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Co-Occurring Psychiatric Disorders in Young People with Eating Disorders: An Multi-State and Real-Time Analysis of Real-World Administrative Data

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Abstract

OBJECTIVE: We aimed to use real-world data to characterize the burden of psychiatric comorbidities in young people with eating disorders (EDs) relative to peers without EDs.

METHOD: This retrospective cohort study used a large federated multi-national network of real-time electronic health records. Our cohort consisted of 124,575 people (14,524 people receiving their index, first-ever, ED diagnosis, compared to 110,051 peers without EDs initiating antidepressants). After 1:1 propensity score matching of the two cohorts by pre-existing demographic and clinical characteristics, we used multivariable logistic regression to compute the adjusted odds ratio (aOR) of psychiatric diagnoses arising in the year following the index event (either first ED diagnosis or first antidepressant script).

RESULTS: Over 50% of people with EDs had prior psychiatric diagnoses in the year preceding the index EDs diagnosis, with mood disorders, generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), specific phobia (SP), attention-deficit hyperactivity disorder (ADHD), and autism spectrum disorder (ASD) being the most common. Adjusted analyses showed higher odds for mood disorders (aOR=1.20 [95% CI=1.14–1.26]), GAD (aOR=1.28 [1.21–1.35]), PTSD (aOR=1.29 [1.18–1.40]), and SP (aOR=1.45 [1.31–1.60]) in the EDs cohort compared to antidepressant-initiating peers without EDs, although rates of ADHD and ASD were similar in both cohorts.

CONCLUSION: This large-scale real-time analysis of administrative data illustrates a high burden of co-occurring psychiatric disorders in people with EDs.

Keywords

Eating disorders; co-occurring disorders; big data; real world data

Introduction

Eating disorders (EDs: anorexia nervosa, bulimia nervosa, binge eating disorder, and other related disorders) are life-threatening mental and physical health conditions, impacting over 55 million people worldwide and accounting for over 6 million disability adjusted life years in 2019 [1]. The total economic burden of EDs ranges from £6–8 billion/year in the United Kingdom (UK) [2] to more than $60 billion from 2018 to 2019 in the United States (US) [3]. Unfortunately, the prevalence of EDs is rising worldwide, including Australia, Western Europe, North America, and East Asia [4]. In the UK, the NHS England data revealed an 84% increase in hospitalizations for eating disorders from 2015 to 2021 [5]. In the US, emergency room visits and inpatient hospitalizations for EDs doubled since the onset of the COVID-19 pandemic[6], with similar findings observed in Canada[7].

Amid mounting acute admissions for pediatric mental health emergencies, US Surgeon General Vivek Murthy announced an urgent need for up-to-date surveillance data on pediatric mental health outcomes, with EDs highlighted as an area warranting urgent attention[8]. The need for real-time surveillance data is dire for EDs, as there is concern that poor ED outcomes are exacerbated by a fragmented and dysfunctional treatment system that may render psychiatric comorbidities un- or undertreated [9]. Concern has also been raised

about EDs treatment and clinical research being siloed and marginalized from mainstream psychiatry [10, 11].

Despite common sentiments that EDs are disconnected from other psychiatric concerns [9, 12], emerging evidence suggests that psychiatric comorbidities are common in patients with EDs. For instance, a recent rapid review of 202 studies related to eating disorders indicated that anxiety disorders, mood disorders, substance use, and PTSD were common psychiatric comorbidities [13], with 95% of people with an ED having a co-occurring affective disorder [11]. In an analysis of 2,400 female inpatients treated for eating disorders, 94% had a comorbid mood disorder, 56% had a comorbid anxiety disorder, and 22% had a comorbid substance use disorder (SUD) [14]. In another analysis of 2,155 people admitted to ED residential treatment, mood disorders occurred in over 75% of participants.[15] Finally, the presence of untreated psychiatric comorbidities may impact treatment and management, as undertreated mental health conditions area negative prognostic factors in people with EDs who are in recovery [16]. Previous studies showed that comorbid diagnoses can lead to increased severity of ED symptoms and poorer treatment outcomes [17–20].

Yet, to our knowledge, there is little multi-state data on the rates of diagnosed co-occurring psychiatric disorders in people with EDs. Amid calls for surveillance and large-scale population based studies [12], there are also few studies analyzing real-time and/or real-world estimates of psychiatric comorbidity in people with EDs, particularly in the post-COVID-19 era. Against this backdrop, the purpose of the present study is to use multi-state, real-time administrative data and compare a cohort of people with EDs to peers without EDs initiating antidepressants, a comparator cohort serving as a proxy for patients receiving routine mental health care who do not have eating disorders. We hypothesized an elevated likelihood of overall psychiatric comorbidities in people with EDs compared to other patients engaged in psychiatric treatment.

Methods

We retrieved data from the TriNetX Research Network (https://trinetx.com/, queried on January 18, 2024). TriNetX is a web-based database of de-identified electronic health records (EHRs) of more than 110 million patients from 84 large health care organizations across 50 US states and 4 countries with a diverse representation of race, ethnicity, age, income, and insurance types. Over 90% of the study's population is from the US; to protect patients' health data, the specific identities of other countries are not provided by TriNetX. TriNetX has previously been used to analyze health outcomes in populations of people underrepresented in the psychiatric clinical trials literature (i.e., children and young adults with externalizing disorders,[21] young people with eating disorders[22], people with gender dysphoria[23]) and its data are linked to ICD-10 codes, visit codes, current procedural terminology (CPT) codes, collection of standardized codes that represent medical procedures, supplies, products and service (HCPCS) codes, and medication codes validated by TriNetX LLC (Cambridge, MA, US) [24, 25]. TriNetX's statistical analyses are built into the analytic platform, and we cannot access detailed geographic locations, treatment settings, and socioeconomic data for individual patients due to privacy protections. The Carilion Clinic institutional review board determined that this study was non-human subjects

research given its use of deidentified secondary data. We followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) and Reporting of Studies Conducted using Observational Routinely Collected health Data statement for Pharmacoepidemiology reporting guideline (RECORD-PE)[26].

Participants

Our population comprised of individuals aged 5–26 years, with age 26 representing a commonly-used upper limit of young adulthood [27]. The primary predictor variable was the diagnosis of EDs, made between January 1, 2022 and December 31, 2022 (i.e., bulimia nervosa, anorexia nervosa, avoidant restrictive food intake disorder, binge eating disorder, eating disorder not otherwise specified), compared to peers without EDs who were receiving antidepressants during the same time period. EDs were ascertained via ICD-10-CM diagnostic codes, from all service settings (e.g. inpatient treatment, outpatient clinics and offices, emergency departments). We elected to combine all EDs into a single category due to concern for misclassification bias arising from diagnostic crossover of individual EDs (i.e., the high prevalence of people changing from AN to BN)[28–30]. We required at least 2 diagnoses for EDs for cohort entry (with no time limit between qualifying ED diagnoses), as past analyses showed that the validity of ICD codes are improved via multiple claims [31]. Our comparator (control) cohort consisted of individuals ages 5–26 years without a diagnosis of EDs who initiated antidepressants (serotonin-selective reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, monoamine oxidase inhibitors, or tricyclic antidepressants) between January 1, 2022 and December 31, 2022 in the TriNetX databases. The decision to select a comparator group of people without EDs who were initiating antidepressants was to address unobserved (and otherwise non-adjustable) confounding factors between people with and without EDs.

Variables

The primary outcome variable was the diagnosis for co-occurring psychiatric disorders within the 365 days ***after*** the "**index" event**, defined as the first ED diagnosis in the year of 2022 for the study group or the first antidepressant prescription in the year of 2022 in the comparator cohort (see eMethods for details). Co-occurring psychiatric disorders included mood disorders (such as major depressive disorder and bipolar disorder), substance use disorders, post-traumatic stress disorder (PTSD), generalized anxiety disorder (GAD), specific phobia (SP), attention-deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), gender dysphoria (GD). Although the DSM-5 classifies major depressive disorder and bipolar disorders separately, we combined the two disorders together under "mood disorders" to decrease misclassification bias [32], given that the misdiagnosis of bipolar disorders as other mood disorders, and vice versa, is common [33]. ICD-10-CM codes for the classification of co-occurring psychiatric disorders are included in the supplementary information.

Covariates were ascertained in the *baseline* period (365 days ***before*** the index event, defined as the first diagnosis of EDs, or first date of antidepressant receipt) and included demographics (age, gender, and race/ethnicity) and past history of co-occurring psychiatric disorders (mood disorders, GAD, ADHD, ASD, SUDs, personality disorders).

Statistical Analysis

First, we computed the descriptive statistics surrounding the prevalence of co-occurring psychiatric disorders in the 365 days ***before*** the index diagnosis of EDs, using chi-square tests to compare the baseline social and demographic characteristics and psychiatric comorbidity burden in people with EDs with peers initiating antidepressants without ED diagnoses.

Second, both cohorts (eating disorder and comparator cohort) were 1:1 propensity score-matched (logistic regression with the built-in Python scikit-learn package) via greedy nearest neighbor matching (with a caliper of 0.1 pooled standard deviations) by demographic characteristics (age, sex, race, and ethnicity) and baseline psychiatric comorbidities before the index date (mood disorders, GAD, SP, SUD, ASD, ADHD, personality disorders) in the 365 days preceding cohort entry (Table 1). Finally, we used logistic regression to estimate the adjusted odds of co-occurring psychiatric diagnoses in the 365 days **after** the index date, comparing the EDs cohort and the antidepressant-initiating cohort without EDs. Because EDs often have their onset after age 12, we conducted a secondary analysis limited to the subgroup of our full cohort (5–26 years) who were 12 years and older.

All statistical analyses were conducted on the TriNetX Analytics Platform.Tests were 2-tailed and results deemed statistically significant at $p < .05$, with the Benjamini-Hochberg correction used to adjust for multiple comparison with a false discovery rate set at 0.1.

Results

Table 1 illustrates baseline demographic information and co-occurring psychiatric disorders between the EDs and comparator group (people initiating antidepressants without ED diagnoses) in the 1 year before the index event both prior to and following propensity score matching. Before matching, our study population encompassed 124,575 people: 1) 14,524 people with EDs in the year of 2022 (2,802 male [19.3%]; mean [SD] age, 15.9 [4.6] years; 9,978 White [68.7%]) and 2) 110,051 people initiating antidepressants in control cohort without ED diagnoses (36,106 male [32.8%]; mean [SD] age, 17.8 [4.7] years; 71,849 White [65.3%]) group respectively.

In univariate comparisons, baseline psychiatric comorbidities preceding the index date were overall more common in people with EDs than peers initiating antidepressants without ED diagnoses. The most common disorders were mood disorders (n=7,435 [51.2%] in ED vs. n=24,840 [22.6%] in the comparator cohort), GAD (n=4,401 [30.3%] vs 8,608 [7.8%]), ADHD (n=2,503 [17.2%] vs 9,210 [8.4%]), PTSD (n=1,484 [10.2%] vs 2,399 [2.2%]), SP (n=1,217 [8.4%] vs 1,217 [1.1%]), ASD (n=1,208 [8.3%] vs 2,515 [2.3%]), and personality disorders (n=601 [4.1%] vs 540 [0.5%]), with $p<.001$ for all comparisons.

Next, we matched the EDs and comparator cohorts by baseline social, demographic, and psychiatric characteristics. The propensity score-matched cohorts included 13,707 patients in each group, with a standardized mean difference threshold of < 0.1 used to mark covariate balance between groups [34, 35] (eTable 1). Table 2 shows that, even after propensity score-matching, the EDs cohort was significantly more likely to receive

diagnoses for mood disorders (adjusted odds ratio [aOR]=1.20 [95% CI=1.14–1.26]), GAD (aOR=1.28 [1.21–1.35]), PTSD (aOR=1.29 [1.18–1.40]), personality disorders (aOR=1.76 [1.53–2.04]), and SP (aOR=1.45 [1.31–1.60]) than peers receiving antidepressants without ED diagnoses following the index event. Yet, rates of ADHD (aOR=1.04 [0.97–1.10]) and ASD (aOR=0.99 [0.91–1.08]) were similar in both cohorts. Subgroup analyses of the cohort (12–26 years) restricted to people 12 years and older did not differ from the parent analyses (eTable 2).

Discussion

To our knowledge, this is the first study using a real-world, multi-state administrative dataset to estimate the prevalence of psychiatric comorbidities in young people, ages 5–26, diagnosed with EDs, illustrating a high burden of psychiatric comorbidity in people with EDs. Although the rates of psychiatric comorbidity in people with EDs are not well-characterized using real-world administrative data, previous literature has consistently suggested that people with EDs suffer from a high burden of co-occurring mental health problems. For instance, a national epidemiologic survey on alcohol and related conditions with 36,000 adult participants found that three EDs (anorexia nervosa, bulimia nervosa, binge eating disorder) were significantly associated with alcohol and drug use disorders, mood disorders, anxiety disorders, and personality disorders [36]. In particular, our analysis shows that comorbid psychiatric diagnoses were more common in the year prior to diagnosis with an ED relative to a general psychiatry population receiving antidepressants. Among the most common diagnoses before the ED diagnosis were mood disorders (51.2%), such as major depressive disorder and bipolar disorder, GAD (30.3%), and ADHD (17.2%), followed by post-traumatic stress disorder (10.2%), specific phobia (8.4%), and autism spectrum disorders (8.3%). After we propensity score-matched people with EDs to antidepressant-receiving peers without ED diagnoses who had a similar degree of psychiatric comorbidity, we found that the EDs cohort was 20%, 28%, 29%, 76%, and 45% more likely to receive subsequent diagnoses for mood disorders, GAD, PTSD, personality disorders, and specific phobia in the year following their first ED diagnosis, respectively. Borderline personality disorder diagnoses, in particular, had the highest aOR, with patients with EDs being 90% more likely to receive this diagnosis than their non-EDs peers.

Intriguingly, we also observed that even though baseline rates of ADHD and ASD were grossly more prevalent in people with EDs than antidepressant-receiving peers without EDs prior to propensity score matching, our adjusted analyses showed that people with EDs were no more likely to receive diagnoses for ADHD/ASD as their peers in the year following the index event. In other words, even though people with EDs are more likely to carry past prior diagnoses for ADHD/ASD preceding their first EDs diagnosis, but they are *subsequently* no more likely to receive ADHD/ASD diagnoses than their peers after being diagnosed with EDs and potentially initiating treatment. The reasons for this finding are perplexing, particularly since studies showed a rising prevalence of ASD and ADHD in people with EDs [37–40]. On one hand, this could represent under-treatment of ADHD and ASD in patients engaged in EDs care (i.e., discontinuation of stimulants while patients undergo weight restoration, lack of ASD-specific therapies in EDs treatment). On the other hand, we are seeing an increased awareness and diagnosis of ADHD/ASD in general healthcare settings

outside of the EDs realm [41, 42], which could explain why the non-EDs comparison cohort had a similar rate of ADHD/ASD diagnosis as the EDs cohort in the 1 year after the index date.

Broadly speaking, the specific reasons underlying the high rates of co-occurring psychiatric diagnoses in people with eating disorders are not fully understood. Several factors are considered possible explanations; for instance, a genome-wide association study revealed that schizophrenia and major depressive disorder polygenic scores were positively associated with anorexia nervosa and binge-eating disorder, the ADHD polygenic score was positively associated with binge-eating disorder, and the bipolar disorder polygenic score was positively associated with anorexia nervosa [43]. Meanwhile, other studies showed that atypical eating behaviors were associated with neurodevelopmental disorders such as ASD and ADHD [44–46]. While our study examined psychiatric comorbidity risk with any eating disorder diagnosis, our report on the magnitude of association based on clinical significance complements the previous literature and strengthens our knowledge and ability to develop and implement evidence-based practices for healthcare professionals caring for patients with psychiatric diagnoses, including ED. Ultimately, these results corroborate these findings and further characterize psychiatric comorbidities in patients with eating disorders across a broad range of diagnoses.

A major strength of our study methodology lies in the TriNetX data's real-time patient health information, allowing us to conduct time-sensitive analyses of the health outcomes of people with EDs that are difficult to achieve with traditional human subjects research. Yet, this analysis of TriNetX data has important limitations. First, there are limitations surrounding generalizability. This study captures a specific year in time (2022) and only includes patients presenting to a healthcare setting, hindering our generalizability to people without consistent access to health care or patients with undiagnosed EDs. More than 90% of healthcare organizations in the TriNetX database are in the US, further limiting the generalizability of these findings, which is a significant consideration given that prevalence of ED is surging worldwide [47]; we are unable to limit our analyses to only the U.S. due to the limited refinement capacities in the TriNetX data. Further, the present study is limited by the lack of detailed race, ethnicity, and socioeconomic data, an important limitation given the structural disparities impacting access to eating disorders treatment in the U.S. Additionally, ASD and ADHD may have presented earlier in childhood for clinical care and are not captured in the observation window.

A second important limitation is that we are unable to reliably differentiate between separate EDs. Diagnostic crossover is also common among anorexia nervosa, bulimia nervosa, and other EDs, and thus the present study did not evaluate individual EDs but rather grouped all subtypes of EDs together. Furthermore, since ICD-10 codes for individual EDs are pending validation studies, we elected to group all eating disorders together to mitigate misclassification bias that is inevitable in analyses of administrative data. Third, we cannot rule out inaccuracies in ICD-10 documentation. Notably, the symptoms of personality disorders often overlap with psychiatric effects of starvation or repeated binge/purge cycles and provisional "personality disorder symptoms" found to quickly improve alongside improvement in ED symptoms [48]. The misdiagnosis of personality disorders in people

with active EDs cannot be ruled out, and we thus elected to report personality disorders in aggregate, although we highlighted diagnoses of borderline personality disorder separately to illustrate its particularly high rate of diagnosis in this population. Further, PTSD may be underdiagnosed in the ED population even though it predicts more severe ED pathology.[49] It is particularly important for future research to evaluate cohorts with co-occurring ED and PTSD, as studies show that the treatment of PTSD may be associated with sustained improvement in ED outcomes.[50] Overall, we would recommend usage of more detailed claims data (i.e., CMS Medicaid databases) to obtain a more precise assessment of mental health outcomes after patients initiating treatment for EDs (i.e., psychotherapy claims, group therapy, residential treatment).

Finally, although we used propensity score-matching to mitigate confounding, we cannot completely eliminate residual confounding. Therefore, we emphasize that the results of such administrative data analyses are preliminary and serve for hypothesis generation, rather than the localization of specific treatment gaps. Future clinical studies are needed to provide mental health professionals with a more complete profile of patients with eating disorders that they will potentially serve.

CONCLUSIONS

In summary, this study draws attention to a high burden of co-occurring psychiatric diagnoses such as mood disorders, GAD, ADHD and ASD in people with EDs in a real-world setting. More research is needed to expand our understanding of the impact of co-occurring psychiatric disorders in people with EDs and inform future evidence-based public health responses.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

[1]. Santomauro DF, Melen S, Mitchison D, Vos T, Whiteford H and Ferrari AJ. The hidden burden of eating disorders: an extension of estimates from the Global Burden of Disease Study 2019. Lancet Psychiatry 2021;8:320–328. [PubMed: 33675688]

[2]. Beat U. The costs of eating disorders: Social, health and economic impacts. Norwich: Author 2015.

[3]. Streatfeild J, Hickson J, Austin SB, Hutcheson R, Kandel JS, Lampert JG, et al. Social and economic cost of eating disorders in the United States: Evidence to inform policy action. International Journal of Eating Disorders 2021;54:851–868. [PubMed: 33655603]

[4]. Li S, Liu J, Ma H, Wang Y and Wu J. Trends in the prevalence and disability-adjusted life years of eating disorders from 1990 to 2017: results from the Global Burden of Disease Study 2017. Epidemiology and Psychiatric Sciences 2020;29:e191. [PubMed: 33283690]

[5]. The Lancet D, amp and Endocrinology. Eating disorders: care not keeping up with demand. Lancet Diabetes Endocrinol 2023;11:625. [PubMed: 37573869]

[6]. Milliren CE, Richmond TK and Hudgins JD. Emergency Department Visits and Hospitalizations for Eating Disorders During the COVID-19 Pandemic. Pediatrics 2022;151:e2022058198.

[7]. Agostino H, Burstein B, Moubayed D, Taddeo D, Grady R, Vyver E, et al. Trends in the Incidence of New-Onset Anorexia Nervosa and Atypical Anorexia Nervosa Among Youth During the

COVID-19 Pandemic in Canada. JAMA Network Open 2021;4:e2137395–e2137395. [PubMed: 34874405]

[8]. Protecting Youth Mental Health & Social Media and Youth Mental Health: The United States Surgeon General's Advisory. 2023.

[9]. Downs J, Ayton A, Collins L, Baker S, Missen H and Ibrahim A. Untreatable or unable to treat? Creating more effective and accessible treatment for long-standing and severe eating disorders. The Lancet Psychiatry 2023;10:146–154. [PubMed: 36697122]

[10]. Marzola E, Panero M, Longo P, Martini M, Fernàndez-Aranda F, Kaye WH, et al. Research in eating disorders: the misunderstanding of supposing serious mental illnesses as a niche specialty. Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity 2022;27:3005–3016.

[11]. Egbert AH, Fitzsimmons-Craft EE, Haynos AF, Levinson CA and Schleider JL. Not niche: eating disorders as an example in the dangers of overspecialisation. The British Journal of Psychiatry 2023:1–4.

[12]. The Lancet P The future of eating disorders research. The Lancet Psychiatry 2023;10:909. [PubMed: 37977693]

[13]. Hambleton A, Pepin G, Le A, Maloney D, Aouad P, Barakat S, et al. Psychiatric and medical comorbidities of eating disorders: findings from a rapid review of the literature. Journal of Eating Disorders 2022;10:132. [PubMed: 36064606]

[14]. Blinder BJ, Cumella EJ and Sanathara VA. Psychiatric Comorbidities of Female Inpatients With Eating Disorders. Psychosomatic Medicine 2006;68.

[15]. Brewerton TD, Gavidia I, Suro G and Perlman MM. Associations between major depressive and bipolar disorders and eating disorder, PTSD, and comorbid symptom severity in eating disorder patients. Eur Eat Disord Rev 2024;32:188–200. [PubMed: 37788327]

[16]. Eskild-Jensen M, Støving RK, Flindt CF and Sjogren M. Comorbid depression as a negative predictor of weight gain during treatment of anorexia nervosa: A systematic scoping review. European Eating Disorders Review 2020;28:605–619. [PubMed: 32886423]

[17]. Lydecker JA and Grilo CM. Psychiatric comorbidity as predictor and moderator of binge-eating disorder treatment outcomes: an analysis of aggregated randomized controlled trials. Psychol Med 2022;52:4085–4093.

[18]. Momen NC, Plana-Ripoll O, Yilmaz Z, Thornton LM, McGrath JJ, Bulik CM, et al. Comorbidity between eating disorders and psychiatric disorders. Int J Eat Disorder 2022;55:505–517.

[19]. Herzog DB, Nussbaum KM and Marmor AK. Comorbidity and outcome in eating disorders. Psychiatr Clin North Am 1996;19:843–59. [PubMed: 9045226]

[20]. Hambleton A, Pepin G, Le A, Maloney D, National Eating Disorder Research C, Touyz S, et al. Psychiatric and medical comorbidities of eating disorders: findings from a rapid review of the literature. J Eat Disord 2022;10:132. [PubMed: 36064606]

[21]. Brown TR, Kablinger AS, Trestman R, Bath E, Rogers C, Lin BY, et al. Psychiatric comorbidities in children with conduct disorder: a descriptive analysis of real-world data. Gen Psychiatr 2024;37:e101501. [PubMed: 38645379]

[22]. Moog D, Xie H, Sun C, Deng W, McDaid E, Liebesny KV, Kablinger AS, Xu KY, Lin BY. The Prevalence of Eating Disorders in Children, Adolescents, and Young Adults, 2017–2022: an Analysis of Real-Time Administrative Data. 2024. General Psychiatry. In Press.

[23]. Sun CF, Xie H, Metsutnan V, Draeger JH, Lin Y, Hankey MS, et al. The mean age of gender dysphoria diagnosis is decreasing. Gen Psychiatr 2023;36:e100972. [PubMed: 37396783]

[24]. Topaloglu U and Palchuk MB. Using a Federated Network of Real-World Data to Optimize Clinical Trials Operations. JCO Clinical Cancer Informatics 2018:1–10.

[25]. Linked EHR and Claims - TriNetX. trinetxcom/real-world-data/linked. 2023.

[26]. Von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC and Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. The Lancet 2007;370:1453–1457.

[27]. Committee on Improving the Health, Safety, and Well-Being of Young Adults; Board on Children, Youth, and Families; Institute of Medicine; National Research Council; Bonnie RJ, Stroud C, Breiner H, editors. Investing in the Health and Well-Being of Young Adults. Washington (DC): National Academies Press (US); 2015 Jan 27.

[28]. Ackard DM, Fulkerson JA and Neumark-Sztainer D. Stability of eating disorder diagnostic classifications in adolescents: five-year longitudinal findings from a population-based study. Eat Disord 2011;19:308–22. [PubMed: 22352971]

[29]. Fichter MM and Quadflieg N. Long-term stability of eating disorder diagnoses. Int J Eat Disorder 2007;40:S61–S66.

[30]. Milos G, Spindler A, Schnyder U and Fairburn CG. Instability of eating disorder diagnoses: prospective study. Brit J Psychiat 2005;187:573–578.

[31]. Rector TS, Wickstrom SL, Shah M, Thomas Greeenlee N, Rheault P, Rogowski J, et al. Specificity and sensitivity of claims-based algorithms for identifying members of Medicare plus Choice health plans that have chronic medical conditions. Health Serv Res 2004;39:1839-+. [PubMed: 15533190]

[32]. Xu KY, Huang V, Williams AR, Martin CE, Bazazi AR and Grucza RA. Co-occurring psychiatric disorders and disparities in buprenorphine utilization in opioid use disorder: An analysis of insurance claims. Drug Alc Depend Rep 2023;9.

[33]. Phillips ML and Kupfer DJ. Bipolar Disorder 2 Bipolar disorder diagnosis: challenges and future directions. Lancet 2013;381:1663–1671. [PubMed: 23663952]

[34]. Patorno E, Glynn RJ, Levin R, Lee MP and Huybrechts KF. Benzodiazepines and risk of all cause mortality in adults: cohort study. BMJ 2017;358:j2941. [PubMed: 28684397]

[35]. Austin PC. Using the Standardized Difference to Compare the Prevalence of a Binary Variable Between Two Groups in Observational Research. Communications in Statistics - Simulation and Computation 2009;38:1228–1234.

[36]. Udo T and Grilo CM. Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. Int J Eat Disord 2019;52:42–50. [PubMed: 30756422]

[37]. Biederman J, Ball SW, Monuteaux MC, Surman CB, Johnson JL and Zeitlin S. Are girls with ADHD at risk for eating disorders? Results from a controlled, five-year prospective study. J Dev Behav Pediatr 2007;28:302–307. [PubMed: 17700082]

[38]. Ziobrowski H, Brewerton TD and Duncan AE. Associations between ADHD and eating disorders in relation to comorbid psychiatric disorders in a nationally representative sample. Psychiat Res 2018;260:53–59.

[39]. Carpita B, Muti D, Cremone IM, Fagiolini A and Dell'Osso L. Eating disorders and autism spectrum: links and risks. Cns Spectrums 2022;27:272–280. [PubMed: 33161925]

[40]. Westwood H and Tchanturia K. Autism Spectrum Disorder in Anorexia Nervosa: An Updated Literature Review. Curr Psychiat Rep 2017;19.

[41]. Li Q, Li YM, Liu BY, Chen QS, Xing XH, Xu GF, et al. Prevalence of Autism Spectrum Disorder Among Children and Adolescents in the United States from 2019 to 2020. Jama Pediatr 2022;176:943–945. [PubMed: 35789247]

[42]. Li YM, Yan XF, Li QS, Li Q, Xu GF, Lu JH, et al. Prevalence and Trends in Diagnosed ADHD Among US Children and Adolescents, 2017–2022. Jama Netw Open 2023;6.

[43]. Hübel C, Abdulkadir M, Herle M, Loos RJF, Breen G, Bulik CM, et al. One size does not fit all. Genomics differentiates among anorexia nervosa, bulimia nervosa, and binge-eating disorder. Int J Eat Disorder 2021;54:785–793.

[44]. Mayes SD and Zickgraf H. Atypical eating behaviors in children and adolescents with autism, ADHD, other disorders, and typical development. Res Autism Spect Dis 2019;64:76–83.

[45]. Nickel K, Maier S, Endres D, Joos A, Maier V, van Elst LT, et al. Systematic Review: Overlap Between Eating, Autism Spectrum, and Attention-Deficit/Hyperactivity Disorder. Front Psychiatry 2019;10.

[46]. Villa FM, Crippa A, Rosi E, Nobile M, Brambilla P and Delvecchio G. ADHD and eating disorders in childhood and adolescence: An updated minireview. J Affect Disorders 2023;321:265–271. [PubMed: 36356347]

[47]. Silén Y and Keski-Rahkonen A. Worldwide prevalence of DSM-5 eating disorders among young people. Curr Opin Psychiatr 2022;35:362–371.

[48]. Cassin SE and von Ranson KM. Personality and eating disorders: a decade in review. Clin Psychol Rev 2005;25:895–916. [PubMed: 16099563]

[49]. Brewerton TD, Perlman MM, Gavidia I, Suro G, Genet J and Bunnell DW. The association of traumatic events and posttraumatic stress disorder with greater eating disorder and comorbid symptom severity in residential eating disorder treatment centers. Int J Eat Disord 2020;53:2061–2066. [PubMed: 33159362]

[50]. Brewerton TD, Gavidia I, Suro G and Perlman MM. Eating disorder patients with and without PTSD treated in residential care: discharge and 6-month follow-up results. J Eat Disord 2023;11:48. [PubMed: 36973828]

Table 1:

Baseline demographic and clinical characteristics of cohorts 1 year preceding first diagnosis of eating disorders or first date of antidepressant receipt (among those without eating disorders)

Variables	Eating Disorders	%	No Eating Disorders, Receiving Antidepressants	%
N	14,524	100%	110,051	100%
Current Age	17.5, 4.6		19.3, 4.7	
Age at Index (years, SD)	15.9, 4.6		17.8, 4.7	
Sex				
Female	11,498	79.2%	71,926	65.4%
Male	2,802	19.3%	36,106	32.8%
Ethnicity				
Not Hispanic or Latino	10,380	71.5%	71,506	65.0%
Unknown Ethnicity	2,272	15.6%	28,682	26.1%
Hispanic or Latino	1,872	12.9%	9,863	9.0%
Race				
White	9,978	68.7%	71,849	65.3%
Black or African American	1,004	6.9%	11,884	10.8%
Other Race	856	5.9%	5,827	5.3%
Asian	452	3.1%	2,300	2.1%
Psychiatric disorders	14,524	100%	51,389	46.7%
Mood disorder [Major depressive disorder; bipolar disorders]	7,435	51.2%	24,840	22.6%
Generalized anxiety disorder	4,401	30.3%	8,608	7.8%
Obsessive-compulsive disorder	1,155	8.0%	1,159	1.1%
Posttraumatic stress disorder	1,484	10.2%	2,399	2.2%
Specific phobia	1,217	8.4%	1,217	1.1%
Panic disorder	911	6.3%	1,709	1.6%
Adjustment disorders	730	5.0%	2,408	2.2%
Attention-deficit/hyperactivity disorder	2,503	17.2%	9,210	8.4%
Autism spectrum disorder	1,208	8.3%	2,515	2.3%
Personality disorders	601	4.1%	540	0.5%
Borderline personality disorder	496	3.4%	378	0.3%
Gender dysphoria	565	3.9%	1,434	1.3%
Substance use disorders	1,176	8.1%	5,431	4.9%
Cannabis use disorders	651	4.5%	2,228	2.0%
Nicotine dependence	462	3.2%	2,664	2.4%
Alcohol use disorders	256	1.8%	919	0.8%
Other stimulant use disorders	62	0.4%	333	0.3%
Opioid use disorders	54	0.4%	390	0.4%

Table 2:

Diagnoses of psychiatric comorbidities, compared between people with eating disorders (n=13,707) matched to peers without eating disorders receiving antidepressants (n=13,707)

Eating disorders cohort versus individuals without eating disorders receiving antidepressants	Adjusted odds ratio	95% confidence interval
Mood disorder [Major depressive disorder; bipolar disorders]	1.20	(1.14, 1.26)
Generalized anxiety disorder	1.28	(1.21, 1.35)
Obsessive-compulsive disorder	1.53	(1.38, 1.70)
Panic disorder	1.63	(1.45, 1.84)
Specific phobia	1.45	(1.31, 1.60)
Posttraumatic stress disorder	1.29	(1.18, 1.40)
Personality disorders	1.76	(1.53, 2.04)
Borderline personality disorder	1.90	(1.61, 2.23)
Gender dysphoria	1.12	(0.99, 1.28)
Attention-deficit/hyperactivity disorder	1.04	(0.97, 1.10)
Autism spectrum disorder	0.99	(0.91, 1.08)
Substance use disorders	1.25	(1.14, 1.37)