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Adult ADHD: Prevalence, Comorbidities, and Patient-Reported Dysfunction in a Tertiary Mental Health Clinic

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Abstract

Background: To investigate patient-reported dysfunction associated with ADHD and three common comorbidities—major depressive disorder (MDD), generalized anxiety disorder (GAD), and bipolar disorder (BPD)—as well as their prevalence.
Methods: Dysfunction was measured using the Sheehan Disability Scale (SDS). Diagnosis was attained by a certified psychiatrist using DSM-5 criteria.
Results: In all, 46.7% of our sample (432/925) was diagnosed with ADHD. The most common comorbidities, in ascending order, were MDD (21.99%), BPD (24.77%), and GAD (31.48%). Sorted by ascending SDS score, the most functionally debilitating diagnoses in our sample were GAD < ADHD < ADHD + GAD < BPD < ADHD + BPD < MDD < ADHD + MDD.
Conclusions: Our sample of adults with ADHD, GAD, BPD, and particularly MDD report significant daily dysfunction. Comorbid ADHD is associated with increased dysfunction, particularly with GAD. The prevalence of adult ADHD in our study was higher than in previous studies.

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INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a heritable, neurodevelopmental disorder marked by difficulties in executive function, impulse control, and emotional dysregulation that can result in distractibility, inattention, impulsivity, and hyperactivity [1, 2]. Though typically regarded as a childhood-onset disorder, ADHD persists into adulthood in approximately half of cases [3, 4] and there is evidence for heterogeneity in the etiology of adult ADHD, a disorder that may be masked in one's childhood by protective factors or mistaken for laziness and rebelliousness [5, 6], or which may be, as some researchers have suggested, neurologically distinct from childhood ADHD [4, 7].

Despite a high symptom burden, adult ADHD is widely under-recognized and under-treated [8, 9]. This shortcoming may be due to a lack of training and familiarity among practitioners with the typical onset and course of adult ADHD, as well as difficulty in distinguishing ADHD from other mental health conditions, particularly borderline personality disorder and bipolar disorder [1, 10]. ADHD can have lasting socioeconomic and health effects that often persist even after symptoms have remitted, which may be attributable, at least in part, to the cumulative effects of social isolation and the development of ADHD-related deficits in self-efficacy and self-esteem [5, 7]. The negative outcomes associated with ADHD are diverse, including physical health problems such as higher rates of obesity, smoking, asthma, migraines, emergency room visits, and injury-related insurance claims and accidental death [7, 11–13]. ADHD is also correlated with lower academic performance, including lower grades and higher rates of academic probation and drop-out [2, 14], and ADHD-associated impulsivity and diminished in-job engagement may precipitate financial problems stemming from higher rates of unemployment and debt, fewer saving behaviours, and lower credit ratings [4, 15, 16].

ADHD is also linked to poorer mental health outcomes, including higher rates of depression, anxiety, and suicidality [17, 18], as well as substance use and addictions, including cannabis, alcohol, illicit drugs, gambling, and problematic technology use [19–21]. Additionally, ADHD is

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associated with poorer psychosocial outcomes, including life satisfaction and subjective wellbeing, psychological distress, social isolation, and difficulties maintaining healthy relationships [4, 22, 23]. All of these outcomes may be linked to the internalizing effects of stigma and perceived health discrimination [6, 10, 24]. Compounding these stressors and risks, ADHD is also associated with insomnia and sleep disorders, nightmares, fatigue, and exhaustion [25–27]. As well as personal ramifications for individuals and their families, work-related impairments and increased healthcare costs present a substantial economic burden on society [1, 18]. The prevalence of adult ADHD in the general population has been estimated at 2.5-5.4% [4, 28–30] and is typically more frequent in high-income than low-income countries [29]. However, the tendency toward underdiagnosis means the overall prevalence could be higher, particularly when using DSM-IV criteria [5, 6, 10].

To date there are few studies of daily functioning in adults with ADHD, with and without comorbidities. Accordingly, the primary aim of this study was to measure and compare self-reported dysfunction in patients diagnosed with pure (mutually exclusive) ADHD, major depressive disorder (MDD), generalized anxiety disorder (GAD), bipolar disorder (BPD), or alcohol use disorder (AUD), and those with MDD, GAD, BPD, or AUD and comorbid ADHD. Our secondary aim was to ascertain the prevalence of ADHD and its comorbidities in a sample of nonpsychotic psychiatric outpatients. A small number of AUD diagnoses precluded a full analysis of AUD dysfunction; as a result we did not generate hypotheses for AUD or comorbid ADHD-AUD dysfunction.

Our hypotheses regarding patient-reported dysfunction in ADHD and non-ADHD patients with and without comorbidities were informed by neurocognitive hypotheses of anxiety, depression, bipolar, and ADHD, according to which deficits in executive function and biases in emotional processing specific to MDD, and to a lesser extent BPD, are more likely to elevate self-reported dysfunction in those patients compared to those with GAD and ADHD [31–34]. Our hypotheses were as follows:

- Patient-reported dysfunction in pure (mutually exclusive) cases would fall into the following quantitative hierarchy: MDD > BPD > GAD > ADHD.

- The addition of comorbid ADHD in patients with MDD would not significantly increase dysfunction scores. We anticipated that the additive effects on dysfunction introduced by comorbid ADHD would be most pronounced in GAD patients, followed by BPD, and least of all in MDD patients.

METHODS

In this single-centre, retrospective, naturalistic study approved by University of Alberta Research Board, we investigated the prevalence of ADHD, its comorbidities, and associated patient-reported dysfunction at an interdisciplinary, referral-based, tertiary care centre in a large city in western Canada. From June 2016 to October 2018, new patients were invited to participate in the study. Participants provided written informed consent before completing the Sheehan Disability Scale (SDS) [35], a short, simple, easy-to-score instrument that assesses functional impairment on a 10-point scale—from 0 (not at all) to 10 (extremely)—in the domains of *work/school, social life,* and *family life*. To our knowledge, while the SDS has been psychometrically validated for use with adults with ADHD [36], it has not been used elsewhere to measure functional impairment in adults with ADHD outside of Morstedt, Corbisiero, Bitto, and Stieglitz's study of impairment due to emotional symptoms [37]. Upon completion of the SDS, patients were seen by one of our centre's certified psychiatrists, who was blinded to the results of the SDS, for face-to-face assessment, diagnosis, and treatment as usual. For this study we logged patients who received a DSM-5 [38] diagnosis, not in remission, of one or more of five common mental disorders observed at our clinic—MDD, GAD, BPD, AUD, and ADHD—as well as their SDS scores (total and work/school, social life, and family life subscales). While there is emerging evidence that adult ADHD may be neurologically distinct from childhood ADHD [4, 7], for our diagnostic purposes in this study, we used established DSM-5 criteria that include adult experiences and require fewer symptoms after age 17 but that nevertheless require the presence of symptoms prior to the age of 12 [38].

Statistical Analysis

Statistical analyses were conducted using the IBM SPSS Statistics for Macintosh, Version 25.0. To test our hypotheses of differential dysfunction levels among pure and comorbid diagnoses, we used factorial ANOVA followed by two-tailed between-group *t* tests. Thirty participants who had missing answers in SDS subscales were excluded from analyses using SDS total score; SDS subscales were analyzed using the full sample. Nine participants who identified as "other" gender were also excluded from factorial ANOVA analysis to prevent confounds in gender analysis (n = 913).

RESULTS

In total, 955 patients participated in our study. Of those who provided a total SDS score from all three subscales (n = 925), 46.70% (n=432) were diagnosed with ADHD by one of our clinic's psychiatrist using DSM-5 criteria. To test our hypotheses, we first conducted a full factorial ANOVA using SDS total score as a dependent variable, and age, gender, and diagnostic label (e.g., MDD), including all pairwise comorbid diagnoses (e.g., ADHD comorbid with MDD) and two or more comorbid diagnoses (e.g., ADHD comorbid with MDD and GAD), as independent variables. We found a null effect of age and gender and their associated interaction effects (see Table 1), so we followed up with a

series of two-tailed independent sample *t*-tests to directly investigate our hypotheses. Our results generally confirm our first hypothesis—MDD > BPD > GAD > ADHD—with the exception that we did not find a statistically significant difference between GAD and ADHD (see Table 2).

Table 1. Summary of factorial ANOVA results.

	df	$F_{(df, 913)}$	p	$\eta_{partial}^2$
Intercept	1	238.52	0.000**	0.38
Diagnosis Label	11	6.01	0.000**	0.15
Age	55	1.23	0.139	0.03
Gender	4	1.59	0.177	0.15
Diagnosis Label x Age	290	0.95	0.673	0.09
Diagnosis Label x Gender	12	0.46	0.936	0.43
Age x Gender	47	0.84	0.759	0.01
Diagnosis Label x Age x Gender	100	0.91	0.709	0.19

Mutually exclusive diagnosis labels used in ANOVA model include the following: ADHD only, BPD only, GAD only, MDD only, AUD only, ADHD and BPD, ADHD and GAD, ADHD and MDD, ADHD and AUD, ADHD with 2 and more comorbidity, two and more comorbidity but no ADHD, and No diagnosed disorders.

Table 2. Between sample t-test summary

Diagnosis 1	Diagnosis 2	t	*df*	*p*
MDD	BPD	2.56	79.72	.013*
BPD	GAD	2.57	98.45	.012*
GAD	ADHD	-.86	119.47	.393
BPD	ADHD	2.19	76.06	.031*

*denotes statistical significance ($p<0.05$)

Our AUD sample size was too small for an analysis of dysfunction. The mean total dysfunction, out of 30, as measured by the SDS for ADHD-only patients (n=163) was 15.85 (Standard Deviation [SD]=7.83), for GAD-only (n=64) was 14.89 (SD=7.53), for BPD-only (n=50) was 18.82 (SD=8.53) , and for MDD-only (n=111) was 22.33 (SD=6.97) (see Figure 1). Mean total dysfunction for comorbid ADHD and GAD-only (only one comorbidity) (n=55) was 18.11 (SD=7.25), comorbid ADHD and BPD-only (n=69) was 20.39 (SD=7.44), and comorbid ADHD and MDD-only (n=36) was 23.36 (SD=4.66) (see also Figure 2).

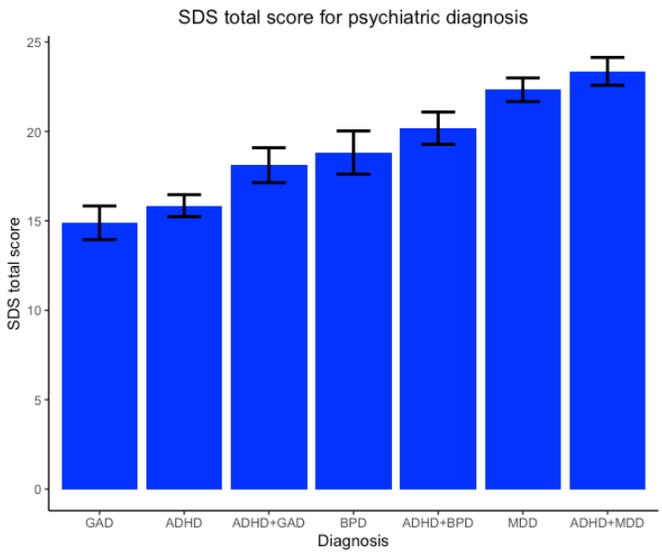

Figure 1. SDS total score as a function of psychiatric diagnosis, sorted by ascending SDS score.

Table 3 contains subscale scores by diagnostic category. While the relative magnitudes of the subscales generally reflect those of the total scores, it may be worth noting that all diagnoses that included ADHD had the highest patient-reported dysfunction in the domain of Work/School.

Table 3. Descriptive statistics of SUS subscales

Diagnosis	Social Life			Work and School			Family Life		
	N	Mean	SD	N	Mean	SD	N	Mean	SD
Single									
GAD	69	5.42	3.08	64	5.08	3.28	69	4.55	2.74
ADHD	166	4.73	3.13	163	5.90	3.02	166	5.22	2.96
BPD	51	6.35	3.06	50	6.36	3.20	51	6.25	3.17
MDD	115	7.59	2.63	111	7.49	2.72	115	7.33	2.53
Comorbid									
ADHD and GAD	57	5.82	2.92	55	6.76	2.45	57	5.63	2.87
ADHD and BPD	71	6.46	2.60	69	7.00	2.90	70	6.69	2.88
ADHD and MDD	39	7.46	2.42	37	8.14	2.02	39	7.69	2.20

SD stands for standard deviation

Of the 432 ADHD-diagnosed patients, 37.73% (n=163) had no comorbidities, 21.99% (n=95) had comorbid MDD, 31.48% (n=136) had comorbid GAD, 24.77% (n=107) had comorbid BPD, and 5.79% (n=25) had comorbid AUD (see Table 4).

Table 4. Comorbidity of psychiatric illness among ADHD patients. Due to some patients having multiple comorbidities, total proportion will not equal 100%.

Diagnosis	Frequency	Proportion (%)
ADHD only	163	37.73
Comorbid with MDD	95	21.99
Comorbid with GAD	136	31.48
Comorbid with BPD	107	24.77
Comorbid with AUD	25	5.79

DISCUSSION

This retrospective, naturalistic study examined patient-reported dysfunction using the Sheehan Disability Scale among individuals with pure (non-comorbid) ADHD, MDD, GAD, or BPD, as well as those with ADHD and comorbid conditions. Consistent with our first hypothesis, non-comorbid patient-reported dysfunction was highest for MDD. We attribute these findings to hippocampal-related executive function, memory, attention, concentration, and motivation deficits common to MDD contributing to actual daily dysfunction, as well as the potential for habitual negative thinking and rumination associated with a hyperactive limbic system [32–34], which could lead to recall bias and inflated reports of dysfunction. Self-reported dysfunction in BPD patients was also relatively high, which makes sense in light of associated neurocognitive deficits around executive function and verbal memory [39, 40]. However, also consistent with our first hypothesis, BPD-associated patient-reported dysfunction was lower than that for MDD; we attribute these findings to the cyclical nature of bipolar disorder, its cognitive heterogeneity, the potential for recent or current hypomanic episodes to ameliorate dysfunction and/or mitigate appraisals of dysfunction, and the tendency for bipolar patients to overestimate their own cognitive performance [31, 41].

Also consistent with our first hypothesis, self-reported dysfunction in pure ADHD and GAD patients was significantly lower than MDD and BPD patients. Compared to MDD and BPD, GAD and ADHD are typically early-onset disorders [3, 4, 32], which we anticipated could lead to inoculation effects whereby dysfunction becomes normalized and less salient to the patient during recall. Moreover, compared to studies of MDD and BPD, the literature on neurocognitive deficits associated with anxiety is less conclusive, with Leonard and Abramovitch [42] finding that elevated primary anxiety may not impact cognitive functioning in the absence of threat or substantial cognitive load. As noted above, ADHD is associated with marked cognitive deficits that may impair daily functioning; we nevertheless hypothesized that self-reported dysfunction in pure ADHD patients would be lower than those with GAD only, due to the particularly inoculating effects of childhood onset and the unlikelihood of mood symptoms and emotional processing biases artificially inflating self-report at the moment of data collection, as may be common with MDD, BPD, and GAD patients. Our findings, which show comparatively low dysfunction scores for pure ADHD patients, are consistent with studies that have found ADHD symptomatology is not always and only debilitating. Some adults with ADHD do function well, thanks to adaptive and compensatory skills and traits such as high intelligence, finding work that is well suited to their symptom profile, and the potential likeability of their chaotic and unpredictable personas, including the tendency for some to divert their attention from individual tasks toward helping behaviours [1, 10, 16]. Contrary to our hypothesis, we did not find a statistically significant difference in patient-reported dysfunction between pure ADHD and GAD patients, but the difference in dysfunction between GAD/ADHD and BPD/MDD was substantive.

In line with our second hypothesis, the compounding effect of a comorbid ADHD diagnosis on dysfunction in patients with MDD and BPD was relatively small compared to those with GAD. We attribute this to a ceiling effect whereby pure MDD and BPD dysfunction scores approach the peak of the numeric scale (total score of 30, or 10 on each subscale) and therefore the additive effects of typically early-onset symptoms of ADHD—which, in line with neurocognitive hypotheses of dysfunction, may also be less likely to emotionally bias recall—might not significantly register. Patients with anxiety, on the other hand, whose self-rated dysfunction is relatively moderate, have more room to experience and report additional dysfunction due to the additive effects of ADHD symptoms. We expect a ceiling effect would have tempered our results using other disability scales as well; the World Health Organization Disability Assessment Schedule (WHODAS 2.0), which has demonstrated internal consistency and reliability, nevertheless offers less granularity than the SDS with its five-point Likert scale [43].

Moreover, the symptom profiles of GAD and ADHD are complementary, and we might expect them to act synergistically to increase dysfunction in comorbid patients. For instance, some patients with both ADHD and GAD may experience enhanced anxiety due to their self-perceived inability to control their impulses and refrain from engaging in risky or socially unacceptable behaviour. While there is evolutionary advantage to approach and avoidance tendencies working in equilibrium to keep behaviours in check, it seems reasonable that an excess of both may create a short circuit of distress and dysfunction. Additionally, as Michelini, Eley, Gregory, and McAdams [44], ADHD may precipitate attention biases (worry, rumination) toward threat stimuli and thereby reduce the ability to attend to everyday activities in already-anxious individuals.

While lower than that of MDD and BPD, the level of dysfunction associated with ADHD among patients at our clinic, as measured using the SDS, still represents significant

impairment in daily life. These findings are consistent with the literature, synthesized above, that suggests ADHD symptoms can make daily tasks difficult. In addition, dysfunction may not tell the whole story. Individuals with ADHD who appear or claim to be functioning at a high level may be chronically struggling to cope, particularly with the exhaustion from lack of sleep and the effort required to focus their thoughts [1, 5]. Moreover, while the early onset of ADHD may inoculate individuals against self-perceived functional impairments due to ADHD symptoms, the chronic course of these symptoms over many years may nevertheless lead to low self-esteem and self-confidence, subjective distress, and substance misuse [10, 20]. Plus, while ADHD symptom severity typically declines with age, adjustment difficulties often persist, even after remission [4, 7].

At 46.7%, the overall prevalence of ADHD in our non-psychotic clinical outpatients sample was higher than that found in similar studies (14-22% [6, 45–48]). These elevated rates may be partly attributable to the local reputation of our clinic's psychiatrists as experts in assessing and treating ADHD and the concomitantly high referral rate from general practitioners in the area seeking assessment for suspected ADHD. These high rates may also reflect an enhanced recognition of and sensitivity to adult ADHD using DSM-5 criteria [5, 6, 10]. Overall, 60.9% of ADHD patients had at least one comorbid condition, which is comparable to the 65-89% found in previous studies [6, 49, 50], particularly considering that other potential comorbidities, such as personality disorders, were not included in our analysis. At 31.48%, GAD was the most common comorbidity, followed by BPD (24.77%), MDD (21.99%), and AUD (5.79%). Our prevalence of comorbid GAD is comparable to other studies in the literature (23-45% [6, 49, 51–54]), while BPD is significantly higher (8-14% [6, 49, 51]) and MDD significantly lower (40-53% [6, 51, 52, 54]). Substance use disorder is difficult to compare, as we only measured for alcohol use.

This study has several limitations. While a naturalistic study is advantageous in its ability to capture dysfunction in the real world without stringent exclusion criteria, the main limitation is the lack of a healthy control group with which to conduct a full factorial analysis. We were also unable to control for confounding variables such as medication use, and our prevalence measures may have been biased by the potential for inflated referral rates from general practitioners with a knowledge of our clinic's specialization in ADHD. This study was also cross-sectional and correlational, and thus temporal factors such as the age of onset of ADHD and its comorbidities could not be accounted for, precluding causal inferences. Additionally, we did not differentiate between subtypes of ADHD and BPD, and this collapsing of subtypes limited the depth of our analysis. As the clinic in which this study was conducted is a non-urgent, tertiary centre, bipolar patients seen at this clinic tend more often to exhibit BPD type-II than type-I symptoms; we would expect BPD-II patients' self-reports to moderate SDS scores, as BPD-II is associated with less severe cognitive deficits compared to BPD-I [31]. We also did not log other, less common comorbidities, such as personality disorders. Finally, while this was accounted for in our hypotheses, self-report may be unreliable. Despite these limitations, to our knowledge this is one of the first studies to use a psychometrically validated scale to measure patient-reported dysfunction in non-psychotic clinical outpatients and to compare dysfunction between pure and comorbid conditions.

CONCLUSION

Canadian adults with one of ADHD, GAD, BPD, or MDD report significant illness-related dysfunction in their daily lives, and this dysfunction is especially pronounced in patients with MDD. Comorbid MDD, GAD, and BPD is common alongside ADHD, and while the presence of ADHD exacerbates dysfunction for those with MDD, GAD, or BPD, these compounding effects appear to be most significant in those with GAD and least pronounced in those with MDD. These disparities may, in part, be attributable to ceiling effects in self-report and the inoculating effects of early-onset ADHD and GAD. With an increased awareness of, and enhanced ability to detect, adult ADHD using DSM-5 criteria, the prevalence of diagnosed adult ADHD may be on the rise.

REFERENCE

[1] Kooij J, Bijlenga D, Salerno L, Jaeschke R, Bitter I, Balazs J, et al. Updated European Consensus Statement on diagnosis and treatment of adult ADHD. European Psychiatry. 2019;56:14–34.

[2] Pinna M, Visioli C, Rago CM, Manchia M, Tondo L, Baldessarini RJ. Attention deficit-hyperactivity disorder adult bipolar patients. Journal of Affective Disorders. 2019;243:391–396.

[3] March P, Cabrera G. Substance abuse and attention deficit hyperactivity disorder (ADHD). Glendale, CA: CINAHL Information Systems; 2018.

[4] Moffitt TE, Houts R, Asherson P, Belsky DW, Corcoran DL, Hammerle M, et al. Is adult ADHD a childhood-onset neurodevelopmental disorder? Evidence from a four-decade longitudinal cohort study. American Journal of Psychiatry. 2015;172:967–977.

[5] Asherson P, Akehurst R, Kooij JS, Huss M, Beusterien K, Sasané R, et al. Under diagnosis of adult ADHD: cultural influences and societal burden. Journal of Attention Disorders. 2012;16:20S–38S.

[6] Belirgan S, Ersoy MA, Ersoy HT. Prevalence of adult attention deficit hyperactivity disorder and comorbid axis-I disorders among first time applied cases of a general psychiatry outpatient clinic and a private psychotherapy centre. Psychiatry and Clinical Psychopharmacology. 2018;28:25–35.

[7] Agnew-Blais JC, Polanczyk GV, Danese A, Wertz J, Moffitt TE, Arseneault L. Evaluation of the persistence, remission, and emergence of attention-deficit/

hyperactivity disorder in young adulthood. JAMA Psychiatry. 2016;73:713–720.

[8] Adamis D, Graffeo I, Kumar R, Meagher D, O'Neill D, Mulligan O, et al. Screening for attention deficit-hyperactivity disorder (ADHD) symptomatology in adult mental health clinics. Irish Journal of Psychological Medicine. 2018;35:193–201.

[9] Adler LA, Faraone SV, Sarocco P, Atkins N, Khachatryan A. Establishing US norms for the Adult ADHD Self-Report Scale (ASRS-v1.1) and characterising symptom burden among adults with self-reported ADHD. International Journal of Clinical Practice. 2019;73:e13260.

[10] Watters C, Adamis D, McNicholas F, Gavin B. The impact of attention deficit hyperactivity disorder (ADHD) in adulthood: A qualitative study. Irish Journal of Psychological Medicine. 2018;35:173–179.

[11] Dalsgaard S, Ostergaar SD, Leckman JF, Mortensen PB, Pedersen MG. Mortality in children, adolescents, and adults with attention deficit hyperactivity disorder: A nationwide cohort study. The Lancet. 2015;385:2190–2196.

[12] Lundervold AJ, Halleland HB, Brevik EJ, Haavik J, Sorensen L. Verbal memory function in intellectually well-functioning adults with ADHD: Relations to working memory and response inhibition. Journal of Attention Disorders. 2019;23:1188–1198.

[13] Sadeghi-Bazargani H, Hasanzadeh K, Salarilak S, Amiri S, Golestani M, Shahedifar N. Evaluating the relationship between adult attention-deficit/hyperactivity disorder and riding behavior of motorcyclists. Injury & Violence. 2019;11:45–52.

[14] Fleming AP, McMahon RJ. Developmental context and treatment principles for ADHD among college students. Clinical Child and Family Psychology Review. 2012;15:303–329.

[15] Das D, Cherbuin N, Butterworth P, Anstey KJ, Easteal S. A population-based study of attention deficit/hyperactivity disorder symptoms and associated impairment in middle-aged adults. PLoS One. 2012;7.

[16] Halbesleben JRB, Wheeler AR, Shanine KK. The moderating role of attention-deficit/hyperactivity disorder in the work engagement-performance process. Journal of Occupational Health Psychology. 2013;18:132–143.

[17] Conejero I, Jaussent I, Lopez R, Guillaume S, Olie E, Hebbache C, et al. Association of symptoms of attention deficit-hyperactivity disorder and impulsive-aggression with severity of suicidal behavior in adult attempters. Scientific Reports. 2019;9.

[18] Gjervan B, Torgersen T, Nordahl HM, Rasmussen K. Functional impairment and occupational outcomes in adults with ADHD. Journal of Attention Disorders. 2012; 16:1177/108.705.4711413074.

[19] Cairncross M, Milosevic A, Struble CA, Ellis JD, Ledgerwood DM. Clinical and personality characteristics of problem and pathological gamblers with and without symptoms of adult ADHD. The Journal of Nervous and Mental Disease. 2019;207:246–254.

[20] Kumar SV, Chate SS, Patil NM, Tekalaki BV, Patil S. Prevalence of undiagnosed and untreated attention deficit hyperactivity disorder in men with alcohol dependence: A case-control study. Archives of Psychiatry and Psychotherapy. 2018;1:26–32.

[21] Marmet S, Studer J, Grazioli VS, Gmel G. Bidirectional associations between self-reported gaming disorder and adult attention deficit hyperactivity disorder: Evidence from a sample of young Swiss men. Frontiers in Psychiatry. 2018;9.

[22] Miranda A, Berenguer C, Colomer C, Rosello R. Influence of the symptoms of attention deficit hyperactivity disorder (ADHD) and comorbid disorders on functioning in adulthood. Psicothema. 2014;26:471–476.

[23] Pond E, Fowler K, Hesson J. The influence of socioeconomic status on psychological distress in Canadian adults with ADD/ADHD. Journal of Attention Disorders. 2019;23:1177/108.705.4716653214.

[24] Stickley A, Leinsalu M, Ruchkin V, Oh H, Narita Z, Koyanagi A. Attention-deficit/hyperactivity disorder symptoms and perceived mental health discrimination in adults in the general population. European Psychiatry. 2019;56:91–96.

[25] Instanes JT, Klungsoyr K, Halmoy A, Fasmer OB, Haavik J. Adult ADHD and comorbid somatic disease: A systematic literature review. Journal of Attention Disorders. 2018;2 2:1177/108.705.4716669589.

[26] Schredl M, Bumb JM, Alm B, Sobanski E. European Archives of Psychiatry and Clinical Neuroscience. 267; 2017.

[27] Wynchank D, Bijlenga D, Beekman AT, Kooij JJS, Penninx BW. Adult attention-deficit/hyperactivity disorder (ADHD) and insomnia: An update of the literature. Current Psychiatry Reports. 2017;19.

[28] De Graaf R, Kessler RC, Fayyad J, ten Have M, Alonso J, Angermeyer M, et al. The prevalence and effects of adult attention-deficit/hyperactivity disorder (ADHD) on the performance of workers: results from the WHO World Mental Health Survey Initiative. Occupational and Environmental Medicine. 2008;65:835–842.

[29] Fayyad J, De Graaf R, Kessler R, Alonso J, Angermeyer M, Demyttenaere K, et al. Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder. The British Journal of Psychiatry. 2007;190:402–409.

[30] Simon V, Czobor P, Bálint S, Mészáros A, Bitter I. Prevalence and correlates of adult attention-deficit hyperactivity disorder: meta-analysis. The British Journal of Psychiatry. 2009;194:204–211.

[31] Bora E. Neurocognitive features in clinical subgroups of bipolar disorder: A meta-analysis. Journal of Affective Disorders. 2018;229:125–134.

[32] Sandi C, Richter-Levin G. From high anxiety trait to depression: A neurocognitive hypothesis. Trends in Neurosciences. 2009;32:312–320.

[33] Vakalopoulos C. Neurocognitive deficits in major depression and a new theory of ADHD: a model of impaired antagonism of cholinergic-mediated prepotent

behaviours in monoamine depleted individuals. Medical Hypotheses. 2007;68:210–221.

[34] Warren MB, Pringle A, Harmer CJ. A neurocognitive model for understanding treatment action in depression. Philosophical Transactions of the Royal Society. 2015;370.

[35] Leon AC, Olfson M, Portera L, Farber L, Sheehan DV. Assessing psychiatric impairment in primary care with the Sheehan Disability Scale. International Journal of Psychiatry in Medicine. 1997;27:93–105.

[36] Coles T, Coon C, DeMuro C, McLeod L, Gnanasakthy A. Psychometric evaluation of the Sheehan Disability Scale in adult patients with attention-deficit/hyperactivity disorder. Neuropsychiatric Disease and Treatment. 2014;10:887–895.

[37] Morstedt B, Corbisiero S, Bitto H, Stieglitz R. Emotional symptoms and their contribution to functional impairment in adults with attention-deficit/hyperactivity disorder. Attention Deficit Hyperactivity Disorders. 2016;8:21–33.

[38] Association AP, others. Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub; 2013.

[39] Kiran DR, Prassana NK, Latha AS, Reddy CG. Comparative study of neurocognitive deficits in bipolar disorder and schizophrenia. International Archives of Integrated Medicine. 2019;6:291–299.

[40] Tournikioti K, Ferentinos P, Michopoulos I, Dikeos D, Soldatos CR, Douzenis A. Sex-related variation of neurocognitive functioning in bipolar disorder: Focus on visual memory and associative learning. Psychiatry Research. 2018;267:499–505.

[41] Camp V, L. S, C BG, Oldenburg JFE. Metacognitive functioning in bipolar disorder versus controls and its correlations with neurocognitive functioning in a cross-sectional design. Comprehensive Psychiatry. 2019;92:7–12.

[42] Leonard K, Abramovitch A. Cognitive functions in young adults with generalized anxiety disorder. European Psychiatry. 2019;56:1–7.

[43] Saltychev M, Katajapuu N, Bärlund E, Laimi K. Psychometric properties of 12-item self-administered World Health Organization disability assessment schedule 2.0 (WHODAS 2.0) among general population and people with non-acute physical causes of disability–systematic review. Disability and Rehabilitation. 2019:1–6.

[44] Michelini G, Eley TC, Gregory AM, McAdams TA. Aetiological overlap between anxiety and attention deficit hyperactivity symptom dimensions in adolescence. Journal of Child Psychology and Psychiatry. 2015;56:423–431.

[45] Montes A, G L, Garcia H, O A, Ricardo-Garcell J. ADHD prevalence in adult outpatients with nonpsychotic psychiatric illnesses. Journal of Attention Disorders. 200 7;11:1177/108.705.4707304428.

[46] Deberdt W, Thome J, Lebrec J, Kraemer S, Fregenal I, Ramos-Quiroga JA, et al. Prevalence of ADHD in nonpsychotic adult psychiatric care (ADPSYC): A multinational cross-sectional study in Europe. BMC Psychiatry. 2015;15.

[47] Nylander L, Holmqvist M, Gustafson L, Gillberg C. ADHD in adult psychiatry: Minimum rates and clinical presentation in general psychiatry outpatients. Nordic Journal of Psychiatry. 2009;63:1080/080.394.808024163 23.

[48] Rao P, Place M. Prevalence of ADHD in four general adult outpatient clinics in North East England. Progress in Neurology and Psychiatry. 2011;15:7–11.

[49] Pineiro-Dieguez B, Balanza-Martinez V, Garcia-Garcia P, Soler-Lopez B. & the CAT Study Group. Psychiatric Comorbidity at the Time of Diagnosis in Adults with ADHD: The CAT Study Journal of Attention Disorders. 20 16;20:1177/108.705.4713518240.

[50] Sobanski E. Psychiatric comorbidity in adults with attention-deficit/hyperactivity disorder. European Archives of Psychiatry and Clinical Neuroscience, 256(suppl1), I/26-I/31. doi:10; 2006.

[51] Chen Q, Hartman CA, Haavik J, Harro J, Klungsøyr K, Hegvik T-A, et al. Common psychiatric and metabolic comorbidity of adult attention-deficit/hyperactivity disorder: A population-based cross-sectional study. PLoS One. 2018;13.

[52] Michielsen M, Comijs HC, Semeijn EJ, Beekman ATF, Deeg DJH, Kooij SJJ. The comorbidity of anxiety and depressive symptoms in older adults with attention-deficit/hyperactivity disorder: A longitudinal study. Journal of Affective Disorders. 2013;148:220–227.

[53] Schatz DB, Rostain AL. ADHD with comorbid anxiety: A review of the current literature. Journal of Attention Disorders. 2006;10:1177/108.705.4706286698.

[54] Van Ameringen M, Mancini C, Simpson W, Patterson B. Adult attention deficit hyperactivity disorder in an anxiety disorders population. CNS Neuroscience & Therapeutics. 2011;17:221–226.