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Review Article

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Alcoholism Research: A Narrative Review

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ABSTRACT

This literature review on alcoholism publications from the last few years suggests that several negative and somewhat disparate effects have been noted for alcoholism use disorder (AUD). These include depression, gastrointestinal conditions, neuroimmune deficits, inferior executive function, greater cortisol regeneration, decreased iron in the hippocampus, accelerated brain age and gait ataxia. A few risks/predictor variables have been noted for AUD in this current literature including lower IQ and cognitive performance, subjective time dilation and greater sleep latency. Only a couple studies in this current literature on AUD have addressed potential underlying biological mechanisms for AUD including less central amygdala neuroimmune signaling and impaired dopamine synthesis. At least four studies on interventions for alcoholism have appeared in this current literature including studies on prevalence data, brain stimulation, medications and ketamine.

Alcoholism, clinically known as alcohol use disorder (AUD), is a chronic, relapsing brain disease characterized by an inability to stop or control alcohol use despite severe social, occupational and/or health consequences. The symptoms include high tolerance, cravings and withdrawal often causing severe, long-term damage to the heart, liver and brain. Although no single cause exists, the risks include genetics, family history, and mental health conditions including depression, PTSD, ADHD and high stress levels.

This narrative review includes current research on the negative effects of alcoholism, the risk factors/predictor variables for experiencing alcoholism, potential underlying biological mechanisms for alcoholism and interventions for alcoholism. This research was found on PubMed and PsycINFO by entering the term alcoholism and the years 2023-2026. Exclusion criteria were protocols, case studies, and non-English language papers.

The 16 papers in this current literature review can be categorized as negative effects of alcoholism (7 papers), risks/predictor variables for alcoholism (3 papers), potential underlying biological mechanisms (2 papers) and interventions (4 papers). These sections are followed by a discussion on methodological limitations of this current literature.

Negative Effects of Alcoholism

Several negative and somewhat disparate effects have been noted for alcoholism in this current literature. These include depression, gastrointestinal conditions, neuroimmune deficits, inferior executive function, greater cortisol regeneration, decreased iron in the hippocampus, accelerated brain age and gait ataxia.

In a paper entitled "Psychological dimensions in alcohol use disorder: Comparing active drinkers and abstinent patients" (N= 150 patients), the active drinkers were more depressed [1]. The active drinkers were also more impulsive, and they experienced more alexithymia, and hopelessness, all potentially confounding the depression effects. Surprisingly, no negative effects were noted for the abstinent patients.

In the study on **gastrointestinal conditions,** the prevalence of GERD (gastroesophageal reflux disease) was as high as 25% [2]. In a paper entitled "Greater **neuroimmune system deficits** in women than men with alcohol use disorder", 41 with mild to moderate alcohol use disorder and 41 controls were given a PET scan [3]. The women were noted to have greater hippocampal and cerebellar neuroimmune deficits than the men. They also had inferior executive function (managing tasks, emotions and goal – directed behavior and less working memory, self-control, and cognitive flexibility). The women may have been more affected both neurologically and cognitively because of their greater exposure time due to their slower alcohol metabolism.

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In a study entitled "Imaging, a putative marker of brain cortisol regulation in alcohol use disorder", PET imaging was conducted on nine individuals with moderate to severe AUD versus 12 controls [4]. In this sample, AUD was defined as 52 drinks per week over six days per week which would mean an extremely high average of as many as 9 drinks per day. Not surprisingly, the results of the PET scans suggested greater brain availability of the **cortisol– regenerating enzyme**, suggesting greater hypothalamic pituitary adrenal activity in this AUD sample.

Table 1: Negative Effects of AUD (and first authors)

Negative Effects	First Authors
Depression	Zizzi
Gastroesophageal disease	Schiel
Neuroimmune system deficits	Zakinaeiz
Cortisol-regenerating enzyme	Verplaetse
Changes in brain metals	Jones
Accelerated brain age	Kamarajan
Gait ataxia	Mistarzet

In research entitled "Alcohol use disorder affects brain metals", 50% of the selected sample (N= 40) had been diagnosed with alcohol use disorder [5]. A decrease in iron was noted in the hippocampus of those with AUD. In females, increases in iron and copper were noted in the substantia nigra. In males, decreases in iron and zinc were noted in the substantia nigra. In the caudate nucleus a doubling of iron was reported for females and an increase in copper was noted in both females and males. The **changes in brain metals** were complex and difficult to interpret. The negative effects were more severe in females which may again relate to greater exposure time due to slower alcohol metabolism in women.

These changes in brain metals may have, in turn, contributed to the **accelerated brain age** noted in another sample with alcohol use disorder [6]. An increase of 1.7 years in brain age was correlated with impulsivity in this sample (N=30 with a history of AUD and 30 healthy controls). This correlation was surprising given that greater impulsivity is more frequently noted in younger people.

Gait ataxia Is still another negative effect that has been noted for individuals with AUD. In a systematic review, as many as 8 of 10 studies on AUD showed gait ataxia (an abnormality that typically results from damage to the cerebellum) [7].

Table 2: Risks/predictor Variables for AUD (and first authors).

Risks/predictor variables	First Authors
Low IQ scores and inferior cognitive performance	Capusan
Long perceived time intervals	Wu
Long sleep latency	Yu

Risks/Predictor Variables

A few risks/predictor variables have been noted for AUD in this current literature. They include lower IQ and inferior cognitive performance, subjective time dilation and greater sleep latency.

Lower scores on general intelligence and cognitive performance tests have been notable risk factors for AUD in at least one study [8]. In this sample from the Swedish military (N= 645,488 males), those with AUD had **lower IQ scores and inferior cognitive performance.** These negative effects could have derived from the changes in brain metals as already noted [5].

In a paper entitled "Subjective time dilation in abstinent patients with AUD", those with AUD **perceived time intervals as longer** (N= 30 abstinent male patients and 30 sex, income, age and education matched controls) [9]. The subjective time dilation was associated with greater craving for alcohol. The subjective time dilation and greater craving may have related to the memory of "waiting for happy hour".

Longer sleep latency has also been a risk factor for AUD in a large sample (N=27,913) [10]. In this research, longer sleep latency was not only correlated with AUD but was also correlated with anxiety and depression which have also been associated with longer sleep latency. Anxiety and depression are likely confounding variables as well as predisposing factors in all of these AUD studies, although they are frequently not measured.

Potential Underlying Biological Mechanisms

Surprisingly, only a couple studies in this current literature have addressed potential underlying biological mechanisms for AUD. These mechanisms include less central amygdala neuroimmune signaling and impaired dopamine synthesis.

Table 3: Potential underlying biological mechanisms for AUD (and first authors).

Mechanisms	First Authors
Less central amygdala neuroimmune signaling	Melkumyan
Impaired dopamine synthesis and release	Bach

In the study on **less central amygdala neuroimmune signaling,** chronic alcohol use was said to disrupt the signaling [11]. The disruption in signaling was noted to alter cytokine expression (regulation of immunity and inflammation) and the activation of glial cells (cells that provide physical and chemical support to neurons).

Impaired dopamine synthesis and release have been noted in molecular imaging studies of AUD including PET and SPECT studies [12]. The authors noted the impaired dopamine synthesis and release as a potential underlying deficiency in a reward mechanism that predisposes to AUD. They further suggested that these were acquired alterations in dopamine signaling. That interpretation is tenuous as this was not a longitudinal study.

Interventions for Alcoholism

At least five studies on interventions for alcoholism have appeared in this current literature. They include studies on treatment prevalence data, brain stimulation, medications and ketamine.

Significant disparities have been noted in the treatment of AUD among US participants (N= 18,692 patients) [13]. In this large sample, as many as **70% were not receiving treatment,** and

only 11% were receiving meds, 14% psychotherapy and 5% a combination of medication and psychotherapy.

Brain stimulation was given in another study [14]. This involved deep brain stimulation of the nucleus accumbens or non-surgical stimulation of the dorsolateral prefrontal cortex or medial prefrontal cortex and anterior cingulate cortex by **transcranial magnetic stimulation.** This noninvasive treatment was effective for AUD just as it has been effective for depression, OCD and migraine headaches.

Table 4: Interventions for AUD (and first authors).

Interventions	First Authors
11% medications, 24% psychotherapy, 5% combination	Le
Transcranial magnetic stimulation	Ygael
Asenapine medication	Rozema
FDA approved medications	Menge
Ketamine	Kelson

In a study entitled "Mining clinical data for novel medications to treat AUD", small effects were noted for most drugs [15]. **Asenapine**, an atypical anti-psychotic, had a large effect. However, this was not among the FDA approved medications.

In a study entitled pharmacological interventions for AUD, a list was given of those **medications that are currently FDA approved** for the treatment of AUD [16]. These included disulfiram, oral naltrexone and comprosate.

Ketamine has been an effective medication for those not responding to FDA – approved first line agents according to a systematic review [17]. In this review, 7 of 11 studies included participants with AUD (N=854 patients with sample sizes ranging from 5 to 211). The most therapeutic effects were noted in those who received ketamine and psychotherapy.

Methodological Limitations of this Literature

Several methodological limitations can be noted for this literature. They include heterogeneity of the definitions of AUD as well as variability in sampling methods, in measures, and in results.

The heterogeneity in AUD criteria has included some researchers defining AUD as mild, moderate or severe. Others sampled participants with self – diagnosed AUD or a history of AUD or patients who were at risk for AUD. The most extreme criterion for AUD was 52 drinks per week for six days per week. Most of the studies involved patients who were being treated for AUD, suggesting that the effects were less negative than expected due to the treatment being received. Sample sizes were also highly variable, ranging from 5 to 645,488.

The control groups varied in terms of the degree of matching. Some AUD groups were simply matched with a non-AUD group. Only one study matched the AUD and non-AUD groups on sex, age and education. Gender differences showed significantly greater effects for women which was thought to be related to slower alcohol metabolism. These consistent gender differences highlighted the need for matching at least for gender.

Heterogeneity was also noted for measures. For example, imaging measures varied from PET to SPECT to fMRI with different brain regions being affected by AUD depending on the type of imaging. Variable contributions of genetics and pre-existing vulnerability traits are also a problem for this area of research. Despite these methodological limitations, this literature highlights the types of future research that are needed on the effects of AUD, risk factors, potential underlying biological mechanisms and effective interventions.

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