

Ibogaine

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1.

"Ibogaine, a natural alkaloid extracted from the root bark of the African shrub *Tabernanthe Iboga*, has attracted attention because of its reported ability to reverse human addiction to multiple drugs of abuse, including alcohol."

Source:

Dao-Yao He, Nancy N.H. McGough, Ajay Ravindranathan, Jerome Jeanblanc, Marian L. Logrip, Khanhky Phamluong, Patricia H. Janak, and Dorit Ron, "Glial Cell Line-Derived Neurotrophic Factor Mediates the Desirable Actions of the Anti-Addiction Drug Ibogaine against Alcohol Consumption," *The Journal of Neuroscience*, Jan. 19, 2005, Vol. 25, No. 3, p. 619.

2.

"Studies also suggest that ibogaine attenuates drug- and ethanol-induced behaviors in rodents. For example, ibogaine reduces operant self-administration of heroin in rats, as well as naloxone-precipitated withdrawal in morphine-dependent rats (Glick et al., 1992; Dworkin et al., 1995). Administration of ibogaine decreases cocaine-induced locomotor activity and reduces cocaine self-administration in rats (Cappendijk and Dzoljic, 1993) and mice (Sershen et al., 1994)."

Source:

Dao-Yao He, Nancy N.H. McGough, Ajay Ravindranathan, Jerome Jeanblanc, Marian L. Logrip, Khanhky Phamluong, Patricia H. Janak, and Dorit Ron, "Glial Cell Line-Derived Neurotrophic Factor Mediates the Desirable Actions of the Anti-Addiction Drug Ibogaine against Alcohol Consumption," *The Journal of Neuroscience*, Jan. 19, 2005, Vol. 25, No. 3, p. 619.

3.

"Despite its attractive properties, ibogaine is not approved as an addiction treatment because of the induction of side effects such as hallucinations. In addition, ibogaine at high doses causes degeneration of cerebellar Purkinje cells (O'Hearn and Molliver, 1993, 1997) and whole-body tremors and ataxia (Glick et al., 1992; O'Hearn and Molliver, 1993) in rats."

Source:

Dao-Yao He, Nancy N.H. McGough, Ajay Ravindranathan, Jerome Jeanblanc, Marian L. Logrip, Khanhky Phamluong, Patricia H. Janak, and Dorit Ron, "Glial Cell Line-Derived Neurotrophic Factor Mediates the Desirable Actions of the Anti-Addiction Drug Ibogaine against Alcohol Consumption," *The Journal of Neuroscience*, Vol. 25, No. 3, Jan. 19, 2005, p. 619.

4.

"Based on anecdotal reports in humans, ibogaine has been claimed [1] to be effective in interrupting dependence on opioids, stimulants, alcohol and nicotine. Preclinical studies in rats have supported these claims: ibogaine has been reported to decrease the i.v. self-administration of morphine [2] and cocaine [3] and the oral intake of alcohol [4] and nicotine [5]. However, studies in rats have also raised concerns regarding potential adverse effects of ibogaine; most notably, high doses have been shown to be neurotoxic to the cerebellum [6,7]."

Source:

Glick, S.D., Maisonneuve, I.M., and Dickinson, H.A., "18-MC Reduces Methamphetamine and Nicotine Self-Administration in Rats," *Neuropharmacology*, Vol. 11, No. 9, June 26, 2000, p. 2013.

5.

"18-MC, a novel iboga alkaloid congener, reduces intravenous methamphetamine and nicotine self-administration in rats. These and previous results with morphine, cocaine and alcohol indicate that 18-MC warrants further development as a potential treatment for multiple forms of drug addiction."

Source:

Glick, S.D., Maisonneuve, I.M., and Dickinson, H.A., "18-MC Reduces Methamphetamine and Nicotine Self-Administration in Rats," *Neuropharmacology*, Vol. 11, No. 9, June 26, 2000, p. 2015.

6.

"Although ibogaine has been reported to effectively reduce drug cravings and withdrawal symptoms in addicts (Sheppard, 1994), its tremorigenic, hallucinogenic, neurotoxic, and cardiovascular side effects (see Alper, 2001) have prevented its

approval as a treatment for addiction. On the other hand, 18-methoxycoronaridine, although not yet tested in humans, has no apparent side effects in rats, presumably because it is more selective pharmacologically than ibogaine."

Source:

Pace, Christopher J., Glick, Stanley D., Maisonneuve, Isabelle M., He, Li-Wen, Jokiel, Patrick A., Kuehne, Martin E., and Fleck, Mark W., "Novel Iboga Alkaloid Congeners Block Nicotinic Receptors and Reduce Drug Self-Administration," *European Journal of Pharmacology*, Vol. 492, 2004, p. 159.

7.

"In summary, repeated administration of drugs of abuse and alcohol induces a common pattern of changes in gene expression and protein levels selectively in the VTA. A subset of these changes is reversed by intra-VTA GDNF, as are some of the drug-induced behavioral effects. Endogenous GDNF systems appear to inhibit drug related behaviors, while repeated drug administration appears to inhibit GDNF signaling itself. Based on these studies, we propose that GDNF is an endogenous anti-addiction agent. This possibility is directly supported by the finding that the activity of the anti-addiction drug, ibogaine, on alcohol consumption is mediated via increased expression of GDNF in the midbrain and the subsequent activation of the GDNF pathway."

Source:

Ron, Dorit, and Janak, Patricia H., *Reviews in the Neurosciences*, Vol. 16, No. 4, 2005, p. 281.