Alcohol consumption is commonly thought to be a major cause of aggression and violence in the American Society. Some correlational evidence supports the notion that alcohol use and aggression are related, although the strength and causal nature of the relationship remains unclear (Dermen & George, 2001). Researchers have been conducting studies on the relationship between alcohol and aggression and the effects of alcohol has on individuals personality. Peter N.S. Hoaken, researcher at the University of Wesern Ontario, presented informative scholarly research on alcohol-related aggression during the Psi Chi regional Sandhills conference. During the lecture Hoaken attempted to identify the role of cognition in alcohol-related aggression and violent crimes. After conducting extensive research Hoaken argues that individual differences in cognition and brain chemistry predict impulsivity, aggression, and alcohol-induced aggression.

Many researchers believed that aggression and alcohol use are strongly related and when under the influence of alcohol the rate that aggressive behaviors will occur is extremely high. Predicting which individuals are likely to exhibit aggression following alcohol consumption is an important and intriguing research problem. In Hoaken’s research he addresses the fact that a lack of focus on the individual is one of the reasons why demonstrating a direct causal relationship between alcohol consumption and
aggression has, at times, yielded mixed results. Hoaken’s view is supported by other researchers who have previously suggested that demonstrating a clear relationship between alcohol intake and aggression is difficult, because alcohol consumption increases aggressiveness in some individuals, but decreases it in others (Gmel & Rehm, 2003). Many of these mixed findings may be related to researchers' lack of focus on differences among individuals. For example, Hoaken’s research indicates a stronger relationship between alcohol consumption and aggression in subjects with certain traits, including antisocial personality, alcohol dependency, previous aggressive episodes, and especially impaired cognitive functions.

The purpose of this research was to relate alcohol and aggression through a cognitive interpretation. In Hoaken’s research a major assumption underlying the cognitive model is that the expression of aggression in a non-intoxicated state is controlled to a large extent by the perception or anticipation of physical and social consequences. Hoaken’s research assumes that, in the intoxicated state, it is difficult to process information regarding behavioral contingencies. This view is supported by some researchers that have suggested that, by alternating cognitive processes, alcohol reduces the drinker’s awareness of the consequences of his or her behavior; for example, individuals inability to process information pertinent to the consequences of his or her behavior (Weisman & Taylor, 2001).

Some people are more likely than others to become aggressive after consuming alcohol because of individual differences. Hoaken’s research supports the fact that
alcohol affects mechanisms by which performance is optimized in situations discarding operations and integration of a set of cognitive processes, in working memory inhibition, abstraction, planning, and active monitoring. However, in my opinion, the level of an individual’s aggressive state depends on how the alcohol affects the brain function by interacting with multiple neurotransmitter systems. Therefore, it is my belief that the neurotransmitter system plays a dominant role in the relationship between alcohol and aggression. My beliefs are supported by Tomkins & Sellers (2001) after they found the following:

Evidence suggests that alcohol affects brain function by interacting with multiple neurotransmitter systems, thereby disrupting the delicate balance between inhibitory and excitatory neurotransmitters. Short-term alcohol exposure tilts this balance in favor of inhibitory influences. After long-term alcohol exposure, however, the brain attempts to compensate by tilting the balance back toward equilibrium. These neurological changes occur as the development of tolerance to alcohol's effects. When alcohol consumption is abruptly discontinued or reduced, these compensatory changes are no longer opposed by the presence of alcohol, thereby leading to the excitation of neurotransmitter systems and the development of alcohol withdrawal syndrome. Long-term alcohol intake also induces changes in many neurotransmitter systems that ultimately lead to the development of craving and alcohol-seeking behavior. (p. 819)

Some neurotransmitters have a more intense affect on the relationship between alcohol and aggression than others. Serotonin is an important brain chemical that acts as a
neurotransmitter to communicate information among nerve cells. Serotonin's actions have been linked to alcohol's effects on the brain and to alcohol abuse. Alcoholics and experimental animals that consume large quantities of alcohol show evidence of differences in brain serotonin levels compared with nonalcoholic (Highley, 2001). Both short- and long-term alcohol exposure also affect the serotonin receptors that convert the chemical signal produced by serotonin into functional changes in the signal-receiving cell (Pihl & Peterson, 1993). Serotonin, along with other neurotransmitters, also may contribute to alcohol's intoxicating and rewarding effects, and abnormalities in the brain's serotonin system appear to play an important role in the brain processes underlying alcohol abuse.

Brain serotonin activity may play a role in governing alcohol intake and in partly determining predisposition to alcohol abuse and dependence and serve as a role in promoting aggressive behavior. Alcohol is known to facilitate aggression in persons with defects in serotonin-related processes. Pihl & Peterson (1993) found alcohol exposure alters several aspects of serotonergic signal transmission in the brain. For example, alcohol modulates the serotonin levels in the synapses and modifies the activities of specific serotonin receptor proteins. Abnormal serotonin levels within synapses may contribute to the development of alcohol abuse, because some studies have found that the levels of chemical markers representing serotonin levels in the brain are reduced in alcoholic humans and chronically alcohol-consuming animals (Higley, 2001).

For my part I agree that alcohol affects an individual role of cognition. Alcohol misuse can harm people other than the drinker, and can have negative consequences for
society as a whole. It is commonly believed to play a role in decreased worker productivity, increased unintentional injuries, aggression and violence against others, and child and spouse abuse (Gmel, 2003). Research findings support the idea that drinking is involved in or associated with many of these social harms, but do not offer evidence that it causes these effects.

After evaluating both sides of the cognitive perspective, alcohol affecting cognitive process which then leads to aggression, and the neurotransmitters perspective, alcohol affecting the pathway of neurotransmitter serotonin which then lead to aggression, I must agree that both views help to influence alcohol-induced aggression. However, in my opinion the serotonin neurotransmitters help influence more alcohol-induced aggression at a more rapid rate than the cognitive perspective. My opinion is supported in a theoretical explanation proposed by Pihl & Peterson (1993), brain serotonin activity may play a role in governing alcohol intake and in partly determining predisposition to alcohol abuse and dependence. Serotonin function may increase temporarily under the influence of alcohol and serotonin levels may subsequently drop below baseline levels. This rebound effect may stimulate aggression among susceptible people exposed to rewarding or punishing stimuli. Alcohol may also facilitate aggression through direct effects on other neurotransmitter systems.
References


