fourth, we constructed a proxy measure to identify students with probable depression and/or anxiety. We are not the ﬁrst to use this approach; Wickens et al established cutoff values in the 12-Item General Health Questionnaire (GHQ-12) to detect probable anxiety and mood disorders in a large national sample.42 We cannot conclude causality using the relationships identiﬁed in this study. Depressed smokers are more likely than nondepressed smokers to report tobacco use as a self-medicating tool to reduce negative affect and increase alertness.43 This selfmedicating behavior seems to be mediated by the dopamine D4 receptor.44 A large, population-based twin study (N 28,000) also demonstrated that the associations between regular tobacco use and major depression are partially mediated by genetics.45 The weight and direction of causality in the association between smoking and mood disturbance remain

unclear. However, evidence suggests a bidirectional relationship, wherein those with depression and/or anxiety use tobacco to reduce mood disturbance during wakefulness, but that tobacco use also impairs sleep, which in turn exacerbates depression and anxiety symptoms.

Conclusions This study is the ﬁrst to explore the interaction of tobacco use, sleep disturbances, and anxiety and depression in a large national sample of college students. Here, we demonstrated that tobacco use and depression/anxiety disorders are both independently associated with more sleep problems in college students, and those students with depression/anxiety are more likely to use tobacco, a behavior that likely exacerbates their sleep problems. These data suggest that treatment of depression and/or anxiety should emphasize tobacco cessation as a way to improve sleep problems associated with these disorders, along with other more established lifestyle modiﬁcations such as implementing stress reduction and improving sleep hygiene. Furthermore, colleges and universities might ﬁnd that focusing on tobacco cessation programs and transitioning to tobacco-free campuses might have secondary positive impacts on mental health and sleep, both of which are initiatives in the Healthy Campus 2020 campaign.

Table 1. Population sample by depression/anxiety status and problematic health factors.

Nondiagnosed and nonsymptomatic 70.5% (nD56,962)

Anxiety (diagnosed or symptomatic) 8.3% (nD6,683)

Depression (diagnosed or symptomatic) 9.9% (nD8,007)

Comorbid anxiety and depression (diagnosed or symptomatic) 11.4% (nD9,198) Sample total (ND80,850) % n % n % n % n % With formal diagnosis 0 0 50.7% 3,390 27.3% 2,185 57.4% 5,279 % Receiving psychological treatment last year 0 0 35.0% 2,341 22.1% 1,770 43.6% 4,013 % Reported tremendous stress in the last year 2.5% 1,417 29.4% 1,962 6.8% 541 38.9% 3,575 % Seriously considered suicide in the last year 0.5% 273 0.7% 45 28.1% 2,253 30.8% 2,836 % Attempted suicide in the last year 0.1% 65 0.2% 13 3.9% 309 4.7% 435 % Diagnosed with insomnia 0.7% 375 4.6% 308 3.7% 299 17.3% 1,588 % Extreme difﬁculty falling asleep >3 days/week 8.3% 4,720 17.5% 1,166 18.3% 1,463 25.8% 2,368 % Reporting sleep as difﬁcult to handle 15.9% 9,010 38.1% 2,541 43.6% 3,484 58.7% 5,385 % Daily tobacco use 3.6% 2,030 5.5% 363 5.9% 468 9.1% 823 % Some tobacco last month (1–29 days) 16.7% 9,399 19.0% 1,250 23.6% 1,865 24.1% 2,182 % Reporting binge drinking in last 2 weeks 35.4% 20,108 36.7% 2,443 37.4% 2,981 38.0% 3,482 % Reporting marijuana use in last month 15% 8,496 18.3% 1,216 23.1% 1,842 26.2% 2,395

### **Article 3.** [DEPRESSION AND ANXIETY STIGMA, SHAME, AND COMMUNICATION ABOUT MENTAL HEALTH AMONG COLLEGE STUDENTS: IMPLICATIONS FOR COMMUNICATION WITH STUDENTS.](http://eds.a.ebscohost.com/eds/viewarticle/render?data=dGJyMPPp44rp2%2fdV0%2bnjisfk5Ie46fKK3%2b%2fjiqzj34HspON88aPqgfTbvk%2btqK9HsKavUJ6suEm3sK5NnsbLPvLo34bx1%2bGM5%2bXsgeKzr020q7RIsa%2b2PurX7H%2b72%2bw%2b4ti7ffPbpIzf3btZzJzfhrunsFGxqLZLr66kfu3o63nys%2bSN6uLyffbq&vid=6&sid=4b14eb9f-e9d5-485c-b2da-f03520e90512@sessionmgr4007" \o "DEPRESSION AND ANXIETY STIGMA, SHAME, AND COMMUNICATION ABOUT MENTAL HEALTH AMONG COLLEGE STUDENTS: IMPLICATIONS FOR COMMUNICATION WITH STUDENTS.)

By: Carmack, Heather J.; Nelson, C. Leigh; Hocke-Mirzashvili, Tatjana M.; Fife, Eric M.. ***College*** ***Student*** Affairs Journal , Spring2018, Vol. 36 Issue 1, p68-79, 12p, 1 Chart, Database: [Education Source](http://libdatab.strayer.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=eue&bquery=DEPRESSION+AND+ANXIETY+STIGMA%2c+SHAME%2c+AND+COMMUNICATION+ABOUT+MENTAL+HEALTH+AMONG+COLLEGE+STUDENTS%3a+IMPLICATIONS+FOR+COMMUNICATION+WITH+STUDENTS.&cli0=FT&clv0=Y&type=0&site=ehost-live&scope=site&custID=strayer&groupID=main&profID=ehost#_blank)

Colleges and universities report an increase in the number of students who seek out counseling for mental health issues, such as depression and anxiety. Although more college students are seeking treatment, there continues to be stigma related to mental health issues. The purpose of this exploratory study was to examine college students’ attitudes about mental health, stigma related to depression and anxiety, and communication about mental health. Two hundred ninety-two college students enrolled in a basic communication course at a large southern university completed a survey about their mental health beliefs, stigmas, and communication about mental health. Students reported more stigma for depression than anxiety. There were also significant differences in perceived personal and perceived public stigma about these issues, with students believing the general public is more likely to stigmatize mental health. Students also reported that as communication about mental health issues increased, their personal stigmas about the health issues decreased. Implications of these findings and recommendations for communication are also discussed. College life is fraught with uncertainty and stress as students negotiate new social and educational experiences. These experiences, ranging from dealing with roommates to adapting their learning styles to succeed in their college courses, may contribute to the increases in reports of depression and anxiety among college students. According to the 2015 National College Health Assessment, approximately 35 % of all college students reported depression or depression-like experiences, 58% reported overwhelming anxiety, and 10% seriously considered suicide (ACHA, 2016). Approximately 14.5% of students reported being diagnosed or treated for depression, 17% reported being diagnosed or treated for anxiety, and 11% reported being diagnosed and treated for both (ACHA, 2016). Depression and anxiety disorders can have a severe impact on students’ ability to complete their undergraduate education; 62 % of college students who dropped out attributed it to mental health issues (Gruttadaro & Crudo, 2012). Linked to the adolescent period during which many students begin college (Eisenberg, Gollust, Golberstein, & Hefner, 2007), these increased of college student mental health distress have become a serious concern for university staff and health providers. Interestingly, a paradox seems to exist: college counseling centers are reporting a rapid increase in students seeking treatment, with many centers reporting being booked in the first few weeks of school (Rector, 2013), but at the same time, there continues to be a stigma associated with depression and anxiety (Gruttadaro & Crudo, 2012). This study focuses on college students’ personal and perceived stigma associated with depression and anxiety and their communication about these issues. Specifically, this study examines the differences between depression stigmas, anxiety stigmas, and the shame associated with those stigmas, and the impact communication about depression and anxiety has on these stigmas. The article begins with a discussion of

stigma, attitudes about mental health, depression, and anxiety. After explaining the methodological approach, the results of the study are presented. Finally, the article concludes with a discussion of the implications and practical application of the findings, focusing on how student affairs professionals and faculty can use these findings to help reduce depression and anxiety stigma with communication.

Literature Review Communication about mental health, especially depression and anxiety, often focuses on the impact of stigma on how individuals talk about the issues (McNair, Highet, Hickie, & Davenport, 2002). Although there continues to be a concerted effort to reduce stigma associated with mental health issues, including working mental health issues into television shows (Fruth & Padderud, 1985; Hoffner & Cohen, 2012; Pirkis, Blood, Francis, & McCallum, 2006), TedTalks about depression and anxiety, and multiple university counseling center workshops (Eustis et al., 2016), stigma is still a primary concern. Stigma remains a major concern because of general attitudes about mental health issues (Kosyluk et al., 2016). An inconsistency exists between perceptions of mental health issues and communication about mental health. Part of this inconsistency stems from a lack of knowledge about mental health issues, including causes, symptoms, and treatment options (Busby Grant, Bruce, & Batterham, 2016; Jorm, 2000). For college students, the lack of knowledge is important because they may have difficultly differentiating between depression and just being “blue” and generalized anxiety disorders and typical anxiety associated with tests and homework. Although the general public is supportive of the use of mental health services for those that have a mental health issue (Leaf, Bruce, Tischler, & Holzer, 1987), whether those services actually get used can depend on the stigma individuals associated with mental health issues (Tucker et al., 2013). For these individuals, embarrassment and perceived negative reactions to seeking treatment continue to be barriers to help-seeking (Barney et al., 2006).

Stigma In his seminal work, Stigma: Notes on the Management of Spoiled Identity, Goffman (1963) discussed the social role of stigma in categorizing and discrediting certain individuals from being considered an unacceptable part of society. Stigma, or the “socialized, simplified, standardized image of the disgrace of a particular social group” (Smith, 2011, p. 455), is a socially constructed phenomenon which socialized societal members into recognizing traits, characteristics, or physical attributes which identify someone as different. As a communicative phenomenon, stigma relies primarily on identifying difference and using communication to devalue and segregate others, which reinforces what is considered (un)acceptable in society (Smith, 2007). Stigma is a multifaceted experience. There are three levels of stigma (or taint) individuals might experience: physical, social, and moral (Ashforth & Kreiner, 1999). Physical stigma is categorized by difference that can be readily identified, such as the loss of a limb or a physical deformity. Social stigma, also known as courtesy stigma (Page, 1984), is separation as a result of association with someone who is stigmatized. Finally, moral stigma occurs when the difference is regarded as sinful or is associated with dubious virtue, such as alcohol and drug. Mental health issues are unique in that they can involve all three types of stigma (Thoits, 2011). There are physical impacts of mental health (physical pain, panic attacks), poor mental health can be seen as a deficiency in character or lack of self-control, and some many find it difficult to associate or have relationships with people with mental health issues. There are three elements of stigma to consider when attempting to understand stigma perceptions: personal stigma, perceived stigma, and shame (Gilbert et al.,

2007; Griffiths, Batterham, Barney, & Parsons, 2011; Griffiths, Christensen, & Jorm, 2008). Personal stigma is concerned with an individual’s personal attitudes toward the stigmatized person (Griffiths et al., 2008). Personal stigma about mental health issues may include not considering the mental health issue a “real” medical disorder, individuals having control over their mental health issues, and associated negative personality or behavioral traits with the individuals. Interestingly, increased knowledge of mental health issues does not seem to impact or lower personal stigma. Wang and Lai (2008) found that individuals stigmatized people with depression as unpredictable and dangerous regardless of whether they had high levels of depression literacy; having family members with depression also did not influence people’s personal stigma of depression. Other factors which have an impact on personal stigma and depression include sex (men report higher levels) and education (those with less education report higher levels; Giffiths et al., 2008). Individuals who disclosed their depression to others reported lower levels of personal stigma (Griffiths et al., 2008). Individuals do report lower levels of personal stigma for anxiety disorders (Griffiths et al., 2011) than for depression. Women reported lower levels of personal stigma toward anxiety, as did individuals who had more exposure to or who had been diagnosed with an anxiety disorder (Batterham et al., 2013). Importantly, personal stigma is negatively related to help seeking; those with higher levels of personal stigma reported seeking out and using therapeutic support than those with lower levels of personal stigma (Eisenberg et al., 2009). Perceived stigma, also known as public stigma, focuses on the perceived beliefs about others’ negative attitudes (Griffiths et al., 2008). Perceived stigma about others’ negative attitudes can include a variety of different groups, including family, friends, the general community, and health providers. The important emphasis here, however, is on the fact that “most people”, or a “majority of society” believes a certain way about an issue. Age is a factor influencing perceived stigma, with younger populations reporting higher levels of perceived stigma (Vaughn-Sandler, Sherman, Aronsohn, & Volk, 2014); this is consistent with Griffiths and colleague (2008) who found that perceived stigma is lower in older populations. Individuals with higher levels of perceived stigma also reported higher levels of depression, lower levels of quality of life, had less social support, and were less likely to seek medical care (Momen, Strychacz, & Viirre, 2012; Vaughn-Sandler et al., 2014). Additionally, family members’ experiences with depression may also impact perceived stigma (Griffiths et al., 2008). Individuals who self-disclosed their depression also reported lower levels of perceived stigma (Griffiths et al., 2008). For anxiety, individuals who had higher exposure to anxiety disorders reported higher levels of perceived stigma (Batterham et al., 2013). H1: There is a difference in depression stigma between personal depression stigma and perceived depression stigma. H2: There is a difference in anxiety stigma between personal anxiety stigma and perceived anxiety stigma. The emotional response to stigma communication is crucial to understanding the entire stigma experience, especially how individuals respond to stigma (Meisenbach, 2010). For stigmatized individuals, a key emotional response is shame. Shame is a reaction to criticism by other people (Shultz, 2000). Shame focuses on others’ perceptions of the individual. Shame is unequivocally tied to embarrassment because attention is focused on the individual (Andersen & Guerrero, 1998) and is the product of being exposed, judged, and deemed different. Shame is a primary emotion associated with stigma because we often view criticism as reflection of self (Luoma & Platt, 2015). The focus is on the individual who has been shamed, in this case stigma

tized, and his or her emotional response is because he or she believes this is how the world sees them. Individuals may experience different types of shame, including external shame, internal shame, and reflected shame. External shame is shame focused on “the negative feelings associated with experiences that others are looking down on the self with desires to reject or harm the self” (Gilbert et al., 2007, p. 128). In other words, external shame focuses on the minds of others. Importantly, external shame is also associated with stigma awareness, especially in relation to attitudes about mental health (Gilbert, 1998; Gilbert et al., 2007). Internal shame focuses on the personal shame an individual experiences. Whereas external shame is concerned with stigma awareness, internal shame is concerned with stigma consciousness, a fear of being stigmatized because of certain traits (Gilbert, 1998). Shame occurs even at the thought of being stigmatized. Finally, a stigmatized individual can experience reflected shame. Reflected shame focuses on the reciprocal nature of stigma and shame. Reflected shame is concerned with the shame an individual can bring to others, especially family, close friends, and the community. Reflected shame assumes a level of stigma will be associated with those close to the stigmatized individual because of the stigma, so the individual feels shameful. For individuals with mental health conditions, there is an assumption that family members may be seen as weak or also having mental health issues and that members of the community may not want to associated with the individual (Gilbert et al., 2007). H3: There is a difference in external shame and internal shame for attitudes toward mental health. H4: There is a difference in reflected shame if a person suffers from mental illness versus reflected shame if a close relative suffers from a mental illness. One way to reduce the stigma and shame associated with mental health issues to focus on increasing positive communication about those mental health issues. Over the past decade, communication scholarship has seen a resurgence in research dedicated to the study of depression and anxiety among college students, especially in how these mental health issues impact disclosure (Scott, Caughlin, Donovan-Kicken, & Mikucki-Enyart, 2013), how they seek out, receive, and perceive social support (Pauley & Hess, 2009; Wright, King, & Rosenberg, 2014; Wright et al., 2013), the role of communication competence and negative messages (Lienemann, Siegel, & Crano, 2013; Wright et al., 2013) and the impact of technology in seeking treatment and social support (Joyce & Weibelzahl, 2011; Wright et al., 2013). What is missing from these examinations is the role of mental health stigma on how students communicate about these mental health issues. Communication about mental health issues can involve a number of different conversations, from openly speaking about a diagnosis in order to dispel myths to the communication between patients and counselors. In this study, communication is conceptualized as general talk about mental health issues with family, friends, and other important people in students’ lives. H5: Talking about anxiety and mental health issues is related to stigmas and attitudes towards mental health problems.

Method Participants and Procedure The participants were 292 students (61 males, 231 females, and 3 missing) enrolled in a basic communication course at a large southern university. The participants’ average age was 18.48; 272 freshmen, 13 sophomores, six juniors, and one senior participated in the study. Thirty-two participants reported being diagnosed with depression by a health provider (11%) and 43 students reported a generalized anxiety disorder diagnosis (15%). At the time of this study, 15 students were currently receiving psychological or counseling treatment (5%) and 28

were taking medication for anxiety or depression (9.5%). Ninety-four students had a close relative who had been diagnosed with depression (32%), 84 students had a family member who had been diagnosed with an anxiety disorder (29%), 124 students had a close friend with depression (42%), and 116 students had a close friend who had been diagnosed with an anxiety disorder (40%). After the researchers obtained university Institutional Review Board approval, the study was listed on the communication department’s research participation system. The research participation system is an online system that helps facilitate research studies and tracks student participation without tying students’ names to the actual study. Students see the study description listed on the site and voluntarily choose to participate in a study. The online survey was administered through Qualtrics web surveying software.

Measures Depression stigma was measured by Griffiths, Christensen, and Jorm’s (2008) Depression Stigma Scale. This 18-item scale has two subscales and responses were measured on a 5-point Likert scale with strongly disagree coded as a 1 to strongly agree coded as a 5. Responses were coded so that a high score represented more stigma. Respondents’ personal attitude towards depression was measured with the 9-item subscale of personal stigma with items such as “Depression is a sign of personal weakness.” Cronbach’s alpha for this subscale was .77. The other 9 items on this scale composed the perceived depression stigma subscale which assesses the respondent’s beliefs about the attitudes of others toward depression. Items such as “Most people believe that depression is not a real medical illness” composed this subscale. Cronbach’s alpha for this subscale was .83. General anxiety stigma was measured with Griffiths, Batterham, Barney, and Parson’s (2011) Generalised Anxiety Stigma Scale (GASS). This 20-item scale has two subscales and responses were measured on a 5-point Likert scale with strongly disagree coded as a 1 to strongly agree coded as a 5. Higher numbers represented more anxiety. The 10-item subscale of personal anxiety had items such as “People with an anxiety disorder should be ashamed of themselves.” Cronbach’s alpha for this subscale was .90. The perceived anxiety subscale had 10 items and had statements such as “Most people think that people with an anxiety disorder are to blame for their problem.” Cronbach’s alpha for this subscale was .91. Attitudes towards mental health were measured with Gilbert, Bhundia, Mitra, McEwan, Irons, and Sanghera’s (2007) Attitudes towards Mental Health Problems scale. This 35-item scale is divided into five subscales. Responses were adapted to a 5-point Likert scale with strongly disagree coded as a 1 to strongly agree coded as a 5 to maintain consistency throughout the questionnaire. General attitudes towards mental health was measured with an 8-item subscale with the word “community” changed to “friends” to better examine the student population perceptions. The subscale was comprised of items such as “My family see mental health problems as something to keep secret.” The subscale had a Cronbach’s alpha of .87. The external shame subscale was measured with 10 items with “community” again changed to “friends” in these items. Items such as “I think my friends (formerly community) would look down on me” were on this subscale which had a Cronbach’s alpha of .94. Internal shame was a 5-item subscale with Cronbach’s alpha of .90. Items such as “I would see myself as inferior” were on this subscale. Items such as “My family would be seen as inferior” were on the 7-item reflected shame 1 subscale, which had a Cronbach’s alpha of .90. Reflected shame 2 was the last subscale of the attitudes towards mental health scale which had a Cronbach’s alpha of .97 and was composed of five items with items such as “I would worry that others will look down on me” if a close relative had a mental health

problems. Talking about anxiety and depression was measured by six items developed for this study and included the following: How often do you talk with your friends about anxiety issues (in general)? How often do you talk with your family about anxiety issues (in general)? How often do you talk with health providers about anxiety issues (in general)? How often do you talk with your friends about depression issues (in general)? How often do you talk with your family about depression issues (in general)? How often do you talk with health providers about depression issues (in general)? Responses were never (coded as a 1), yearly (coded as a 2), monthly (coded as a 3), weekly (coded as a 4), 2-3 times a week (coded as a 5), and daily (coded as a 6). Responses were then summed so that a higher number represented more talking about anxiety and depression issues in general. Sex, age, and year in school were also assessed for demographic information. See Table 1 for means and standard deviations of all of the scales.

Results Hypothesis one examined whether there were significant differences among personal stigmas of depression (M = 2.13, SD = .54) and perceived stigmas of depression (M = 3.41, SD = .61). To test this hypothesis, a paired-sample t-test was conducted and was significant t(291) = -26.43, p<.0005. Hypothesis one was supported. To see if there were differences among personal stigmas of anxiety (M = 1.81, SD = .60) and perceived stigmas of anxiety (M = 3.08, SD = .73) as hypothesized in Hypothesis two, a paired-samples t-test was conducted and was significant t(291) = -22.36, p<.0005. Hypothesis two was supported. Hypothesis three suggested there would be differences in external and internal shame. To examine whether there was a difference between external shame (M = 1.70, SD = .60) and internal shame (M = 3.14, SD = .98) a paired sampled t-test as conducted and was significant t(291) = -25.54, p<.0005. Hypothesis three was supported. Finally, Hypothesis four posited a difference between whether reflected shame was significantly different if you suffered from mental illness (M = 2.45, SD = .90) or a family member suffered from a mental illness (M = 2.26, SD = 1.17) a paired-sample t-test was executed and was significant t(291) = 2.70, p=.007. Hypothesis four was supported. Hypothesis five examined whether talking about depression and anxiety issues was related to personal and perceived stigmas of depression and anxiety and attitudes towards mental health problems. Using

Pearson’s correlation coefficients there were no significant correlations among talking about depression and anxiety and attitudes toward mental health problems. However, there were significant negative correlations between personal depression stigmas and communication r(237) = -.31, p<.0005 and between personal anxiety stigmas and communication r(237) = -.29, p<.0005. Thus, as talk increased, personal stigmas went down. Hypothesis five was partially supported.

Discussion With 92% of college and university counseling centers reporting an increase in students utilizing their services for treatment of depression and anxiety (Rector, 2013), the need to study issues of stigma and communication connected to student perceptions of mental health is paramount. This study sought to examine the differences that might exist between stigmas and mental health shame and how communication about depression and anxiety might impact stigma and shame. Most of the hypotheses were supported. Students reported significantly higher levels of personal stigma than perceived stigma, which is consistent with previous research (e.g., Griffiths et al., 2008; Kosyluk et al., 2016; Vogel, Shechtman, & Wade, 2010). Students conceptualized shame differently based on whether they or a family member had a mental health issue. Additionally, communication had a positive impact on reducing personal depression and anxiety stigmas. One of the most important findings was that students reported higher perceived personal stigma for depression than for anxiety. Why would students report more personal stigma for depression than anxiety? For students, it is possible this difference might stem from the perceived educational situationality of anxiety. Students constantly encounter someone proclaiming test anxiety or public speaking anxiety, and might do so themselves. Anxiety is communicatively framed as “necessary routine evils” in college life, not a legitimate mental health issue. In contrast, depression is clearly talked about as a mood disorder. Everyday communication even supports these differences. Students might declare themselves anxious about school or personal events, but when they feel sad or tired, might not classify themselves as feeling “depressed” because they understand the seriousness of depression. Anxiety, then, becomes a temporary diagnosis (only on test day or before an exam). These classifications further enhance the view that depression is a legitimate disorder, whereas anxiety is viewed as a temporary state. Therefore, being diagnosed with depression incurs more personal

stigma than anxiety. Importantly, the findings of this study suggest that communicating with others about depression and anxiety lowers college students’ perceived personal stigma about those mental health issues. Although communicating about mental health did not reduce the shame students reported, communicating with others, be it friends, family, or a mental health counselor, helps students to feel more comfortable and accepting of depression and anxiety. It is important to note, however, that quantity of communication is not the only important factor. Quality of communication is also important. Barney and colleagues (2006) found that when individuals believed people would respond negatively to communicating about mental health, they were less likely to seek help from mental health counselors. Talk about depression and anxiety needs to be positive in order to encourage students to seek help. Increasing positive communication about mental health could be the first step in helping students feel less shame about having depression and anxiety and seeking treatment. The findings from this study point to practical applications and suggestions for faculty and student affairs professionals working with college students. Faculty and student affairs professionals are in unique positions of having multiple roles in which they interact with students. They teach, educate, advise, supervise, and assist students, sometimes taking on multiple role interactions. These close interactions with students mean that student affairs professionals and faculty may be among the first to notice changes or problems students may be experiencing, be it acting up in class, slipping grades, or changes in social interactions. Encouraging students to seek assistance may help to activate their help seeking attitudes As the data showed, students experience more internal, personal shame about mental health issues, especially if they suffered from a mental health issue. It is important to address the emotional needs of students, especially when students find themselves associating shame with depression or anxiety diagnoses. Only after assuring the student that how they are feeling (perceived stigma) is acceptable or normal is it possible to provide information or solution-focused communication. When addressing the stigma, staff and faculty should reassure the student that stigma is much less prevalent, that mental health issues are common, normal, and it is good to seek professional assistance. However, it is also important for staff and faculty to know their limits. Especially when staff and faculty members are not trained in mental health counseling, assistance should focus on helping to reduce stigma by talking about the how mental health issues are common for college students and encouraging treatment seeking behaviors. Universities can create campus campaigns to communicate the commonality of depression and anxiety in college. These campaigns could be used to reduce the disparity in personal stigma about depression and anxiety. The more students are exposed to personal stigma-reducing messages, the more personal stigma should be reduced (Busby Grant, Bruce, & Batterham, 2016), which in turn should lead to the increased use of appropriate communication strategies to talk about mental health issues. Campaigns might also focus not only on long-term or severe cases of depression and anxiety but could also address those factors that increase the chances of either illness manifesting. Campaigns and other university interventions can be extremely helpful because they can show students how to appropriately communicate about difficult topics, like depression and anxiety, with family and friends (Browning, Meyer, Truog, & Solomon, 2007). By providing more campaigns and interventions, mental health concerns can become normalized (Eustis et al., 2016), reducing stigma and increasing positive communicative behaviors.

Limitations and Future Directions One obvious limitation of the study was the age of participants and the fact that all of the participants were from one university. In general, most of the participants in this study were first-year students and female. However, the participants were from a variety of disciplines because the fundamental human communication course is required of all students at this university. The age range of participants was also typical for a first-year general education course. Additionally, there were few non-traditional and upper division students who participated in the study. How non-traditional students talk about mental health and their perceptions of stigma may be very different than traditional college-aged students, especially when it comes to depression and anxiety which manifest because of college stressors. Additionally, students who have been in college for several years may respond differently in how they communicate about mental health. Future research needs to examine the different communication strategies that would help decrease stigma and shame as well as what influencers (peers, parents, university staff, etc.) are the more effective groups for those communication strategies. Future studies should also examine a larger population and more diverse age groups from different areas of the country to determine if mental health stigmas and attitudes vary in different parts of the country. A more cross-sectional population of college students (freshmen, sophomores, juniors, and seniors) may provide a more complete picture of college students’ perceptions and stigmas of mental health. Moreover, a more focused analysis of specific college-aged groups could provide insight into how to help different groups. Students who stay longer than the traditional four years (“super” seniors) may encounter depression and anxiety for unique reasons associated with an extended college stay. Likewise, the study of graduate students would also be an interesting group on which to focus. The communication experiences of these more seasoned groups could contribute to what we know about how college students communicate about mental health.

1. **Article 4.** Expectations of the university to post-graduation transition of students with mental health conditions. By: Lucas, Rebecca; Cage, Eilidh; James, Alana I.. Psychology Teaching Review , 2018, Vol. 24 Issue 1, p79-81, 3p, Database: [Education Source](http://libdatab.strayer.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=eue&bquery=Expectations+%26quot%3bof%26quot%3b+the+university+%26quot%3bto%26quot%3b+post-graduation+transition+%26quot%3bof%26quot%3b+students+with+mental+health+conditions.&cli0=FT&clv0=Y&type=0&site=ehost-live&scope=site&custID=strayer&groupID=main&profID=ehost#_blank)

Expectations of the university to post-graduation transition of students with mental health conditions Rebecca Lucas, Eilidh Cage & Alana I. James

The number and percentage of students with mental health conditions (MHC) attending university is increasing. Students with MHC can be well supported during their time at university, but receive less support with the transition from university to post-graduation. This time is characterised by great change and can be accompanied by mixed feelings. This paper presents preliminary data on the transition expectations and perceptions of 35 students with MHC. High levels of fear and low levels of preparedness regarding the transition were reported. Only 55–60 per cent of students had accessed transition-related or careers-related support, but those who had found it productive. Students suggested that more condition-specific support, and support in the form of workshops, lectures and one-to-one meetings would be beneficial. This knowledge can be used to better support students with MHC with the university to post-graduation transition. MENTAL HEALTH CONDITIONS (MHC) affect mood, thinking and behaviour. In the 2015–2016 academic year, over 10,000 first year UK domiciled undergraduate students declared a clinically diagnosed MHC (HESA, 2017), although the actual number of students meeting diagnostic criteria for a MHC is likely to be much higher (Eisenberg et al., 2007). Students with MHC can access support for their time at university, such as specialist mentoring to enhance academic skills, social relationships and wellbeing (Lucas & James, 2018). However, specialist mentoring does not typically focus on preparing students for the university to post-graduation transition. The transition not only involves leaving university but also moving into employment or further study. Accordingly, the transition is characterised by departure from routine, loss of existing support networks and loss of a ‘safe-space’, as well as adaption to a new environment, with a new role and new people. Thus the transition may be especially challenging for students with MHC. However, there is a paucity of empirical literature on the university to post-graduation transition for students with MHC. This is of great concern given that six months post-graduation, graduates with MHC are twice as likely as their peers without a disability to be unemployed and 10 per cent less likely than their peers without a disability to be in full time employment (Association of Graduate Careers Advisory Services, 2016). We are therefore undertaking a study examining the university to post-graduation transition for students with MHC. This paper reports preliminary findings from the first wave of participants in Phase One, which focuses upon students’ expectations and preparation for this transition. Method Participants Participants were 35 (29 female) final year undergraduate students from two universities who had declared to their university a diagnosis of a mental health condition. The mean age of the participants was 22.24 (SD = 3.10). Whilst 31.43 per cent of students reported having just one MHC, 48.57 per cent reported having two mental health conditions, most commonly anxiety and depression, and 20.00 per cent reported having anxiety, depression plus another mental health condition. Participants’ current level of psychological wellbeing was assessed via the Warwick-Edinburgh Mental Well-being Scale (Tennant et al., 2007). The mean score was 37.60 (SD = 12.24), which is >1SD below norms seen on population surveys (Braunholtz et al., 2007), suggesting low wellbeing.

Measures Participants’ expectations of the transition from university to post-graduation were assessed via an online questionnaire, lasting around 20 minutes. Participants rated eight emotions (based on Plutchik’s Wheel of Emotions) regarding the transition out of university, and how well supported they felt by the university with the transition and with preparing for their career, on a five point Likert scale ranging from one (strongly disagree) to five (strongly agree). They were also asked whether they had accessed support related to this transition or preparing for their career, and if there was any additional support for these issues the university should consider providing for students with mental

health conditions. The study followed the BPS ethical guidelines, and received ethical approval from both universities. Participants provided informed written consent and received a £5 Amazon voucher.

Results Current feelings The emotion felt most strongly about the transition out of university was fear, with students also feeling low levels of preparedness (see Table 1).

Transition support Overall, participants were neutral regarding how well supported they felt with the transition out of university, with a mean score of 2.62 (SD = 1.01). Sixty per cent had accessed emotional support related to the transition from either a disability advisor, specialist mentor, welfare officer or personal tutor and this support was considered helpful (see Table 2).

Career support Participants were also neutral regarding how well supported they felt in terms of preparing for their future career, with a mean score of 2.68 (SD = 1.25). Half (55.89 per cent) had accessed career-related support from either a disability advisor, specialist mentor, welfare officer, personal tutor or the careers service, and rated these sources of support as helpful (see Table 2).

Additional support Content analysis of students’ responses determined that seven students suggested specific support for students with MHC (e.g. workshops, lectures) would be beneficial. Six students suggested providing more of, and greater promotion of, existing resources, and four suggested regular and compulsory meetings regarding the transition/careers, such as with personal tutors or the careers service.

Discussion The undergraduate students with MHC who completed our study reported high levels of fear and low levels of preparedness regarding the transition. Just over half the students had accessed transition-related support and career-related support, however, those who did access support found it helpful. Whilst specialist professional service staff may be best placed to deliver certain support, our preliminary data show that more students had contacted their personal tutor. This emphasises the importance of university departments’ involvement in helping prepare students for the transition. Students suggested that increasing pre-existing support, as well as providing specialist support for students with MHC, would be beneficial. In the full project we will examine in more depth the expectations and preparations of a wider range of students. In addition, we will examine the perceptions of professional support staff. Phase Two will examine why all students are not accessing available support and how any barriers to accessing support could be addressed. By increasing our understanding of the university to post-graduation transition for students with MHC, we can better support these students, and ultimately make the experience more positive and successful.

1. **Article 5.** Academic procrastination in college students: The role of self-reported executive function.”  Journal of Clinical and Experimental Neuropsychology. By: Rabin, Laura A.; Fogel, Joshua; Nutter-Upham, Katherine E. ***Journal*** of ***Clinical*** & ***Experimental*** ***Neuropsychology***. Mar2011, Vol. 33 Issue 3, p344-357. 14p. 4 Charts. DOI: 10.1080/13803395.2010.518597. , Database: [Academic Search Complete](http://libdatab.strayer.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=a9h&bquery=XY+%26quot%3bprocrastination%26quot%3b%5b100%5d+AND+(XY+%26quot%3bneuropsychology%26quot%3b%5b85%5d+OR+XY+%26quot%3bexecutive%26quot%3b%5b58%5d+OR+XY+%26quot%3bclinical%26quot%3b%5b49%5d+OR+XY+%26quot%3bfunction%26quot%3b%5b46%5d+OR+XY+%26quot%3bexperimental%26quot%3b%5b44%5d+OR+XY+%26quot%3breported%26quot%3b%5b39%5d+OR+XY+%26quot%3bself%26quot%3b%5b29%5d+OR+XY+%26quot%3bacademic%26quot%3b%5b26%5d+OR+XY+%26quot%3brole%26quot%3b%5b24%5d+OR+XY+%26quot%3bcollege%26quot%3b%5b23%5d+OR+XY+%26quot%3bjournal%26quot%3b%5b16%5d+OR+XY+%26quot%3bstudents%26quot%3b%5b16%5d)&cli0=FT&clv0=Y&type=0&site=ehost-live&scope=site&custID=strayer&groupID=main&profID=ehost#_blank)

Academic procrastination in college students: The role of self-reported executive function

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Procrastination, or the intentional delay of due tasks, is a widespread phenomenon in college settings. Because procrastination can negatively impact learning, achievement, academic self-efﬁcacy, and quality of life, research has sought to understand the factors that produce and maintain this troublesome behavior. Procrastination is increasingly viewed as involving failures in self-regulation and volition, processes commonly regarded as executive functions. The present study was the ﬁrst to investigate subcomponents of self-reported executive functioning associated with academic procrastination in a demographically diverse sample of college students aged 30 years and below (n = 212). We included each of nine aspects of executive functioning in multiple regression models that also included various demographic and medical/psychiatric characteristics, estimated IQ, depression, anxiety, neuroticism, and conscientiousness. The executive function domains of initiation, plan/organize, inhibit, self– monitor, working memory, task monitor, and organization of materials were signiﬁcant predictors of academic procrastination in addition to increased age and lower conscientiousness. Results enhance understanding of the neuropsychological correlates of procrastination and may lead to practical suggestions or interventions to reduce its harmful effects on students’ academic performance and well-being.

INTRODUCTION

Academicprocrastination—theintentionaldelayinthe beginning or completion of important and timely academic activities (Schouwenburg, 2004; Ziesat, Rosenthal, & White, 1978)—is a widespread phenomenon in college settings. Approximately 30% to 60% of undergraduate students report regular postponement of educational tasks including studying for exams, writing term papers, and reading weekly assignments, to the point at which optimal performance becomes highly unlikely (Ellis & Knaus, 1977; Janssen & Carton, 1999; Kachgal, Hansen, & Nutter, 2001; Onwuegbuzie, 2004; Pychyl, Lee, Thibodeau, & Blunt, 2000a; Pychyl, Morin, & Salmon, 2000b; Solomon & Rothblum, 1984). While occasional delays are acceptable and may even be advantageous, what distinguishes problematic or habitual procrastination from merely deciding to perform an activity atsomelatertimeistheaccompanyinginternalsubjective discomfort (Lay & Schouwenburg, 1993). This discomfort may manifest as anxiety, irritation, regret, despair, or self-blame (Burka & Yuen, 1983; Pychyl et al., 2000a; Rothblum,Solomon,&Murakami,1986).Therearealso external consequences to chronic academic procrastination such as compromised performance and progress, decreased learning, lost opportunities, increased health risks, and strained relationships (Beswick, Rothblum, & Mann, 1988; Burka & Yuen; Burns, Dittman, Nguyen, & Mitchelson,2000;Moon&Illingworth,2005a;Rothblum et al., 1986; Tice & Baumeister, 1997). Due to these signiﬁcant negative aspects, researchers have studied procrastination and have proposed various cognitive, emotional, and personality variables as possible predictors. Frequently cited cognitive correlates include a tendency toward self-handicapping, low selfesteem, low academic self-efﬁcacy, fear of failure, and distorted perceptions of available and required time to complete tasks (Ferrari, Parker, & Ware, 1992; Ferrari & Tice, 2000; Judge & Bono, 2001; Kachgal et al., 2001; Lay, 1988, 2004; Pychyl, Coplan, & Reid, 2002). With regard to emotional functioning, several studies have found that anxiety, depression, and worry are associated with procrastination (Antony, Purdon, Huta, & Swinson, 1998; Ferrari, Johnson, & McCown, 1995; Stoeber & Joormann, 2001; van Eerde, 2003),buttheempiricalevidenceconcerningtherelationship between mood and procrastination is not deﬁnitive (Steel, 2007). In terms of personality features, research has consistently shown that lower conscientiousness and, to a lesser extent, higher neuroticism are related to trait procrastination (Johnson & Bloom, 1995; Lee, Kelly, & Edwards, 2006; Milgram & Tenne, 2000; Schouwenburg & Lay, 1995; van Eerde, 2003). Various demographic and medical/psychiatric variables have also been examined in relation to procrastination. Research is inconclusive with regard to whether academic procrastination is related to students’ gender (Steel, 2007; van Eerde, 2003). Although several studies have reported higher levels in males (Milgram, Marshevsky, & Sadeh, 1994; Ozer, Demir, & Ferrari, 2009;Senécal,Koestner,&Vallerand,1995),manyothers have reported no such gender differences (Ferrari, 2001; Ferrari et al., 1992; Haycock, McCarthy, & Skay, 1998; Hess,Sherman,&Goodman,2000;Kachgaletal.,2001). Research is similarly inconclusive regarding age, with some studies reporting negative correlations (Beswick et al., 1988; Prohaska, Morrill, Atiles, & Perez, 2000; van Eerde, 2003) and others reporting no meaningful correlations (Haycock et al., 1998; Howell, Watson, Powell, & Buro, 2006) between age and procrastination. Ethnicity (Clark & Hill, 1994; Kachgal et al., 2001; Prohaska et al., 2000) and level of intelligence (Ferrari, 1991a; van Eerde, 2003) do not seem to be related to procrastination, though there is little extant research on the relation between these variables. Students with drug and alcohol problems(Jamrozinski,Kuda,&Mangholz,2009),learning disabilities (Klassen, Krawchuk, Lynch, & Rajani, 2008a), and attentional problems (Safren, 2006; Steel, 2007; Weiss & Murray, 2003) have also been reported to exhibit heightened levels of procrastination. Procrastination is increasingly recognized as involving a failure in self-regulation such that procrastinators, relative to nonprocrastinators, may have a reduced ability to resist social temptations, pleasurable activities, and immediate rewards when the beneﬁts of academic preparation are distant (Ariely & Wertenbroch, 2002; Chu & Choi, 2005; Dewitte & Schouwenburg, 2002; Ferrari, 2001; Schouwenburg & Groenewoud, 2001; Tan et al., 2008; Van Eerde, 2000; Wolters, 2003). These individuals also fail to make efﬁcient use of internal and external cues to determine when to initiate, maintain, and terminate goal-directed actions (Senécal et al., 1995). Associated characteristics include reduced agency, disorganization, poor impulse and emotional control, poor planning and goal setting, reduced use of metacognitive skills to monitor and control learning behavior, distractibility, poor task persistence, time and task

management deﬁciencies, and an intention–action gap (Dewitte & Lens, 2000; Dewitte & Schouwenburg, 2002; Ferrari & Emmons, 1995; Shanahan & Pychyl, 2007; Steel, 2007; Steel, Brothen,& Wambach, 2001; Tan et al., 2008; Wolters, 2003). Research implicates frontal brain systems in selfregulatory and related processes, and this is generally referred to as executive functioning (Roth, Randolph, Koven,&Isquith,2006;Stuss&Benson,1986).Executive functions comprise various neurocognitive processes that enable novel problem solving, modiﬁcation of behavior in response to new information, planning and generating strategies for complex actions, and the self-regulation of cognition, behavior, and emotion (Roth et al., 2006; Williams, Suchy, & Rau, 2009). Given the role of executive functioning in the initiation and completion of complex behaviors, it is surprising that little research examines the relationship between executive functioning and academic procrastination. Extant research is limited and/or indirect. For example, Schouwenburg (2004) found that procrastination was inversely correlated with adoption of a systematic and disciplined approachto one’swork and withplanning and managing of one’s time, suggestive of poor organization. Wolters (2003) showed that procrastination correlated with students’ self-efﬁcacy and self-regulated learning strategies. HowellandWatson(2007)foundthatlowercognitiveand metacognitive strategy usage and disorganization predicted procrastination in a sample of college students. Strub(1989)describedthecaseofa60-year-oldmanwho developed chronic procrastination following a cerebral hemorrhagethatresultedinfrontallobesyndrome.Thus, some evidence of associations between frontal system network dysfunction and procrastination has emerged, though it remains unclear which speciﬁc aspects of executive functioning are most implicated in delay behaviors. Toourknowledge,theonlydirectinvestigationofexecutive dysfunction as a source of procrastination is an unpublished doctoral thesis, which did not ﬁnd a signiﬁcant relationship between neuropsychological tests of executive functioning and severe academic procrastination (Stone, 1999). To address this gap in the literature and to capture more everyday aspects of executive functioning,thecurrentstudyinvestigatedtheextenttowhich procrastination was predicted by self-reported executive functioning in a sample of college undergraduates. We carriedoutseparateanalysesforeachofnineclinicalsubscales of a self-report measure of executive functioning, the Behavior Rating Inventory of Executive Function– Adult Version (BRIEF-A; Roth, Isquith, & Gioia, 2005). In our ﬁrst model, we included each of the nine BRIEFA subscales plus demographic and medical/psychiatric variables. In a second model, we included the ﬁrst set of variables plus estimated IQ and relevant personality and mood variables. We hypothesized that BRIEF-A subscales tapping inhibitory control/impulsivity, selfmonitoring, planning and organization skills, and task initiation would be signiﬁcant predictors of academic procrastination. Conscientiousness, neuroticism, and mood symptoms were also hypothesized to be signiﬁcant predictors of academic procrastination. We did not generate speciﬁc hypotheses concerning the relation between procrastination and gender, age, ethnicity, alcohol or drug use, or various medical/psychiatric conditions, given the equivocal nature of ﬁndings or paucity of research on these variables.

METHOD

Participants and procedure

Data were collected from February 2007 through February 2009. Participants (n = 212) were drawn from various undergraduate psychology courses at a four-year public college that is part of a large, urban university system. Students were offered partial class credit or the chance to win a $50 gift certiﬁcate as compensation for participation. Students were informed that the study entailed completing a series of paper-and-pencil questionnaires that would take approximately 30–40 min on the general topic of academic motivation. Participation was voluntary and conﬁdential, and informed consent was obtained according to an institutional review board (IRB)-approved protocol. Our sample was obtained from a larger sample of 243 individuals. Data were collected in a psychology laboratory and in classrooms. Due to signiﬁcant missing data, three questionnaires were excluded from statistical analysis. We also excluded individuals with invalid BRIEF-A protocols (n=2) and those who were more than 30 years of age (n = 26) to allow for a similar young adult age proﬁle, resulting in the ﬁnal sample of 212 individuals.

Measures and scoring

Participants ﬁrst completed a demographic questionnaire, which asked them to report their age (in years), sex (male or female), and race (African American, Asian, Caucasian, Hispanic/Latino, Native American, or “Other”). Participants also reported any medical or psychiatric conditions (scored dichotomously) including: diagnosis of learning disability, diagnosis of attention-deﬁcit/hyperactivity disorder (ADHD), current use of alcohol, diagnosis of psychiatric/neurological illness(es), diagnosis of chronic/major medical problem(s), and current illicit drug use. Participants then completed the following study measures in the order in which they are listed: (a) Lay General Procrastination (GP) Scale, Student Version (Lay, 1986); (b) Beck Depression Inventory–II (BDI–II, Beck, Steer, & Brown, 1996); (c) Beck Anxiety Inventory (BAI; Beck & Steer, 1993), Shipley Institute of Living Scale (Shipley, 1991), BRIEF-A, and NEO Five Factor Inventory (NEO-FFI; Costa & McRae, 1992). The Lay GP is a 20-item measure of trait procrastination that examines behavioral tendencies to delay the start or completion of everyday tasks. Participants rate various statements on a 5-point Likert scale

(1=extremely uncharacteristic; 5 = extremely characteristic). Sample items include: “I often ﬁnd myself performing tasks that I had intended to do days before” and “I usually start an assignment shortly after it is assigned.” Ten items are reverse-keyed, and scores range from20to100withahighertotalscoreindicatinggreater procrastination. The Lay GP is considered unidimensional, and it has good validity and reliability in a variety of contexts (Diaz-Morales, Ferrari, Diaz, & Argumedo, 2006; Ferrari, 1989; 1991b; Lay, 1988; Lay & Burns, 1991). The BRIEF-A is a self-report measure of executive functions or self-regulation in the everyday environment, which includes nine nonoverlapping theoretically and empirically derived clinical scales. Participants rate the frequency of 75 problematic behaviors over the past month on a 3-point scale (1 = never; 2 = sometimes; 3 = often), and higher scores indicate greater degrees of executive dysfunction. Mean raw scores and standard T scores can be calculated for each of the clinical scales, and there are also three validity scales (Negativity, Inconsistency, and Infrequency); as mentioned above, we excluded participants with elevated scores on one or more of the validity scales (deﬁned as a T score of 65 or greater). The BRIEF-A has demonstrated reliability, validity, and clinical utility as an ecologically sensitive measure of executive functioning in healthy individuals and also those presenting with a range of psychiatric and neurological conditions (Roth et al., 2005). The BRIEF-A Inhibit scale contains 8 items that measure behavioral regulation or the ability to not act on an impulse (e.g., “I have problems waiting my turn”). The Self-Monitor scale contains 6 items that measure the extent to which a person keeps track of his/her behavior and its impact on others (e.g., “When people seem upset with me, I don’t understand why”; “I say things without thinking”). The Plan/Organize scale contains 10 itemsthatassesstheabilitytomanagecurrentandfutureoriented task demands within their situational contexts (e.g., “I don’t plan ahead for tasks”; “I have trouble organizing work”), The Shift scale contains 6 items that measure the ability to shift behaviorally or cognitively from one situation, activity, or aspect of a problem to another, as the circumstances demand (e.g., “I have trouble thinking of a different way to solve a problem when stuck”). The Initiate scale contains 8 items related to the ability to begin a task and to independently generate ideas, responses, or problem-solving strategies (e.g., “I start things at the last minute such as assignments, chores, tasks”). The Task Monitor scale contains 6 items that measure the extent to which an individual keeps track of his/her problem-solving success or failure (e.g., “I misjudge how difﬁcult or easy tasks will be”). The Emotional Control scale contains 10 items related to a person’s ability to modulate emotional responses (e.g., “I overreact to small problems”; “I get emotionally upset easily”). The Working Memory scale contains 10 items that tap the capacity to hold information in mind for the purpose of generating a response or completing a task (e.g., “I have trouble with jobs or tasks that have more than one step”). The Organization of Materials scale has 8 items that assess orderliness in one’s everyday environment and the ability to keep track of everyday objects, including homework (e.g., “I have trouble ﬁnding things in my room, closet, or desk”). The NEO-FFI is a used to measure the “Big Five” domains of adult personality (openness, conscientiousness, extraversion, agreeableness, and neuroticism). The 60 items include questions about typical behaviors or reactions. Participants rate themselves using a 5point Likert scale (1 = strongly disagree; 5 = strongly agree). The current study analyzed participants’ scores on the neuroticism and conscientiousness subscales because of their known association with procrastination. Neuroticism refers to a person’s stress reactivity or emotional responsiveness to challenge and proclivity for negative mood states such as anxiety or worry. Conscientiousness denotes the extent to which a person istaskoriented,achievementstriving,deliberate,dependable, careful, and organized, and possesses self-control. We utilized NEO-FFI raw scores (range 0 to 48 per scale), which can be used to derive percentiles from a college-age normative data sample. Previous research has demonstrated the reliability and validity of this measure (Caruso, 2000; Costa & McCrae, 1992; Holden, 1992) and its association with a variety of psychological and health variables (John & Srivastava, 1999; Matthews, Deary, & Whiteman, 2003). The Shipley Institute of Living Scale (Shipley) is a test of general intellectual functioning for adults and adolescents, which contains two subscales—Vocabulary and Abstraction. The 40-item Vocabulary section requires individuals to select the word closest in meaning to a target word from among four alternatives. The 20-item Abstraction section requires individuals to complete a series of numbers, letters, or words with the next item that should follow in the sequence. A total score is calculated, which is used to derive a Full Scale IQ estimate. The Shipley has shown strong psychometric properties in both healthy and clinical populations (Nixon, Parsons, Schaeffer, & Hale, 1995; Phay, 1990; Smith & McCrady, 1991; Zachary, Crumpton, & Spiegel, 1985). The Beck Depression Inventory–II (BDI–II; Beck et al., 1996) and Beck Anxiety Inventory (BAI; Beck & Steer, 1993) are among the most widely used selfadministered measures of emotional functioning, and there is strong support for their reliability and validity with young adults (Arnou, Meagher, Norris, & Branson, 2001; Carmody, 2005; Osman, Kopper, Barrios, Osman, & Wade, 1997). The BDI–II consists of 21 items that assess the intensity of depression experienced in the past two weeks. Each item contains a list of four statements arranged in increasing severity about a particular symptom; total scores range from 0 to 63, with higher scores indicating stronger severity of depressive symptoms. The BAI evaluates both physiological and cognitive symptoms of anxiety and consists of 21 self-administered items, each describing a common symptom. The BAI is rated on a scale of 0 to 3 (0= not at all bothered; 3 = I could barely stand it), indicating the degree to

which the individual has been bothered by each symptom during the past week. The BAI has been found to reliably discriminate anxiety from depression while displaying convergent validity (Beck & Steer). Item scores are summed to obtain a total score that can range from 0 to 63, with higher scores indicating higher levels of anxiety.

Statistical analyses

Descriptive statistics were calculated for all variables. For the multivariate analyses, the continuous variable of academic procrastination was used as the outcome variable. Linearregressionanalysiswasusedtodeterminethevariables associated with the outcome of academic procrastination. Two models were conducted for each of the nine BRIEF-A executive functioning clinical scales. Model 1 consisted of the independent variables of demographic variables (i.e., age, sex, race/ethnicity dichotomized as Caucasian versus minority), presence of a number of medical and psychiatric diseases or conditions (i.e., diagnosis of learning disability, diagnosis of ADHD, current use of alcohol, diagnosis of psychiatric/neurological illness(es), diagnosis of chronic/major medical problem(s), and current illicit drug use), and the particular executive functioning category. Model 2 consisted of the independent variables of those in Model 1 plus estimated IQ, depressive symptoms, anxiety symptoms, NEO-Neuroticism, and NEO-Conscientiousness. SPSS Version 17 was used for all analyses. Analyses in Model 2 had 24 fewer participants because of missing data on the BDI–II.

RESULTS

Table 1 shows the descriptive statistics. The sample had anaverageageofmorethan21years,andmorethanthree quarters were women. In terms of ethnic/racial composition, 60% identiﬁed as Caucasian, 14% Asian, 13% African American, 5% Hispanic, and 8% as “Other.” For the purposes of the statistical analyses, race was treated as a dichotomous variable (i.e., Caucasian/minority) in order to prevent possible statistical over adjustment. Almost half the sample reported drinking alcohol, with an average of 2.7 drinks per week (SD = 2.9) among those who drank. The other ﬁve conditions/diseases all were less than 5%. Though not shown in Table 1, the psychiatric/neurological illnesses included borderline personality disorder, obsessive compulsive disorder, generalized anxiety disorder, various eating disorders, depression, epilepsy, and traumatic brain injury. The reported medical conditions included asthma, hyperthyroidism, diabetes, cardiovascular disease, and polycystic ovary disease. Average procrastination scores were within the neutral range, while scores ranged from minimal to severe. Average BRIEF-A scores were within the normal range, while approximately 12.5% of scores were within the clinically elevated range (deﬁned as a T score of 65 or greater). Average NEO Neuroticism and Conscientiousness scores were within the average to high range, with scores that ranged from low to high. Average anxiety and depressive symptoms were within the minimal to mild range, with scores that ranged from minimal to severe. Table 2 shows the linear regression analyses for the executive functioning categories of Inhibit, Shift, and Emotional Control. For Inhibit, Model 1 shows significance for both increasing age and Inhibit with increasing procrastination. Model 2 shows similar signiﬁcant resultsforageandInhibit.Also,NEO-Conscientiousness approachedsigniﬁcance(p=.054)withdecreasingscores associated with increasing procrastination. For Shift, Model 1 shows signiﬁcance for both increasing age and Shift with increasing procrastination, and increasing BDI–IIhadap-valueof.098forassociationwithincreasing procrastination. Model 2 shows signiﬁcance for increasing age and decreasing NEO-Conscientiousness scores associated with increasing procrastination while Shift was no longer signiﬁcantly associated with procrastination. For Emotional Control, Model 1 shows signiﬁcance for both increasing age and Emotional Control with increasing procrastination. Model 2 shows signiﬁcance for increasing age and decreasing NEOConscientiousness scores associated with increasing procrastination while Emotional Control was no longer signiﬁcantly associated with procrastination. Table 3 shows the linear regression analyses for the executivefunctioningcategoriesofSelf-Monitor,Initiate, and Working Memory. For Self-Monitor, Model 1 shows signiﬁcance for both increasing age and Self-Monitor with increasing procrastination. Model 2 shows similar signiﬁcant results for age and Self-Monitor. Also, decreasing NEO-Conscientiousness scores were significantly associated with increasing procrastination, and increasing BDI–II had a p-value of .098 for association with increasing procrastination. For Initiate, Model 1 shows signiﬁcance for Initiate with increasing procrastination. Model 2 shows similar signiﬁcant results for Initiate. Also, increasing age approached signiﬁcance (p = .061) with increasing procrastination. For Working Memory, Model 1 shows signiﬁcance for both increasingageandWorkingMemorywithincreasingprocrastination. Model 2 shows similar signiﬁcant results for age and Working Memory. Also, decreasing NEOConscientiousness scores were signiﬁcantly associated with increasing procrastination. Table 4 shows the linear regression analyses for the executive functioning categories of Plan/Organize, Task Monitor, and Organization of Materials. For Plan/Organize, Model 1 shows signiﬁcance for Plan/Organize with increasing procrastination. Model 2 shows similar signiﬁcant results for Plan/Organize. Also, those without learning disabilities approached signiﬁcance (p=.050) for increasing procrastination. For Task Monitor, Model 1 shows signiﬁcance for Task Monitor with increasing procrastination. Also, increasing age approached signiﬁcance (p = .055) with increasing procrastination. Model 2 shows similar signiﬁcant results for Task Monitor, age now was also signiﬁcantly associated, and decreasing NEO-Conscientiousness scores approached signiﬁcance (p=.088) with increasing procrastination. For Organization of Materials, Model 1 shows signiﬁcance for Organization of Materials with increasing procrastination. Also, increasing age had a p-value of .09 for association with increasing procrastination. Model 2 shows similar signiﬁcant results for Organization of Materials, increasing age approached signiﬁcance (p = .057) with increasing procrastination, and increasing BDI–II had a p-value of .089 for association with increasing procrastination.

DISCUSSION

A substantial body of research reveals the prevalence of academic procrastination as a self-perceived problem for college students, with consequences ranging from reduced academic achievement and progress to increased stress and poor quality of life. To our knowledge, no research has investigated the subcomponents of selfreported executive functioning most related to procrastination in undergraduate students despite the obvious overlap between these constructs. In a diverse sample of college undergraduates aged 30 years and below, we found that all nine clinical subscales of executive functioning were signiﬁcantly associated with increasing academic procrastination in a model that included personal and medical demographic characteristics. Also, in most of the analyses increasing age showed a signiﬁcant or trendlevelassociationwithincreasingprocrastination.In a second model that also included various demographics and psychological variables (i.e., intellectual, personality, mood), lower conscientiousness also showed a signiﬁcant or trend-level association with increasing procrastination in the majority of analyses. As hypothesized, the ﬁndings showed the importance of various aspects of self-reported executive functions in predicting a tendency toward academic procrastination in college students. In the second model, which included all the demographic, medical, and psychological variables, the BRIEF-A subscales of Initiate, Plan/Organize, Organization of Materials were independently associated with academic procrastination. Close attention to the abilities tapped by these subscales is warranted with an eye towards implications for remediation of problematic delaybehaviors.Individualswithinitiationproblemstypically want to succeed but have difﬁculty getting started and may require extensive prompts or cues to begin an activity. Those with planning and organization difﬁculties may fail to begin academic tasks in a timely fashion or fail to function efﬁciently because they do not have required objects or materials available when they ﬁnally sitdowntowork.Theyalsomayapproachtasksinahaphazardfashionorbecomeoverwhelmedbylargeamounts of information (Roth et al., 2005). While it is not surprising that initiation, planning, and organizational skills were predictive of academic procrastination, these results speak to the importance of working with students to improve these abilities. Effective strategies may involve teaching students to set proximal subgoals for their academic work along with reasonable expectations about the amount of effort required to complete a given task (Ariely & Wertenbroch, 2002; Brooke & Ruthven, 1984; Lamwers & Jazwinski, 1989; Tuckman, 1998). The use of contracts for periodic work completion, administration of weekly or repeated quizzes until topic mastery is achieved, and development of short assignments that build on one another with regular deadlines and feedback are helpful strategies. These goal-setting and achievement experiences enable students with procrastination tendencies to try out what it is like to complete assignments on time (Ackerman & Gross, 2005; Seo, 2008). They also serve to enhance

self-efﬁcacy and self-satisfaction with performance and may diminish the perceived burden or aversiveness associated with task completion (Stock & Cervone, 1990). In turn, procrastination may be reduced as deadlines seem less distant and the intention–action gap narrows (Steel, 2007). Four other BRIEF-A clinical scales were signiﬁcant predictors of procrastination—those of Inhibit, Self Monitor, Working Memory, and Task Monitor. The Inhibit and Self-Monitor subscales measure the ability to not act on impulse and to keep track of and maintain appropriate regulatory control over behavior. It is widely known that procrastinators tend to choose short-term beneﬁt sover long-termgains, reﬂecting a core component of poor self-regulation (Tice & Baumeister, 1997). How then can students learn to overcome their natural preference for impulsive gratiﬁcation through self-control and engagement in behaviors that facilitate attainment of long-term academic goals? Pychyl et al. (2000a) draw upon previous research by Ainslie and Haslam (1992) and suggest that students who procrastinate are seeking temporary relief from the negative or anxious affect associated with unpleasant academic tasks. Through counseling, such individuals should be less inclined towards this short-term affective improvement, which comes at the expense of long-term goal attainment and self-management. In working with such students, a ﬁrst step might be to help them develop an awareness of these emotions and their role in jeopardizing achievement. Subsequently, students are trained in volitional skills(i.e.,how to maintain action against competing goal tendencies while managing intrusive effects of negative affect). Related competencies include gaining control over immediate impulses through the establishment of ﬁxed daily routines for learning and leisure activities, task persistence, and time management (Dietz, Hofer, & Fries, 2007). In addition, the skills necessary to initiate a task often need to be isolated and broken into small, attainable steps. Students can learn to be mindful of the resources required to carry out these steps and identifyandtroubleshootproblemsthatarisewhiledrawing on both working memory and task monitoring skills (Haycock et al., 1998). Procrastination is conceptually and empirically linked to conscientiousness, a trait reﬂecting responsibility, selfdiscipline, achievement motivation, and the careful and diligent fulﬁllment of obligations. In addition, conscientiousness shows an increase in young adulthood corresponding to the maturation of frontal brain regions thatsubservevariousexecutivefunctions(Robins,Fraley, Roberts, & Trzesniewski, 2001; Welsh, Pennington, & Groisser, 1991). Our results indicated that low conscientiousness was an important predictor of procrastination, and it overrode the signiﬁcance of several components of executive functioning—namely, the ability to shift from one situation or activity to another and to modulate emotional responses. While it might be tempting to target this characteristic directly, personality traitsare relatively stable and enduring, andnot easily modiﬁable. Nonetheless, some have suggested that the negative effects of low conscientiousness can be ameliorated through techniques focused on organization and the stimulation of self-control. An easy way to increase self-control and prevent distraction is to block access to short-term temptations—for example, by training oneself to study in a library, work with a clean desktop, and/or study with the door closed (Schouwenburg, 2004). Others have highlighted the value of achievement motivation, which can be enhanced by setting more difﬁcult academic goals and learning to enjoy performance for its own sake, reducing the aversive nature of due tasks (Costa & McCrae, 1992; Spence & Helmreich, 1983). Peer monitoring with accountability and consequencesforbehavioralfailureandself-appraisalmethods (e.g., self-tests with criteria for mastery included) also may improve academic conscientiousness (Tuckman & Schouwenburg, 2004). Questions remain, however, about the long-lasting or sustainable nature of the behavior change with these interventions. Furthermore, conscientiousness may be negatively related with ﬂexibility and creativity(Feist,1998;vanEerde,2003;Wolfradt&Pretz, 2001), which may be problematic in academic situations that require innovative solutions. Only a few other predictors yielded signiﬁcant or trend-level ﬁndings. First, even within this restricted sample of undergraduates, increasing age showed a consistently strong association with higher levels of procrastination. This goes against most reported ﬁndings (Haycock et al., 1998; Howell et al., 2006; Steel, 2007; vanEerde,2003),butmayreﬂectthepreviousﬁndingthat academic procrastination increases as students advance through the educational process (Rosário et al., 2009), with college seniors procrastinating more than ﬁrst-year students (Hill, Hill, Chabot, & Barrall, 1978; McCown & Roberts, 1994, as cited in Ozer et al., 2009). Perhaps the longer a student remains in school, the less enthusiastic and motivated he/she becomes or the more entrenched bad habits become. It is also possible that familial and work responsibilities increasingly limit the time one can devote to academic tasks, or students may acquire additional bad academic habits over time. These possibilities, however, need to be further explored empirically. What is clear is that older undergraduate students in the current study were at greater risk for academic procrastination and possibly represent an at-risk group for negative consequences based on dilatory behaviors. Second,althoughdepressionhasseveralcharacteristics that make it a likely suspect for causing procrastination (e.g., low energy, poor concentration), we saw only minimal, trend-level evidence for the association between increasing depressive symptoms and greater procrastination.Thisisconsistentwithrecentmeta-analyticﬁndings, which concluded that depression’s connection appears to beduemostlytowaningenergylevels,whichmakestasks more aversive to pursue (Steel, 2007). Additionally, as notedbyPychyletal.(2000a),whenstudentsareprocrastinating, they may not concurrently experience negative affect because they are engaged in pleasant activities to the neglect of those found more aversive. In summarizing the effects of mood on procrastination, van Eerde (2003) observed that mood variables are just as likely to be precursors as outcomes of procrastination, and extant

research provides no indication of whether to consider them as antecedents or consequences. Clearly the link between procrastination and depression is complex, and futureresearchmightbeusefultofurtherunderstandthis relationship. Third, there was one model in which presence of a learning disability was associated with lower procrastination, perhaps due to the tendency of this studentpopulationtoseekoutacademichelporremediation (Trainin & Swanson, 2005). This ﬁnding is in contrast to the one previously published study (Klassen, 2008a), which found that college students with learning disabilities reported higher levels of procrastination than their peers and lower levels of metacognitive self-regulation and self-efﬁcacy for self-regulation. It is unclear what accounts for this discrepancy, but it is important to note that both the depression and learning disability ﬁndings wereattrendlevelsandneedtobereplicatedandclariﬁed before conclusions can be drawn. Contrary to our hypotheses, we did not ﬁnd significance for the predictors of neuroticism and anxiety, two closely related traits. This is consistent with results of a recent meta-analyses of procrastination’s possible causes and effects (Steel, 2007), which suggested that neuroticism’s connection to procrastination was primarily due to impulsiveness and added little unique variance over conscientiousness (Johnson & Bloom, 1995; Lee et al., 2006; Schouwenburg & Lay, 1995). Similarly, Haycock et al. (1998) found that anxiety did not contributesigniﬁcantlytothevarianceinprocrastinationand concluded that anxiety should be examined and interpretedinthecontextofitsrelationshiptoothervariables. Steelalsonotedthatmoodsarepronetochange,andthat procrastinators may feel remorse for their inactions at any time, perhaps after the academic semester has ended. Consequently, researchers might need to test mood at more than one time point or over longer time periods, in order to detect a relationship with procrastination. Research employing repeated measures of state anxiety overanacademicsemestersupportstheideathatprocrastinators tend to experience less stress early on, but more stress later on and overall (Tice & Baumeister, 1997). Additional support comes from research that found a relationship between procrastination and anxiety but only as an increase during the last week of the course (Lay & Schouwenburg, 1993) or as a decrease at the course beginning (Towers & Flett, 2004, as cited by Steel, 2007). Clearly, the relationships between procrastination and neuroticism/mood are complex and may not be best described in a general linear fashion.

Limitations

Our ﬁndings, while suggestive that aspects of selfreported executive dysfunction are related to academic procrastination, warrant consideration in the context of study limitations. Despite its advantage as a comprehensive, ecologically sensitive measure of executive functioning, the BRIEF-A, along with all self-report instruments, is open to the criticism that it may have produced socially desirable responses or other biases. The present ﬁndings are therefore preliminary and would be strengthened if future research were to use behavioral measures of task postponement in addition to self-report instruments(e.g.,Howelletal.,2006;Milgram,Dangour, & Raviv, 1992). External correlates of executive function are also needed in the form of clinical neuropsychological measures. Seven of the nine BRIEF-A subscales showed a signiﬁcant association with procrastination, and while we achieved ample sensitivity, speciﬁcity could be improved. To this end, objective neuropsychological instruments should be selected that assess domains of executive functioning indentiﬁed as important in the current study with the goal of improving our speciﬁcity and further delineating the relationship between academic procrastination and various executive functions. Given the overrepresentation of females in social science participant pools as well as data collection time constraints, we could not recruit more males, though this would have been preferable. We attempted to address this sampling limitation by using gender and other demographic characteristics as covariates in all analyses, with no signiﬁcant ﬁndings pertaining to these variables. A methodological limitation is that we did not counterbalance the ordering of tests, though we did separate the Lay from the BRIEF-A by placing one at the beginning and one at the end of the test battery. Because the Lay and BRIEF-A tap similar behaviors and cognitive styles, it might have been preferable to counterbalance to minimize the possibility that participants’ responses were inﬂuenced by ordering of critical questions. This study was correlational and cross-sectional in nature, and we therefore cannot draw conclusions pertaining to directionality or predictive value of executive function difﬁculties to long-term procrastination and associated negative outcomes. While it is plausible that procrastinators lack executive control skills, it is also possible that both procrastination and executive dysfunction are caused by another variable or variables, and the current study was not designed to address this possibility. Similarly, it is possible that BRIEF-A scores served as a proxy for either diagnosed or undiagnosed ADHD, which by deﬁnition leads to these symptoms. However, 2% of our sample reported a diagnosis of ADHD, and the prevalence in adults is about 4% (Kessler et al., 2006). Thus, due to underreporting or underdiagnosis, we may have overlooked another 4–6 individuals, which would not have altered our overall pattern of ﬁndings. Furthermore, the current study was intended to investigate the relation between mild executive dysfunction and procrastination in a generally healthy adult sample rather than in a clinical sample for whom the pattern and overall severity of BRIEF-A scores would likely differ. Finally, we measured various aspects of task postponement but did not inquire directly about the adverse impact of such deferment on functioning. An increased understanding of the antecedents, motivational dynamics, and effects of procrastination will help to identify the appropriate strategies to remediate the problematic aspects of this behavior.

Future directions

Our ﬁndings are consistent with the conceptualization of executive functioning as central to the ability to engage in independent, goal-oriented behavior, especially in the context of unstructured, novel, or complex tasks, and suggest that procrastination could be an expression of subtle executive dysfunction—even in this group of neuropsychologically healthy young adults. Executive functions rely on a number of cortical and subcortical brain regions including prefrontal cortices, anterior cingulate gyrus, basal ganglia and diencephalic structures, cerebellum, deep white matter tracks, and parietal lobes areas. These brain areas are richly interconnected and are also linked with many additional regions that together subserve virtually all cognitive processes (Funahashi, 2001; Greene, Braet, Johnson, & Bellgrove, 2008; Roth et al., 2006). While executive dysfunction is observed in variouspsychiatric,neurological,andsystemicdisorders,our research suggests that there may be problems within cognitively healthy individuals that contribute to a vulnerability to procrastination. Future research might identify subtle neuroanatomic or functional brain abnormalities associated with procrastination. As training of cognitive strategies has been found to alter brain activity or neurochemistry (Olesen, Westerberg, & Klingberg, 2004; Roth et al., 2006; Valenzuela et al., 2003), pre–post intervention studies using neuroimaging paradigms might also provide evidence of neurobiological mediation of procrastination. Researchers are also exploring the degree to which individual differences in executive capacity may be attributed, at least in part, to genetic variation (Goldberg & Weinberger, 2004; Kempf & Meyer-Lindenberg, 2006). Individual differences in executive function are being considered at multiple levels of analysis, including potential genotypes, proposed endophenotypes (e.g., performance on cognitive tasks that involve executive functions), and relevant phenotypes associated with executive functioning,suchastemperament,personality,andpsychopathology(Williamsetal.,2009).Iffeasible,thecombinationof neuroimaging techniques with behavioral measures, self report, and genetics may help reﬁne the phenotype of procrastination and inform the development of strategic individualized treatments. It will also be important to validate self-reported procrastination with external measures such as grade point average, such as number of missing or late assignments, incomplete grades, and so on. Our results revealed increased age as a signiﬁcant predictor of academic procrastination, which suggests the need to target at-risk upper level students who may be struggling to remain productive. The additional ﬁnding of low conscientiousness among procrastinating students is consistent with their characterization as less self-regulated and disciplined and suggests avenues for remediation(described above).Such interventions should account for a recent ﬁnding, which demonstrates that in addition to self-regulation skills, students must also possess the conﬁdence to implement effective learning strategies, resist distractions, complete schoolwork, and participate in class learning (referred to as “self-efﬁcacy for self-regulation of learning”; Klassen, Krawchuk, & Rajani, 2008b). Thus, cognitive and behavioral strategies to improve higher order executive processes should be delivered in conjunction with attempts to build students’ conﬁdence in their ability to achieve academic success.

CONCLUSION

College students are faced with multiple tasks and deadlines that need to be accomplished within designated time frames, while much of their time is unstructured and unregulated. Because delay behaviors can have serious negative consequences, much research has focused on identifying the factors that produce and sustain academic procrastination so that effective interventions may be implemented. To our knowledge, this study was the ﬁrst to investigate subcomponents of self-reported executive functioning associated with procrastination in a demographically diverse sample of college students. We found that the domains of initiation, plan/organize, organization of materials, inhibition, working memory, and task monitoring signiﬁcantly predicted academic procrastination in addition to increased age and lower conscientiousness. Overall, the conceptualization of academic procrastination as a problem of executive dysfunction holds promise for researchers, educators, and practitioners who seek to understand this behavior and apply focused, strategic interventions to help alleviate its negative consequences.

**Article 6.** Internet Addiction and Depression among College Students in Malaysia

Internet Addiction and Depression among College Students in Malaysia

Zahiruddin Othman", Chung Wah Lee2>

ABSTRACT

Introduction: Internet has revolutionized the information age. However, excessive internet use has led to health issues among users and the most commonly reported psychological problem is depression. Yet, there is dearth of research in this area among college students in Malaysia. Objective: The aim of the present study was to examine the internet addiction (IA) and its association with depression and anxiety among college students. Methods: Students age 18-24 from allied health colleges who were doing attachment and posting in Hospital Tengku Ampuan Rahimah, Klang were recruited into the study. IA was assessed using the internet addiction test (1AT), whilst depression and anxiety by using the hospital anxiety and depression scale (HADS) Results: IA was significantly associated with depression (p < 0.001) and male gender (p = 0.047). In addition, IA was also associated with internet use characteristics such as using computer outside home (p = 0.008), using the internet for surfing (p =

0.016) and e-mailing (p = 0.025), and spending more time online during the weekends (p = 0.003). IA was not associated with anxiety (p = 0.365). Conclusions: Internet addiction is associated with depression and male gender among Malaysian college students. Further study on psychological factors such as personality traits and coping styles is recommended in order to understand the underlying mechanism in IA and propose possible interventions.

INTRODUCTION

The internet is a global system of interconnected computer networks that has revolutionized the information age, allowing human to gain access to unlimited amount of information as well as changing the way human communicates with each other'-”. Affordable access to internet allows for rapid growth of users worldwide. It can have a positive impact, for example high internet usage (more than 6 hours/day) among medical students was associated with higher academic performance31. Concurrently, many studies have shown that excessive internet use has led to negative impact on the health of users41. The idea that problematic computer use meets criteria for an addiction was first proposed by Kimberly Young51. Since then internet addiction (IA) has been extensively studied by other researchers using terms, such as pathological internet use61, problematic internet use71, excessive internet use and compulsive internet use and internet dependence\*\* 1 2 \*. In DSM-V, internet gaming disorder, also commonly referred to as IA, has been included under Section 3- Emerging Measures and Models, which warrant further clinical research before it can be considered as a formal disorder. IA has nevertheless now been recognized as a public health issue, with behavior according to literatures, similar to that of gambling disorder, and has shown to cause dysfunction in the many aspect of the individual's life4-91. A systematic review of scientific studies on IA indicated strong

associations between IA, symptoms of ADHD, and depression. The strongest association was found between IA and depression. Otherwise anxiety, social phobia, obsessive-compulsive symptoms, and aggression did not appear to be significant factors of I A101. A number of studies have been conducted in this field among Malaysian young adults"141. Depression was associated with male gender and pathological internet use among Malaysian undergraduates"1. In a cross-sectional online survey among young adults, compulsive internet use was associated with GHQ scores indicating presence of mental health problem121. Otherwise, there is still scarcity of research focusing on the association between IA and depression or anxiety in Malaysia. The aim of the present study, therefore, was to examine the IA and its association with depression and anxiety among college students.

METHODS

Study setting and subjects

This cross-sectional study was approved by the USM Human Research Ethics Committee (HREC) and Malaysia Medical Research and Ethics Committee (MREC). It was conducted in November 2015 to January 2016 at Hospital Tengku Ampuan Rahimah (HTAR), a govern ent hospital situated in Klang where students from nearby allied health colleges came to do their attachments and postings as part of their training requirement of their respective courses. The name list of Malaysian students aged 18-24 was obtained from the training unit in the administrative office of HTAR. All of the subjects were engaged after obtaining verbal consent from their clinical supervisor, in small groups of five to ten students at a time, due to their postings being in small groups in various different departments. Information related to the study and questionnaires were briefed to them

and sufficient time was given allowing them to ask questions pertaining to the study. Those with previous history of mental illness or on psychiatric medications were excluded. A written consent form which had been distributed to each subject was signed and returned, before the questionnaires were allocated to them. All the questionnaire sets were tagged with a serial number for easy reference during data entry. The completed questionnaires were detached and separated from the consent form so that they remained anonymous.

Me a s u re m e n ts

a. Socio-demographic and internet use information

Information on socio-demography and internet use of respondents was collected using a self-made questionnaire. These include duration of internet use in hours during the weekdays and weekends, vehicle for internet use, such as smartphone, home computer or computer outside home, and purpose of internet use, whether it is used for social networking, chatting, surfing, games, e-mailing, downloading, or shopping.

b. The internet addiction test (IAT)

The original IAT was created by Kimberly Young and by far the most widely translated and used tools for the assessment of IA globally. It comprises a total of 20 items rated on a 5-point Likert scale which takes about 5 minutes to complete. 8 items were adapted from the DSM- IV pathological gambling criteria and the remaining 12 items assessed the areas of life affected by the excessive internet use. It has good internal consistency and concurrent validity and is a reliable instrument to assess the addictive use of the internet151. Scores of 0-19, 20-49, 50-79, and 80-100 indicate limited use, mild/ average user, moderate/regular user/occasional or frequent problems secondary to internet use, and severe/significant problematic use of internet. The validated Malay version of IAT was available with good internal consistency (Cronbach's a = 0.91), parallel reliability (intraclass coefficient = 0.88, p < 0.001) and concurrent validity with the Com pulsive Internet Use Scale (Pearson's correlation = 0.84, p < 0.001) 16'.

c. The hospital anxiety and depression scale (HADS)

The HADS was designed to identify the caseness, which is possible and probable diagnosis of depression and anxiety among patients in non-psychiatric hospital clinics. The somatic symptoms of anxiety and depression were omitted to suit the hospital population so that the somatic symptoms originating from the patient's medical condition would not give a false positive on either the anxiety or depression component of the scales, making the scales suitable for hospital use17’. A systemic review concluded that not only HADS is a questionnaire that performs well in screening for the dimensions of anxiety and depression component separately for caseness in a non-psychiatric hospital clinic setting, it also has the same properties when applied to the general population, in the general practice setting as well as among the psychiatric patients'\*’. The scale has a total of 14 items, with 7 items in HADS-A and the other 7 items in HADS-D, all intermingled to form a complete questionnaire. It is rated from 0-3 in each item, thus scoring from a range of 0 to 21 for each component, that is the anxiety component and the depression component. It was found that a cut-off point of 8/21 for both the anxiety or depression component provides the most optimum sensitivity and specificity when using this tool. The anxiety component of HADS has a specificity of 0.78 and a sensitivity of 0.9 while the depression component has a specificity of 0.79 and a sensitivity of 0.83. A validated Malay version is available. The optimum cut-off point for both HADS-A and HADS-D is 8/9 with anxiety subscale sensitivity 90.0% and specificity 86.2% and depression subscale sensitivity 93.2% and specificity 90.8%'”.

Statistical a n alyses

The data were entered into the Statistical Package for Social Sciences (SPSS) version 22. The preliminary information on socio-demography, duration spent online and activities involved regularly online, of the study population were explored and analyzed using descriptive analysis. The associations between anxiety and depression component of the HADS and IA were analyzed using the chi square test. P-value of less than 0.05 was taken as significant at 95% confidence interval for all variables.

RESULT

A total of 267 college students who fulfilled the inclusion and exclusion criteria, and answered all the questions were included into the study. They comprised of the age group 18 to 24 years old with the mean age 20.9 with a standard deviation of 1.4. The majority were Malay (88.4%) and female (86.1%). During the weekdays 123 (46.1%), 84 (31.5%), 31 (11.6%), 14 (5.2%) and 15 (5.6%) of subjects spent time online for less than 3 hours, 3 to < 7 hours, 7 to < 9 hours, 9 to < 12 hours, and 12 hours or more, respectively. While during the weekend 13 (4.9%), 73 (27.3), 42 (15.7), 81 (30.3) and 58 (21.7) of subjects spent time online for less than 3 hours, 3 to < 7 hours, 7 to < 9 hours, 9 to < 12 hours, and 12 hours or more, respectively. The data demonstrated increased use of internet during the weekends in which majority use it for 9 hours or more. As shown in table 1, the socio-demographic and internet use factors significantly associated with IA were male gender (p = 0.047), students who uses computer outside their home (p = 0.008), students who uses the internet for surfing (p = 0.016) and e-mailing (p = 0.025), and more time online during the weekends (p = 0.003). Table 2 showed that depression (p < 0.001) was significantly associated with IA whiie anxiety (P = 0.365) was not.

DISCUSSIO N

In this study, depression was significantly associated with IA that is consistent with other studies from South Korea20 22’, China23 28’, Norway2”, UK301 and US3". A local study also had a similar finding"’. Anxiety was not associated with A1 that is consistent with 3 cross-sectional stud- jes2s.26.3oi Ma|e gen(jer was significantly associated with IA that is consistent with other studies22 24-2W,-3<”. The biological explanation regarding the associations between IA and depression is still inconclusive. Genetic polymorphisms similarity between AI and depressed patients may explain the association. In a study by Lee et al,32’, 91 male adolescents with excessive internet use (EIU) and 75 healthy comparison subjects were compared on their genetic polymorphisms of the serotonin transport gene. The EIU group had higher homozygous short allelic variant of the serotonin transporter gene (SS-5HTTLPR) frequencies suggesting that EIU subjects may have genetic polymorphisms similar to depressed patients. Structural alterations in the prefrontal cortex may mediate the relationship between internet gaming disorder (IGD) and depressed mood. In a study by Choi et al.n\ lower gray matter (GM) density in the left dorsolateral prefrontal cortex (DLPFC) in the IGD group than in the internet gaming control group and non-gaming control group, and the GM density was associated with lifetime usage of internet gaming, depressed mood, craving, and impulsivity in the gaming users. Striatal volumetric analysis detected a significant reduction in the right nucleus accumbens in the IGD group and its association with lifetime usage of gaming and depression. These findings suggest that alterations in the brain structures involved in the reward system are associated with IGD- related behavioral characteristics. Furthermore, the DLPFC, involved in cognitive control, was observed to serve as a mediator in the association between prolonged gaming and depressed mood. Although the cross-sectional design is reliable in determining associations, it is unable to determine the causal relationship between AI and depression. It is possible that subjects with 1A are at higher risk for comorbid depression. On the other hand, it is also possible that depression leads to IA or both shared similar underlying biological causes. Therefore, it is recommended that prospective cohort studies be performed within this scientific field to determine the causality.

C O N C L U S IO N

Internet addiction is associated with depression and male gender among Malaysian college students. Further in depth study is recommended which may include psychological factors such as personality traits and coping styles in order to investigate the dependent users and also to take measures to rehabilitate them if necessary.

### **Article 7** [**Addictive potential of novel treatments for refractory depression and anxiety**](http://eds.b.ebscohost.com.libdatab.strayer.edu/eds/viewarticle/render?data=dGJyMPPp44rp2%2fdV0%2bnjisfk5Ie46fKK3%2b%2fjiqzj34HspON88aPqgfTbvlCtp7ZHsKavUJ6uuEmysK5OnsbLPvLo34bx1%2bGM5%2bXsgeKztkywp7JJtqqvPurX7H%2b72%2bw%2b4ti7feLp4ofonPJ55bO%2fZqTX7FXj2vF87eCsSrau43viqeFO36a0TLLa4FGv2ORO4dmueeDZsE61q7I%2b5OXwhd%2fqu37z4uqM4%2b7y&vid=12&sid=cdaaa108-901b-4052-97db-f01d65fa3498@sessionmgr102)

*Detail Only Available*

Academic Journal

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**Abstract |** [**Full Text**](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-article-NDT)

Dusan Kolar Mood Disorders Research and Treatment Service, Department of Psychiatry, Queen&rsquo;s University, Kingston, Ontario, Canada Abstract: Treatment-resistant mood disorders and anxiety disorders require intensive treatment, but treatment options should balance benefits and adverse effects or other potential detrimental effects on patients, including the risk of developing prescription medication addiction. Some of the newer treatment modalities for mood and anxiety disorders may have similar properties to benzodiazepines. The goal of this review was to identify the potential for developing dependence on the novel treatment approaches to treatment-resistant depression and refractory anxiety disorders. PubMed, MEDLINE, PsycINFO, Ovid, Cochrane Library, and Google Scholar were searched. Ketamine is effective in improving symptoms of major depressive disorder, but with no sustained benefits. Long-term use of oral or intranasal ketamine formulations may be associated with the risk of developing dependence. Augmentation of stimulant medication is usually effective for residual symptoms of depression, but the effects are usually short lasting and there is a potential for abuse. Synthetic cannabinoids and medicinal cannabis are increasingly being prescribed for a number of medical conditions, including anxiety disorders, without enough evidence about their efficacy and with the risk of patients developing dependence. In summary, benzodiazepines, ketamine, stimulant medications, and cannabinoids have some common characteristics, including short-lasting benefits and the risk of developing prescription medication addiction with longer use. All of these treatments may raise ethical dilemmas about the appropriateness of prescribing these medications in the long run for patients with depression and anxiety disorders. Keywords: treatment-resistant depression, anxiety, addiction, ketamine, cannabinoids

**Introduction**

Major depressive disorder and anxiety disorders are the most prevalent mental conditions in psychiatric practice. There has been an increase in the number of patients diagnosed with treatment-resistant depression and anxiety.

These people with treatment refractory conditions may have higher rates of comorbidity with other psychiatric disorders and more general medical comorbidity, inability to work, higher rates of absenteeism, and more frequent hospitalizations and incur higher costs on the health care system.

Treatment-resistant depression and anxiety usually require more intensive treatment and multimodal treatment including different treatment modalities, but medication remains the major treatment component. There are not many new antidepressant medications and clinicians sometimes rely on off-label treatment options. Some of these new treatments have not been sufficiently clinically investigated in terms of safety and possible detrimental effects such as addictive potential. Some of these new agents for refractory depression and anxiety are effective in short-term treatment, but, similarly to benzodiazepines, may be associated with developing tolerance and dependence with long-term use. The goal of treatment should not be obtaining instant and short-lasting benefits without taking into account possible negative effects, such as the potential for developing dependence. The aim of good clinical balance is to weigh the benefits and possible negative effects of treatment. Patients may expect from medical professionals instant solutions for their suffering, and this often entails the prescribing of medications with rapid onset of action such as analgesics or benzodiazepine anxiolytics. However, medical professionals should follow ethical principles when prescribing medications and should distinguish agents with the potential to temporarily improve patient symptoms from agents that are genuinely therapeutic.

The patient’s interests will be best served by obtaining evidence-based treatments and medications with well-established efficacy and safety.

It is well known that benzodiazepines are addictive, but we should not forget that these medications were prescribed for decades as a main treatment option for patients with anxiety disorder. Was there a lack of evidence about the addictive potential of benzodiazepines or was this evidence simply ignored as there were no better alternatives? Currently, a significant number of patients is addicted to benzodiazepines and for many of them benzodiazepines are no longer a sustainable treatment option because of restrictions in prescribing benzodiazepines. Many of these patients were also informed about the increased risk of dementia with long-term use of benzodiazepines,[1](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref1) which is an additional burden on patients. Nine studies have demonstrated this increased risk of dementia in patients on long-term treatment with benzodiazepines,[2](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref2) and this risk is higher in elderly people.[3](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref3)

The introduction and easy acceptance of newer treatment options for refractory depression and anxiety, such as ketamine and stimulant medication augmentation in treatment-resistant depression, and synthetic cannabinoids and medicinal cannabis in anxiety disorders and depression, have many characteristics in common with the uncritical prescribing of benzodiazepines for anxiety disorders in the past.

Research studies on the addictive potential of ketamine, stimulant medications, and nabilone are very limited and the literature on this topic is sparse. The goal of this literature review is to explore the available data and provide a critical opinion about using potentially addictive agents in clinical practice.

**Method**

Searches of PubMed, MEDLINE, PsycINFO, Ovid, Cochrane Library, and Google Scholar were conducted for placebo-controlled randomized clinical trials, meta-analyses, non-randomized controlled studies, naturalistic studies, case reports, and treatment guidelines published in the past 10 years (from 2008 to 2017/2018) on the potential for abuse and dependence of new and not well-investigated medications in the treatment of refractory depression and refractory anxiety disorders. As the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) classification was used, post-traumatic stress disorder (PTSD) and obsessive-compulsive disorder were excluded from the category of anxiety disorders and therefore new agents in treating these conditions, besides nabilone, will not be included. Nabilone will be included simply because the off-label use of this medication for anxiety, insomnia, and depression started after the publication of studies on the use of nabilone in PTSD-associated nightmares. This review will not include other common augmentation strategies in treatment-resistant depression, such as atypical antipsychotics and other newer medications such as pramipexole, as the focus of this review is only medication with potential addictive potential.

**Ketamine in the treatment of major depressive disorder**

Ketamine is a non-selective N-methyl-D-aspartate (NMDA) receptor antagonist. Ketamine has both opiate and stimulant effects. It is a strong promoter of dopamine turnover and ketamine’s monoaminergic properties are similar to those of cocaine or amphetamine. Ketamine also has mu-opioid receptor properties.[4](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref4)

Ketamine was used as an anesthetic agent primarily in pediatric surgery and veterinary medicine. Pharmacologically, ketamine can be classed as a dissociative anesthetic medication, hallucinogen, and psychotomimetic drug. Ketamine is also well known as a recreational drug because of its psychedelic and dissociative effects.

More recently, ketamine has been used for treating depressive symptoms refractory to other treatments. Clinical trials of ketamine in patients with treatment-resistant depression demonstrated that it has rapid antidepressant effects within 2–4 h, but these benefits of ketamine infusions are short lived, being sustained for only 4–7 days.[5](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref5)

A total of 7 trials with 147 ketamine-treated participants showed that ketamine produced a rapid but transient antidepressant effect, accompanied by brief psychomimetic and dissociative effects.[6](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref6)

Ketamine is effective in the rapid treatment of unipolar and bipolar depression, both as monotherapy and in combination with other medications.[7](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref7),[8](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref8)

Kellner and colleagues reported a series of severely depressed patients, some of whom were suicidal, who received ketamine infusions before being referred for electroconvulsive therapy. These patients had either no antidepressant effects or transient antidepressant benefit from ketamine with unpleasant side effects, mostly dissociative effects.[9](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref9) The authors concluded that evidence-based treatments, including electroconvulsive therapy, should be offered before experimental and unproven treatment approaches.

A meta-analysis which included 7 randomized controlled trials (RCTs) with small sample size reported no serious adverse events in short-term treatment. The authors concluded that the potential for misuse is negligible, although this statement did not come from the results of any of studies included in this meta-analysis.[8](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref8)

The most common adverse events after ketamine infusion are dizziness, blurred vision, headache, nausea or vomiting, dry mouth, poor coordination, poor concentration, and restlessness, and these side effects are usually short lasting. About 17% of patients in one study developed significant dissociative symptoms.[10](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref10) Cardiovascular adverse events were reported, requiring a higher dose of antihypertensive medication, and caution is needed in patients with cardiovascular conditions.[7](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref7) Side effects during and after each ketamine infusion were generally mild in research studies and ketamine caused minimal positive psychotic symptoms.[11](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref11)

The potential for abuse and other safety concerns associated with ketamine mean that caution is required when applying ketamine outside the research setting.[10](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref10)

Concerns over cognitive impairment, cystitis, liability for abuse, and a potential increase in iatrogenic ketamine disorder related to more frequent use of ketamine suggest that the use of ketamine should be restricted until additional research data are available.[12](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref12)

The main concern about the clinical application of ketamine trials is the conclusion that treatment response is short lasting.[8](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref8) In a 4 week placebo-controlled study, the efficacy and safety of an intravenous single dose of ketamine as augmentation of escitalopram in major depressive disorder were examined.[13](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref13) This augmentation strategy was safe and effective in speeding up oral antidepressant efficacy. Effects lasted for 2 weeks, but this study does not provide evidence of sustained benefits as continuous therapy with escitalopram served as a maintenance treatment after discontinuing ketamine.[13](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref13)

Another study which failed to demonstrate any long-lasting effects of ketamine was an RCT which included 47 patients with treatment-resistant depression. At day 7 after stopping ketamine infusions, only 21 patients still met the criteria for response.[10](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref10) About 89% of patients relapsed, on average 19 days after receiving 6 infusions of ketamine.[11](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref11)

This research evidence of the lack of sustained benefits of ketamine is consistent with clinical experience of short-lived therapeutic effects. The only study demonstrating some sustained benefits of intranasal esketamine is an RCT published by the Janssen Research and Development team.[14](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref14) This study included 67 patients with treatment-resistant depression. Among 22 participants in the follow-up phase, only 10 had improvements in depressive symptoms that persisted up to 2 months after stopping esketamine.[14](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref14) This small sample size in the follow-up phase and a small percentage of participants maintaining benefits do not provide convincing evidence of sustained therapeutic benefits of ketamine.

As the benefits of ketamine are short lasting, ongoing treatment with ketamine would be needed for a long time, similarly to selective serotonin reuptake inhibitors, serotonin–norepinephrine reuptake inhibitors, and other antidepressants. Such regular treatment with ketamine would make it the first time in the history of medicine that an anesthesia medication for short-term use was used for the long-term treatment of a chronic condition, without enough knowledge about the possible consequences and risks of this prolonged therapy.[15](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref15)

The literature on using oral and intranasal ketamine formulations is sparse, but there are established ketamine clinics around the world which provide off-label ketamine to patients for extended periods. As ketamine is a well-known recreational drug that can lead to ketamine dependence, treating depression with oral or intranasal ketamine formulations may increase the risk of developing dependence over an extended period, despite the use of much lower doses. There is no available research on the addictive potential of ketamine used in the treatment of depression.

At the current level of knowledge, we can only hypothesize that long-term use of ketamine in the treatment of depression may result, as in drug addicts, in patients developing tolerance, dependence, drug craving, and withdrawal symptoms.

There is some research evidence of neurotoxicity in animals,[6](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref6) and this concern should not be neglected in considering long-term ketamine treatment trials. There is research evidence of abnormalities of white matter in bilateral frontal and left temporoparietal regions following chronic ketamine use.[16](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref16) Detrimental effects of ketamine are evident when used in uncontrolled circumstances.

Recreational ketamine users may develop memory deficits, delusions, hallucinations, ulcerative cystitis, elevated liver enzymes, and biliary tract dilatation in the absence of an obstructing lesion.[17](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref17)

A significant number of ketamine abusers developed dependence after regular use for 1 year, and 53.5% of these abusers reported withdrawal symptoms including fatigue, excessive yawning, aggressive or hostile behavior, anger, irritability, and depression.[18](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref18)

Ketamine is not recommended as a treatment option for treatment-resistant depression in any treatment guidelines. The Canadian Network for Mood and Anxiety Treatments (CANMAT) guidelines for the management of adults with major depressive disorder emphasize that ketamine is associated with psychotomimetic side effects and the potential for abuse, and that there are very limited data on its safety and efficacy with longer term use.[19](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref19)

It seems that the conclusion of the group of authors from Australia, the UK, the USA, and Canada, in an editorial published in the British Journal of Psychiatry in late 2016, is probably the best recommendation on using ketamine in clinical practice: “Much more needs to be learnt about the maintenance of response and long-term outcome before using ketamine more widely in clinical practice.”[20](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref20)

**Stimulant medication augmentation in treatment-resistant depression**

Augmentation is a common strategy in addressing treatment resistance in depression. Among available augmentation strategies, stimulant augmentation is a treatment with less available evidence compared to other augmentations in treatment-resistant depression. The use of methylphenidate and amphetamines for augmentation is limited as there are no guidelines or expert opinion/consensus on the optimal duration of treatment, stimulant dosage, and treatment goals.

The cognitive-enhancing properties of stimulant medications, increased energy level, and general improvement in morning functioning make stimulant medications more acceptable to patients than atypical antipsychotics. Some clinicians unreasonably prescribe stimulant medications in the belief that they are serving the patients’ best interests, although there is no strong evidence for the efficacy of stimulant augmentation.[21](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref21) Stimulant augmentation in the treatment of major depressive disorder is a third-line treatment option with a level 3 of evidence in the CANMAT guidelines for Major Depressive Disorder.[19](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref19)

Some researchers have tried to demonstrate that psychostimulants may have overall antidepressant effects beyond augmentation, such as in an Australian open study from 2013. Stimulants, including methylphenidate and dextroamphetamine administered as augmentation, were very effective in treating melancholic symptoms for 20% of patients among the group of 50 patients with unipolar or bipolar depression. For 50% of patients in each group, stimulants were “somewhat effective” and for 30% ineffective.[22](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref22)

Corp and colleagues carried out a literature review on using stimulants and stimulant alternatives in treating depression and concluded that modafinil and armodafinil are effective treatments for treatment-resistant unipolar and bipolar depression.[23](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref23) Data from randomized clinical trials on methylphenidate and amphetamines are too limited to support the use of stimulants as a first-line augmenting strategy for depression, with the exception of one RCT demonstrating the effects of lisdexamfetamine.[23](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref23) There is limited evidence on the efficacy of lisdexamfetamine in improving executive dysfunctions and depressive symptoms in patients with mild major depressive disorder.[24](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref24)

Stimulant medications could address certain residual depressive symptoms, including decreased energy, lack of concentration, decreased alertness, and daytime sleepiness, but unfortunately these benefits are not sustained and patients lose all of these positive effects of stimulants after medications are discontinued. However, it is not uncommon to see patients using stimulants continuously for several years or even longer.[25](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref25) As a result of this long-term use, the patients may develop dependence on stimulants, particularly amphetamines. There is no research evidence on long-term use of stimulant medications in treatment-resistant depression, but in clinical practice there are patients with depression receiving stimulants for a few years or longer.

There is little research evidence about the potential for addiction of stimulant medications, probably because these medications are mostly used in the treatment of attention-deficit hyperactivity disorder (ADHD) in children and adolescents, and the prevailing hypothesis in child psychiatry is that treating ADHD will prevent the development of substance use disorder. On the other hand, trials on the use of stimulant medication (lisdexamfetamine) in treating cocaine dependence[26](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref26) and methylphenidate in the treatment of amphetamine/metamphetamine dependence[27](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref27) may imply the potential of prescription stimulant medications to be used as substitution therapy.

There is concern over the non-medical use and misuse of stimulant medications in individuals without ADHD, particularly the misuse of short-acting agents,[28](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref28),[29](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref29) and the potential for abuse is equated with illicit central nervous system stimulants such as amphetamine and cocaine.[30](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref30) However, there is no research evidence for patients being dependent on prescription stimulant medications in the context of long-term treatment. A group of authors from France emphasized that the risk factor for dependence on methylphenidate in children who receive normal doses is the duration of treatment.[31](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref31)

In the case of stimulant augmentation in major depressive disorder, discontinuing stimulants after long-term use could cause sudden and significant declines in energy level, motivation, and cognitive performance, as well as high anxiety and restlessness. These types of withdrawal symptoms provide the best evidence of dependence on stimulants.

However, if Parker et al concept of using stimulants as antidepressants[22](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref22) became widely accepted, then individuals with depression could be taking stimulant medications for most of their life, similarly to patients with ADHD.

**Medicinal cannabis and synthetic cannabinoids in anxiety and depression**

Medicinal cannabis is considered nowadays as a treatment for various medical conditions such parkinsonism, hepatitis C, pain management in cancer, spasticity in patients with multiple sclerosis, neuropathic pain, refractory epilepsy, glaucoma, and some psychiatric conditions (anxiety and depression).

Cannabis and synthetic cannabinoids exert their effects via the endocannabinoid system. Cannabinoid receptors (CB1 and CB2) are present in the central nervous system and throughout the body. They are activated by cannabis, synthetic cannabinoids, or endogenous endocannabinoids.[32](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref32) This presumed mechanism of action is an explanation for possible the therapeutic effects of medicinal cannabis. However, it is still difficult to understand the wide range of therapeutic indications for medicinal cannabis.

It also seems that public approval is driving the medicinal cannabis legalization process without going through the standardized procedures for new drug development and approval.[33](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref33)

Medicinal marijuana and recreationally used marijuana differ in how the drug is used in terms of the amount and goals of ingestion, although there is a significant overlap between medicinal users and recreational users.[33](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref33)

Cannabis is the most widely used illicit drug in the USA. Among the 20 million Americans who used marijuana in 2013, it was found that up to 9% of users may become dependent and this number goes up to 17% if marijuana use is started during adolescence.[34](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref34) Long-term and regular use of cannabis for medical conditions could be associated with an even higher rate of cannabis dependence and this medical use is particularly problematic in psychiatric conditions such as anxiety disorders.

The results of a systematic review and meta-analysis on the use of cannabinoids in medicine did not provide any evidence for the efficacy of cannabis and cannabinoid drugs in treating anxiety. There is some evidence of efficacy in the treatment of chronic pain and spasticity.[35](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref35)

The evidence to support using cannabis in the treatment of anxiety disorders and mood disorders is very limited and includes a few single-dose studies.[36](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref36) On the other hand, cannabis is associated with adverse effects including increased anxiety, psychosis, cognitive impairment, and addiction, which pose significant limitations to using cannabis as a treatment in psychiatry.[36](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref36)

The unjustified belief that medicinal cannabis may have anxiolytic effects comes from reports about decreased anxiety levels after taking cannabis. This transitory decrease in anxiety level after a single dose, with a subsequent increase in baseline anxiety, is the best evidence that medicinal marijuana is not a sustainable treatment option for anxiety disorders. Psychiatric patients in general and patients with a history of substance use disorders are not candidates for medicinal cannabis at all.[37](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref37)

Frequent cannabis users have a higher prevalence of anxiety disorders, but there is not enough evidence of increased risk for developing long-lasting anxiety disorders in cannabis users.[38](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref38)

There are single reports of marijuana being used successfully to treat PTSD symptoms.[39](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref39) This is somewhat surprising because substance abuse, including cannabis use disorder, is a common psychiatric comorbidity/complication in patients with PTSD, but now we have the situation where cannabis is being used for PTSD treatment.

A synthetic cannabinoid, nabilone, has been used lately for treating nightmares associated with severe PTSD. However, nabilone has been approved only for the treatment of cancer therapy-induced nausea.

An open-label clinical trial on using nabilone in 47 patients diagnosed with PTSD demonstrated that adjunctive use of nabilone (Cesamet™) resulted in a significant reduction in nightmare intensity and improvement in the quality of sleep.[40](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref40) In 2015, a group of Canadian military psychiatrists published a randomized placebo-controlled study on the efficacy of nabilone in the treatment of PTSD-associated nightmares. They found a significant reduction in nightmares in the population of military patients with PTSD. The major limitation of this study is the small sample size of only 10 patients.[41](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref41)

There have been no studies on the addictive potential of nabilone. There is only one study, published in 2010, saying that reports of nabilone abuse are rare, but the authors recommended a follow-up of patients, including assessment of tolerance and dependence.[42](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref42)

Nabilone is currently a commonly prescribed medication for nightmares associated with various clinical conditions, not only PTSD, and long-term use of nabilone is, unfortunately, not uncommon. There is a tendency for nabilone or even medicinal cannabis to be prescribed for patients with anxiety, insomnia, and depression, without research evidence of therapeutic effects and with unknown long-term consequences.[37](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref37)

Since cannabis induces a sense of euphoria, it has been reported that many people begin cannabis use during depressive episodes.[36](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref36) However, it would be a pitfall for clinicians to prescribe medicinal cannabis for patients with depression as a transitory experience of euphoria is not a therapeutic effect.

**Discussion**

Benzodiazepines, ketamine, stimulant medications, and cannabinoids have some common characteristics which are elaborated on in this paper. All of these agents may help patients with mood and anxiety disorder symptoms to a certain degree, but it is very important to make a distinction between agents that may make patients feel better and agents that are genuinely therapeutic.

These agents could alleviate patients’ suffering in the short run, but their longer use may have undesirable effects, including the possibility of developing tolerance and dependence, and the potential for toxicity with long-term administration.

We learned this lesson with benzodiazepines, but this knowledge does not prevent many physicians from prescribing or even overprescribing off-label treatment options such as stimulant medications and ketamine for patients with major depressive disorder, or synthetic cannabinoids and medicinal cannabis for patients with anxiety disorders. Benzodiazepines were uncritically overprescribed for decades as a main treatment option for anxiety disorders, while at present there is a strong recommendation in many countries to avoid prescribing benzodiazepines whenever possible.

Ketamine has rapid antidepressant effects, but these effects are short lived and usually last from only a few days to a maximum of 7 days.[5](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref5) Therefore, if clinicians are aiming to treat major depressive disorder with ketamine, regular use is needed to achieve a therapeutic effect. However, prescribing oral or intranasal formulations of ketamine, a well-known recreational drug, can easily foster an addiction to prescription medication. Ethical dilemmas about prescribing benzodiazepines and ketamine are very similar with regard to their potential for the development of dependence.

Generations of medical students and psychiatrists were taught about the detrimental effects of cannabis on mood disorders and anxiety disorders, but we are now witnessing the process of the gradual introduction of medicinal marijuana in treating various medical conditions and, possibly, in treating psychiatric disorders. Is this due to a change in the psychiatric paradigm or temporary confusion in psychiatric practice influenced by the lack of new and effective evidence-based treatments in psychiatry?[43](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref43)

Stimulant augmentation in the treatment of major depressive disorder is a third-line (level 3 of evidence) treatment option/augmentation strategy in the Canadian guidelines for mood and anxiety disorders[19](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref19) for cases of very refractory depression. Stimulant augmentation is supposed to help patients with residual symptoms to reach remission. However, patients may carry on receiving continuously stimulant medications for a few years, or usually longer, in spite of gaining only some partial benefits from this add-on treatment.[21](https://www.dovepress.com/addictive-potential-of-novel-treatments-for-refractory-depression-and--peer-reviewed-fulltext-article-NDT#ref21) Augmentation with stimulant medications should not be a long-term treatment option.

Clinicians should refrain from prescribing all of these off-label treatment options in regular clinic practice until more research evidence on their efficacy and safety is available. The risk of dependence with regular use of oral or intranasal ketamine, long-term use of stimulant medication, and synthetic cannabinoids and medicinal cannabis is significant. Psychiatrists should recall negative previous experiences with benzodiazepines to avoid potential pitfalls in clinical practice using new medications with similar properties.