The Alcohol Blackout

Amy Du Beau, PhD October 2022

Exposé exploring the mechanisms of ethyl alcohol induced memory lapses from a neuroscientific and legal perspective.

Contents

Abbreviations and symbols: BAC, blood alcohol content; BrAC, breath alcohol concentration; C, carbon; dl, deciliter; GABA, γ-aminobutyric acid; g, gram; H, hydrogen, ml, milliliter; O, oxygen; ≈ approximate; ≥ greater than or equal to

Metric units: deci = 10^{-1}, milli = 10^{-3}

Keywords: Behavior, Chemistry, Criminality, Forensic, Genetics, Investigation, Law, Memory, Neuroscience, Police

Introduction

Because alcohol is so commonplace in our society, we might take for granted that it is a powerful drug. Alcohol-induced amnesia known as 'blackouts' are associated with an array of negative consequences, both biomedical and legal. The use of alcohol is reportedly implicated in greater than half of all violent crimes (Pressman & Caudill, 2013). If an alcohol blackout coincides with the commission of crime, the outstanding issue is determining culpability and intentionality as well as accurately reconstructing events. Making inferential assessments about a drinker's mental state can be a challenging task for legal experts. Therefore, understanding the underlying mechanisms of alcohol blackouts is requisite. This exposé investigates how alcohol affects the brain's memory and decision-making processes from a neuroscientific perspective with respect to legal considerations.

Methods, limitations and scope

Current literature was reviewed using the PubMed National Center for Biotechnology Information database, focusing on modern research articles. This exposé is not intended to be an exhaustive review, rather addressing salient aspects of alcohol-induced blackouts with forensic application. Figures were created using Microsoft's PowerPoint software program (2016 version). **Figure 1** is from an online source. For **Figure 3**, a standard acrylic anatomical brain model was digitally photographed, then cropped and labelled using Adobe Photoshop Elements 13 Expert software.

Ethyl alcohol

Alcohol is an organic compound having at least one hydroxyl functional group (oxygen plus hydrogen, OH) bound to a saturated carbon (C) atom, inclusive of compounds for various industrial and pharmacological uses, e.g., propanol, isopropyl, isobutyl, phenols, ethylene, etc., as well as ethanol or ethyl alcohol (C_2H_6O), the active ingredient in beverages. Ethyl alcohol is produced by fermentation of sugars from fruits and yeasts from grains.

Throughout this exposé, 'alcoholic beverage' or 'alcohol' refers to ethyl alcohol. Refer to **Figure 1** and **Table 1**.

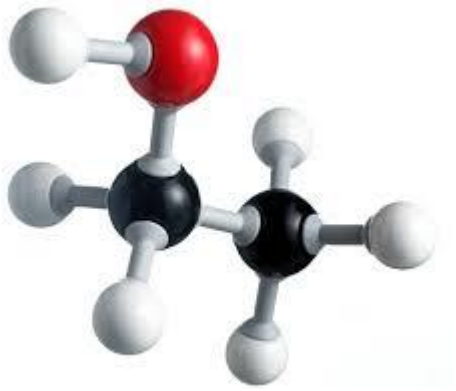

Figure 1 | Ethyl alcohol molecule

Three-dimensional ethanol molecule, C_2H_6O, the active ingredient in alcoholic beverages.

Due to the physicality of this small molecule (molecular weight 46 grams/mol), ethanol infiltrates the brain, absorbed across the blood-brain barrier, resultantly altering neurotransmission, memory processes and behavior.

Image courtesy of online source.

The literature reviews cited in this exposé are consistently reported as BrAC, measured in grams (g)/deciliter (dl). While BrAC and blood alcohol content (BAC) are relational, BAC is a measurement of how much alcohol is present in the bloodstream (g/milliliter), recognizing that some alcohol is diffused both metabolically and via the respiratory system. Individuals invariably differ in how they physiologically process alcohol, depending on factors such as their sex, weight, muscle to fat ratio, genetic predisposition, food intake during the drinking episode, level of hydration, mood and various situational and environmental factors (Kalat 2019).

Enjoying the occasional alcoholic beverage is regarded as sociably normal and safe for most healthy adults in moderation. However, quantity and frequency are critical factors as ethyl alcohol is actually a neurotoxin with physiologic effects, acting as a depressant in the central nervous system. An episode of intoxication can last approximately 2-6$^+$ hours as alcohol is metabolized, with effects gradually tapering off over time (Refer to **Special section**). Steep increases in BrAC are especially implicated in alcohol blackouts (Kalat 2019; Wetherill & Fromme 2016; Merrill *et al.*, 2014).

Table 1 | Alcohol content of beverages

Alcohol beverage (1 serving)	Percent (%) ethyl alcohol	Ounces per glass
Beer	5	12
Wine	12	5
Distilled spirits	40	1.5

Approximate content (%) and quantity (ounces) of ethyl alcohol per serving of common beverages. There is considerable variability depending on the type and quality of beverages and how they are served. Distilled spirits include whiskey, rum, vodka, gin, tequila, etc. (National Institute of Alcohol Abuse and Alcoholism).

Ethyl alcohol and the brain

Alcohol is permeable via the blood-brain barrier (**Figure 2**), disrupting brain activity at the molecular, cellular, anatomical, organismic and behavioral level. At the molecular level, alcohol acts on neurotransmitter receptors in the brain, notably the inhibitory transmitter γ-aminobutyric acid (GABA), the excitatory transmitter glutamate and dopamine associated with mood and motivation (Pimentel *et al.*, 2020). Alcohol's relatively small size, molecular weight 46 g/mol, approximately three times smaller than these neurotransmitters, means that it can effectively infiltrate neural processes, markedly altering perception and behavior. Alcohol consumption has the same effect as activation of GABA receptors in the brain. GABA's neurochemical properties include anxiolytic (anti-anxiety) and hypnotic activation, altering motor coordination, decision-making processes and behavioral control (Kalat 2019; Wetherill & Fromme 2016; Chitty *et al.*, 2014).

Alcohol-induced blackouts may be erroneously confused with losing consciousness from excessive alcohol intake, but 'blacking out' and 'passing out' actually describe two very different states. A drinker experiencing a blackout is conscious, volitionally interacting with their environment, physically active, socially aware and otherwise cognitively functional, albeit with deficits. Conversely, a drinker who has lost consciousness at extreme BrAC levels

is physiologically incapacitated and unresponsive (Pressman & Caudill 2013; Mundt & Zakletskaia 2012). Alcohol blackouts can happen to any drinker, not necessarily only those suffering from alcoholism, as had historically been assumed, although alcoholics do have a proclivity for excessive drinking habits that elicit blackouts (Barnett *et al.*, 2014; White 2003).

Notably, acute personality changes have been reported during an alcohol blackout (Mezquita & Ibáñez 2021; Hakulinen & Jokela 2019). Beyond alcohol being just a drug of disinhibition (Gladwell 2019), drinkers' behavior may become uncharacteristically erratic. Alcohol is implicated in the downregulation of the prefrontal cortices and affective nucleus accumbens (**Figure 2**), brain regions associated with fear stimuli, motivation, reasoning and behavioral control (Pressman & Caudill 2013; Cima *et al.*, 2004), accounting for why intoxicated subjects are primed to make grossly bad decisions.

Our developed prefrontal cortices confer conscientiousness, affording accountability for our actions, so alcohol's dampening effects of this brain region can translate into profound behavioral deficits. For a notable example, up to 50% of homicides are reportedly preceded by alcohol consumption by the perpetrator (United States Department of Justice). Taken together, deficits in the ability to form memories along with dysregulation of behavioral control are a dynamite combination for potentiating aggression or violent criminality (Du Beau 2018; Wetherill & Fromme 2016).

An historic local study reported that drinkers given prescribed quantities of alcohol in controlled settings experienced a "deepening of mood," ostensibly coinciding with blackouts (Wolf 1980). However, based on modern research, there is no evidence that mood is associated with the physiological onset of blackouts. Also, the drinker's ethnicity is irrelevant to the blackout phenomenon. Refer to **References**.

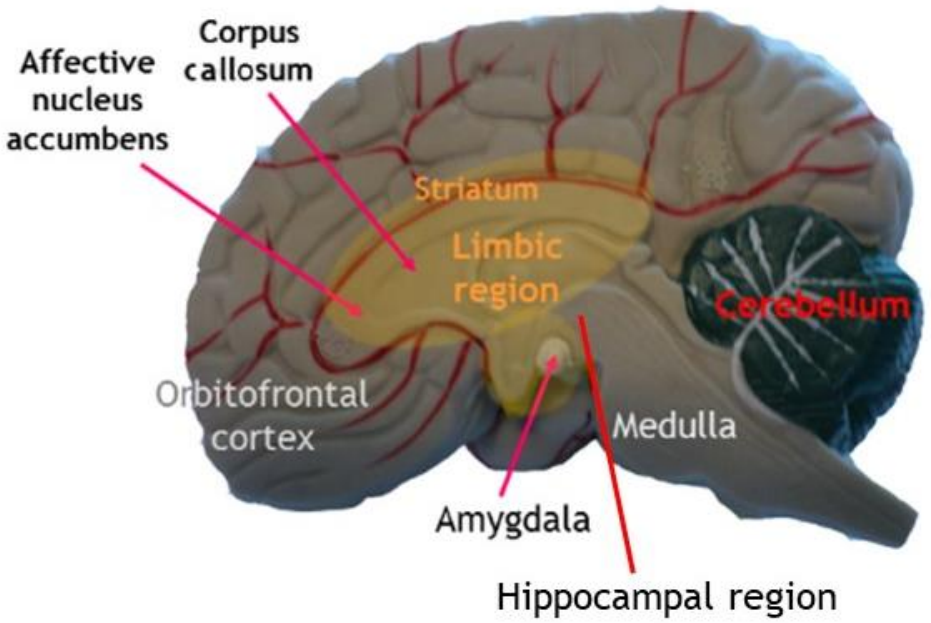

Figure 2 | Relevant anatomy of the human brain

Anatomically distinct regions of the human brain, shown in the sagittal plane of axis in this figure, are identified by pastel shading and corresponding colored text. The sagittal section divides the brain into left and right hemispheres along the corpus callosum. Vasculature associated with the blood-brain barrier is shown as red lines.

Alcohol affects the hippocampi within the limbic region, altering memory processes. Neurons from the limbic region project to the orbitofrontal cortices, involved in working memory and transference of information to long-term memory storage. The amygdala is associated with emotional regulation. Alcohol also affects the affective nucleus accumbens, which seats motivation, reinforcement learning and fear stimuli.

The cerebellum, associated with balance and coordination, is among the first regions of the brain to be influenced by alcohol. The medulla extends towards the spinal cord. Image modified from Du Beau (2018).

Field sobriety tests

Early symptoms of alcohol intoxication are the impetus for field sobriety tests conducted by police officers on site (Rubenzer 2008). Walking in a heel-to-toe straight line, touching the index finger to the tip of the nose, the horizontal gaze test and reciting the alphabet backwards are effective in determining if a subject may be drunk. Conducting field sobriety tests, police are actually checking the basic neurological functioning of the cerebellum, the brain region associated with balance and coordination, and the medial temporal lobe (Broca's area) producing speech, respectively (**Figure 2**). Field sobriety tests are an effective means of detecting intoxication. And the scent of alcohol on the subject's breath can be readily detected by police too, quantifiable by the breathalyzer device, recorded as BrAC (g/dl).

Investigative procedures generally preclude interviewing and interrogating intoxicated witnesses or suspects. However, in practice, this may be problematic because they may be the only subjects present at an incident. Importantly, police may not necessarily know if the subject is intoxicated, especially if they are experiencing an alcohol blackout where typical symptoms may be subtle or even absent (Hagsand *et al.*, 2022). Further, certain perfectly normal, sober behaviors present for those who may be neuroatypical (such as high-functioning autism, hard-of-hearing or deafness, etc.) or those merely dealing with a managed medical issue or disability may be misinterpreted as intoxication by the police. Refer to **Figure 3**.

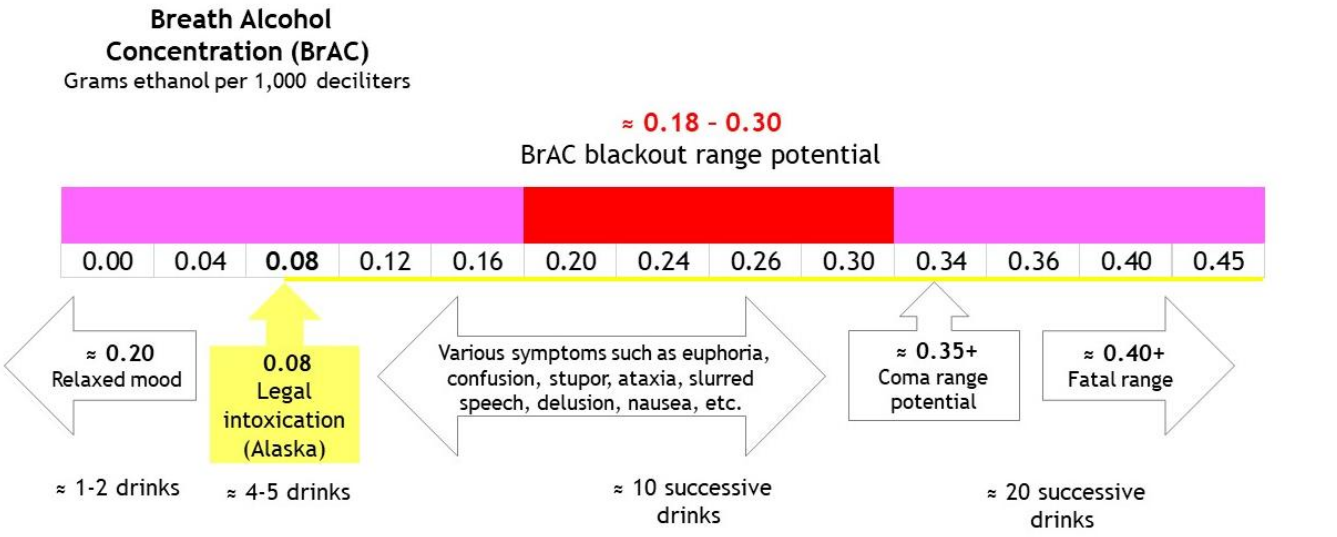

Figure 3 | Breath alcohol concentration and blackout range

Blackouts can be experienced between the approximate range of BrAC 0.18 to 0.30 g/dl, represented by the horizontal red bar along the continuum of intoxication. Approaching BrAC 0.35, the drinker may lose consciousness and could become comatose. Reaching a BrAC of approximately 0.40 may be fatal. In Alaska, the legal level of intoxication is ≥ BrAC 0.08 g/dl (and less for commercially licensed drivers). 1 dl = 0.1 liter.

Memory processes

Experimental studies of alcohol blackouts report a marked impact on selective memory processes, although other cognitive processes may be effectively intact. Because the alcohol blackout is a distinct neurological phenomenon occurring within relatively narrow parameters, it does not present with the usual indices of severe intoxication (Hagsand *et al.*, 2022; Pressman & Caudill 2013).

Investigators know that memories may not be perfectly infallible reflections of actual events; the brain, after all, does not operate like a video recorder. Indeed, flawed eyewitness memory can be a key reason for wrongful convictions (Morgan *et al.*, 2013; Patihis *et al.*, 2013). Alcohol and other mind-altering substances are known to impair

memory encoding, retrieval and transference. A confounding issue is that bystanders and investigators cannot decisively know if they are encountering someone experiencing an alcohol blackout since there is no demonstrable evidence (Hagsand *et al.*, 2022; Pressman & Caudill 2013). Analogously, a person may feel hunger or pain, but we usually cannot know this just by observing them. This section discusses how alcohol backouts alter memory processes.

Short-term versus long-term memory

Sensory memory is a prequel to short-term memory, relying on the five senses for input. Sensory memory is a transient mental state that entails the integration and processing of sensory inputs, lasting for only a brief period of time. Short-term memory differs from long-term memory in that it decays over time and is relatively limited in the amount of information stored (known as memory chunking). Working memory is short-term memory applied to cognitive tasks (Wang 2021; Kalat 2019). Examples of short-term working memory include remembering where you put your car keys, remembering a one-off passcode, remembering the menu number of a dish while ordering at the restaurant or recounting what happened last evening; processes which require attention involving prefrontal regions of the brain.

The brain can selectively shift such information into long-term memory storage, which is integrated and consolidated into a panoply of brain regions as axons carry electrochemical signals (neurotransmission), projecting and connecting proximally (close to) or distally (far away from). Declarative long-term memories are available consciously, expressed via language, quantities or figures. Similarly, tacit knowledge entails learned behavioral repertoires; skills you know such as driving a car, playing the piano, doing a surgical procedure or knowing how to bake a pie. So, what is happening during an alcohol blackout? Refer to **Figure 4**.

Anterograde versus retrograde amnesia

Alcohol can interfere with the retrieval and transference of information between short and long-term memory processes. That is, signalling between brain regions is stuck as alcohol interacts with the functioning of prefrontal cortices and cerebellum (**Figure 3**). During a blackout episode, the drinker is unable to form long-term memories, experiencing anterograde amnesia (Wetherill & Fromme 2016). The inability to recount events that happened after the drinking episode is an example of anterograde amnesia. Meanwhile, during a blackout, the subject can remarkably engage in social interactions, carrying on detailed conversations and performing such physical activities as driving, etc. (Miller *et al.*, 2018; Wetherill & Fromme 2016; White 2003).

Research reports that intoxicated individuals are able to recall information immediately after it is presented and can hold such information in short-term memory for at least one minute. Drinkers can typically recall long-term memories formed before they became intoxicated. However, after only one or two drinks, drinkers begin to show substantial impairments in the ability to transfer information into long-term memory (Wang 2021; Miller *et al.*, 2018; Wetherill & Fromme 2016; Nash & Takarangi 2011).

Alcohol blackouts do not involve retrograde amnesia, which is a relatively rare neurological state caused by a traumatic brain injury, lesion/tumor, onset of disease (such as encephalitis, dementia, feverish delirium) or neurosurgery. Retrograde amnesia negatively impacts declarative memory while procedural memory may be intact. Retrograde amnesiacs, for example, may not know their name or be able identify family members or recount what happened yesterday. Selectively inexplicable gaps in memory, anterograde or retrograde, without due cause are suspicious, presenting a red flag for investigators.

Fragmentary versus en bloc blackout

Researchers have identified and characterized two qualitatively distinct types of blackouts: fragmentary and en bloc. Both types invariably occur when BrAC reaches 0.06 g/dl or greater (Hartzler & Fromme 2003). Investigators should know that blackouts represent a liminal mental state potentiating aggressive criminality.

Fragmentary blackouts (also known as 'gray outs' or 'brown outs') involve partial amnesia during a drinking episode, although the drinker may be prompted to recall salient events with relevant situational cues. En bloc blackouts typically occur at higher BrAC than fragmentary blackouts, presenting with a distinctly acute onset (Miller *et al.*, 2018; White *et al.*, 2004; Hartzler & Fromme 2003).

According to a study of arrestees charged with alcohol related offenses, the probability of a fragmentary blackout was 50% at a BrAC of 0.22 g/dl and 50% for a BrAc of 0.31 g/dl for an en bloc blackout (Perry *et al.*, 2006). The practice of 'drinking to get drunk' involving drinking games (Pennies, Back-to-Back, etc.) as popularized in college dormitories (Gladwell 2019), gulping drinks or taking shots in rapid succession, marked by a rapid spike in BrAC, is strongly implicated in the phenomenon of blackouts (Wetherill & Fromme 2016; Merrill *et al.*, 2014).

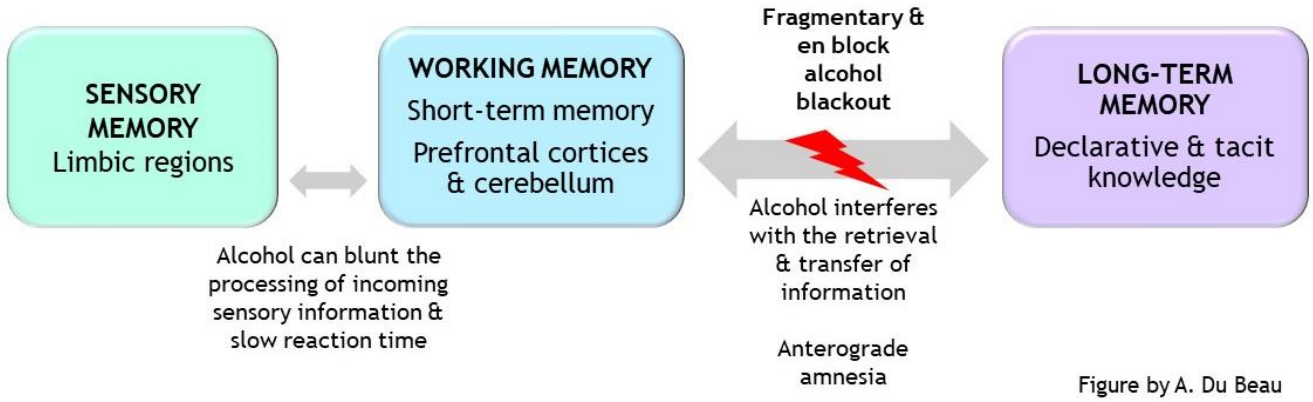

Figure 4 | Memory stages and blackout range

Schematic representing the stages of memory, revealing the effects of alcohol on the associated brain regions. Sensory memory (green box) via the limbic regions is transient as information is relayed to working short-term memory (blue box) held generally in prefrontal cortices and the cerebellum. Declarative and tacit knowledge that defines long-term memory (purple box) is consolidated in various brain regions. Alcohol interferes with memory processes and is implicated in amnesiac blackouts.

Mens rea and legal considerations

Criminal behavior leading to a conviction necessitates the commission of an unlawful act, *actus reus*, 'guilty act', coincidentally occurring by a state of mind, *mens rea*, 'guilty mind', that implies culpability for the action. Within the law, the concept of intentionality has many synonyms, referred to as voluntarily, purposely, knowingly or willfully, etc. However, in practice, these all mean the same in legal proceedings. The alcohol blackout, existing in the legal gray zone, challenges the conventional criteria of intentionality. Therefore, being well informed about the underlying science is requisite for legal counsel to best strategize their casework.

Automatisms are behaviors performed without conscious awareness or intentionality. In the absence of intention, *mens rea*, an automatism could provide a defense for criminal acts (Du Beau 2018; Pressman & Caudill 2013; Fenwick 1990). However, because cognitive faculties are still present during a blackout, drinkers do not meet the full criteria for experiencing automatisms. From historical research, "He/she knew right from wrong at the time of the act" is the critical factor regarding the law (van Oorsouw *et al.*, 2004; Goodwin 1995). Now, whether *animus nocendi*, 'intention to cause harm', is absent or not during a blackout depends on particular case circumstances, to the discretion of triers of the law.

Alcohol blackouts can be misconstrued as an absence of higher cognitive function during an amnesiac drinking episode. While the formation of long-term memories is absent during a blackout, existing scientific literature strongly suggests that cognitive processes associated with consciousness remain sufficiently intact. Memory lapses associated with alcohol backouts do not negate *mens rea* (Pressman & Caudill 2013; Cima *et al.*, 2004). The psychiatric literature offers three overarching explanations for the phenomena of crime related amnesia (Cima *et al.*, 2002), as detailed in this section.

1/ The commission of a violent crime is inherently stressful, suggesting that the emotively charged context could somehow undermine memory for criminal acts. However, negating this argument, studies have demonstrated that survivors of World War II concentration camps, for notable example, rarely report amnesia for their horrifying experiences (Cima *et*

al., 2004; Porter *et al.*, 2001; Kuck & Cox 1992). Related, the Reid Technique teaches that gross inconsistencies in memory recall are indicative of deceit (Reid Technique).

Whereas there is legal precedent to prevent voluntary intoxication and blackouts from being viable defenses against committing a crime (Marlowe *et al.*, 1999; Cunnien 1986), an outstanding issue is the extent to which alleged victims in a blackout should be culpable for their actions, despite their lack of memories. Unquestionably, when a victim is incapacitated from alcohol and unable to provide consent, there are still grounds for a conviction of sexual assault (Pressman & Caudill 2013).

2/ According to forensic research, most cases of crime-related amnesia are malingering (Cima *et al.*, 2004, 2002). The notion that by feigning a memory deficit, offenders might not be culpable for their actions is not supported by neuroscientific evidence (Cima *et al.*, 2004; Kopelman 1995). If a defendant should claim retrograde amnesia (the inability to recall events well before the incident in question), which is relatively rare, then the defense would need to provide substantiating medical evidence, e.g., traumatic brain injury, brain lesion, neurosurgery, etc.

3/ Many offenders who claim amnesia report being intoxicated during the commission of the given crime (Marlowe *et al.*, 1999). However, experiments testing this notion have demonstrated that alcohol inhibits memory process as well as complex behavioral repertoires, although basic motor functions may be intact (Cima 2004). Specifically, some crimes entail a good deal of strategic planning, coordination, attention and concentration, precluding the offender being intoxicated during the commission of the crime.

Examining this argument further, suspects' statements made during an alcohol blackout, inclusive of putative confessions, are factually unreliable. Intoxicated suspects may be especially vulnerable to suggestion, complying with leading investigative questioning (Hagsand *et al.*, 2022). Alcohol is certainly not a 'truth serum' and spurious confessions elicited during such an altered mental state must be considered artefactual. While a drinker's motor functions may be working during an alcohol blackout, they cannot successfully perform coordinated complex skilled movements. For example, a high-functioning drinker may be able to passably dance at the barroom (although not at their

graceful best), but could never possibly execute a ballet performance (Du Beau 2020; Gladwell 2019; Wetherill & Fromme 2016).

During an alcohol blackout, drinkers may appear discombobulated (or even stupid) as they are experiencing a dissociative state of consciousness coupled with functional motor activity. Unlike someone who has lost consciousness, the drinker experiencing a blackout is engaging with their environment and necessarily behaving volitionally. Regarding a criminal act, an alcohol blackout is not exculpatory (Du Beau 2020; Gladwell 2019; Wetherill & Fromme 2016; Pressman & Caudill 2013). For a cogent discourse on state of consciousness and criminality, please refer to *Sleep and Violent Behavior* (Du Beau 2020).

Admissibility standards for scientific evidence vary among jurisdictions (and even among courts within a particular jurisdiction). In *Frye* jurisdictions, a party proffering expert scientific testimony must show that the methodology would be accepted in that particular scientific field. Alternatively, in accordance with *Daubert* standards, general acceptance is just one reliability factor. As to testability, due to the very nature of alcohol blackouts, there is no generally accepted scientific method to detect a blackout while it is occurring or afterward (Daubert v Frye 2022; Pressman & Caudill 2013; Lustre 2009).

The fallibility of memory, even in the absence of alcohol blackouts, has been well documented (Morgan *et al.*, 2013; Patihis *et al.*, 2013), subject to provision of misinformation, individual points of view, the passage of time, subjective perceptions and even ulterior motives. Resultantly, the credibility of memories recalled following an alcohol blackout may be especially suspect.

Forensic researchers examined a quadruple murder case in which the defendant claimed to have no memory of committing the crime because he was in an alcohol-induced blackout at the time (Pressman & Caudill 2013). Applying *Daubert* legal standards to this case (Daubert v Frye 2022), the researchers concluded that the defendant's blackout could not be used as a viable defense. Equally important, however, may be the memory of the alleged victim of a crime (Pressman & Caudill 2013; van Oorsouw *et al.*, 2004) wherein circumstantial and physical evidence emerge to be the deciding factor.

When examining the impacts of alcohol blackouts, triers of the law must ask the accused, victim or witness to remember not remembering. This critical challenge also raises questions about the credibility of such recollections. Pursuant to filling in gaps in memory, people may rely on the account of others who were present at the incident in question (apart from ever-present digital cell tower data, etc.). This tack can be fraught with misinformation, however, which can lead to fallacious reconstruction of events (Nash & Takarangi 2011). Making such inferences with a reliable degree of accuracy remains a formidable task; triers of the law must do their best to ascertain the truth, and why sound scientific expertise guides and optimizes such legal decision-making processes.

Special section | Chemistry and genetics of alcohol misuse

Alcohol blackouts can happen to anybody and are not necessarily correlated to alcoholism. Adjunct to the present exposé, this special section describes the genetics underlying the chemistry of alcohol catalysis (metabolic breakdown) and alcoholism. Refer to **Figure 5**.

After anybody drinks an alcohol beverage, enzymes in the liver metabolize it to acetaldehyde, which is then converted to acetic acid by enzymatic action.

Figure 5 | Chemistry of alcohol catalysis

Figure by A. Du Beau

Acetaldehyde is responsible for the familiar misery of hangover symptoms: nausea, vomiting, flushing of the face, head achiness, malaise, sensory sensitivities, etc. Hangover symptoms that are a sequala of acetaldehyde accumulation are a powerful behavioral deterrent to developing alcoholism.

There is a genetic component to alcoholism that predisposes certain individuals to misuse alcohol and become dependent, which is why alcoholism tends to run in families (Kalat,

2019). Under normal circumstances, the enzyme acetaldehyde dehydrogenase catalyzes acetaldehyde more slowly than those who have the alcoholism gene, identified as *ADH1B/ADH1C* (Janiak *et al.*, 2020; Tolstrup *et al.,* 2008).

Prescription drugs to treat alcoholism, e.g., Antabuse, mimic the toxic effects of acetaldehyde, making the experience of drinking nearly intolerable, the premise being that negative reinforcement is a powerful demotivator. The downside is that patients must be consistently compliant with taking the drug on schedule for effective treatment outcomes.

Greater than 1/3 of people in Asia, for example, lack this gene, hence alcohol misuse is relatively low in Asian countries. Conversely, certain ethnicities, such as American and Alaskan indigenous populations, tend to have the *ADH1B/ADH1C* gene, predisposing them to developing alcoholism (Tolstrup, *et al.,* 2008). Some societies outright forbid the use of alcohol, notably the Muslim culture, which also precludes alcoholism (Kalat, 2019). For those with a family history and genetic predisposition for alcoholism, awareness and precautionary lifestyle measures can be especially helpful to avoid alcohol misuse.

Conclusion

Alcohol blackouts occur within narrow physiological parameters, marked by a rapid spike in BrAC, acutely altering memory processes and behavior. And alcohol blackouts can intersect with an array of criminal acts. Because cognition can be surficially intact during an alcohol blackout, detecting such an episode can be confounding for investigators. We judge intentional attempts to cause harm to someone, even if the attempt fails, with more blameworthy gravity than harm inflicted inadvertently or recklessly, so adjudicating criminal cases where alcohol blackouts are a factor can be especially challenging for triers of the law. This rigorously researched exposé identifies and characterizes the underlying mechanisms of alcohol blackouts, revealing the legal ramifications associated with blackouts.

> *"Here's to alcohol, the rose-colored glasses of life."* — F. Scott Fitzgerald (1922), *The Beautiful and Damned.*

Statement

I am the sole author of this report and declare no conflict of interest. The contents of this report are compiled, reported and portrayed to the best of my knowledge and are not intended to be exhaustive research review, recognizing that information may change over time. Questions and comments are welcome. All contents referenced from this document must be cited directly to me.

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I am a neuroscientist (PhD), an independent consultant with expertise in both forensic behavioral analysis and bloodstain pattern analysis.

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