Gamma-hydroxybutyrate (GHB)

Clinical name: gamma-hydroxybutyrate / gamma-hydroxybutyric acid
Brand name: none
Street name(s): GHB, Georgia Home Boy, Grievous Bodily Harm, liquid X, liquid ecstasy, organic quaalude, scoop, goop, G

Miscellaneous:

- Synthetically discovered in the 1920s.
- Researched for use as an anesthetic in the early 1960s.
- Previously sold OTC in health food stores. Marketed as a performance-enhancer in weight-training formulations until it was banned in 1989 by the FDA.
- Currently utilized in various European countries as an anesthetic adjuvant.
- Received Schedule I distinction in March, 2000 under the Hillory J. Farias and Samantha Reid Date-Rape Prohibition Act.

Pharmaceutical Properties:
GHB is initially found as a colorless and odorless solution that may or may not have a slightly bitter taste (depending on purity). The solution may be dried or salted to form a light-colored, highly soluble powder. It is common to see this powder form of GHB compressed into tablets. The solution form of GHB is seen mixed with water or other liquids in a multitude of containers.

Uses:
CNS depressant, anesthetic adjuvant. Has shown potential for treating narcolepsy, chronic fatigue syndrome, fibromyalgia and alcohol/opiate withdrawal.

Administration:
Oral ingestion

Mechanism/Pharmacology:
As a compound that naturally occurs in the body, GHB is a breakdown by-product of gamma-amino-butyric acid (GABA). GHB acts as a partial agonist of the $GABA_b$ receptor and can display either depressant or stimulatory effects. Recently, Cammalleri et. al. have discovered that GHB also "reduces GABA(A)-mediated inhibitory postsynaptic potentials in the CA1 region of the hippocampus."[1] Upon ingestion, GHB exhibits expeditious absorption from the gut in approximately 10-15 minutes. Gamma-butyrolactone (GBL), a precursor that is converted to GHB via dehydrogenase in the GI tract, is absorbed even faster. GHB has the ability to cross the blood brain barrier (BBB), and demonstrates a 30-minute half-life.[2]

Side effects:

Short-term: Sedation, dizziness, euphoria, nausea/vomiting, seizures, respiratory depression, coma, bradycardia, hypotension and visual disturbances.[2-4]

Long-term: Insomnia, tachycardia, tremors, anxiety, delirium/confusion and speech alterations.[2-4]

*A number of the side effects are potentiated with the simultaneous use of alcohol and other CNS depressants, threatening the user with a significant likelihood of morbidity/mortality. Respiratory depression is the chief concern regarding these circumstances.

Testing:

Current testing is normally performed via a urine test.[5] A blood test, however, is possible. It should be noted that "Date-rape prevention" kits are available that provide buyers with test strips that may be dipped in beverages and change to a designated indicator color (i.e. red) if a drink has been "spiked" with a drug commonly used in cases of date-rape (GHB, Rohpnol®, ketamine, etc.).

Treatment:

Treatment is largely symptomatic and, thus, greatly depends on whether the exposed individual is a long-term or short-term user. A barbiturate or benzodiazepine could be utilized to control anxiety and insomniatic tendencies. If needed, a beta-blocker may be used to help stabilize an erratic blood pressure.[6] Propranolol, in particular, also has indications for the treatment of anxiety and could help synergistically in that respect. The beta-blocker would most likely require only short-term use (3-4 days) to reestablish rhythmic normalcy. The barbiturate/benzodiazepine, however, may be scheduled consistently for the first three days to treat the often-severe initial symptoms, and be tapered to an "as needed" medication afterwards.

Synthesis:

There are several internet resources that currently produce "kits" for homemade use that contain GBL, either sodium hydroxide or potassium hydroxide and litmus paper to monitor the resulting solution's pH. GBL is combined with one of the aforementioned hydroxide solutions in a general cooking pot.[7] That is essentially all that is required. GBL proceeds through a dehydrogenase reaction and the result is GHB. The pH level must be strictly maintained to ensure reactive success and end product quality/purity.[7] Adding slightly more of either of the potently basic hydroxides will lower the pH to its desired range. Producing heat as they react, the chemicals involved generate all the energy needed for the reaction. Thus, an external heat source is not usually required. The final product is a clear liquid, though the individual producing the drug may proceed to "salt" or dry the drug into a powder form for ease of administration/distribution purposes.

User Identification:

Physical: Hallucinating, vertigo, spontaneous unconsciousness, amnesia and obvious interference with speech and motor control/coordination.

Citation References:

1. Cammalleri M, Brancucci A, Berton F, Loche A, Gessa GL, Francesconi W. Gamma-hydroxybutyrate reduces GABA(A)-mediated inhibitory postsynaptic potentials in the CA1 region of the hippocampus. Neuropsychopharmacology 2002;27(6):960-9.
2. Li J, Stokes SA, Woeckner A. A tale of novel intoxication: a review of the effects of γ-hydroxybutyric acid with recommendations for management. Ann Emergency Med 1998;31:729-35.
3. Freese TE, Miotto K, Reback CJ. The effects and consequences of selected club drugs. J Subst Abuse Treat 2002;23(2):151-6.
4. Smith KM. Drugs used in acquaintance rape. J Am Pharm Assoc 1999;39(4):519-25.
5. Stillwell ME. Drug-facilitated sexual assault involving gamma-hydroxybutyric acid. J Forensic Sci 2002;47(5):1133-4.
6. Teter CJ, Guthrie SK. A comprehensive review of MDMA and GHB: two common club drugs. Pharmacotherapy 2001;21(12):1486-513.
7. DEA drug intelligence brief. Club drugs: an update. (2001). Retrieved December 4, 2002, from http://dea.gov/pubs/intel/01026/index.html

General References:

1. Cammalleri M, Brancucci A, Berton F, Loche A, Gessa GL, Francesconi W. Gamma-hydroxybutyrate reduces GABA(A)-mediated inhibitory postsynaptic potentials in the CA1 region of the hippocampus. Neuropsychopharmacology 2002;27(6):960-9.
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8. Palatini P, Tedeschi L, Frison G, Padrini R, Zordan R, Orlando R, et al. Dose-dependent absorption and elimination of gamma-hydroxybutyric acid in healthy volunteers. Eur J Clin Pharmacol 1993;45:353-6.
9. Chin R, Sporer K, Cullison B, Dyer JE, Wu T. Clinical course of γ-hydroxybutyrate overdose. Ann Emergency Med 1998;31:716-22.