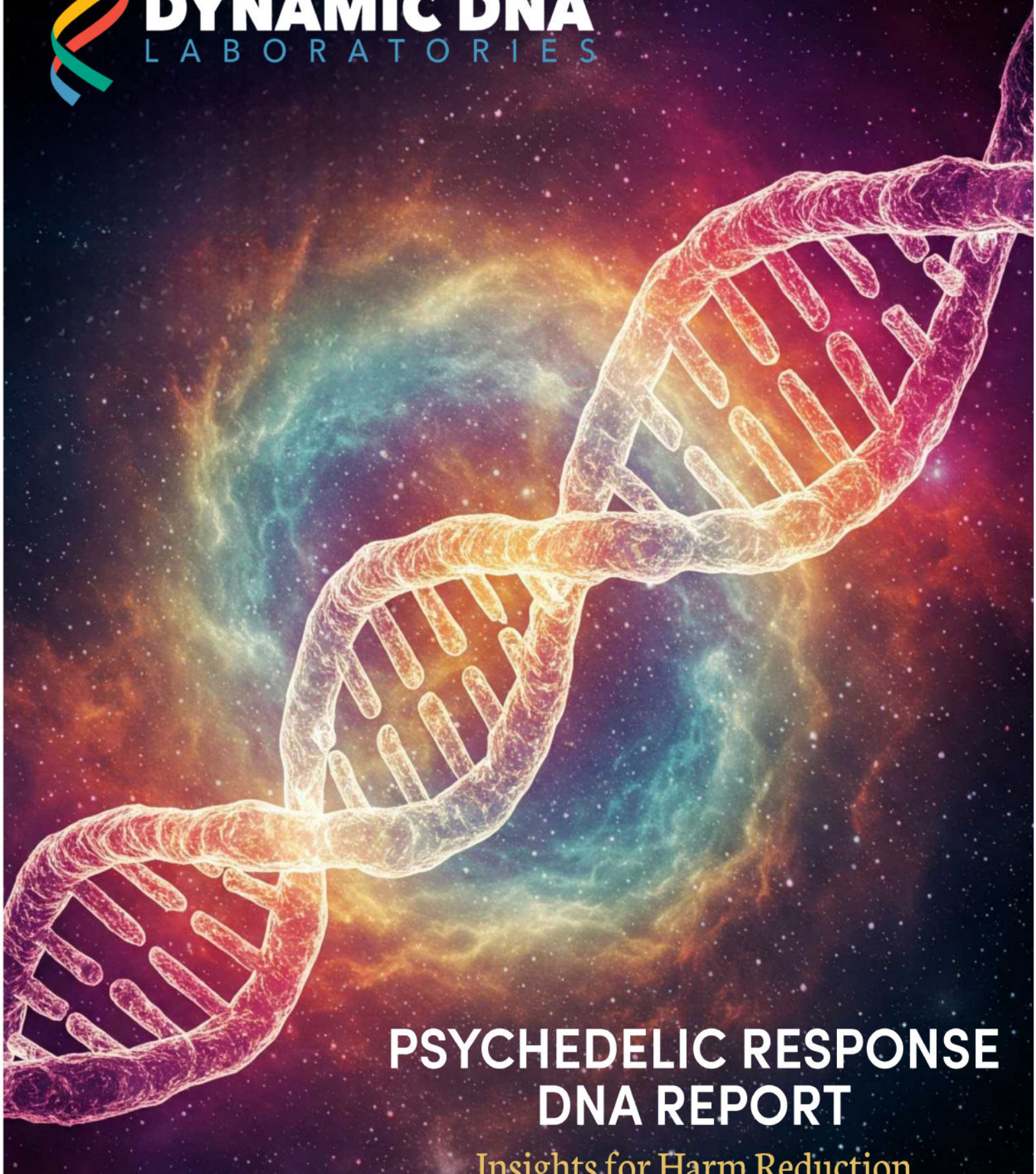




DYNAMIC DNA
LABORATORIES

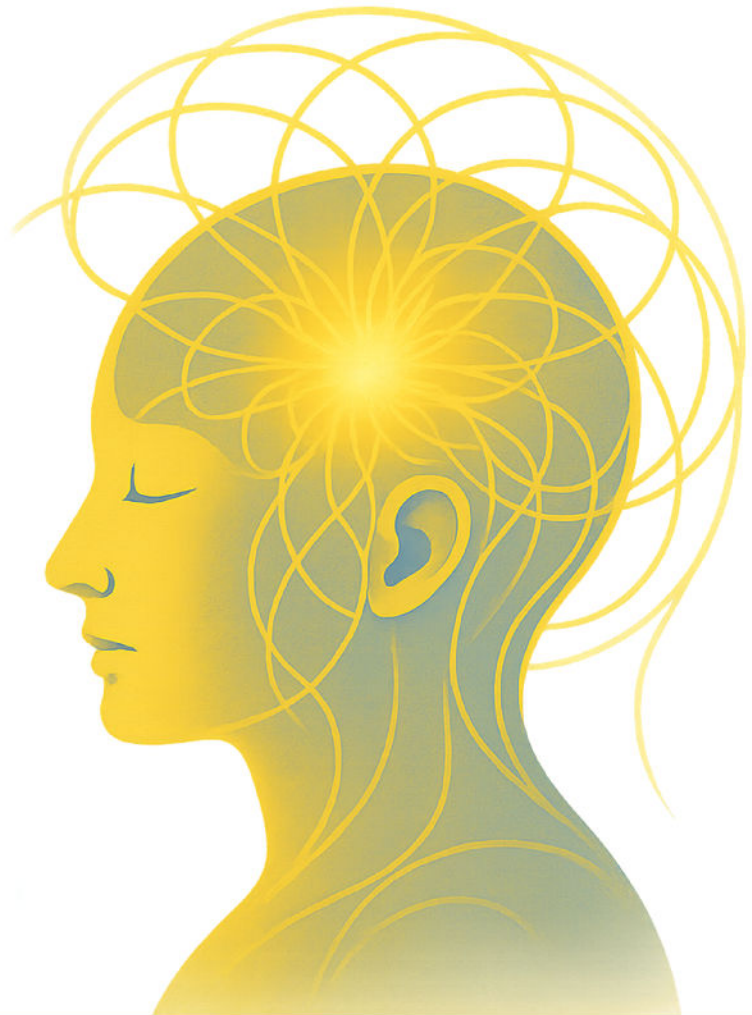


PSYCHEDELIC RESPONSE DNA REPORT

Insights for Harm Reduction
& Therapeutic Use

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About This Report

This report synthesizes a detailed DNA analysis by Dynamic DNA Labs, identifying key genetic variants relevant to the efficacy, safety, and nuances of metabolism and affinity of psychedelic substances. It offers a comprehensive evaluation of genetic responses for personalized psychedelic therapy. The insights provided, including pharmacogenomic counsel derived from research and educational data for non-FDA-approved substances, aim to enhance therapeutic outcomes by aligning psychedelic use with the customer's unique genetic makeup, fostering safer and more effective experiences.

CUSTOMER'S DATA

ID	614210
Customer	Rev. XXXXXX
Date of Birth	XX/XX/XX
Sex	Male
Received	04/30/24
Processed	28/30/24

Broad Overview

Psychoactive substances, often referred to as psychedelics, entheogens, or hallucinogens, have played a significant role in human history. From ancient rituals to modern therapeutic practices, they've shaped our understanding of consciousness and the human psyche. In the modern age, as the intersection of genetics and pharmacology becomes clearer, we're beginning to understand how our genetic makeup can influence our response to these powerful compounds.

With the emergence of personalized medicine, there's an increasing recognition of the importance of tailoring treatments to the individual. By understanding how genetics affects our response to psychoactive substances, we can pave the way for safer, more effective, and more personalized therapeutic interventions, as well as enhance recreational experiences.

Principle of the Test

Pharmacogenetics is an evolving field that seeks to understand the relationship between an individual's genetic makeup and their response to drugs. Every individual carries a unique genetic code, and even minor variations in this code can lead to significant differences in drug metabolism, efficacy, and risk of side effects. By analyzing specific genetic markers, pharmacogenetic tests can offer insights into how an individual might react to a particular substance.

The relevance of this test in the context of psychoactive substances cannot be understated. Given the profound and often unpredictable effects of these substances, having a predictive tool can be invaluable. For individuals seeking therapeutic benefits or even just a recreational experience, understanding one's genetic predispositions can make the difference between a positive, transformative experience and a challenging, potentially harmful one.

Predictable Experiences

The world of psychoactive substances is vast and varied, with each substance offering a unique journey. While the experience is influenced by numerous factors, from mindset to environment, genetics can play a pivotal role. For instance, certain genetic variants might make an individual more prone to a longer or stronger experience.

Understanding these genetic markers can provide a roadmap for individuals, helping them select substances and dosages that align with their desired outcomes. While it's important to note that no experience can be entirely predicted, having insights into one's genetic predispositions can offer a degree of assurance and preparation, enhancing the overall experience.

Harm Reduction

When it comes to psychoactive substances, safety and harm reduction are of paramount importance. These substances can be powerful, and while they offer potential benefits, they also come with risks. By understanding one's genetic makeup, individuals can be better equipped to make informed decisions about dosages, such as reducing the risk of adverse reactions.

Moreover, certain genetic markers can indicate a heightened risk of negative psychological reactions or even long-term mental health challenges following the use of specific substances. For individuals with these markers, foreknowledge allows for informed decision-making, potentially avoiding substances that carry higher risks for them. In the broader context of public health, pharmacogenetic insights can be a valuable tool in harm reduction strategies, promoting safer and more responsible use.

Uses

The applications of psychoactive substances are vast, ranging from therapeutic interventions to personal growth and recreation.

Psychedelic Assisted Therapy: Recent research has shown promising results in using psychedelics like psilocybin and MDMA to assist in therapy for conditions like depression, PTSD, and anxiety. Under controlled settings, these substances can catalyze profound therapeutic breakthroughs, often in cases where conventional treatments have failed.

Recreational and Personal Growth: Beyond therapy, many individuals seek out psychoactive substances for personal growth, introspection, or simply for recreation. When used responsibly, these experiences can offer insights, enhance creativity, or foster a deeper connection with oneself and the world.

Possible Side Effects

While psychoactive substances can offer profound experiences, they are not without risks. It's essential to be aware of potential side effects and to approach their use with caution.

Physical and Psychological: Depending on the substance and dosage, users might experience nausea, increased heart rate, or changes in blood pressure. Psychologically, while many experiences are positive, some individuals might face challenging or distressing experiences, characterized by anxiety, paranoia, or feelings of losing control.

Long-Term Considerations: While many psychoactive substances have a low potential for addiction, repeated use can lead to tolerance, requiring higher doses to achieve the same effects. Furthermore, individuals predisposed to certain mental health conditions might be at risk of exacerbating their symptoms or triggering latent issues.

Legality

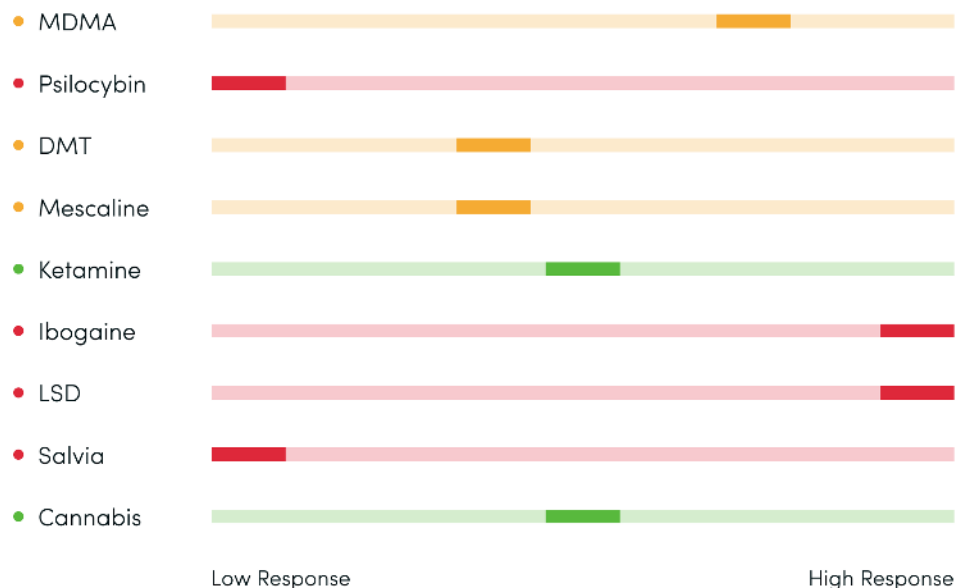
The legal status of psychoactive substances varies widely across countries, often influenced by cultural, historical, and political factors.

In the US: Many psychoactive substances are classified as Schedule I drugs, indicating a high potential for abuse and no accepted medical use. However, recent shifts in public perception and promising research results are leading to changes, with some jurisdictions decriminalizing or even legalizing certain substances for therapeutic use.

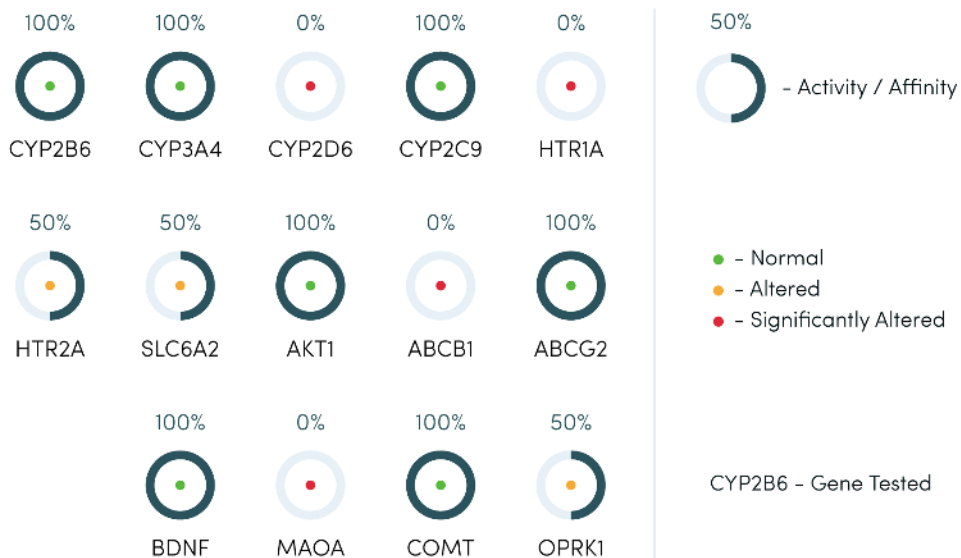
Internationally: Globally, the legal landscape is diverse and is changing amidst continuous emerging research on the various benefits of therapeutic uses. While some countries have strict prohibitions, others have more lenient policies or even traditional exceptions. For instance, Ayahuasca is recognized as a cultural heritage in parts of South America. As of 2024, some psychedelics have been given a legal or decriminalised foothold in: parts of Central Africa (Equatorial Guinea, Cameroon and Republic of the Congo), South Africa, Australia, the Bahamas, Brazil, the British Virgin Islands, Canada, the Czech Republic, Costa Rica, Israel, Jamaica, Mexico, Nepal, the Netherlands, Panama, Peru, Portugal, Samoa, Spain, and Switzerland.

Individual Response

Anticipated response to psychoactive compounds based on this genetic analysis.



Genetic Profile



Genotyping Information

Alleles Tested: ABCB1: rs1128503, rs1045642, rs2032582; ABCG2: rs12505410, rs13120400, rs2231142; BDNF: rs6265; CYP2B6: rs3745274, rs34223104, rs3211371, rs28399499; CYP2C9: rs56165452, rs9332131, rs72558189, rs7900194, rs28371686, rs28371685, rs1799853, rs1057910; CYP2D6: rs5030867, rs28371706, rs72549354, rs5030865, rs774671100, rs1135822, rs201377835, rs28371710, rs28371696, rs5030862, rs35742686, rs28371725, rs5030863, rs5030655, rs769258, rs59421388, rs3892097, rs1080985, rs16947, rs1135840, rs1065852, EXON9/INTRON2; CYP3A4: rs55785340, rs4986910, rs35599367, rs2740574, rs4987161, rs12721629, rs12721627; HTR2A: rs7997012; OPRK1: rs6473797; AKT1: rs2494732; HTR1A: rs6295, rs10042486; MAOA: rs1137070; SLC6A2: rs12708954, rs1861647, rs2242446, rs5569; COMT: rs4680.

MDMA

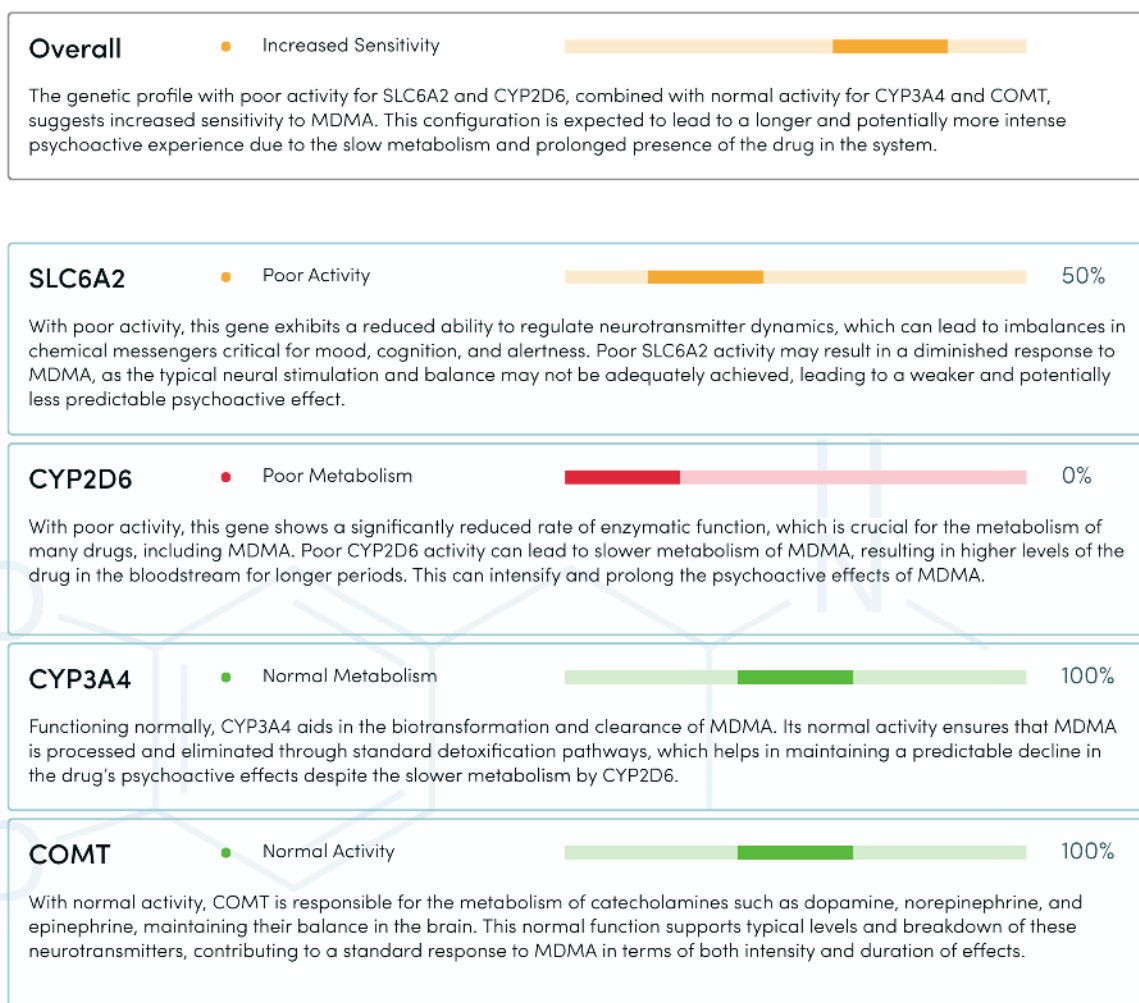
Introduction

MDMA (3,4-methylenedioxymethamphetamine), commonly known as ecstasy or molly, was first synthesized by the pharmaceutical company Merck in 1912 in Germany, originally intended as a precursor in chemical research rather than for direct therapeutic use. By the late 1970s and early 1980s, a small group of psychiatrists and therapists began exploring MDMA's potential in psychotherapy, particularly for its ability to enhance emotional openness and facilitate deeper connections between patients and therapists. However, in 1985, the Drug Enforcement Administration (DEA) placed MDMA under an emergency ban, classifying it as a Schedule I substance, meaning it was deemed to have no accepted medical use and a high potential for abuse. This decision forced many therapists to abandon its use, despite growing anecdotal evidence of its therapeutic benefits, particularly for trauma-related disorders.

MDMA is classified as an empathogen-entactogen, meaning it enhances feelings of emotional connection, empathy, and openness by increasing the release of neurotransmitters such as dopamine and serotonin. This makes it particularly promising in therapeutic settings, especially for treating PTSD and other mental health conditions. However, outside of clinical use, MDMA gained widespread popularity in the 1980s and 1990s as a recreational drug, often sold in pressed pill form as ecstasy and later in powder or crystalline form as molly. Its ability to produce euphoria, heightened sensory perception, and emotional bonding made it a staple in the electronic music scene, particularly at raves, dance clubs, music festivals, and social gatherings.

MDMA's effects typically begin within 30 to 60 minutes of ingestion and can last three to six hours, depending on the dose and individual metabolism. As research on its therapeutic applications continues, there is renewed interest in its potential medical benefits, particularly in controlled settings for psychedelic-assisted therapy.

Genetic Profile & Pharmacogenetic Implication^{1, 2}



Psilocybin Introduction

Psilocybin is a naturally occurring psychedelic compound found in over 200 species of mushrooms, commonly referred to as “magic mushrooms.” These mushrooms have been used for thousands of years by various cultures worldwide, particularly in spiritual ceremonies and religious rituals. Ancient cave paintings suggest that their use predates recorded history, with indigenous groups in Mesoamerica incorporating them into sacred traditions. References to psilocybin-containing mushrooms can be found in Mayan and Aztec texts, such as the Popol Vuh, where they were revered for their ability to induce visions, facilitate healing, and provide wisdom from the spirit world.

Psilocybin’s effects vary widely depending on dose, mindset, and environment, a concept known as “set and setting.” Even at low doses, users may experience hallucinations, enhanced perception, and altered cognition, while higher doses can lead to profound mystical experiences, ego dissolution, and deep introspection. These characteristics make psilocybin particularly promising in therapeutic settings, where its ability to induce spiritual awakenings and psychological breakthroughs has shown potential in treating mental health disorders.

Despite being classified as a Schedule I controlled substance, psilocybin has a low potential for abuse and is associated with few adverse effects, most of which are mild. Its active metabolite, psilocin, has a half-life of approximately three hours, and its therapeutic effects can persist long after the drug has left the system. Research suggests that just two or three psilocybin-assisted therapy sessions may lead to long-term or even permanent improvements in conditions such as substance use disorders (SUDs), depression, and end-of-life anxiety. Clinical trials have already demonstrated promising results in treating nicotine and alcohol addiction, paving the way for further research into its transformative potential in mental health care.

Genetic Profile & Pharmacogenetic Implication¹³

Overall

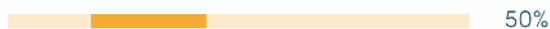
● Low Sensitivity



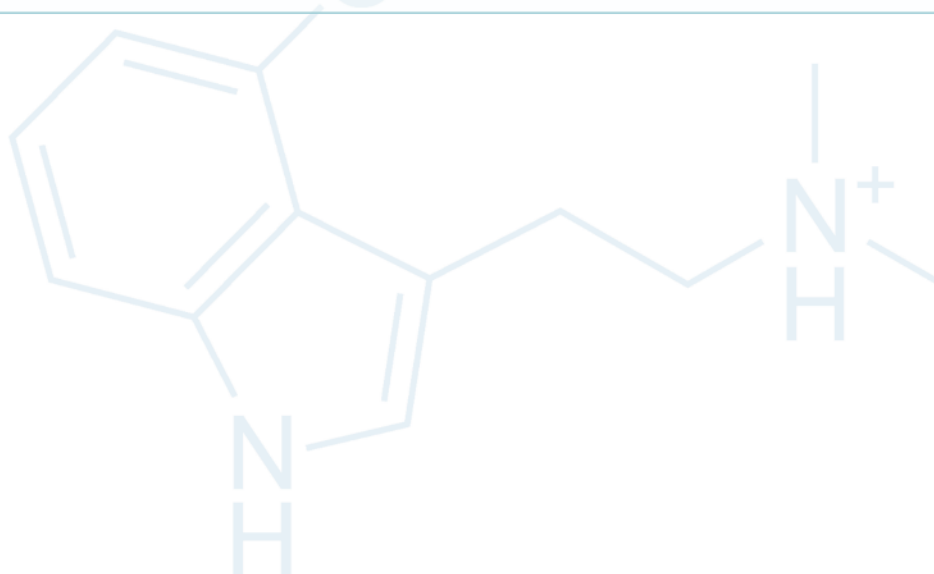
This profile suggests the potential for a weaker or less pronounced experience with psilocybin. Poor activity in the HTR2A gene implies a reduced sensitivity to the way psilocybin interacts with this brain receptor. Due to this lowered responsiveness, individuals might find that the typical visual, emotional, and cognitive effects of psilocybin are less intense or noticeable compared to those with normal HTR2A activity.

HTR2A

● Poor Activity



Poor activity in the HTR2A gene suggests decreased sensitivity to psilocybin. Individuals with this profile may experience weaker or less pronounced psychedelic effects than those with normal HTR2A activity. There might be a need for higher doses to achieve the desired effects, but this carries its own risks.



DMT

Introduction

Dimethyltryptamine (DMT) is a naturally occurring psychedelic compound that has been used for centuries in spiritual, religious, and medicinal ceremonies by indigenous cultures. It is best known as the active ingredient in ayahuasca, a traditional plant-based brew used by indigenous groups in South America, particularly in Ecuador, Colombia, Peru, and Brazil. In these cultures, DMT-containing brews have been revered for their healing properties, their ability to facilitate communication with the spirit world, and their role in deep introspection and self-discovery.

DMT has gained scientific interest due to its potential rapid-acting and long-lasting antidepressant effects, making it a promising candidate for psychedelic-assisted therapy. One of its most distinctive features is its short duration of action—when inhaled (smoked or vaped), the experience typically lasts between 5 to 20 minutes, while in ayahuasca, it can extend up to six hours. This brief yet intensely immersive psychedelic state allows for shorter treatment sessions, offering potential advantages for supervised therapeutic use.

The primary effects of DMT are psychological and sensory, often described as inducing intense visual and auditory hallucinations, euphoria, and an altered perception of time, space, and identity. Many users report entering otherworldly realms or experiencing what feels like a near-death or out-of-body experience. Depending on the dose and setting, DMT can facilitate deep personal insight, ego dissolution, and profound shifts in consciousness. As research into psychedelic medicine continues to expand, DMT remains at the forefront as a potential transformational tool for mental health treatment and personal exploration.

Genetic Profile & Pharmacogenetic Implication ^{7, 8}

Overall

● Complex Metabolism



The genetic profile with poor activity for both CYP2D6 and MAOA, combined with decreased HTR1A receptor affinity, suggests a prolonged but muted sensitivity to DMT. The slow initial and subsequent metabolic processes due to poor CYP2D6 and MAOA activity extend the presence and duration of DMT's effects. However, the decreased affinity of HTR1A receptors results in a reduced interaction with DMT, leading to a less intense and possibly less profound psychoactive experience.

CYP2D6

● Poor Metabolism

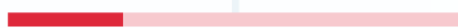


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This gene encodes an enzyme that is essential for the metabolic processing of various psychoactive agents. Poor activity of CYP2D6 leads to a reduced pace in the breakdown of substances such as 5-MeO-DMT, resulting in a more extended retention of the entheogen in the body. This sluggishness may cause a longer duration of the psychoactive effects, potentially deepening the ayahuasca experience. The individual with this genetic profile could be predisposed to a more drawn-out journey, experiencing the effects of the entheogen for an extended period, thus enhancing the opportunity for profound spiritual encounters but also raising the specter of prolonged discomfort or other adverse reactions.

MAOA

● Poor Activity

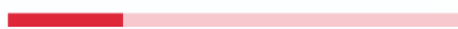


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This enzyme is crucial in the metabolic pathway for deactivating monoamine neurotransmitters. Poor activity in MAOA indicates a similar reduction in the metabolic degradation of monoamines, which could compound the effects of a slow CYP2D6 metabolism, allowing for an even more prolonged presence of 5-MeO-DMT and its effects. The synergistic effect of compromised MAOA and CYP2D6 activities could significantly extend and intensify the psychoactive experience elicited by ayahuasca.

HTR1A

● Decreased Affinity



0%

Decreased affinity at the HTR1A receptors translates into a less responsive serotonergic system. While this might ordinarily mitigate the intensity of the psychoactive experience, in the presence of poor enzymatic activity, it could lead to a nuanced situation. The individual may not feel the effects of ayahuasca as intensely or as quickly as others might, which could influence the dosing requirements and the overall intensity of the experience.

Mescaline Introduction

Mescaline is a classic psychedelic found in peyote (*Lophophora williamsii*) and San Pedro (*Trichocereus* species) cacti, both of which have been used for thousands of years in spiritual and healing ceremonies. Peyote is central to Native American religious practices, while San Pedro, known as huachuma, has long been revered in Andean shamanism for its medicinal and visionary properties. These traditions emphasize mescaline's role in fostering introspection, spiritual insight, and emotional healing.

The mescaline experience is mystical and visually immersive, often described as bright and colorful, with effects including visions, altered perception, deep introspection, and heightened emotional connection. Many report feelings of empathy, love, and physical euphoria, though consumption can cause nausea and vomiting due to its bitter taste. Unlike other psychedelics such as LSD or psilocybin, mescaline is known for its gentler, dreamlike quality and its ability to induce a strong sense of unity with nature.

Research suggests mescaline may have therapeutic potential, particularly in treating addiction, anxiety, and depression when combined with psychotherapy. Studies indicate that mescaline's effects on neuroplasticity and serotonin receptors could contribute to lasting mental health benefits. However, its long duration (8+ hours) poses challenges for clinical use, requiring extended monitoring in therapeutic settings. Despite the renewed interest in psychedelics, mescaline remains a Schedule I substance in the U.S., limiting its accessibility for research and treatment.

Genetic Profile & Pharmacogenetic Implication¹⁵

Overall

● Lower Sensitivity



The genetic profile with decreased activity for both CYP2D6 and HTR2A suggests a prolonged and muted Mescaline experience. The decreased CYP2D6 activity leads to slower metabolism and extended presence of Mescaline in the body, resulting in a longer duration of effects. The decreased HTR2A activity reduces the intensity of the psychoactive effects due to diminished interaction with serotonin receptors. The combination of these factors results in a Mescaline experience that begins slowly with extended effects but is less intense overall.

CYP2D6

● Poor Metabolism



0%

An enzyme essential for the metabolism of numerous psychoactive compounds, decreased activity in CYP2D6 leads to a slower processing rate of Mescaline. This diminished metabolic capacity can result in Mescaline remaining in the body for an extended period, potentially prolonging its psychoactive effects. Individuals with this genetic profile may experience a sustained duration of Mescaline's effects, which could deepen the intensity of spiritual or therapeutic sessions but also heighten the risk of enduring adverse effects.

HTR2A

● Decreased Activity



50%

This gene codes for the serotonin 2A receptor, a critical mediator of Mescaline's psychedelic effects. Decreased activity in HTR2A may result in a reduced sensitivity to Mescaline, potentially dulling its psychedelic impact. The dual decrease in both metabolic rate and receptor sensitivity could lead to a scenario where Mescaline's effects are prolonged yet less intense, offering a unique but possibly subdued psychedelic experience.


Ketamine Introduction

Ketamine (ketamine hydrochloride) is a dissociative anesthetic and analgesic (painkiller) that was developed in the 1960s and FDA-approved in the 1970s for medical use. It has since become widely used in both human and veterinary medicine. By the 1980s, its dissociative and psychedelic properties led to increasing recreational use, earning it a reputation as both a club drug and a powerful therapeutic tool.


Unlike classic psychedelics, ketamine works through the NMDA receptor, inducing a dreamlike, out-of-body state known as the "K-hole" at higher doses. Its effects can include euphoria, dissociation, visual and auditory hallucinations, and a reduced sensitivity to pain. These properties make ketamine unique among psychedelic therapies, as it produces rapid antidepressant effects that can last for days to weeks after a single dose.

Ketamine is one of the few psychedelics legally available for mental health treatment. Ketamine-assisted psychotherapy (KAP) combines ketamine's effects with guided psychotherapy to help patients process trauma, depression, anxiety, and PTSD. Research continues to explore its potential in treating substance use disorders, chronic pain, and suicidal ideation, positioning ketamine as a groundbreaking tool in modern mental health care.


Genetic Profile & Pharmacogenetic Implication ^{11, 12}

Overall ● Standard Response 


This profile suggests a standard ketamine experience, including its effects on the body and its potential therapeutic benefits. With normal activity across BDNF, CYP2B6, and CYP2C9, ketamine is likely to be metabolized, interact with the brain, and be eliminated in a typical manner. This genetic predisposition might not necessitate significant dosage adjustments, assuming there are no other individual health factors or medications involved that could interfere with ketamine therapy.

BDNF ● Normal Activity  100%

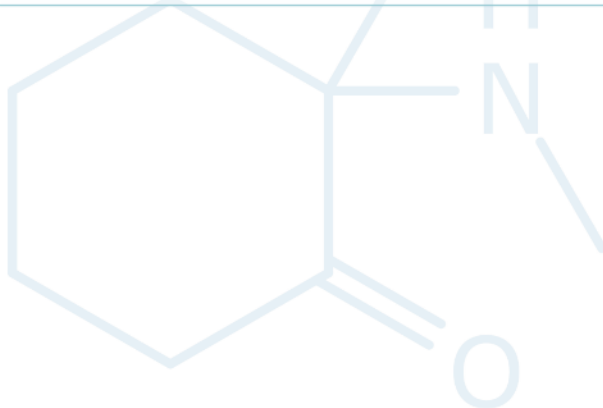
Brain-derived neurotrophic factor (BDNF) plays a vital role in neuroplasticity, influencing how the brain adapts and changes. Normal activity in this gene suggests a standard baseline for ketamine's potential effects on mood regulation and its possible therapeutic actions.

CYP2B6 ● Normal Metabolism  100%

A key player in ketamine metabolism, normal activity in CYP2B6 implies typical initial processing of ketamine. This standard metabolic function suggests that ketamine will be broken down at a consistent pace, contributing to a predictable timeframe for its dissociative and possible antidepressant effects.

CYP2C9 ● Normal Metabolism  100%

Also involved in ketamine metabolism, normal activity in CYP2C9 reinforces the expectation of standard processing and elimination of ketamine. This indicates that the drug's duration of effects and clearance from the body should align with established pharmacological data.



Ibogaine Introduction

Tabernanthe iboga is a shrub native to Central West Africa, particularly Gabon, Cameroon, and the Democratic Republic of Congo. Its root bark contains ibogaine, a psychoactive alkaloid that has been used for centuries in Bwiti spiritual and initiation ceremonies. In traditional settings, high doses of iboga root induce intense hallucinogenic experiences that serve as rites of passage and are believed to facilitate ancestral communication and healing. At lower doses, iboga has been used to combat fatigue, hunger, and thirst.

Beyond its traditional use, ibogaine has gained attention for its anti-addictive properties, showing promise in treating opioid use disorder (OUD), alcohol use disorder (AUD), and other substance dependencies by reducing cravings and withdrawal symptoms. Research is also exploring its potential applications in mental health conditions such as depression and PTSD. However, ibogaine remains a Schedule I-controlled substance in the United States, meaning it is not approved for medical treatment. Despite this, ibogaine treatment clinics operate in countries like Mexico, Canada, and Costa Rica.

While ibogaine offers potential therapeutic benefits, it also carries significant risks, particularly concerning cardiovascular health. The substance can cause irregular heart rhythms (arrhythmias), bradycardia (dangerously slow heart rate), seizures, and gastrointestinal distress. Individuals with a history of heart conditions, severe obesity, or certain psychiatric disorders (e.g., schizophrenia) should avoid ibogaine. Additionally, certain medications, including SSRIs, antipsychotics, and heart-related drugs, may interact dangerously with ibogaine. Due to these risks, ibogaine should always be administered under medical supervision to ensure safety.

Genetic Profile & Pharmacogenetic Implication^{9, 10}

Overall

● High Sensitivity



This genetic profile suggests a potentially prolonged and intense Ibogaine experience. Poor CYP2D6 activity will greatly slow down Ibogaine metabolism, leading to much higher levels of the drug remaining in the body. Poor ABCB1 activity at the blood-brain barrier will further compound this effect.

CYP2D6

● Poor Metabolism

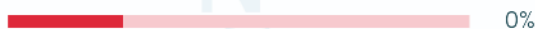


0%

Reduced activity of this key metabolic enzyme indicates a less efficient breakdown of Ibogaine, leading to prolonged circulation of the drug within the system. The consequent increased exposure may amplify the psychoactive effects and extend the duration of therapeutic impact, potentially heightening the risk of adverse events. Such a metabolic profile suggests that the individual may require careful dosing, with extended intervals to accommodate the slower processing and to ensure safety.

ABCB1

● Poor Activity



0%

Poor activity of the ABCB1 gene, coding for the efflux transporter P-glycoprotein, could result in a diminished clearance of Ibogaine from the brain. This inefficiency may cause an accumulation of the substance in neural tissues, possibly intensifying the central effects or leading to unexpected neurotoxicity. The careful modulation of Ibogaine's entry to and exit from the central nervous system becomes critical under these circumstances.

ABCG2

● Normal Activity



100%

With normal activity, the ABCG2 transporter is anticipated to handle the cellular efflux of Ibogaine effectively, likely maintaining a balance in tissue distribution and aiding in the overall elimination of the alkaloid. This standard activity may provide some equilibrium to the altered dynamics imposed by the other two genes, helping to prevent excessive peripheral accumulation of Ibogaine.

LSD

Introduction

LSD (lysergic acid diethylamide), commonly known as acid, is one of the most potent psychedelic substances. It was first synthesized in 1938 by Swiss chemist Albert Hofmann, but its psychedelic effects remained unknown until 1943, when Hofmann accidentally ingested a small amount and experienced its mind-altering properties. Throughout the 1950s and 1960s, LSD was widely studied for its potential in psychotherapy, with psychiatrists exploring its ability to enhance self-awareness and treat mental health disorders. At the same time, the CIA conducted secret experiments investigating its use in mind control and interrogation.

By the 1960s, LSD became deeply intertwined with the counterculture movement, embraced for its supposed ability to expand consciousness and challenge societal norms. Its association with anti-war activism and rebellion led to increasing government restrictions, culminating in its classification as a Schedule I drug in 1968, effectively banning its use and research.

LSD is typically synthesized as a crystalline powder, dissolved in liquid, and absorbed onto blotter paper ("tabs") for ingestion. Its effects can last six hours or longer, with users experiencing intense visual and auditory hallucinations, altered perceptions of time and reality, heightened emotions, and deep introspection. Many also report long-lasting positive mood shifts that persist for days or weeks. Common physical effects include dilated pupils, increased heart rate, sweating, and appetite suppression. Despite its prohibition, modern research is once again exploring LSD's potential in treating depression, PTSD, and anxiety, sparking renewed interest in its therapeutic applications.

Genetic Profile & Pharmacogenetic Implication³

Overall

● High Sensitivity



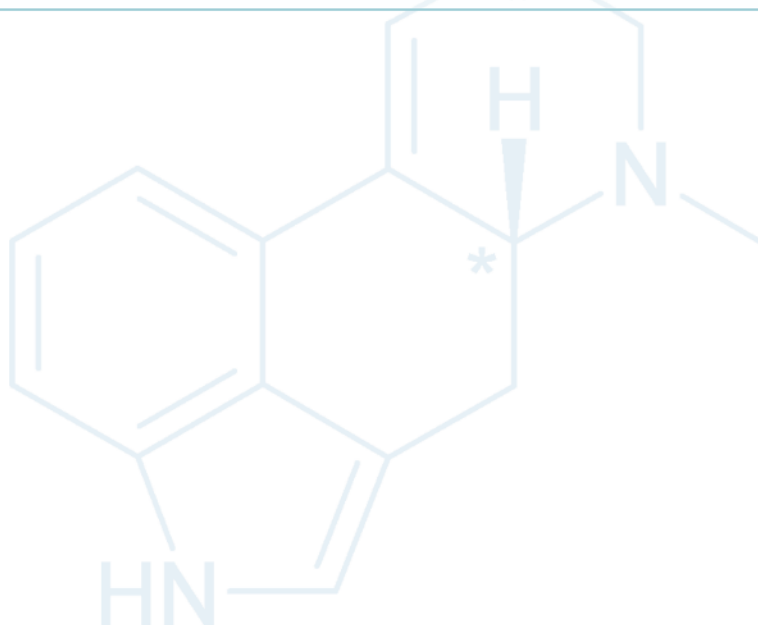
This profile suggests a potentially more intense and longer-lasting LSD experience. Poor CYP2D6 activity indicates slower metabolism of LSD within the body. This could lead to higher concentrations of LSD remaining active for an extended period, resulting in both amplified effects and a prolonged duration of the experience.

CYP2D6

● Poor Metabolism



Poor activity in the CYP2D6 gene indicates a slower metabolic breakdown of LSD. This may result in a more intense and prolonged LSD experience compared to individuals with normal CYP2D6 activity. With slower metabolism, there's a higher chance of LSD accumulating in the system, potentially increasing the likelihood of both desired and undesired effects. Those with poor CYP2D6 activity might be more sensitive to LSD's psychoactive effects and could have an increased risk of side effects.



Salvia

Introduction

Salvia divinorum, commonly known as diviner's sage or magic mint, is a psychoactive plant native to the Mazatec regions of Mexico, where it has been used for centuries in shamanic rituals and traditional medicine. The Mazatec people have long employed Salvia divinorum for spiritual visions, healing ceremonies, and treating ailments such as insect bites, skin conditions, menstrual cramps, depression, and even alcohol addiction.

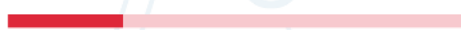
The plant's psychoactive effects come from Salvinorin A, the most potent natural hallucinogen known, which uniquely targets kappa opioid receptors (KOR) rather than the serotonergic system like most psychedelics. Unlike LSD or psilocybin, Salvia induces intense dissociative and perceptual distortions, often leading to geometric visions, dream-like experiences, out-of-body sensations, and altered perceptions of time and space. Users may feel as if they are merging with objects or transported to other dimensions. The effects vary depending on dosage and method of ingestion, with traditional tea preparations producing gentler experiences, while smoking or chewing the leaves leads to rapid, more intense hallucinations.

While some users report spiritual insight, synesthesia, and enhanced appreciation of the environment, others experience panic, paranoia, loss of motor control, and difficulty integrating the experience—particularly with potent extracts. Despite its therapeutic potential in treating depression, substance use disorder (SUD), and pain, Salvia divinorum remains federally legal in the U.S. but is banned in 29 states. It is also not classified under United Nations drug control conventions, allowing for continued research into its clinical applications and unique pharmacology.

Genetic Profile & Pharmacogenetic Implication¹⁴

Overall

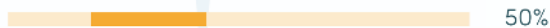
● Low Sensitivity



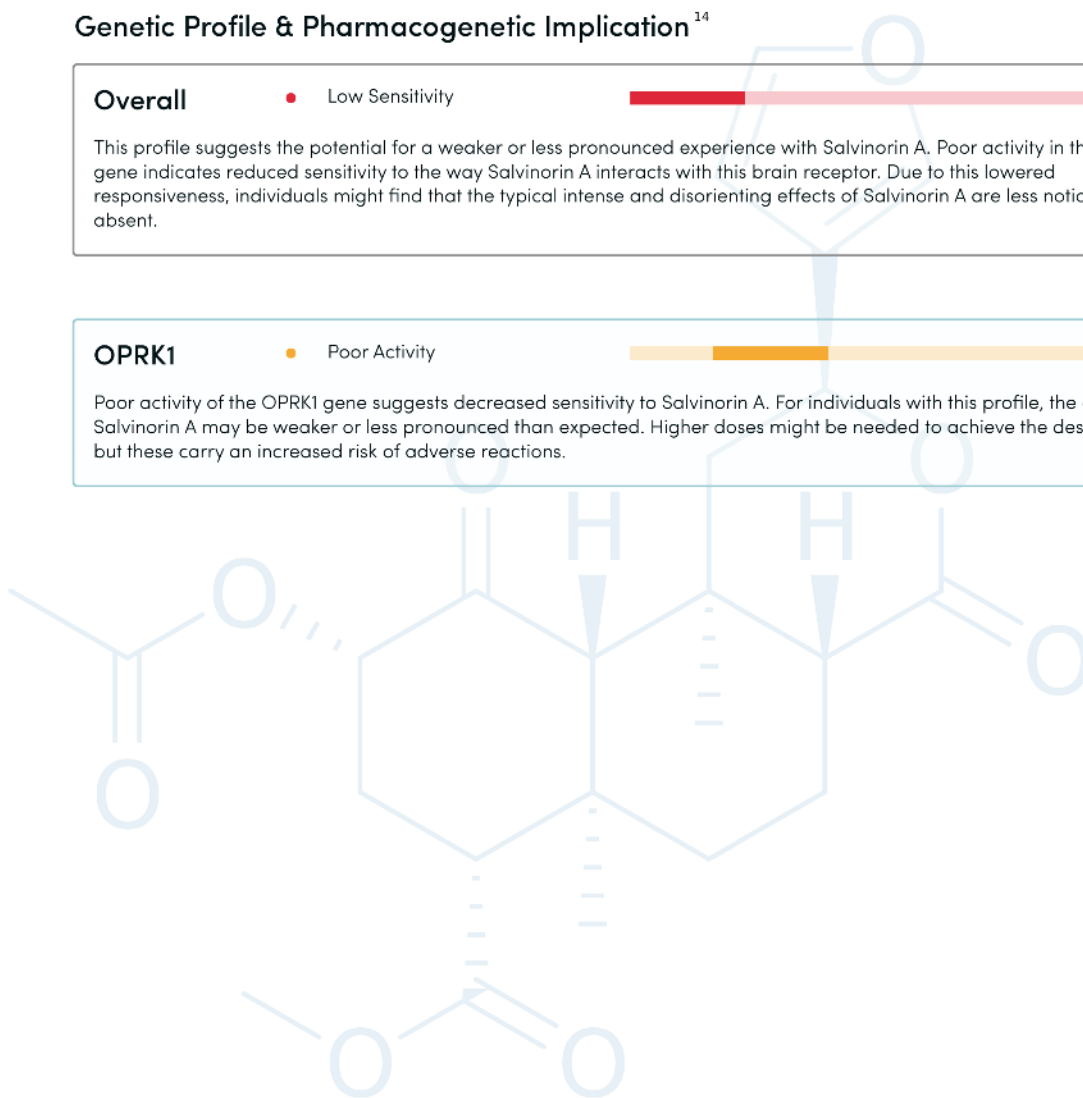
This profile suggests the potential for a weaker or less pronounced experience with Salvinorin A. Poor activity in the OPRK1 gene indicates reduced sensitivity to the way Salvinorin A interacts with this brain receptor. Due to this lowered responsiveness, individuals might find that the typical intense and disorienting effects of Salvinorin A are less noticeable or absent.

OPRK1

● Poor Activity



Poor activity of the OPRK1 gene suggests decreased sensitivity to Salvinorin A. For individuals with this profile, the effects of Salvinorin A may be weaker or less pronounced than expected. Higher doses might be needed to achieve the desired effects, but these carry an increased risk of adverse reactions.



Cannabis Introduction

Cannabis is a flowering plant native to Asia that has been cultivated for its mind-altering properties for over 12,000 years. Historically, it has been used across various cultures, particularly in China, India, the Middle East, and the Mediterranean, for religious, medicinal, and recreational purposes. Cannabis plants that lack significant levels of psychoactive compounds, known as hemp, have been valued for their industrial applications, including fiber and rope production.

The plant contains numerous cannabinoids, the most notable being tetrahydrocannabinol (THC) and cannabidiol (CBD). THC is primarily responsible for cannabis's psychoactive effects, while CBD is known for its therapeutic potential without intoxicating properties. Methods of consumption include smoking, edibles, and topicals, with effects varying in onset and duration.

Cannabis is increasingly recognized for its therapeutic benefits. Research suggests it may be effective in managing chronic pain, inflammation, epilepsy, anxiety, depression, and post-traumatic stress disorder (PTSD). It is widely used to alleviate chemotherapy-induced nausea, stimulate appetite in patients with conditions like HIV/AIDS, and provide relief for individuals with multiple sclerosis and neurodegenerative diseases. Additionally, CBD has gained attention for its potential role in reducing seizures in treatment-resistant epilepsy. As research continues, cannabis is being explored for its neuroprotective properties and possible applications in mental health and addiction treatment.

Its legal status varies globally, with increasing acceptance for both medicinal and recreational use in many regions. As policies evolve, cannabis-based therapies are becoming more accessible, offering new treatment options for a variety of medical conditions.

Genetic Profile & Pharmacogenetic Implication^{4, 5, 6}

Overall

● Standard Response



This profile suggests a standard cannabis experience, both in terms of how it acts in the body and the duration of its effects. Standard activity across AKT1, CYP2C9, and CYP3A4 indicates that cannabis will likely be processed and eliminated in a typical manner.

AKT1

● Normal Activity



This gene plays a role in cell signaling and growth, and its normal activity suggests standard initial processing of cannabis within the body. This baseline function likely leads to a typical onset and distribution of cannabis's effects.

CYP2C9

● Normal Metabolism



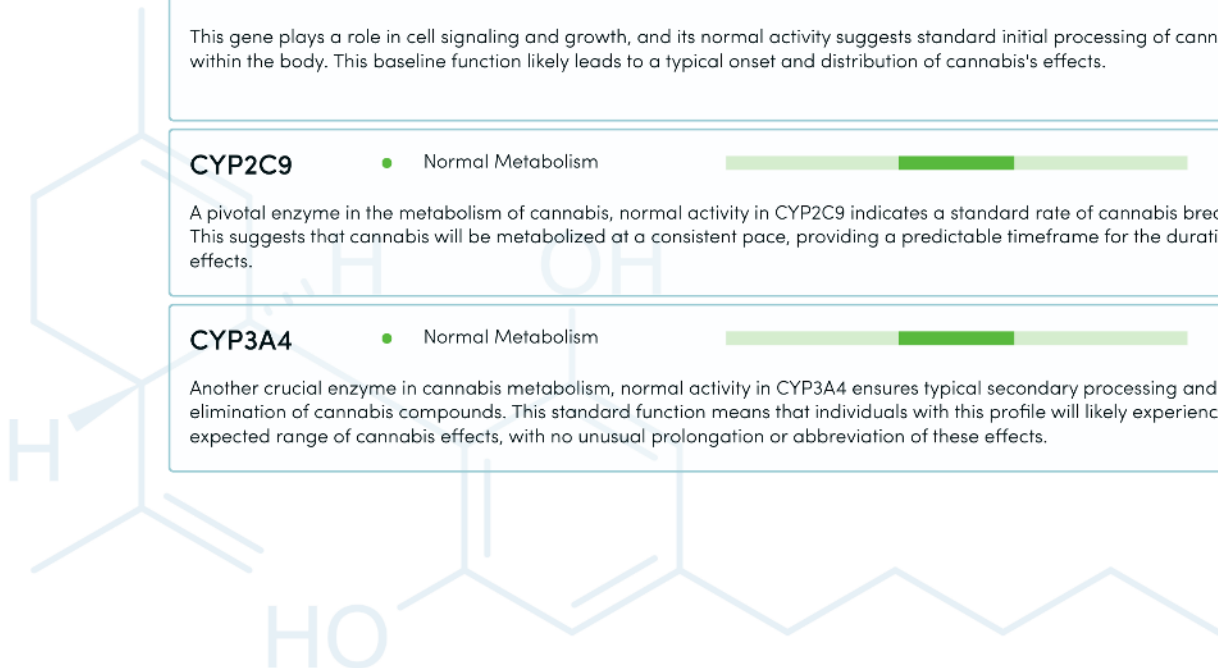
A pivotal enzyme in the metabolism of cannabis, normal activity in CYP2C9 indicates a standard rate of cannabis breakdown. This suggests that cannabis will be metabolized at a consistent pace, providing a predictable timeframe for the duration of its effects.

CYP3A4

● Normal Metabolism



Another crucial enzyme in cannabis metabolism, normal activity in CYP3A4 ensures typical secondary processing and elimination of cannabis compounds. This standard function means that individuals with this profile will likely experience the expected range of cannabis effects, with no unusual prolongation or abbreviation of these effects.



Methodology

Laboratory specimens associated with this report were analyzed using a DNA microarray. Genomic DNA was extracted from the submitted specimen and amplified using whole genome amplification techniques. The polymorphisms assayed in this report were targeted through the use of oligonucleotide primers. Single nucleotide polymorphisms were determined by fluorophore-based detection of a labeled probe hybridized to the complementary target sequence.

Limitations

This test detects polymorphisms other than those listed in this report. Polymorphisms not detected in this analysis include known mutations that result in an altered predisposition to the conditions discussed in this report. The absence of a detectable gene variant or polymorphism does not rule out the possibility that the test subject has an increased chance of developing any conditions discussed here. In very rare circumstances, polymorphisms in the primer or probe binding site may affect genotyping results. This test does not identify non-genetic factors that may contribute to an individual's predisposition to developing any of the conditions discussed in these findings. This test has not been approved by the United States Food and Drug Administration (FDA) and should not be used as the sole evidence of diagnosis. Genetic screening does not replace the need for regular clinical screenings for any of the conditions or analytes mentioned in this report.

Disclaimer

The content provided in this report, and any of Dynamic DNA Laboratories affiliated websites, is not intended to promote or endorse the use of illegal substances. While acknowledging that psychedelics are utilized by individuals globally, the educational information presented in this report is aimed at encouraging safe and responsible use of psychedelic substances, should individuals choose to engage with them.

We do not offer mental health or medical services, and our DNA analysis and report services are not a substitute for professional medical, psychological, or psychiatric diagnosis, treatment, or advice. All content created and disseminated by Dynamic DNA Laboratories is intended for informational and educational purposes only.

This report does not offer medical advice and does not replace the guidance of a qualified and licensed therapist, healer, counsellor, guide, or shaman.

Considering the nature of the psychedelic experience, there are individuals who should abstain from using psychedelics entirely. For those dealing with conditions such as major depressive disorder, substance addiction, end-of-life anxiety, or an eating disorder, it is strongly advised to only engage with psychedelics under the supervision of a qualified professional.

References

1. Vizeli P, Meyer Zu Schwabedissen HE, Liechti ME. No major role of norepinephrine transporter gene variations in the cardiostimulant effects of MDMA. *Eur J Clin Pharmacol*. 2018 Mar;74(3):275–283. doi: 10.1007/s00228-017-2392-2. Epub 2017 Dec 2. PMID: 29198060; PMCID: PMC5808057.
2. Aitchison KJ, Tsapakis EM, Huezó-Díaz P, Kerwin RW, Forsling ML, Wolff K. Ecstasy (MDMA)-induced hyponatraemia is associated with genetic variants in CYP2D6 and COMT. *J Psychopharmacol*. 2012 Mar;26(3):408–18. doi: 10.1177/0269881111434624. Epub 2012 Feb 1. PMID: 22303032.
3. Vizeli P, Straumann I, Holze F, Schmid Y, Dolder PC, Liechti ME. Genetic influence of CYP2D6 on pharmacokinetics and acute subjective effects of LSD in a pooled analysis. *Sci Rep*. 2021 May 25;11(1):10851. doi: 10.1038/s41598-021-90343-y. PMID: 34035391; PMCID: PMC8149637.
4. Di Forti M, Iyegbe C, Sallis H, Koliakou A, Falcone MA, Paparelli A, Sirianni M, La Cascia C, Stilo SA, Marques TR, Handley R, Mondelli V, Dazzan P, Pariante C, David AS, Morgan C, Powell J, Murray RM. Confirmation that the AKT1 (rs2494732) genotype influences the risk of psychosis in cannabis users. *Biol Psychiatry*. 2012 Nov 15;72(10):811–6. doi: 10.1016/j.biopsych.2012.06.020. Epub 2012 Jul 24. PMID: 22831980.
5. Nasrin S, Watson CJW, Perez-Paramo YX, Lazarus P. Cannabinoid Metabolites as Inhibitors of Major Hepatic CYP450 Enzymes, with Implications for Cannabis-Drug Interactions. *Drug Metab Dispos*. 2021 Dec;49(12):1070–1080. doi: 10.1124/dmd.121.000442. Epub 2021 Sep 7. PMID: 34493602.
6. Doohan PT, Oldfield LD, Arnold JC, Anderson LL. Cannabinoid Interactions with Cytochrome P450 Drug Metabolism: a Full-Spectrum Characterization. *AAPS J*. 2021 Jun 28;23(4):91. doi: 10.1208/s12248-021-00616-7. PMID: 34181150.
7. Shen HW, Jiang XL, Winter JC, Yu AM. Psychedelic 5-methoxy-N,N-dimethyltryptamine: metabolism, pharmacokinetics, drug interactions, and pharmacological actions. *Curr Drug Metab*. 2010 Oct;11(8):659–66. doi:10.2174/138920010794233495. PMID: 20942780; PMCID: PMC3028383.
8. Reckweg JT, Uthaug MV, Szabo A, Davis AK, Lancelotta R, Mason NL, Ramaekers JG. The clinical pharmacology and potential therapeutic applications of 5-methoxy-N,N-dimethyltryptamine (5-MeO-DMT). *J Neurochem*. 2022 Jul;162(1):128–146. doi: 10.1111/jnc.15587. Epub 2022 Mar 8. PMID: 35149998; PMCID: PMC9314805.
9. Glue P, Winter H, Garbe K, Jakobi H, Lyudin A, Lenagh-Glue Z, Hung CT. Influence of CYP2D6 activity on the pharmacokinetics and pharmacodynamics of a single 20 mg dose of ibogaine in healthy volunteers. *J Clin Pharmacol*. 2015 Jun;55(6):680–7. doi: 10.1002/jcph.471. Epub 2015 Feb 25. PMID: 25651476.
10. F Martins ML, Heydari P, Li W, Martínez-Chávez A, Venekamp N, Lebre MC, Lucas L, Beijnen JH, Schinkel AH. Drug Transporters ABCB1 (P-gp) and OATP, but not Drug-Metabolizing Enzyme CYP3A4, Affect the Pharmacokinetics of the Psychoactive Alkaloid Ibogaine and its Metabolites. *Front Pharmacol*. 2022 Mar 4;13:855000. doi: 10.3389/fphar.2022.855000. PMID: 35308219; PMCID: PMC8931498.
11. Woelfer M, Li M, Colic L, Liebe T, Di X, Biswal B, Murrough J, Lessmann V, Brigadski T, Walter M. Ketamine-induced changes in plasma brain-derived neurotrophic factor (BDNF) levels are associated with the resting-state functional connectivity of the prefrontal cortex. *World J Biol Psychiatry*. 2020 Nov;21(9):696–710. doi: 10.1080/15622975.2019.1679391. Epub 2019 Nov 4. PMID: 31680600.
12. Langmia IM, Just KS, Yamoune S, Müller JP, Stingl JC. Pharmacogenetic and drug interaction aspects on ketamine safety in its use as antidepressant - implications for precision dosing in a global perspective. *Br J Clin Pharmacol*. 2022 Dec;88(12):5149–5165. doi: 10.1111/bcp.15467. Epub 2022 Aug 5. PMID: 35863300.
13. Catlow BJ, Song S, Paredes DA, Kirstein CL, Sanchez-Ramos J. Effects of psilocybin on hippocampal neurogenesis and extinction of trace fear conditioning. *Exp Brain Res*. 2013 Aug;228(4):481–91. doi: 10.1007/s00221-013-3579-0. Epub 2013 Jun 2. PMID: 23727882.
14. Butelman ER, Mandau M, Tidgewell K, Priszczano TE, Yuferov V, Kreek MJ. Effects of salvinorin A, a kappa-opioid hallucinogen, on a neuroendocrine biomarker assay in nonhuman primates with high kappa-receptor homology to humans. *J Pharmacol Exp Ther*. 2007 Jan;320(1):300–6. doi: 10.1124/jpet.106.112417. Epub 2006 Oct 23. PMID: 17060493
15. Kolaczynska KE, Luethi D, Trachsel D, Hoener MC, Liechti ME. Receptor Interaction Profiles of 4-Alkoxy-3,5-Dimethoxy-Phenethylamines (Mescaline Derivatives) and Related Amphetamines. *Front Pharmacol*. 2022 Feb 9;12:794254. doi: 10.3389/fphar.2021.794254. PMID: 35222010; PMCID: PMC8865417.