

Factors that Increase the Potential for Alcohol Blackout.

- If the person is a novice drinker.
- If a person has experienced blackouts in the past.
- Drinking on an empty stomach. (Goodwin 1969, White 2003, see the “*Toxicology*” section in this handout)
- “*Gulping*” drinks (White 2003, p. 5)
- Proof of the alcohol - Proof is 2x the alcohol content (80% proof is 40%) (White 2003, p.7)
- Females do not metabolize alcohol as quickly due to body weight, proportion of body fat, and levels of key enzymes. (White 2002, Dettler, 2009, Jones 2010). There is also evidence that females are more susceptible than males to milder forms of alcohol-induced memory impairments. (Mumenthler 1999).
- Some Asians and Native Americans lack a critical enzyme to better process alcohol. (ADH & ALDH)
- Alcohol combined with benzodiazepine sedatives (White 1997, Saum and Inciardia 1997, Silvers 2003)
- THC – marijuana (Ciccocioppo 2002).

Aaron White (2003) explains:

Drinking large quantities of alcohol often precedes blackouts, but several other factors also appear to play important roles in causing such episodes of memory loss. As Goodwin and colleagues (1969a) stated with regard to subjects in one of their studies, “Although blackouts almost always were associated with heavy drinking, this alone seemed insufficient to produce one. On many other occasions, subjects said they had drunk as much or more without memory loss” (p. 195). Among the factors that preceded blackouts were gulping drinks and drinking on an empty stomach, each of which leads to a rapid rise in BAC.

Subsequent research provided additional evidence suggesting a link between blackouts and rapidly rising BACs. Goodwin and colleagues (1970) examined the impact of acute alcohol exposure on memory formation in a laboratory setting. The author recruited 10 male subjects for the project, all but one through the unemployment office in St. Louis, Missouri. Most subjects met diagnostic criteria for alcoholism and half had a history of frequent blackouts. The men were asked to consume roughly 16 to 18 ounces of 86-proof bourbon in approximately 4 hours. Beginning 1 hour after subjects began drinking, memory was tested by presenting subjects with several different stimuli, including a series of children’s toys and scenes from erotic films. Subjects were asked to recall details regarding these stimuli 2 minutes, 30 minutes, and 24 hours after the stimuli were shown. Half of the subjects reported no recall for the stimuli or their presentation 30 minutes and 24 hours after the events, though most seemed to recall the stimuli 2 minutes after presentation. Lack of recall for the events 24 hours later, while sober, represents clear experimental evidence for the occurrence of blackouts. The fact that subjects could remember aspects of the events 2 minutes after they occurred but not 30 minutes or 24 hours afterward provides compelling evidence that the blackouts stemmed from an inability to transfer information from short-term to long-term storage. For all but one subject in the blackout group, memory impairments began during the first few hours of drinking, when BAC levels were still rising. The average peak BAC in this group, which was roughly 0.28 percent, occurred approximately 2.5 hours after the onset of drinking.

In a similar study, Ryback (1970) examined the impact of alcohol on memory in seven hospitalized alcoholics given access to alcohol over the course of several days. All subjects were White males between the ages of 31 and 44. Blackouts occurred in five of the seven subjects, as evidenced by an inability to recall salient events that occurred while drinking the day before (e.g., one subject could not recall preparing to hit another over the head with a chair). Estimates of BAC levels during blackout periods suggested that they often began at levels around 0.20 percent and as low as 0.14 percent. The duration of blackouts ranged from 9 hours to 3 days. Based on his observations, Ryback concluded that a key predictor of blackouts was the rate at which subjects consumed their drinks. He stated, "It is important to note that all the blackout periods occurred after a rapid rise in blood alcohol level" (p. 622). The two subjects who did not black out, despite becoming extremely intoxicated, experienced slow increases in blood alcohol levels.

Aaron M. White, *What Happened? Alcohol, Memory Blackouts, and the Brain* (2003).
Alcohol Research & Health. 2003 Vol 27(2) p. 189

Identifying factors that increase the likelihood for alcohol-induced blackouts in the prepartying context.

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Abstract:

The present study examined risk factors related to 'blacking out' (e.g., temporary periods of memory loss during drinking) during preparty drinking events (i.e., pregameing, predrinking). Participants were students from two universities on the West Coast who reported past month prepartying (N = 2,546) in online surveys administered in the fall of 2008. Among these students, 25% (n = 636) reported blacking out during at least one occasion in which they prepartied in the past month. A logistic regression model underscored that Greek student affiliation, family history of alcohol abuse, frequency of prepartying, and both playing drinking games and consuming shots of liquor while prepartying increased the likelihood of blacking out. Limitations and implications for future research and collegiate prevention strategies are discussed.