endocannabinoid system plays in alcohol addiction and suicidal tendencies. Such influence extends to various mood disorders.

Long-term alcohol abuse depletes endocannabinoid tone, and this, in turn, has an adverse impact on a plethora of physiological processes that are modulated by the endocannabinoid system. The endocannabinoid system interacts with other neurotransmitters (serotonin, dopamine, glutamate, etc.) to protect the brain against alcohol-induced harm. While the effects of alcohol on brain function would block the brain’s crucial neuroprotective response during alcohol consumption, the vicious cycle of addiction feeds on it—thereby creating a self-sustaining cycle that is difficult to break.

Cannabis and Other Drugs
By Amanda Reiman, Harm Reduction Journal, Dec. 3, 2009

Marijuana is SAFER

By Steve Fox, Paul Armentano, Marijuana is SAFER

The struggle between weed and wine continues to unfold in 21st century America, where the alcohol industry funds organizations that seek to maintain marijuana prohibition. Such influence peddling by Booze, Inc. is not only a zero-sum game against an recreational competitor; war drug posturing is also smooth public-relations for liquor companies given the well-documented, deleterious health and social costs of their products. Alcohol is a pivotal factor in two some-two-thirds of all cases of violence between intimates in the United States, and booze is responsible for 100,000 sexual assaults among young people each year and 100,000 annual deaths. Alcohol is a potent factor in the harmful effects of alcoholism are well known, scientists have only recently begun to investigate and understand the critical role that the endocannabinoid system plays in alcohol addiction and various mood disorders.

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Scientists believe that alcohol has a dose-dependent effect on 2-AG levels in humans as well as rodents. In other words, when a person gets a little tipsy from drinking booze, his or her 2-AG levels rise slightly; when someone gets drunk on alcohol, 2-AG slashes around the brain; and as inebriation fades, 2-AG returns to its normal, baseline level.

Parsons and a team of researchers also documented that heroin administration triggered a corresponding rise in anandamide (the other key endocannabinoid in the rats’ nucleus accumbens, but had no effect on 2-AG levels. The nucleus accumbens is one of the brain’s crucial reward centers; hence the spike in the endocannabinoid levels during alcohol intoxication makes perfect sense.

The human brain is a delicate organ, stymied by a thick skull and a blood-brain barrier primed to keep foreign substances from penetrating. The endocannabinoid system is a crucial component of the brain’s overall protective apparatus. Parsons put it this way: “Endocannabinoids buffer stress... An increase in endocannabinoids serves as a buffer to physiological and behavioral stress.”

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Alcoholism and the ECS

Many references in the medical literature support the use of marijuana in the treatment of drug and alcohol addiction.

poisoning. From a medical perspective, that would be malpractice.

Enhancing Endocannabinoid Tone

Researchers focusing on alcoholism are currently exploring the possibility of “enhancing endocannabinoid tone” by manipulating the enzymes that control 2-AG and anandamide metabolism. One approach relies on URB-597, an experimental drug that inhibits fatty acid amidase hydrolase (FAAH), an enzyme that breaks down endocannabinoids. Fore-stalling the enzymatic degradation of 2-AG and anandamide raises endogenous cannabinoid levels in the brain. FAAH inhibitors indirectly bolster CB1 receptor signaling.

Parson’s rat brain microdialysis research suggests that FAAH inhibitors, by facilitating increased CB1 activity, can reduce anxiety-like behavior associated with alcohol dependence. FAAH inhibitors have also demonstrated therapeutic benefit in animal models of other severe disorders, including neuropathic pain, neural degenerations, conditions related to epileptic seizures, hypertension, depression, and inflammatory bowel disease, as well as against the proliferation and migration of cancer cells, according to Stephan Petrosino and Vincenzo Di Marzo at the University of Naples.

Additional studies indicate that genetic mutations may contribute to the dysregulation of endocannabinoid signaling. Scientists have linked a predilection for excessive alcohol intake to “polymorphisms” (atypical amino acid sequence repeats) in FAAH and CB1 receptor genes. A naturally-occurring “single nucleotide polymorphism” in the gene encoding the endocannabinoid inactivating enzyme FAAH is often found in people who engage in problem alcohol and drug use. This same FAAH gene polymorphism, according to German researchers, is often present in patients with obesity and irritable bowel disease.

Of course, there is another way to enhance CB1 signaling and adjust end-enzymatic processes — one could smoke, vaporize or eat cannabis, a natural, non-toxic herb, and thereby influence gene expression. THC, as noted earlier, activates both the CB1 and CB2 receptors. And CB2, the second most prominent cannabinoid in marijuana, inhibits FAAH! What’s more, THC and CBD work best in tandem, synergistically, so to speak, along with dozens of other phytocannabinoids, terpenes and flavonoids found in cannabis.

Endocannabinoid deficiency?

If alcoholism is an endocannabinoid deficiency syndrome, then it makes perfect sense that people might successfully

Many references in the medical literature support the use of marijuana in the treatment of drug and alcohol addiction.

wean themselves from booze by smoking marijuana, which triggers cannabinoid receptor signaling. In 1891, Dr. J.B. Mattison, writing in the St. Louis Medical and Surgical Journal, described can-nabis as a “remarkable” treatment for drug and alcohol dependence. Many references in subsequent medical literature support the use of marijuana in the treatment of drug and alcohol addiction.

There is compelling evidence that alcohol consumption diminishes among those who “self-medicate” with cannabis. A NIDA-funded investigation in Jamaica in the mid-1970s concluded that ganja smokers drank much less alcohol than non-smokers, lending credence to the notion that widespread marijuana use was the main reason for significantly lower levels of alcoholism in Jamaica than anywhere else in the Caribbean.

Other surveys have shown that a reduction in marijuana use leads to increased alcohol consumption among the stressed-out masses. After medical marijuana was legal in California in 1996, Dr. Tod Mikurya and several like-minded physicians successfully treated hundreds of alcoholic patients who got their lives back after switching to pot.

Parson’s brain research implicitly validates cannabis substitution as a harm reduction strategy for treating alcoholism. But the idea of substituting mari-juana for alcohol and other addictive substances is still strictly taboo in NIDA-contracted scientific laboratories, where synthetic enzyme-tweakers are favored over the “kind bud.”

FAAH-inhibitors are still years away from FDA approval for those who are unable or disinclined to stop using psychoactive substances completely. Marijuana may provide a safe and effective alternative to pharmaceuticals, which emphasizes complete abstinence.

To assess the extent to which medical marijuana patients are using the herb as a replacement for alcohol and/or prescription pharmaceuticals, Amanda Reiman, a lecturer at the University of California’s School of Social Welfare in Berkeley, surveyed 350 members of the Berkeley Patients Group (BPG), a city-licensed medical marijuana dispensary. Reiman, BPG’s research director, presented her findings at the 2009 ICRS conference, which was attended by a BPG activist contingent. Forty percent of respondents said they used marijuana as a substitute for alcohol.

“When addressing the efficacy of cannabis as a substitute for alcohol, all participants reported cannabis substitution as very effective or effective,” Reiman noted. Twenty-six percent of those surveyed said they used marijuana to replace more dangerous illegal drugs. Fifty-seven percent asserted that mari-juana provided better relief for their symptoms than conventional medica-tions, and 66 percent said they used can-nabis as a replacement for prescription pills.

Rather than being a so-called gateway to hard drugs and addiction, marijuana is an exit drug for hard self-medicating. Reiman and others have found that can-nabis enables people to minimize or eliminate their use of more harmful sub-stances, including prescription meds, opioids, and alcohol.

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