**Alcohol use among populations with Autism Spectrum Disorder:**

**A narrative systematic review**

**William Barbera  
ORCID = 0000-0002-0728-4734**

**Betul Aslanb  
ORCID = 0000-0002-7421-5245**

**Dr. Tim Meynena  
ORCID = 0000-0003-0661-5391**

**Prof. John Marsdena  
ORCID = 0000-0002-1307-2498**

**Prof. Samuel R. Chamberlainbc  
ORCID =** **0000-0001-7014-8121**

**Vigneshwar Paleria  
ORCID = 0000-0002-2223-9210**

**Prof. Julia Sinclair\*b  
ORCID = 0000-0002-1905-2025**

a Addictions Department, School of Academic Psychiatry, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK.

b Department of Psychiatry, Faculty of Medicine, University of Southampton, Southampton, UK.

c Southern Health NHS Foundation Trust, Southampton, UK.

\*Corresponding Author

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**Declaration of interest:**

WB: This review was completed in part fulfilment of Doctorate of Clinical Psychology thesis, King’s College London (1); SRC: receives a stipend from Elsevier for Associate Editor work; JM: In the past three years, JM declares research grants for the following clinical trials: King’s College London [KCL]); Indivior (trial of extended-release pharmacotherapy for opioid use disorder; sponsor: KCL and South London & Maudsley NHS Trust); and Beckley PsyTech (phase 2a trial of 5-MeO-DMT for alcohol use disorder; sponsor: Beckley PsyTech). He is the senior academic advisor for the Office for Health Improvement and Disparities, English Department of Health and Social Care, and a clinical academic consultant for the US National Institute on Drug Abuse, Clinic for Clinical Trials Network. JM declares honoraria and travel support from PCM Scientific, OPEN Health, and Indivior to contribute to scientific and educational meetings. He holds no stocks in any company; BA, TM, VP, JS: report no declarations of interest.

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**Author contribution:**

Concept and design of review: WB, BA, TM, JM, JS; Data acquisition and quality appraisal: WB, VP, TM; Analysis: WB, TM, JM, SC, JS; Drafting of manuscript: WB, BA, TM, JM, SC, JS

**Transparency declaration:**

All authors declare the manuscript is honest, accurate, and transparent of the review findings. The review was pre-registered on PROSPERO (No.: CRD42023430291). The protocol was edited to clinical samples only, as described in the main text.

**Data availability:**

Not applicable as this is a review article.

**Abstract:**

*Background:* Alcohol use in autism spectrum disorder (ASD) is under-researched. Previous reviews have explored substance use as a whole, but this neglects individual characteristics unique to different substances. Alcohol use in non-clinical samples is associated with diverse responses. To advance practice and policy, an improved understanding of alcohol use among people with ASD is crucial to meet individual needs.

*Aims:* This was a narrative systematic review of the current literature on the association between alcohol use and ASD, focusing on aetiology (biological, psychological, social, and environmental risk factors) and implications (consequences and protective factors) of alcohol use in autistic populations who utilise clinical services. We sought to identify priority research questions and offer policy and practice recommendations.

*Method:* PROSPERO Registration: CRD42023430291. The search was conducted across five databases: CINAHL, EMBASE, MEDLINE, PsychINFO, and Global Health. Included studies explored alcohol use and ASD within clinical samples.

*Results:* A total of 22 studies were included in the final review. The pooled prevalence of alcohol-use disorder in ASD was 1.6% and 16.1% in large-population registers and clinical settings, respectively. Four components were identified as possible aetiological risk factors – age, co-occurring conditions, gender, and genetics. We identified 10 implications for co-occurring alcohol use disorder in ASD, summarised as a concept map.

*Conclusion:* Emerging trends in the literature suggest direction and principles for research and practice. Future studies should use a standardised methodological approach—including psychometrically validated instruments and representative samples—to inform policy and improve the experience for autistic populations with co-occurring alcohol use.

**Introduction**

Autism spectrum disorder (ASD) is a neurodevelopmental condition characterised by social communication/interaction challenges, restrictive and repetitive behaviour, inflexible behaviour patterns, and atypical or excessive interests or hobbies (2). ASD is usually taken to capture a broad range of disorders, which has been previously referred to as Asperger’s syndrome, pervasive developmental disorder, and autism spectrum disorder. There is no universally accepted consensus on the terminology used to describe ASD. Here, we use identity-first language (“autistic person/people” (3)) as this has been recommended by the stakeholders of the Substance use, Alcohol, and Behavioural Addictions in Autism partnership (SABA-A (4)).

A recent UK study has suggested progressive increases in ASD diagnoses over time, with the greatest increase in adults and females (5). The apparent growth in the prevalence of ASD has been attributed to changes in diagnostic criteria (6), reporting practices (7), and increased awareness (8). Despite suggested increases in ASD diagnoses, waiting time for assessment continues to rise across Europe (9). Consequently, many autistic people continue to access healthcare services unrecognised and as incident rates increase (10, 11), so does the need to further explore co-occurring conditions to improve treatment outcomes.

Within ASD populations, research indicates alcohol is the most reported substance used (12-14). Previous reviews and meta-analyses of substance-use disorder (SUD) in autistic populations reported a wide prevalence range of Alcohol-use Disorder (AUD; 0%-16%), attributed to heterogeneity across study samples and the diagnostic procedures used (12, 13, 15, 16). Several studies have found when compared to neurotypical peers, autistic adults report lower alcohol use and higher rates of abstinence (17-19). Further findings from two showed that the rate of alcohol use in autistic people increases with age (19, 20).

AUD is a spectrum disorder characterised by maladaptive patterns of alcohol use and related impairments of personal and social functioning that are clinically assessed as mild, moderate, or severe (21). As criterion symptoms accumulate, the risk of harm and adverse psychosocial consequences increases (22). Of all SUDs, AUD is the most prevalent within global populations, requiring targeted interventions and policies to reduce alcohol-related harm (23, 24). Of note, the language used to describe AUD may be stigmatising, leading to significant barriers to receiving or seeking support (25). Here, we extracted the same phraseology used in studies, but we omitted stigmatising labels such as addict, misuse, and abuse within the main body.

*Biopsychosocial factors of AUD and ASD*

A greater understanding of both risk and protective factors in ASD could improve translational opportunities for research and clinical practice (26). Despite the estimates mentioned previously, the prevalence and nature of AUD among autistic people are underexplored (4, 12). From a biopsychosocial lens, identifying shared factors between conditions can offer avenues to promoting resilience and intervention of risk factors.

Typically, the developmental onset and course of both ASD and AUD are diverse. Early conceptualisations position ASD as emerging in childhood (27), while AUD routinely develops across the lifespan from adolescence and early adulthood (28, 29). Both conditions possess an element of genetic heritability (30, 31). Two reviews found an overlap of neurological circuitry between ASD and SUD (32), as well as several independent studies which observed overlapping genes in the susceptibility to ASD or AUD (33). These findings suggest the possibility of shared genetic pathways between both conditions. However, the identification of specific risk variants for each remains inconclusive and emphasises consideration for epigenetic interactions (34-36).

Several environmental factors are known to contribute to the development of AUD; including parental alcohol use and supply (37), low prosocial behaviours (38), social norms (39), peer substance use (40), and adverse childhood experiences (41). Research has found some autistic people may have an equal or greater likelihood of experiencing traumatic events compared to neurotypical peers (42-44). This connection could be attributed to interpersonal victimisation and bullying, emotional dysregulation in processing traumatic stress, lack of support, and social isolation common in ASD (42-44). Exposure to traumatic events, as a common factor for both ASD and AUD, may increase the overall risk of harmful alcohol use. Whereas social capital - referring to the level of community attachment, closeness, and supportiveness experienced by an individual - has the potential to reduce the risk of alcohol use (45). However, as aforementioned, social isolation and lack of support are common in autistic people, presenting social capital as a possible area of vulnerability.

Characteristic features of ASD may be protective, influencing how an autistic person interacts with their environment. For example, developmental and communication challenges, as well as unsettled peer relationships in ASD adolescents were negatively associated with alcohol and substance use (46). In addition, certain factors such as parental involvement, household rules, and monitoring can limit the availability and opportunity for alcohol use among autistic individuals, thus reducing the risk of developing AUD (40, 47).

Bowri et al. (18) examined factors associated with alcohol use within a high-functioning community sample of autistic adults. Dividing the sample into three groups by alcohol use, non-drinkers and hazardous drinking patterns were predictors for higher scores of autistic traits, depression, social anxiety, and generalised anxiety, in comparison to non-hazardous drinkers. Non-hazardous drinkers reported the highest scores of wellbeing among the three groups, whereas hazardous use was associated with a higher frequency of co-occurring psychiatric conditions. However, these findings did not distinguish whether alcohol use acts as a protective factor against co-occurring psychiatric conditions or if reduced co-occurring conditions led to less harmful alcohol use.

Antecedents to alcohol use have focused on positive and negative motivations for use (48-50). Social facilitation, mood enhancement, symptom management, coping mechanisms for difficulties such as social anxiety, sensation seeking, and ‘self-medication’ of sensory processing difficulties are themes positively associated with alcohol use (17, 48-52). In contrast, factors such as fear of addiction, disinhibition, olfactory sensitivity, and decreased access to alcohol limit the risk and decrease motivation for alcohol use in ASD populations (18, 48, 53). Given the diverse levels of functioning and severity, inherent within the spectrum of both ASD and AUD, current research lacks a validated measure that simultaneously captures elements of both conditions. This absence of standardisation across studies impairs the ability to establish consistent conclusions from findings (4). Overall, there is a greater need to increase screening and prevention, and to reduce barriers to support for autistic people with AUD (48, 54-56).

*Current review*

The Substance use, Alcohol, and Behavioural Addictions in Autism partnership (SABA-A), funded by the Society for the Study of Addiction, brought together a range of experts to identify key policy, research, and clinical practice questions for ASD and addiction. In 2023, the project published the top ten priorities, outlining the most urgent issues impacting the lives of autistic individuals with substance use, problematic alcohol use, or behavioural addictions (4). The highest-ranked priority was the identification and prevention of specific triggers, risk factors, and facilitators of substance, alcohol, or behavioural addictions in autistic people. Additional priorities included enhancing awareness, reducing stigma, adaptations to current approaches, how other conditions or traits impact the development and maintenance of addiction, and differences in vulnerability between autistic and non-autistic populations. SABA-A has published one review on ASD and gambling and the current review explored ASD and alcohol use (57).

Existing reviews have generally focused on SUD, rather than exclusively on AUD. Different substances can serve different purposes for autistic adults and given that alcohol use is common within ASD populations, it is of interest that there is limited information on how ASD and AUD present in clinical services.

Accordingly, our aim was to:

(1) Systematically identify and collate findings from studies which have examined the association between AUD and ASD;

(2) Explore the current knowledge of clinical samples on aetiology – including biological, psychological, social, and environmental risk factors associated with alcohol use among autistic people; and implications – including the protective factors and consequences of co-occurring alcohol use in autistic people;

(3) Identify priority research questions and offer recommendations for policy, and clinical practice.

**Methods**

*Protocol registration*

This was a pre-registered systematic review. The protocol was prospectively registered with the International Prospective Register of Systematic Reviews (PROSPERO) on 31st May 2023 (identifier CRD42023430291).

Records identified from\*:

Databases (n = 5)

* CINAHL (n = 299)
* EMBASE (n= 4,145)
* Global Health (n = 388)
* MEDLINE (n = 1,574)
* PsychInfo (n = 1,250)

Total n = 7,611

Records removed *before screening*:

Duplicate records removed   
(n = 2,338)

Records screened:

(n = 5,273)

Records excluded:

(n = 4,890)

Reports sought for retrieval:

(n = 383)

Reports not retrieved:

(n = 0)

Reports assessed for eligibility:

(n = 383)

Reports excluded:

* Did not explore both ASD and alcohol use (n = 72)
* ASD and alcohol use were studied independently and intersection was not reported (n = 78)
* Grouped ASD with other disorders (e.g., ADHD, intellectual disabilities, etc.) (n = 15)
* Combined alcohol use with SUD (n = 14)
* Excluded study type (n = 101)
* Grey literature (n = 11)
* Study protocol (n = 2)
* Limited sample size (n = 15)
* Non-quantified alcohol use definition (n = 20)
* Non-clinical population (n = 25)
* Not available in English (n = 8)

Studies included in review:

(n = 22)

**Identification of studies via databases and registers**

**Identification**

**Screening**

**Included**

***Figure 1****: PRISMA flow diagram.*

*Design and search strategy*

We conducted a search of the extant literature using five databases: CINAHL, EMBASE, MEDLINE, PsychInfo, and Global Health. The search string consisted of: ((ASD) OR (Autis\*) OR (Asperger\*) OR (ASC) OR (PDD) OR (Pervasive Developmental Disorder) OR (Neurodevelopmental) OR (Kanner) OR (Developmental Disabilities / OR Autism Spectrum Disorders / OR Child Developmental Disorders [MeSH Major Topic])) AND (Alcohol\*) OR (Alcohol addiction) OR (Alcohol misuse) OR (Alcohol dependen\*) OR (Drinking) OR (Alcoholism / OR Alcohol abuse / OR Alcohol Use Disorder / OR Alcohol related disorders [MeSH Major Topic])). This string was adapted from an initial scope conducted by one of the authors (B.A) and finalised with current authors.

The search was completed as planned with the exception at full-text screening to narrow the search to clinical samples only. Clinical samples were defined as participants recruited from healthcare settings or patient register databases. Given the heterogeneity of existing literature, this approach was taken to provide future clinical services and research recommendations as a meta-analysis would not be feasible at this stage. The search was repeated in August 2023 to identify any additional papers since the initial search in May 2023. This review adhered to narrative synthesis and PRISMA guidelines (see supplementary material for checklist) (58, 59).

*Inclusion and exclusion criteria*

Primary inclusion required studies to explore broadly both ASD and alcohol use in a clinical setting or population. Studies that measured autistic traits were also included. We applied no limits on the date of publication or age of participants, accounting for potential longitudinal studies. Both qualitative and quantitative articles were included to capture reported life experiences alongside statistical inferences and associations between ASD and alcohol use. Included studies were required to be peer-reviewed and published in English. We excluded studies of other reviews, meta-analyses, studies with an ASD sample of less than five, genome-wide association studies, book chapters, and grey literature.

*Data extraction and synthesis*

A narrative synthesis was the preferred method of analysis as the literature is limited. Articles were initially screened by title and abstract, using the criteria specified, before retrieval for full-text review. Where studies did not solely focus on ASD and alcohol use, only related data was extracted. The first data extraction took place on 16th June 2023 and final data extraction on 18th August 2023, following the repeated search. Two raters (WB, VP) were used at both steps of the screening process and quality appraisal. Any discrepancies were discussed amongst the authors until a consensus was reached. The overall approach was guided by the Popay et al., (59) framework for narrative synthesis. Tabulation was used to infer similarities across studies and themes were grouped across aetiological factors and potential implications of ASD and alcohol use. The summary of findings is presented as a concept map, to capture key themes and inform the third aim, identifying priority areas and recommendations (59).

*Quality assessment*

The Mixed Methods Appraisal Tool (MMAT (60)) was used to assess the methodological quality of selected studies. Each study was evaluated by design and rated across five criteria, individual to each study design (yes = 1, no = 0), with an overall quality score calculated as a percentage of total criteria met (presented in **Table 1** (61)). We did not set a minimum threshold for quality criteria and, therefore, did not exclude any studies. Full quality description can be found in the supplementary material (**Table 2**).

**Results**

Twenty-two studies were included in the final review following the full-text screening of 383 articles (**Figure 1**). Full study characteristics and a summary of individual findings are presented in **Table 1**. Detailed findings are reported below, and the data are synthesised into main components for aetiological risk factors and implications for co-occurring AUD in ASD in **Box 1** as a concept map (59). Four components of biological, psychological, and social/environmental aetiological risk factors emerged alongside 10 potential implications of co-occurring AUD in ASD.

| **Table 1:** Summary of studies included in the review. | | | | | | | | | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Study** | **Quality assessment a** | **Population/ Setting** | **Design and sample size** | **AUD measure** | **Age b** | **Sex** | **Race/ Ethnicity** | **Socio- economic c** | **Key findings**  **(Sig. is statistical significance)** |
| *AUD service* | | | | | | | | | |
| Hildebrand Karlén et al. (2021) | 40% | Alcohol treatment settings (inpatient and outpatient) | Longitudinal, quantitative non-randomised  (*n = 153; n=91 completed 2.5 year follow-up)* | Previous dx. of AUD based on DSM-IV criteria. Additional structured interview of ASI | M = 50.2  (8.56)  29-71 | 80.8% male | NR | 11.01 years in school;  87.05% in current employment;  57.3% in current relationship. | * Sig. but weak correlations between autistic personality traits (APTs) and baseline consumption (positive) and   APTs and age at entering treatment (negative).   * Post-treatment, the number of APTs was not correlated with 1) how much participants lowered or maintained consumption, 2) whether they normalised drinking, or 3) were neither more nor less prevalent in abstainers. * Problematic drinking patterns 2.5 years after treatment were sig. related to idiosyncratic features of ASD. |
| Kronenberg et al. (2014) | 100% | Outpatient dual diagnosis service. Treatment-seeking individuals with ASD or ADHD and comorbid SUD. | Qualitative interviews  (N = 23; ASD + SUD *n = 12)* | Previous dx. of SUD based on DSM-IV criteria. | ASD+ SUD sample: M = 37  (NR) | ASD+ SUD sample: 100% male | NR | ASD+SUD sample: Employment: 50% employed; Living: 42% living alone, 58% living with others. | * Three main themes: (1) jumbled thoughts and emotions, (2) ambiguity of substance use, and (3) structure. |
| Narita et al. (2016) | 80% | Hospital patients with AUD. | Case-control, quantitative non-randomised cohort  (N = 139, AUD *n = 64, Unrelated controls n = 75)* | Previous dx. based on DSM-IV criteria | AUD:  M = 57.3  (10.18)  NR | AUD: 78.1% male | NR | All patients lived in Yamagata prefecture Japan. | * No sig. difference between the AUD group and control group for autism susceptibility candidate 2 gene (AUTS2) polymorphisms. * Distribution of A-A haplotype combinations were sig. different and higher in frequency within the AUD group compared to controls. |
| Walhout et al. (2022) | 60% | Addiction Treatment Centre. | Naturalistic, quantitative non-randomised cohort  (T0 N *= 57,  T1 n = 30,  T2 n = 27)* | Previous SUD dx. based on DSM-5 criteria. Additional measure of MATE 2.1 | M=36.8 (11.65)  19-64 | 86% male | Country of birth:  93% Netherlands, other European 3.5%, Non-European 3.5% | Education: 33.3% ‘Low’, 43.9% ‘Medium’, 21.1% ‘High’; Employment:75.4% unemployed/ social benefit, 24.6% Job/ student loan; Marital status:  71.9% single, 17.5% married, 10.5% divorced/ widow/ widower | * Sig. decrease in alcohol consumption from baseline to time 1and time 2 following adapted group cognitive behavioural therapy (CBT) for SUD. Cannabis use remained unchanged at both time points. * Sig. decrease in cravings, passive coping style, and symptoms of depression, anxiety, and stress at time 1 and 2. * Sig. increase in seeking social support as a coping mechanism, overall feelings of control, and self-empowerment at time 1 and 2. * At time 2, there was a sig. increase in using reassuring thoughts, learning new potentials, and spirituality. |
| Yoshimura et al. (2022) | 100% | Addiction Treatment Centre. | Naturalistic longitudinal, quantitative non-randomised cohort  (N *= 637)* | Clin. ax. using ICD-10 criteria | M=53.9 (12.9)  20-85 | 85.2% male | NR | NR | * The presence of ASD traits (*n=29)* did not affect abstinence rates during follow-up compared to those without traits (*n = 461).* * When divided into three groups based on autism-spectrum quotient (AQ, Low, moderate, High), no differences were observed in abstinence rates. |

| *ASD service* | | | | | | | | | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Study** | **Quality assessment a** | **Population/ Setting** | **Design and sample size** | **AUD measure** | **Age b** | **Sex** | **Race/ Ethnicity** | **Socio- economic c** | **Key findings**   * **(Sig. is statistical significance)** |
| Miles et al.  (2003) | 20% | Unrelated patients with infantile ASD were recruited from university hospital and clinics. | Family history interview, quantitative non-randomised (*n = 167; split high alcoholism n = 65, low alcoholism n = 102)* | Semi-Structured Assessment for Genetics of Alcoholism (SSAGA) | High alcoholism family:  1.1-39.8  Low alcoholism:  1.0-41.2 | 81.4% | NR | Socioeconomic status (Hollingshead scale):  High alcoholism:  36% group I & II, 28% group III, 36% group IV & V; low alcoholism: 50.6% group I & II, 25.8% group III, 23.6% group IV & V | * Prevalence of alcoholism in first- and second-degree relatives was 13.7% of ASD probands. * Males (20.3%) were sig. more likely to have a history of alcoholism than females (6.6%). * 39% (n = 65/167) met the criteria for alcoholism pattern consistent with the study criteria for genetic trait. * In high alcoholism families, females were 18 times more affected (38.2% v 8.7%) and males four times (15.9% v .9%) than in low alcoholism families. * Ratio of female to male alcoholism was sig. higher in high-alcoholism families compared to low alcoholism. |
| Roy et al. (2015) | 60% | Outpatient clinic seeking AS diagnosis | Cross-sectional, quantitative non-randomised cohort  (*N = 50)* | SCID-I  (DSM-IV) | M=36.5 (NR)  20-62 | 68% male | NR | Income: 42% own income, 50% financial support, 8% disability pension; Education:  4% ‘none’, 4% ‘special school’, 12% ‘low’, 22% ‘intermediate’, 58% ‘high’; Employment: 46% currently employed, 52% not employed; Living: 48% live alone, 22% with a partner and/or child, 28% live with parents, 2% live in psychiatric nursing | * Alcohol abuse or dependence was present in 18% of the population *(n=*9). * Harmful AUD/dependence prevalence was unequal across genders (males 20%: females 12%). * Prevalence of alcohol dependence (8%) was higher than a German population sample (6.3%). * Age differences were also found, with alcohol dependence only present in elderly individuals (≥40 years). |
| Yule et al. (2023) | 100% | Medical chart review from specialised ambulatory program for ASD | Retrospective case-control, quantitative non-randomised  (N = 679; ASD *n = 230, ADHD n = 219, controls n = 230)* | Clin. ax. using DSM-III-R and DSM-IV for SUD | ASD sample: M=20.0  (10.3)  12-59 | 79% male | 93% Caucasian, other ethnicities NR | Socioeconomic status was measured using the Hollingshead scale.  ASD sample:  M = 2.0  (1.0) | * ASD sample had sig. higher rates of bipolar disorder, major depressive disorder, multiple anxiety disorders, oppositional defiant/antisocial personality disorder, and conduct disorder compared to controls. * Non sig. prevalence of AUD in comparison to controls but sig. lower than participants with ADHD. * Sig. lower risk of developing AUD in ASD than ADHD and non-sig. but a downward trend with controls. * ASD participants were sig. older when they developed AUD compared to ADHDor controls. |

| *ASD and AUD service* | | | | | | | | | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Study** | **Quality assessment a** | **Population/ Setting** | **Design and sample size** | **AUD measure** | **Age b** | **Sex** | **Race/ Ethnicity** | **Socio- economic c** | **Key findings**   * **(Sig. is statistical significance)** |
| Clarke et al. (2016) | 80% | Asperger syndrome service; Drug and alcohol service | Qualitative interviews  (*n = 8)* | SUD dx. based on DSM-IV criteria; combined with DAST and AUDIT | M=35.4  (14.65)  21-55 | 87.5% male | NR | NR | * Six themes for ‘factors contributing to substance use’: social facilitation, self-medication, recreational use of substances, substance use of peers, defining problematic substance use, and a discrepancy between need and support. * Subthemes: substance used to increase social confidence; substances used to facilitate communication. |
| *Forensic service* | | | | | | | | | |
| Anckarsäter et al. (2008) | 40% | Group 1 – Special psychiatric hospital inpatients;  Group 2 – forensic psychiatry;  Group 3 – special institution for adolescents | Case-series, Quantitative non-randomised  (Total *n = 42;* Group 1 *n = 4,* Group 2 *n = 18,* Group 3 *n = 20)* | Medical note review (group 1&3) and structured interviews (group 2) | Group 1: Mdn = 27 (19-46) Group 2:  Mdn = 25.5 (18-47) Group 3:  Mdn = 15 (11-18) | Group 1:  50% male Group 2: 83.33% male Group 3: 70% male | NR | NR | * Prevalence of AUD in individual ASD diagnoses: autism *n = 2/5,* atypical autism n = 3/10. * Prevalence within group 2 only = 27.78%. |
| Chaplin et al. (2021) | 60% | Male prisoners | Cross-sectional, Quantitative non-randomised  (n = 240; n=46 screening positive for ASD traits, of these n = 12 meeting Autism Diagnostic Observation Schedule (ADOS) criteria) | MINI for dx. based on ICD-10 criteria. | Positive ASD traits (n = 46):  20-29: 47.8%  30-39: 21.7% 40-49: 28.3% 50+: 2.2% | 100% male | 80.4% White, 15.2% Black, 4.3% Asian | NR | * Of the 12 who were screened using the ADOS, only 2 were known to the prison to have ASD (83.5% unidentified). * 8/37 with positive ASD traits and 1/11 ADOS confirmed met criteria for alcohol dependency. There were no sig. differences between groups. * There was no reported alcohol abuse across both ASD traits and ADOS confirmed groups. * ASD positive trait sample had sig. more suicide-related behaviours and self-harm compared to prisoners with no neurodevelopmental difficulties. However, there were no differences in the ADOS-confirmed sample. * In terms of comorbid mental health problems compared to no neurodevelopmental difficulties:   ASD positive traits were sig. different across depression, major depression with psychotic features, mania or hypomania, generalised anxiety disorder, social phobia, obsessive compulsive disorder (OCD), and antisocial personality disorder.  In the ADOS confirmed group, social phobia and OCD were sig. different from controls. |

| **Study** | **Quality assessment a** | **Population/ Setting** | **Design and sample size** | **AUD measure** | **Age b** | **Sex** | **Race/ Ethnicity** | **Socio- economic c** | **Key findings**   * **(Sig. is statistical significance)** |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Haw et al. (2013) | 40% | Specialist,  low-secure psychiatric unit for adults | Matched cohort, quantitative non-randomised (*n = 88; ASD n = 45, control n = 43)* | Retrieved from electronic case notes (ICD-10 criteria) | ASD:  Mdn = 27 (NR)  19-57 | 100% male | ASD:  Ethnicity: 88.9% white British, 11.1% other ethnic group | ASD:  Educational support: 53.5% yes, 46.7% no; History of childhood abuse/neglect: 22.2% yes, 77.8% no; No school qualifications: 66.7% yes, 33.3% no; previous employment: 44.4% yes, 55.6% no; marital status: 97.8% single, 2.2% married/divorced; children: 6.7% yes, 93.3% no. | * Prevalence of alcohol use or dependence was sig. lower in ASD compared to controls. * Lifetime history of alcohol abuse was lower, but not sig., in ASD compared with controls. * Intoxicated at the time of index offence was lower in ASD but not sig. different than controls. |
| *Population-based cohorts* | | | | | | | | | |
| Abdallah et al. (2011) | 60% | Danish nationwide health registers. | Matched cohort, quantitative non-randomised (n = 1,234; ASD n = 414, control n = 820) | Register dx. based on ICD-8, ICD-10 codes. | ASD:  M = 16.28  (4.55)  NR | ASD:  80.9% male | NR | NR | * Lower prevalence of alcohol-related disorders (ARD) in ASD compared to controls. * Non sig. crude odds ratio and adjusted odds ratio of ASD having a lower risk of having ARD compared to controls. |
| Butwicka et al (2017) | 80% | Swedish longitudinal, population-based registers. | Longitudinal matched cohort, Quantitative non-randomised  (N *= 1,376,286; ASD n = 26,986; controls n = 1,349,300)* | Registers dx. based on ICD-8, ICD-9 and ICD-10 codes. | ASD:  Year of birth:  17.4% 1970-79; 49.5% 1980-89; 33% 1990-99; (18-47) | ASD: 70.4% male | ASD:  Mother’s region of birth:  82.8% Sweden, 4.2% other Nordic, 12.8% outside Nordic, 0.1% unknown.  Father’s region of birth:  81.8% Sweden, 3.8% other Nordic, 13.8% outside Nordic, 1.2% unknown | ASD:  Family income (percentile): 23.7% <20; 71.5% 20-79; 4.8% ≥ 80.  Education: 59.3% Primary and lower; 31.6% upper secondary; 5.9% post-secondary; 3.2% postgraduate | * Probands had a substantially increased risk of somatic disease linked to alcohol use. * AUD was the third highest-risk substance. Sig. difference between ASD and non-ASD controls for Crude OR which was maintained when adjusted for parental education, family income, and SUD dx before ASD dx. * Sig. difference within ASD of AUD dx compared to non-ASD when using different International Classification of Diseases systems (ICD-8/9 vs 10). * Risk of AUD in ASD compared to non-ASD controls in descending risk order of comorbidity: ADHD, ADHD + intellectual disability (ID), none, ID. Risk order is maintained when accounting for additional multivariate analysis. * Risk of AUD in ASD related to non-ASD controls, who received a neuropsychiatric disorder dx. prior to SUD, descending risk order of comorbidity: none, ADHD, ADHD + ID, ID. Risk order is maintained when accounting for additional multivariate analysis. * Shared AUD liability between ASD group and relatives, descending highest odds ratio: parents, half-siblings, full siblings. |

| **Study** | **Quality assessment a** | **Population/ Setting** | **Design and sample size** | **AUD measure** | **Age b** | **Sex** | **Race/ Ethnicity** | **Socio- economic c** | **Key findings**   * **(Sig. is statistical significance)** |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Chen et al. (2017) | 60% | Taiwan’s National Health Insurance (NHI) | Matched cohort, quantitative non-randomised cohort (N = 28,090; ASD n = 5,618, control n = 22,472) | Medical records using dx codes from ICD-9-CM. | ASD:  Age at enrolment  M = 17.2 (4.58)  12-29 | ASD:  78.2% male | NR | ASD:  Taiwan urbanisation:  18.7% 1 (most urbanised), 30.4% 2, 9.5% 3, 8.6% 4, 32.8% 5 (most rural).  Income-related insurance amount (NTD, New Taiwan Dollars):  86.5% ≤ 15,840 /mo, 12.4% 15,841 – 25,000 /mo, 1.1% ≥ 25,001 /mo. | * Prevalence of AUD in the ASD sample was non-sig. different from controls. * Cox regression analysis found autistic males with AUD were more likely to attempt suicide, stratified by age and sex, during follow-up compared to controls. * Overall, AUD in ASD was related to a sig. increase of suicide attempts compared to controls. |
| Croen et al. (2015) | 80% | Kaiser Permanente Northern California integrated healthcare insurance | Matched cohort, quantitative non-randomised cohort (N = 16,577, ASD *n = 1507; control n = 15,070)* | Medical records using dx codes from ICD-9. | ASD:  M = 29.0  (12.2)  18-65+ | ASD:  73.1% male | ASD:  65.6% White, 3.9% Hispanic, 7.6% Black, 11.1% Asian, 11.7% Other | ASD:  Type of insurance: 73.5% Kaiser Permante, 24.9% Medicaid, 1.7% self-pay | * Sig. difference between ASD adults and controls for the prevalence of harmful alcohol use and dependence. * Self-reported alcohol use was lower in the ASD group. * Autistic females were diagnosed less often than men for alcohol abuse and dependence. * Higher odds ratio for harmful alcohol use and alcohol dependence across male and female ASD participants compared to controls. |
| Hermens et al. (2013) | 60% | Youth Mental Health cohort | Cross-sectional, quantitative non-randomised cohort (N = 2112; ASD n = 22; Alcohol Use Disorders Identification Test (AUDIT) sample *n = 522, of AUDIT sample, ASD  n = 5* | Clin. ax. using DSM-IV criteria, WHO-ASSIST, AUDIT | ASD (n=5)  M = 15.8  (2.3)  12-30 | ASD:  100% male | NR | NR | * The proportion of weekly alcohol use in the total sample: 25% (n=1/4) females and 5.5% (n=1/18) males. * Prevalence of AUDIT categories within the total ASD sample: Abstainers 13.64%, low risk 4.5%, high risk 4.5%. * Prevalence of AUDIT categories within samples with ASD:   Abstainers 1.89%, low risk .56%, high risk 2.17%. |

| **Study** | **Quality assessment a** | **Population/ Setting** | **Design and sample size** | **AUD measure** | **Age b** | **Sex** | **Race/ Ethnicity** | **Socio- economic c** | **Key findings**   * **(Sig. is statistical significance)** |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Huang et al. (2021) | 60% | Taiwan National Health Insurance Programme | Retrospective, matched cohort, Quantitative non-randomised  (N = 32,995, ASD *n = 6599, controls n = 26,396)* | Medical records using dx codes from ICD-9-CM criteria | ASD:  M = 11.9  (5.1)  (<6->18) | ASD: 77.2% male | NR | ASD:  Years of education: 15.5% ≥ 12; Marital status – married: 1.7%; Level of care: 45.9% Hospital centre, 47.5% Regional hospital, 6.7% Local hospital; Charlson Comorbidity Index (CCI): 93.5% 0, 6.1% 1, 0.4% ≥ 2; Urbanicity of residence: 45.8% 1 (highest), 40.1% 2, 13.4% 3, .7% 4; Monthly income: 73.7% <18,000, 16.3% 18,000 – 34,999, 10% ≥ 35,000. | * Adjusted hazard ratios (aHRs) for AUD were sig. higher for the ASD group than non-ASD controls. * Crude incidence of AUD per 100,000 person-years was higher in the ASD group in comparison to non-ASD controls. * Subgroup analysis of AUD ASD subgroups aHRs receiving one psychotropic agent and multiple psychotropic agents were lower than the group receiving no psychotropic agents. * 8 psychiatric comorbidities were found to be associated with an increase in aHRs for AUD compared to the absence of these comorbidities within the ASD group compared to non-ASD controls. * Comparing these comorbidities with the non-ASD sample, aHRs were substantially higher in ASD patients with either anxiety disorder or impulsive control disorder. |
| Langley et al. (2023) | 60% | Secure Anonymised Information Linkage (SAIL) databank. | Matched cohort; Quantitative non-randomised  (N = 43,698, ASD *n =* 5001, ASD controls *n =* 11,427, ADHD *n =* 7738, ADHD controls *n =* 19,532) | Medical records using ICD-10 and NHS READ codes. | ASD (end of follow-up):  M = 19.4  (2.6)  NR | ASD:  78.9% male | NR | ASD:  Welsh Index of Multiple Deprivation (WIMD) quintile (start of follow-up):  M = 3.2  (1.4)  1 = 15%  2 = 16%  3 = 19%  4 = 24%  5 (most deprived areas) = 32% | * In the total sample, 0.9% (n = 5001) had ASD, with diagnosis more likely in males (1.5%) than females (0.4%). * Similar levels of AUD to matched controls. These associations were robust when controlling for sex, record availability, and deprivation. * Higher Cox regression model for males compared to females. |

| **Study** | | **Quality assessment a** | **Population/ Setting** | **Design and sample size** | **AUD measure** | **Age b** | **Sex** | **Race/ Ethnicity** | **Socio- economic c** | **Key findings**   * **(Sig. is statistical significance)** |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Roux et al. (2021) | | 80% | Medicare & Medicaid Services across a range of clinical settings. | Matched cohort, quantitative non-randomised cohort  (N = 2,892,718; ASD only *n = 209,795; ID only n = 790,719; ASD + ID n = 123,705; Control n = 1,768,499)* | Medicaid data using dx codes based on ICD-9 criteria | ASD + SUD only:  51.4% 11-17, 24.19% 18-29, 11.02% 30-45, 13.39% 46-64.  ASD, ID, + SUD: 35.82% 11-17, 32.29% 18-29, 16.13% 30-45, 15.76% 46-64. | ASD + SUD = 71.46% male  ASD, ID + SUD = 71.55% male | ASD + SUD: 64.8% White, 18.7% Black, 1% Asian/Pacific Islander, 7.2% Hispanic/Latino, 8.4% Other.  ASD, ID, + SUD:  62% White, 21% Black, 1.6% Asian/Pacific Islander, 7.3% Hispanic/Latino, 8% Other. | Medicaid enrolment:  ASD + SUD:  32.77% poverty, 55.5% disability, 11.73% other  ASD, ID, + SUD: 8.07% poverty, 85.84% disability, 6.1% other. | * SUD – Alcohol type: all groups were sig. different across chi-square tests for ASD only, ASD and ID, and controls. * Overall AUD prevalence across ASD samples = 1.45%. |
| Underwood et al. (2019) | | 40% | Welsh National Centre for Mental Health | Matched cohort, quantitative non-randomised cohort  (N = 181; ASD *n = 105, control n = 76)* | Case-note review using ICD-10 criteria | ASD:  M= 37.8 (12.3) | ASD:  76.2% | 100% Caucasian | ASD:  Employment: 32% currently working, 49.5% not currently working due to sickness or disablement; Marital status: 50.5% married or cohabiting. | * 9.5% (*n = 10)* met ICD-10 criteria for alcohol use disorder and 36.2% had problems due to alcohol use (*n =* 21). * Sig. increase alcohol-related problems in ASD samplecompared to controls. |
| Yu et al. (2019) | | 80% | Swedish longitudinal, population-based registers. | Matched cohort, quantitative non-randomised cohort  (Major sample with controls *n = 3,859,497;  ASD n = 9,529, ASD controls n = 186,017)* | Dx. from inpatient or outpatient setting based on ICD-10 criteria. | ASD:  Follow-up start age:  M = 17.7  (10.4) | 100% male | NR | 39.5% low income, 99.3% single, 8.4% born abroad, previous intimate partner violence (IPV) .2% | * The crude hazard ratio of perpetrated IPV was higher in the ASD sample with comorbid AUD compared to non-AUD. * Sig. adjusted hazard ratio perpetrated IPV against women by men with ASD and comorbid AUD. * Non sig. hazard ratio of perpetrated IPV against women by men with ASD without comorbid AUD. * Sig. ratio of hazard ratios of perpetrated IPV against women in men with mental health diagnoses, between adjusted hazard ratios, after adjustment of prior alcohol use disorder, between individuals with mental health disorders and unaffected siblings. |
|  | Abbreviations: ADHD: attention deficit hyperactivity disorder; aHRs: adjusted hazard ratios; ARD: alcohol-related disorders; ASD: autism spectrum disorder; AUD: alcohol-use disorder; AUTS2: autism susceptibility candidate 2 gene; CBT: cognitive behavioural therapy; GAD: generalised anxiety disorder; ICD: international classification of diseases; ID: intellectual disability; M: mean; Mdn: median; NHI: national health insurance; NR: not reported; NTD: New Taiwan Dollar; OCD: obsessive compulsive disorder; SUD: substance-use disorder; WIMD: Welsh index of multiple deprivation.  Measures: ADOS: Autism Diagnostic Observation Schedule (85), adapted for prison use (89); AQ: autism-spectrum quotient (90); AUDIT: Alcohol Use Disorders Identification Test (87); CCI: Charlson Comorbidity Index (91); Hollingshead scale: Hollingshead four factor index of social status (92); Taiwan urbanisation (93).  a Quality assessment summarised by Mixed-Methods Appraisal Tool (59, 60) total domain score. Percentage of quality criteria met are presented as star (\*) ratings: \*\*\*\*\* = 100%, \*\*\*\* = 80%, \*\*\* = 60%, \*\* = 40%, \* = 20%.  bAge is reported in years. Mean, (standard deviation), and (range) reported where available.  cSocioeconomic variables reported for ASD samples only where available. In absence, whole sample characteristics were reported. Variables include: education, employment, income, living arrangement, marital status, social support. | | | | | | | | | |

**Study characteristics**

The publication year of included studies ranged from 2003 to 2023, with 45% of studies being published in the past 5 years (2019 to 2023). Studies were primarily conducted in European countries (*n =* 13), followed by the USA (*n =* 4), Japan (*n =* 2), Taiwan (*n =* 2) and Australia (*n =* 1). Study designs consisted of 20 quantitative (case-control *n =* 12, cross-sectional *n = 5,* and cohort studies *n = 3*) and 2 qualitative (interviews *n = 2)*. Overall, samples were derived from a range of settings: drug and alcohol services (*n =* 5), ASD services (*n =* 3), a combination of both ASD/AUD services (*n =* 1), forensic (*n =* 3), and large, population-based registers (*n =* 10).

**Quality appraisal**

Overall, the average quality score of included studies was 66%, with the majority of studies 60% or above (n = 17). Studies that scored below 60% were retained due to limited available literature. The main quality issues within clinical settings were insufficient descriptions of study samples, utilisation of non-validated measures, and absence of controls for confounding variables. In large population-based studies, diagnoses were primarily sourced from medical records, with a large proportion of missing data, and potential recruitment bias within samples due e.g., paid healthcare insurance.

* 1. **Participant characteristics**

A total of 750 ASD participants were represented from clinical settings and 389,281 from large-population registers. The pooled mean of ASD gender was 83.4% male. The ages of included participants ranged from 1.1 years to 85 years, with a median of mean 32.2 years (S.D = 14.58) reported by 16 (72.7%) studies. Of studies that reported socioeconomic status (count = 16) and race and ethnicity (count = 8), records were incompatible for comparison across categories. For studies that reported demographics, participants were primarily White/Caucasian, achieved primary and secondary education qualifications, were unemployed, single, possessed lower socioeconomic scores, and lived in urbanised areas (See **Table 1** for individual breakdown).

* 1. **Prevalence and risk**

Excluding studies with exclusive ASD-AUD samples (i.e. samples that only included individuals with both by deliberate design), prevalence in clinical settings ranged from 6.7% to 39%; while in large population-based registers, the rate varied between 0.7% to 9.5%. Pooled prevalence was higher for clinical settings (121/750, 16.13%) than large-population registers (6,124/389,281, 1.57%).

In large population-based registers, the associated average risk of AUD within ASD patients compared to non-ASD controls, varied significantly. Two studies suggested a decreased AUD risk (62, 63), three studies found similar or no difference in AUD risk (64-66), and four studies indicated increased AUD risk (67-69).

* 1. **Aetiological risk factors**

Risk factors involved in the development of AUD in ASD included genetics, co-occuring conditions, gender, and age.

Three studies of varying quality found evidence for a genetic pattern and increasing average risk between ASD and AUD (67, 70, 71). As both conditions are heterogeneous in origin, a lack of genetic specificity within the literature can only conclude an overall association. The indicated risk of AUD is substantially compounded by co-occurring disorders, such as ADHD (67, 68, 72). However, in comparison to ADHD alone, ASD was associated with a lower average risk of AUD (72). Furthermore, when compared to non-ASD controls, ASD-AUD patients present with higher occurrences of anxiety disorders accentuating the complexity between disorders (68, 73). Gender differences contribute to this complexity, as autistic females were underrepresented and diagnosed less frequently than autistic males in included studies (62, 66, 70). Yet findings present a higher risk for AUD in autistic females, indicating a nuanced relationship given the unequal distribution across samples and overlapping confidence intervals with male counterparts, which requires further study. In addition, there was an emerging pattern of alcohol use developing in older ASD patients when compared to controls (72, 74).

Two qualitative studies explored the development and everyday life consequences of SUD in ASD (75, 76). Emerging themes indicated two interrelated alcohol use coping motives: self-medication and social facilitation. Proactive strategies position alcohol use as a means to self-medicate symptoms associated with cognitive and emotional distress, as well as idiosyncratic features of ASD such as sensitivity to sensory processes (75, 76). Reactive strategies occur in the context of social situations, to facilitate interactions, or cope with associated negative appraisals. ASD patients described the inability to express themselves and anticipated social rejection, leading to feelings of loneliness and social exclusion (75, 76). Through alcohol use, insecurities and oversensitivity can be mitigated by feeling more confident and comfortable in social situations (75, 76). This reinforces the use of alcohol due to elevated mood, social gains, connection, and alleviation of social anxiety (75, 76).

* 1. **Implications of alcohol use**

From this review, the literature suggests a continued pattern of alcohol use could impact general functioning and increased harmful experiences, such as somatic disease, intimate partner violence, and suicide attempts (65, 67, 76, 77). Further analysis of suicide attempts, stratified by age and sex, found only autistic males had a significant increase in average risk, compared to controls, lending further support to gender differences (65). The included study of intimate partner violence only investigated the perpetration against women by men with comorbid ASD and AUD. Hazard ratios of engaging in intimate partner violence against women by men were significantly greater in autistic people with comorbid AUD (77).

The consequences of alcohol-related problems can be maintained by specific ASD traits (78). A series of exploratory Pearson chi-square tests found traits of rigidity, social avoidance and withdrawal, were significantly related to harmful drinking patterns. For example, rigidity in thought could create a barrier to implementing lifestyle change. The authors questioned whether these specific traits were indicative of long-term difficulties, such as anxiety and inflexibility to adapt to social cues, or the overlap of these traits in the spectrum of alcohol use. Although, ASD traits were not found to be related to rates of abstinence at follow-up (78, 79). If certain characteristics of ASD sustain harmful patterns of alcohol use, current intervention methods may not adequately address patient needs, and greater accommodation for ASD traits may yield greater outcomes (78). As such, adapted ASD-AUD treatment, with a focus on core ASD symptoms, social skills and ASD-related stress, presented promising results requiring further study (80).

Potential protective factors of developing AUD in ASD included psychotropic medication use and a prior diagnosis of ASD. Huang et al. (68) found a reduction in overall AUD average risk if autistic patients had received one or multiple psychotropic medications, even in the context of additional co-occurring disorders. Psychotropic medication was classified as antidepressants, second-generation antipsychotics, and mood stabilisers. The risk of AUD was decreased amongst patients who had received a prior diagnosis of ASD, for both patients with no co-occurring conditions and co-occurring ADHD (67). However, one forensic study found the majority of cases, that met ASD diagnostic criteria, were previously unknown to providers (73).

A diagram with text and images

Description automatically generated with medium confidence

**Box 1 – Concept map of findings for alcohol use in clinical, autistic populations**

1. **Discussion**

This narrative systematic review aimed to identify relevant clinical studies, explore the aetiology and implications of alcohol use in ASD, and suggest future research, policy, and clinical recommendations. The final review included 22 studies, extracting data from healthcare settings and large, population-based registers. The findings of this review stress the variable nature of studies investigating AUD in ASD. Due to divergent quality and heterogeneous parameters, our overall findings are speculative.

Of the included studies, the prevalence of AUD in ASD appears to range between 1.6% in large-population registers and 16.1% in clinical settings. Within global populations, irrespective of neurodiversity status, the lifetime prevalence of AUD is estimated to be 8.6% (81). This is substantially greater than the rate found in this review, potentially supporting the evidence that autistic populations generally report lower rates of alcohol use (17-19). This is further compounded given the low global prevalence rates of ASD (8), despite the recent suggested increase in ASD diagnoses (5). With regard to clinical settings, the observed prevalence in this review is greater than that of one study (11.8%) which compared rates of AUD in European primary care to a general population (82). However, Manthy (82) did not differentiate for neurodiversity status. It is possible AUD is exacerbated by ASD symptoms, and given the higher observed rate in this review, screening within clinical services is important to explore the intersection between disorders and improve the precision of prevalence rates.

This review identified four possible risk factors for the development of AUD in ASD: age, co-occurring conditions, gender, and genetics. Based on the broader literature and this review, both conditions may share genetic vulnerability (33, 67, 70, 71). Although specific genes are not clearly identified, studies considered family history and ancestry (67, 70). Adopting an epigenetic viewpoint, considering parental monitoring and alcohol use can influence the development of AUD (37, 40, 47), it would be interesting to investigate the associations between genetics, family environment, and AUD development across the severity and lifespan of ASD.

Findings suggest the risk of AUD increases with age in ASD and in the presence of co-occurring difficulties in clinical samples, an observation found in other literature within non-clinical samples (19). However, the risk of AUD could be decreased with a prior diagnosis of ASD (67). We hypothesise that a prior diagnosis of ASD explains some daily life experience and potential availability for support to manage symptoms. In the absence of appropriate support, ASD patients may seek other means to manage or alleviate symptoms. This may also offer an explanation as to why the risk of AUD was reduced for ASD populations receiving psychotropic medication (68).

Alcohol use may lead to reduced sensory perception and inhibit cognitive processing, such as accessibility to self-critical memories, leading to greater symptom control (75, 76). Such motivations have been explored in non-clinical ASD samples yielding similar results (17, 49-52). In comparison to general populations, motivations for alcohol use can fall into two categories: enhancement and coping (83). Both of these predictors can lead to alcohol use problems. Yet, using alcohol to cope can lead directly to alcohol use problems, whereas enhancement is indirectly associated with use through alcohol use problems. This is comparable to the interrelated factors of self-medication (coping) and social facilitation (enhancement) found in this review for ASD-AUD (75, 76).

The processes of how harmful alcohol use develops in ASD are unclear. Within wider literature, Cho et al. (84) found longitudinal associations for two reinforcement cycles related to alcohol dependence, with a stronger association for negative reinforcement. In the context of ASD and this review, improved social interaction (positive) and relief from sensory processes (negative) could form reinforcing maintenance loops (See **Box 1**). Therefore, the function of alcohol use for ASD people could influence how AUD develops and provide a theoretical target for intervention. However, existing approaches would require appropriate adaptation to reduce barriers, create shared understanding, and meet specific ASD population needs (48).

* 1. *Principles of care and research*

The findings of this review highlight the significant need for research to improve clinical practice for ASD-AUD patients. In **Box 2**, we have provided guiding principles for clinicians and researchers to consider and take forward, based on the review findings. Seven principles are outlined across assessment, consideration for co-occurring difficulties and life course, prevention, function of use, education and training, and adapted treatment. We offer a further four novel research recommendations.

|  |  |  |
| --- | --- | --- |
| **Box 2**  **Seven guiding principles related to aetiology and implications of alcohol use and Autism. Included recommendations for clinical practice and novel research ideas.** | | |
| **Domain** | **Guiding principle** | **Recommendation** |
| Assessment | AUD in ASD is not routinely assessed within clinical services or research using universal, standardised measures. Autistic people usually receive a diagnosis later in life, increasing potential harm from alcohol use. | Clinical: - Screen for alcohol use in ASD using gold standard measures (e.g., ASSIST-Lite).  - Measure alcohol use using standardised measures (e.g., AUDIT).  - Use of existing ‘gold-standard’ assessments for ASD (e.g., ADOS) and AUD (e.g., AUDIT or SCID) for diagnosis.  Research:  - Establish minimum dataset and measures to use in ASD and AUD research.  - Development of a diagnostic tool to assess AUD in ASD, accounting for the severity of ASD, harm from AUD, and motivations for alcohol use. This tool would benefit from an alternative approach to existing measures based on gender-specific criteria. |
| Co-occurring difficulties | Patients who have psychological comorbidities, such as  ADHD, ID, or low mood, increases the risk of AUD in ASD. | Clinical:  - Patients should be offered a comprehensive assessment accounting for co-occurring mental health difficulties.  - Working alongside co-occuring conditions. Targeted treatment for the function of alcohol use or most influential comorbid disorder (e. ADHD).  Research:  - Impact of comorbidity on development and life course of AUD in ASD. |
| Life course | Emerging pattern of AUD  developing later in life for ASD  patients. | Clinical:  - Clinicians should screen for ASD and hold ‘sensible’ conversations with AUD patients. Appreciation of neurodiversity as a consideration of the patient’s story.  - Existing services should develop links across ASD and drug and alcohol services to increase joined-up care.  Research:  - Risk factors, triggers, and facilitators in the development of AUD.  - Timing of diagnosis and impact on life (potential harms). |
| Prevention | Normalising the attitudes and beliefs of alcohol use in  younger ASD patients. | Clinical:  - To educate neurodiverse young adults on the associated risks of alcohol use.  - To provide realistic expectations as an aspect of education from a non-stigmatising stance.  - Early intervention for emerging adulthood ASD and AUD patients. Adapted to allow for time for generalisation-flexibility in the length of time to see a client.  Research:  - Development of co-produced programs designed to educate neurodiverse young adults on alcohol use and reduce stigma based on behavioural principles. |
| Function of use | The use of alcohol for social facilitation or self-medication is more likely to reinforce harmful use. | Clinical:  - Bespoke formulation of motivators and functions-including specific ASD experiences.  - Develop healthy coping strategies.  Research:  - Research investigating the link between social facilitation and self-medication.  - Consideration for severity and cross-spectrum differences within ASD and AUD. |
| Adapted treatment | Adapted treatment has the potential to be effective. Features to include an aim to increase a sense of control in daily life, and change dysfunctional beliefs, and coping strategies. | Clinical:  - Emphasis on understanding how autism impacts the patient (function/features) and adapting interventions. These considerations extend to alcohol-based interventions such as detox or rehabilitation. Creating neurodiverse friendly environments (quiet zones etc). - Proactive role in intervention planning such as providing extra time for therapy or support to access ‘safe’ recovery spaces e.g., AA for neurodivergence. - Psychoeducation on possible connections between AUD and ASD. Coping with ASD-related stress (e.g., sensory overload). Research: - Manualised intervention package to be tested.  - Efficacy in comparison to treatment as usual.  - Active ingredients. - Use of buddies in treatment programs. |
| Education and training | Neurodiverse patients have the potential to go unidentified in forensic populations. | Clinical:  - Staff training to understand how to identify and work with ASD.  - Increased support for forensic patients with ASD. A potential pathway for screening and referring to local, psychologically informed environments.  Research:  - Development of a cross-clinical setting training package for working with ASD, with consideration for AUD. |
| Novel research recommendations:   1. Autistic traits did not impact abstinence rates or whether ASD and AUD patients changed consumption.    * Exploratory research to understand why abstinence rates are not related to ASD. Initial findings suggest this is related to rigidity and social deficits. Factors which are associated with maintenance of harmful use. 2. Biomarkers for risk of AUD in ASD.    * Following genetic patterns and early evidence for shared links between ASD and AUD. Additional biological and genetic research should be completed. 3. Family support    * To explore systemic approaches for change/skill development and to help implementation in social environments. Working alongside family, support networks, and clinical teams. 4. Gender differences. | | |
| * + Females are underrepresented in existing research. There is a dearth of research on gender differences which requires examination. | | |
| Abbreviations: AA: alcoholics anonymous; ADHD: attention deficit hyperactivity disorder; ADOS: Autism Diagnostic Observation Schedule (85); ASD: autism spectrum disorder; ASSIST-LITE: Alcohol, Smoking and Substance Involvement Screening Test short-form (86); AUD: alcohol-use disorders; AUDIT: Alcohol Use Disorders Identification Test (87); ID: Intellectual disability; SCID: Structured Clinical Interview for the DSM (88). | | |

To outline some of our principles and recommendations, we draw on the findings of this review and the wider literature. As highlighted, autistic people may be accessing services undiagnosed (73), and if a timely diagnosis could protect autistic people from developing AUD (67, 72), routine assessment could prevent future harm. However, as the waiting time for ASD assessment grows (9), current services could implement screening tools to inform clinical formulations. Hence, the development of a specific ASD-AUD screening tool may benefit future research. Subsequent studies should consider the overrepresentation of male participants, as indicated by this review, and the potential bias of some existing measures towards males (85). In addition, greater awareness is a promising sign of advocacy for the needs of autistic people, yet this does not necessarily translate to available services (8). Cross-service collaborations may prove fruitful for developing individual pathways to share resources. Similar to harm-reduction strategies, co-produced research, education, and training could inform future prevention strategies.

A further interest is the emerging pattern that AUD develops later in life for autistic people (72, 74). This poses the question of whether this could be attributed to the change in diagnostic criteria over time (67) or to the limited resources to diagnose autistic adults (9). We could also question whether it could be a result of the function or motivation to use alcohol as a coping strategy, as availability increases in adulthood (75, 76). Future longitudinal studies could explore the development of AUD and functions of use over time in autistic adults. This in turn could direct adapted treatment, such as one included study (80), and future randomised controlled trials to test for efficacy.

* 1. *Limitations*

Due to the inconsistency of reporting demographics and severity of both spectrum disorders, comparisons between studies are difficult to establish. Without knowing the specificities across the spectrum of ASD with AUD present, it is likely to be difficult to meet the needs of this patient group adequately. The findings of this review should be taken with caution due to varied sample sizes, absence of control groups, and lack of consideration for confounding variables. Furthermore, diagnoses sourced from medical records do not specify how assessments were conducted or which diagnostic tools were used. These issues are deepened by the differences in conceptualisation across classification manuals. In addition, this review focused on clinical samples only, excluding research on non-clinical samples, which could disregard existing applicable findings. Furthermore, the use of the MMAT to appraise the quality of studies may overlook methodological concerns which include more variables with non-standardised measures. None of the included studies were randomised controlled trials, and despite identifying possible associations, this review lacks an the exploration of casual relationships.

This review, the first of its kind, highlights emerging trends and areas for future development in research and clinical practice. Included studies have identified some possible factors that may be associated with the development of AUD in ASD, yet further research is required. Future research would benefit from carefully-defined variables, such as those identified by reviews like this, with the aim to longitudinally identify both causative factors and effective management strategies.

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**Supplementary Materials**

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| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Table 2: Mixed Methods Appraisal Tool (MMAT) ratings for each study with quality appraisal comments on strengths, weaknesses, and relevance to review** | | | | | | | | | | | |
| **Studies** | |  | **MMAT Criteria** | | | | | | **Comments** | | |
| Author | Year | Type | 1 | 2 | 3 | 4 | 5 | % | Main strengths | Main weaknesses | Study relevance to question |
| Hildebrand et al. | 2021 | 3. Quantitative non-randomised | 1 | 0 | 0 | 0 | 1 | 40 | • Long period of data collection (2004-2012) across two treatment centres with a clear description of the study sample, including exclusion criteria. • Differences between participating and non-participating groups compared. | • Autistic personality traits (APTS) were conceptualised as items on the Structured Clinical Interview of Personality Disorders (SCID-II) and Temperament and Character Inventory (TCI) to represent domains of Wing's Triad (unvalidated). • Significant drop-out at post-treatment (40.5%). • No method to control for confounders. | Study aimed to explore whether APTs were related to alcohol-use disorder (AUD) characteristics and their impact on treatment outcomes. |
| Kronenberg et al. | 2014 | 1. Qualitative | 1 | 1 | 1 | 1 | 1 | 100 | • Appropriate approach for study.  • Data reached saturation and findings consistent with quotes used.  • Wide range of topics to explore everyday behaviour across different life domains. |  | Studied patient perspectives of everyday consequences living with substance-use disorder (SUD) and co-occurring autism spectrum disorder (ASD). |
| Narita et al. | 2016 | 3. Quantitative non-randomised | 0 | 1 | 1 | 1 | 1 | 80 | • Clearly defined method for polymerase chain reaction for elucidating frequencies of polymorphism.  • Complete dataset.  • Case-control design with healthy controls. | • Poor description of sample with some data unavailable. | Autism Susceptibility Candidate 2 (AUTS2) gene is implicated in the development of ASD. Exploring genetic factors of AUD could illicit a link. |
| Walhout et al. | 2022 | 3. Quantitative non-randomised | 1 | 1 | 0 | 0 | 1 | 60 | • Clearly defined sample with similarities to other research ASD samples.  • Validated measures used to capture variables of interest. | • Significant discontinuation of participation at time 1 (47.4%) and time 2 (52.6%).  • No control group. | Adapted cognitive behavioural therapy (CBT) treatment for ASD and co-occurring SUD from an Addictions Treatment Centre. |
| Yoshimura et al. | 2022 | 3. Quantitative non-randomised | 1 | 1 | 1 | 1 | 1 | 100 | • Representative clinical sample.  • Validated measure.  • 19.6% drop-out.  • Use of multiple cox proportional hazard analyses to examine confounding variables. |  | Study sought to investigate comorbidities and neurodevelopmental characteristics of dependent AUD after hospital treatment. |
| Miles et al. | 2003 | 3. Quantitative non-randomised | 0 | 0 | 0 | 0 | 1 | 20 | • Participants with full data only. | • Family history interview to ascertain alcoholism.  • Of 333 referrals, only 167 had complete family history data.  • Some control comparisons but overall results do not account for confounders. | Use of family history method to determine the prevalence of alcoholism in ASD. |
| M. Roy et al. | 2015 | 3. Quantitative non-randomised | 0 | 1 | 1 | 0 | 1 | 60 | • Use of validated measures to explore study variables.  • Complete dataset.  • No changes in observations. | • Small sample that could represent milder forms of Asperger’s Syndrome due to the method of diagnosis.  • Confounders not accounted for. | Study examined comorbidities and the course of Asperger’s Syndrome across different areas of participant’s lives. |
| Yule et al. | 2023 | 3. Quantitative non-randomised | 1 | 1 | 1 | 1 | 1 | 100 | • Wide sample with limited exclusion criteria.  • Use of structured, systematic assessments to examine diagnoses.  • Minimal data loss with no changes in exposure.  • Two control groups accounting for confounders with related samples. | • Of note, study did not use validated clinical diagnostic measures such as ADOS/ADI-R. However, process of diagnosis from structured interview is detailed, accounting for rater reliability. | Investigates risk factors related to developing AUD within ASD, ADHD, and controls. |
| Clarke et al. | 2016 | 1. Qualitative | 0 | 1 | 1 | 1 | 1 | 80 | • Consistent data collection methods in line with general practice.  • Use of negative case analysis to ensure representation of experiences.  • Interpretation and analysis coherence. | • The sample was limited and did not reach saturation. Predominantly male and lacked diversity. | Explored whether the experiences of having Asperger Syndrome contributed to development of SUD and facilitative mechanisms. |
| Anckarsater et al. | 2008 | 3. Quantitative non-randomised | 1 | 0 | 0 | 0 | 1 | 40 | • Multiple forensic settings. | • Medical chart review of alcohol use.  • Missing data (group 3).  • Confounders not accounted for. | Prevalence of ASD in forensic institutions alongside an overview of co-existing problems and other clinical features. |
| Chaplin et al. | 2021 | 3. Quantitative non-randomised | 1 | 1 | 0 | 1 | 0 | 60 | • Clear inclusion/exclusion criteria.  • Wide range of measures used to assess.  • Matched within with non-positive ASD traits. | • Missing data.  • Change in screening tool during observation.  • Literature on the use of screening tools with prisoners is limited. | Investigated ASD vulnerabilities and methods of screening in a prison population. |
| Haw et al. | 2013 | 3. Quantitative non-randomised. | 0 | 0 | 1 | 0 | 1 | 40 | • Limited missing data (*n = 6).*  • No changes in patient status. | • Tertiary referral service preventing generalisation.  • Majority of diagnoses retrieved from clinical notes.  • A control sample was used but was significantly different to the ASD group. | To describe characteristics of adult male ASD patients compared to non-ASD controls admitted to low-secure units. |
| Abdallah et al. | 2011 | 3. Quantitative non-randomised | 1 | 0 | 1 | 1 | 0 | 60 | • Nationwide dataset.  • Complete dataset.  • Frequency-matched cases based on gender and year of birth. | • Short study follow-up period.  • Coded diagnoses in databases using two different International Classification of Diseases systems (ICD-8/10). | Brief report to estimate psychiatric comorbidity rates of ASD |
| Butwicka et al. | 2017 | 3. Quantitative non-randomised | 1 | 0 | 1 | 1 | 1 | 80 | • Large, representative sample.  • Complete outcome data.  • Stratified regression models accounting for sex, birth year, and country of birth.  • Multivariate analyses adjusted for family income, parental education, and country of origin. | • Coded diagnoses in databases. | Investigates the risk of alcohol-related problems in ASD and associated comorbidities. |
| Chen et al. | 2017 | 3. Quantitative non-randomised | 0 | 0 | 1 | 1 | 1 | 60 | • Complete outcome data.  • Matched control sample (1:4 ratio) based on age, sex, and time of enrolment. | • Help seeking sample only.  • Diagnosis retrieved from medical records (diagnosed by board-certified psychiatrists). | Explores the risk of suicide of young adults with ASD considering confounding factors such as alcohol use. |
| Croen et al. | 2015 | 3. Quantitative non-randomised | 1 | 0 | 1 | 1 | 1 | 80 | • Large, ethnically diverse study population.  • Complete outcome data.  • Multivariate, logistic regression model controlled for sex, age, race/ethnicity. | • Non-validated diagnoses. | Determines prevalence of psychiatric and medical conditions among large population of ASD sample across ages. |
| Hermens et al. | 2013 | 3. Quantitative non-randomised | 1 | 0 | 0 | 1 | 1 | 60 | • Large sample across five years.  • Use of logistic regressions to account for diagnosis and age. | • Diagnosis made by variety of different assessing professions.  • Only sub-sample of participants completed self-report alcohol measure. | Determined rates of alcohol use in young people entering mental healthcare. |
| Huang et al. | 2021 | 3. Quantitative non-randomised | 0 | 0 | 1 | 1 | 1 | 60 | • Randomly selected control at 1:4 ratio, matched by sex, age, and index date.  • Adjusted hazard ratios accounting for a wide range of variables. | • Relies on medical records for diagnosis.  • Relatively small sample in comparison to original cohort, with a proportion excluded due to original diagnosis date and missing data. | Explores risk of SUD, associated comorbidities, and mortality risk amongst ASD patients compared to non-ASD controls. |
| Langley et al. | 2023 | 3. Quantitative non-randomised | 1 | 0 | 0 | 1 | 1 | 60 | • Large, representative sample.  • Matched control sample.  • Feasibility tested. | • Diagnosis drawn from medical records.  • One of the combined databases had 19% missing data. | To establish the feasibility of a nationwide e-cohort of ADHD and ASD for future longitudinal research. |
| Roux et al. | 2022 | 3. Quantitative non-randomised | 1 | 0 | 1 | 1 | 1 | 80 | • Very large sample of Medicaid enrollees.  • Sample matched to control sample by same-age enrolees. | • Diagnosis drawn from medical records. | To characterise the population of ASD, ASD+ID with and without SUD to estimate the prevalence of SUD and adjusted risk. |
| Underwood et al. | 2019 | 3. Quantitative non-randomised | 0 | 0 | 0 | 1 | 1 | 40 | • Use of a control group. | • Recruitment bias.  • Use of non-validated measures.  • Missing data. | Examines demographic, social, psychiatric, and physical health characteristics of the cohort presenting with ASD in adulthood compared with controls. |
| Yu et al. | 2019 | 3. Quantitative non-randomised | 1 | 0 | 1 | 1 | 1 | 80 | • Large, varied sample.  • Minimal missing data.  • Confounders accounted for in regression analysis and comparisons across demographics e.g. income, marital status, immigration status etc. | • Diagnosis drawn from medical records. | To investigate the risk of IPV against women among men, including ASD and SUD. |