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Effects of Lysergic Acid Diethylamide as a Drug Dose Among Youth

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ABSTRACT: Lysergic acid diethylamide, commonly known as LSD (from German Lysergsäure-diethylamid), also known colloquially as acid, is a potent psychedelic drug.[12] Effects typically include intensified thoughts, emotions, and sensory perception.[13] At sufficiently high dosages LSD manifests primarily mental, visual, as well as auditory, hallucinations.[14][15] Dilated pupils, increased blood pressure, and increased body temperature are typical.[16] Effects typically begin within half an hour and can last for up to 20 hours (although on average the trip lasts 8-12 hours).[16][17] LSD is also capable of causing mystical experiences and ego dissolution.[15][18] It is used mainly as a recreational drug or for spiritual reasons.[16][19] LSD is both the prototypical psychedelic and one of the "classical" psychedelics, being the psychedelics with the greatest scientific and cultural significance.[12] LSD is typically either swallowed or held under the tongue.[13] It is most often sold on blotter paper and less commonly as tablets, in a watery solution or in gelatin squares called panes.[16]LSD is considered to be non-addictive with low potential for abuse. Despite this, the US government made it illegal as part of the 'War on Drugs' after experimenting on people with it during MKULTRA. [20][21] Adverse psychological reactions are possible, such as anxiety, paranoia, and delusions.[7] LSD is active in small amounts relative to other psychoactive compounds with doses measured in micrograms.[22] It is possible for LSD to induce either intermittent or chronic visual hallucinations, in spite of no further use. Common effects include visual snow and palinopsia. In cases where this causes distress or impairment it is diagnosed as hallucinogen persisting perception disorder (HPPD).[23][24] While overdose from LSD is unknown, LSD can cause injury and death as a result of accidents stemming from psychological impairment.[12][16] The effects of LSD are thought to stem primarily from it being an agonist at the 5-HT_{2A} (serotonin) receptor, and while exactly how LSD exerts its effects by agonism at this receptor is still not fully known, corresponding increased glutamatergic neurotransmission and reduced default mode network activity are thought to be key mechanisms of action.[7][12][21][25][26] In addition to serotonin, LSD also binds to dopamine D_1 and D_2 receptors, which is why LSD tends to be more stimulating than compounds such as psilocybin.[27][28] In pure form, LSD is clear or white in color, has no smell, and is crystalline.[13] It breaks down with exposure to ultraviolet light.[16]

KEYWORDS: LSD, recreational, drug abuse, hallucinations, psychedelic, spiritual, mystical, ego dissolution

I.INTRODUCTION

LSD was first synthesized by Swiss chemist Albert Hofmann in 1938 from lysergic acid, a chemical derived from the hydrolysis of ergotamine, an alkaloid found in ergot, a fungus that infects grain.[16][23] LSD was the 25th of various lysergamides Hofmann synthesized from lysergic acid while trying to develop a new analeptic, hence the alternate name LSD-25. Hofmann discovered its effects in humans in 1943, after unintentionally ingesting an unknown amount, possibly absorbing it through his skin.[29][30][31] LSD was subject to exceptional interest within the field of psychiatry in the 1950s and early 1960s, with Sandoz distributing LSD to researchers under the trademark name Delysid in an attempt to find a marketable use for it.[30]

LSD-assisted psychotherapy was used in the 1950s and early 1960s by psychiatrists such as Humphry Osmond, who pioneered the application of LSD to the treatment of alcoholism, with promising results.[30][32][33][34] Osmond coined the term "psychedelic" (lit. mind manifesting) as a term for LSD and related hallucinogens, superseding the previously held "psychotomimetic" model in which LSD was believed to mimic schizophrenia. In contrast to schizophrenia, LSD induces transcendental experiences with lasting psychological benefit.[12][30] During this time, the Central Intelligence Agency (CIA) began using LSD in the research project Project MKUltra, which used psychoactive substances to aid interrogation. The CIA administered LSD to unwitting test subjects in order to observe how they would react, the most well-known example

of this being Operation Midnight Climax.[30] LSD was one of several psychoactive substances evaluated by the U.S. Army Chemical Corps as possible non-lethal incapacitants in the Edgewood Arsenal human experiments.[30]

In the 1960s, LSD and other psychedelics were adopted by, and became synonymous with, the counterculture movement due to their perceived ability to expand consciousness. This resulted in LSD being viewed as a cultural threat to American values and the Vietnam war effort, and it was designated as a Schedule I (illegal for medical as well as recreational use) substance in 1968.[35] It was listed as a Schedule 1 controlled substance by the United Nations in 1971 and currently has no approved medical uses.[16] As of 2016, about 10% of people in the United States have used LSD at some point in their lives, while 0.7% have used it in the last year.[36] It was most popular in the 1960s to 1980s.[16] The use of LSD among US adults increased 56.4% from 2015 to 2016.[37]

LSD is commonly used as a recreational drug. LSD can catalyze intense spiritual experiences and is thus considered an entheogen. Some users have reported out of body experiences. In 1966, Timothy Leary established the League for Spiritual Discovery with LSD as its sacrament.[39][40] Stanislav Grof has written that religious and mystical experiences observed during LSD sessions appear to be phenomenologically indistinguishable from similar descriptions in the sacred scriptures of the great religions of the world and the texts of ancient civilizations.[41] LSD currently has no approved uses in medicine.[42][43] A meta analysis concluded that a single dose was effective at reducing alcohol consumption in alcoholism.[34] LSD has also been studied in depression, anxiety,[44][45] and drug dependence, with positive preliminary results.[46][47] LSD is exceptionally potent, with as little as 20 µg capable of producing a noticeable effect. LSD can cause pupil dilation, reduced appetite, profuse sweating and wakefulness. Other physical reactions to LSD are highly variable and nonspecific, some of which may be secondary to the psychological effects of LSD. Among the reported symptoms are elevated body temperature, blood sugar, and heart rate, alongside goose bumps, jaw clenching, mouth dryness, and hyperreflexia. In negative experiences, numbness, weakness, nausea, and tremors have also been exhibited.[16] The most common immediate psychological effects of LSD are visual hallucinations and illusions (colloquially known as "trips"), which vary depending on how much is used and how the dosage interacts with the brain. Trips usually start within 20–30 minutes of taking LSD orally (less if snorted or taken intravenously), peak three to four hours after ingestion, and can last up to 20 hours in high doses. Users may also experience an "afterglow" of improved mood or perceived mental state for days or even weeks after ingestion in some experiences.[50] Good trips are reportedly deeply stimulating and pleasurable, and typically involve intense joy or euphoria, a greater appreciation for life, reduced anxiety, a sense of spiritual enlightenment, and a sense of belonging or interconnectedness with the universe.[51][52] Negative experiences, colloquially known as "bad trips," evoke an array of dark emotions, such as irrational fear, anxiety, panic, paranoia, dread, distrustfulness, hopelessness, and even suicidal ideation.[53] While it is impossible to predict when a bad trip will occur, one's mood, surroundings, sleep, hydration, social setting, and other factors can be controlled (colloquially referred to as "set and setting") to minimize the risk of a bad trip.[54][55] LSD causes an animated sensory experience of senses, emotions, memories, time, and awareness for 6 to 20 hours, depending on dosage and tolerance.[17] Generally beginning within 30 to 90 minutes after ingestion, the user may experience anything from subtle changes in perception to overwhelming cognitive shifts. Changes in auditory and visual perception are also typical.[56][57]

Some sensory effects may include an experience of radiant or more vibrant colors, objects and surfaces appearing to ripple, "breathe," or otherwise move, spinning fractals superimposed on one's vision, colored patterns behind closed eyelids, an altered sense of time, geometric patterns emerging on walls and other textured objects, and morphing objects.[56] Some users also report a strong metallic taste for the duration of the effects.[58] Food's texture or taste may be different, and users may also have an aversion to foods that they would normally enjoy. Similar effects have also been found in rats.[59]

Some report that the inanimate world appears to animate in an inexplicable way; for instance, objects that are static in three dimensions can seem to be moving relative to one or more additional spatial dimensions.[60] Many of the basic visual effects resemble the phosphenes seen after applying pressure to the eye and have also been studied as form constants. Sometimes these effects and patterns can be changed when concentrated on, or can change based on thoughts, emotions or music.[61] The auditory effects of LSD may include echo-like distortions of sounds, changes in ability to discern concurrent auditory and visual stimuli, and a general intensification of the experience of music. Higher doses often cause intense and fundamental distortions of sensory perception such as synesthesia, the experience of additional spatial or temporal dimensions, and temporary dissociation.

II.DISCUSSION

Out of the 20 drugs ranked in order of individual and societal harm by David Nutt, LSD was third to last, or approximately one-tenth as harmful as alcohol. The most significant adverse effect of LSD was impairment of mental functioning while intoxicated.[63]

Mental disorders

LSD may trigger panic attacks or feelings of extreme anxiety, known colloquially as a "bad trip". Although population studies have not found an increased incidence of mental illness in psychedelic drug users overall, with psychedelic users actually having lower rates of depression and substance abuse than the control group,[64][65] there is evidence that people with severe mental illnesses like schizophrenia have a higher likelihood of experiencing adverse effects from taking LSD.[66]

Suggestibility

While publicly available documents indicate that the CIA and Department of Defense have discontinued research into the use of LSD as a means of mind control,[67] research from the 1960s suggests that both mentally ill and healthy people are more suggestible while under its influence.[68][69][70]

Flashbacks

"Flashbacks" are a reported psychological phenomenon in which an individual experiences an episode of some of LSD's subjective effects after the drug has worn off, persisting for days or months after hallucinogen use.[71][72] Individuals with hallucinogen persisting perception disorder experience intermittent or chronic flashbacks that cause distress or impairment in life and work.[24]

The etiology of the "flashback" phenomenon appears to be varied. Some researchers such as Krebs and Johansen (2015[73]) attribute at least some of the cases to be related to somatic symptom disorder, when people fixate on normal somatic experiences and perceptions that they weren't aware of before consuming the drug. Other researchers relate it to an associative reaction to a contextual cue akin to what people that have faced trauma or strongly emotional experiences face when receiving a triggering stimulus (Holland and Passie 2011[74]). There is no consensus on what are the risk factors but some researchers theorize that pre-existing psychopathologies may be a significant contributor (Abraham and Duffy 1996[75])

The prevalence of HPPD is difficult to estimate but appears to be very rare, with estimates ranging from 1 in 20 users for the transitory and less serious type 1 HPPD, to 1 in 50,000 users for the more concerning type 2 HPPD.[24]

Contrary to rumors circulating the internet that LSD is stored in the spinal cord or other parts of your body long-term,[76] the pharmacological evidence shows LSD has a short half-life of 175 minutes, undergoing enzymatic metabolism into more polar and therefore water-soluble compounds such as 2-oxo-3-hydroxy-LSD that are eliminated through the urine. No evidence of long term storage of LSD in the body exists.[7]

Cancer and pregnancy

The mutagenic potential of LSD is unclear. Overall, the evidence seems to point to limited or no effect at commonly used doses.[77] Studies showed no evidence of teratogenic or mutagenic effects.[7][78]

Addiction and tolerance

LSD exhibits significant tachyphylaxis with tolerance manifesting 24 hours after a one-time administration, however tachyphylaxis at intervals shorter than 24 hours is largely unknown.[79] Tolerance to LSD builds up with consistent use[80] and cross-tolerance has been demonstrated between LSD, mescaline,[81] psilocybin,[82] and to some degree DMT.[a] Researchers believe that tolerance returns to baseline after two weeks of not using psychedelics.[85]

The NIH states that LSD is addictive,[23] while most other sources state it is not.[20][86] A 2009 textbook states that it "rarely produce[s] compulsive use."[5] A 2006 review states it is readily abused, but does not result in addiction.[20] There are no recorded successful attempts to train animals to self-administer LSD in laboratory settings.[21]

III.RESULTS

A report in 2008 stated that, though there was no "comprehensive review since the 1950s" and "almost no legal clinical research" since the 1970s, there had been "no documented human deaths from an LSD overdose". Eight individuals who accidentally consumed very high amounts by mistaking LSD for cocaine developed comatose states, hyperthermia, vomiting, gastric bleeding, and respiratory problems—all survived, however, with hospital treatment and without residual effects.[7] According to more recent reports, several behavioral-related fatalities and suicides have occurred due to LSD.[87][88] Reassurance in a calm, safe environment is beneficial. Agitation can be safely addressed with benzodiazepines such as lorazepam or diazepam. Neuroleptics such as haloperidol are not recommended because they may have adverse effects. LSD is rapidly absorbed, so activated charcoal and emptying of the stomach is of little benefit, unless done within 30–60 minutes of ingesting an overdose of LSD. Sedation or physical restraint is rarely required, and excessive restraint may cause complications such as hyperthermia (over-heating) or rhabdomyolysis.[89]

Massive doses "should be treated with supportive care, including respiratory support and endotracheal intubation if needed. Hypertension [high blood pressure], tachycardia [rapid heart-beat], and hyperthermia should be treated symptomatically. Hypotension [low blood pressure] should be treated initially with fluids and subsequently with pressors if required." "Intravenous administration of anticoagulants, vasodilators, and sympatholytics may be useful" when treating ergotism.[89]

Most serotonergic psychedelics are not significantly dopaminergic, and LSD is therefore atypical in this regard. The agonism of the D_2 receptor by LSD may contribute to its psychoactive effects in humans.[28][90]

LSD binds to most serotonin receptor subtypes except for the 5-HT_3 and 5-HT_4 receptors. However, most of these receptors are affected at too low affinity to be sufficiently activated by the brain concentration of approximately 10–20 nM.[86] In humans, recreational doses of LSD can affect 5-HT_{1A} (K_i=1.1nM), 5-HT_{2A} (K_i=2.9nM), 5-HT_{2B} (K_i=4.9nM), 5-HT_{2C} (K_i=23nM), 5-HT_{5A} (K_i=9nM [in cloned rat tissues]), and 5-HT_6 receptors (K_i=2.3nM).[91][92] Although not present in humans, 5-HT_{5B} receptors found in rodents also have a high affinity for LSD.[93] The psychedelic effects of LSD are attributed to cross-activation of 5-HT_{2A} receptor heteromers.[94] Many but not all 5-HT_{2A} agonists are psychedelics and 5-HT_{2A} antagonists block the psychedelic activity of LSD. LSD exhibits functional selectivity at the 5-HT_{2A} and 5HT_{2C} receptors in that it activates the signal transduction enzyme phospholipase A2 instead of activating the enzyme phospholipase C as the endogenous ligand serotonin does.[95]

Exactly how LSD produces its effects is unknown, but it is thought that it works by increasing glutamate release in the cerebral cortex[86] and therefore excitation in this area, specifically in layers IV and V.[96] LSD, like many other drugs of recreational use, has been shown to activate DARPP-32-related pathways.[97] The drug enhances dopamine D_2 receptor protomer recognition and signaling of D_2–5-HT_{2A} receptor complexes,[27] which may contribute to its psychotropic effects.[27] LSD has been shown to have low affinity for H1 receptors, displaying antihistamine effects.[98][99][100]

LSD is a biased agonist that induces a conformation in serotonin receptors that preferentially recruits β-arrestin over activating G proteins.[101][102] LSD also has an exceptionally long residence time when bound to serotonin receptors lasting hours, consistent with the long lasting effects of LSD despite its relatively rapid clearance.[101][102] A crystal structure of 5-HT_{2B} bound to LSD reveals an extracellular loop that forms a lid over the diethylamide end of the binding cavity which explains the slow rate of LSD unbinding from serotonin receptors.[103][104][105] The related lysergamide lysergic acid amide (LSA) that lacks the diethylamide moiety is far less hallucinogenic in comparison.[105]

The effects of LSD normally last between 6 and 12 hours depending on dosage, tolerance, and age.[107] The Sandoz prospectus for "Delysid" warned: "intermittent disturbances of affect may occasionally persist for several days."[108] Aghajanian and Bing (1964) found LSD had an elimination half-life of only 175 minutes (about 3 hours).[91] However, using more accurate techniques, Papac and Foltz (1990) reported that 1 µg/kg oral LSD given to a single male volunteer had an apparent plasma half-life of 5.1 hours, with a peak plasma concentration of 5 ng/mL at 3 hours post-dose.[109]

The pharmacokinetics of LSD were not properly determined until 2015, which is not surprising for a drug with the kind of low-µg potency that LSD possesses.[6][9] In a sample of 16 healthy subjects, a single mid-range 200 µg oral dose of LSD was found to produce mean maximal concentrations of 4.5 ng/mL at a median of 1.5 hours (range 0.5–4 hours) post-

administration.[6][9] Concentrations of LSD decreased following first-order kinetics with a half-life of 3.6±0.9 hours and a terminal half-life of 8.9±5.9 hours.[6][9]

The effects of the dose of LSD given lasted for up to 12 hours and were closely correlated with the concentrations of LSD present in circulation over time, with no acute tolerance observed.[6][9] Only 1% of the drug was eliminated in urine unchanged, whereas 13% was eliminated as the major metabolite 2-oxo-3-hydroxy-LSD (O-H-LSD) within 24 hours.[6][9] O-H-LSD is formed by cytochrome P450 enzymes, although the specific enzymes involved are unknown, and it does not appear to be known whether O-H-LSD is pharmacologically active or not.[6][9] The oral bioavailability of LSD was crudely estimated as approximately 71% using previous data on intravenous administration of LSD.[6][9] The sample was equally divided between male and female subjects and there were no significant sex differences observed in the pharmacokinetics of LSD.[6][9]

Implications

LSD is a chiral compound with two stereocenters at the carbon atoms C-5 and C-8, so that theoretically four different optical isomers of LSD could exist. LSD, also called (+)-D-LSD, has the absolute configuration (5R,8R). The C-5 isomers of lysergamides do not exist in nature and are not formed during the synthesis from d-lysergic acid. Retrosynthetically, the C-5 stereocenter could be analysed as having the same configuration of the alpha carbon of the naturally occurring amino acid L-tryptophan, the precursor to all biosynthetic ergoline compounds.

However, LSD and iso-LSD, the two C-8 isomers, rapidly interconvert in the presence of bases, as the alpha proton is acidic and can be deprotonated and reprotonated. Non-psychoactive iso-LSD which has formed during the synthesis can be separated by chromatography and can be isomerized to LSD.

Pure salts of LSD are triboluminescent, emitting small flashes of white light when shaken in the dark.[107] LSD is strongly fluorescent and will glow bluish-white under UV light. LSD is an ergoline derivative. It is commonly synthesized by reacting diethylamine with an activated form of lysergic acid. Activating reagents include phosphoryl chloride[110] and peptide coupling reagents.[100] Lysergic acid is made by alkaline hydrolysis of lysergamides like ergotamine, a substance usually derived from the ergot fungus on agar plate; or, theoretically possible, but impractical and uncommon, from ergine (lysergic acid amide, LSA) extracted from morning glory seeds.[111] Lysergic acid can also be produced synthetically, although these processes are not used in clandestine manufacture due to their low yields and high complexity.[112][113] "LSD," writes the chemist Alexander Shulgin, "is an unusually fragile molecule ... As a salt, in water, cold, and free from air and light exposure, it is stable indefinitely."[107]

LSD has two labile protons at the tertiary stereogenic C5 and C8 positions, rendering these centers prone to epimerisation. The C8 proton is more labile due to the electron-withdrawing carboxamide attachment, but removal of the chiral proton at the C5 position (which was once also an alpha proton of the parent molecule tryptophan) is assisted by the inductively withdrawing nitrogen and pi electron delocalisation with the indole ring.[citation needed]

LSD also has enamine-type reactivity because of the electron-donating effects of the indole ring. Because of this, chlorine destroys LSD molecules on contact; even though chlorinated tap water contains only a slight amount of chlorine, the small quantity of compound typical to an LSD solution will likely be eliminated when dissolved in tap water.[107] The double bond between the 8-position and the aromatic ring, being conjugated with the indole ring, is susceptible to nucleophilic attacks by water or alcohol, especially in the presence of UV or other kinds of light. LSD often converts to "lumi-LSD," which is inactive in human beings.[107]

A controlled study was undertaken to determine the stability of LSD in pooled urine samples.[121] The concentrations of LSD in urine samples were followed over time at various temperatures, in different types of storage containers, at various exposures to different wavelengths of light, and at varying pH values. These studies demonstrated no significant loss in LSD concentration at 25 °C for up to four weeks. After four weeks of incubation, a 30% loss in LSD concentration at 37 °C and up to a 40% at 45 °C were observed. Urine fortified with LSD and stored in amber glass or nontransparent polyethylene containers showed no change in concentration under any light conditions. Stability of LSD in transparent containers under light was dependent on the distance between the light source and the samples, the wavelength of light, exposure time, and the intensity of light. After prolonged exposure to heat in alkaline pH conditions, 10 to 15% of the parent LSD epimerized to iso-LSD. Under acidic conditions, less than 5% of the LSD was converted to iso-LSD. It was also demonstrated that trace amounts of metal ions in buffer or urine could catalyze the decomposition of LSD and that this process can be avoided by the addition of EDTA. LSD may be quantified in urine as part of a drug abuse testing program, in plasma or serum to

confirm a diagnosis of poisoning in hospitalized victims or in whole blood to assist in a forensic investigation of a traffic or other criminal violation or a case of sudden death. Both the parent drug and its major metabolite are unstable in biofluids when exposed to light, heat or alkaline conditions and therefore specimens are protected from light, stored at the lowest possible temperature and analyzed quickly to minimize losses.[122]

Maximum plasma concentrations were found to be 1.4 and 1.5 hours after oral administration of 100µg and 200µg respectively with a plasma half-life of 2.6 hours (ranging from 2.2–3.4 hours among 40 human test subjects).[123]

LSD can be detected using an Ehrlich's reagent and a Hofmann's reagent.

LSD was first synthesized on November 16, 1938[125] by Swiss chemist Albert Hofmann at the Sandoz Laboratories in Basel, Switzerland as part of a large research program searching for medically useful ergot alkaloid derivatives. The abbreviation "LSD" is from the German "Lysergsäurediethylamid".[126]

LSD's psychedelic properties were discovered 5 years later when Hofmann himself accidentally ingested an unknown quantity of the chemical.[127] The first intentional ingestion of LSD occurred on April 19, 1943,[128] when Hofmann ingested 250 µg of LSD. He said this would be a threshold dose based on the dosages of other ergot alkaloids. Hofmann found the effects to be much stronger than he anticipated.[129] Sandoz Laboratories introduced LSD as a psychiatric drug in 1947 and marketed LSD as a psychiatric panacea, hailing it "as a cure for everything from schizophrenia to criminal behavior, 'sexual perversions', and alcoholism."[130] Sandoz would send the drug for free to researchers investigating its effects.[29]

Beginning in the 1950s, the US Central Intelligence Agency (CIA) began a research program code named Project MKUltra. The CIA introduced LSD to the United States, purchasing the entire world's supply for $240,000 and propagating the LSD through CIA front organizations to American hospitals, clinics, prisons and research centers.[131] Experiments included administering LSD to CIA employees, military personnel, doctors, other government agents, prostitutes, mentally ill patients, and members of the general public in order to study their reactions, usually without the subjects' knowledge. The project was revealed in the US congressional Rockefeller Commission report in 1975.

In 1963, the Sandoz patents on LSD expired[119] and the Czech company Spofa began to produce the substance.[29] Sandoz stopped the production and distribution in 1965.[29] Several figures, including Aldous Huxley, Timothy Leary, and Al Hubbard, had begun to advocate the consumption of LSD. LSD became central to the counterculture of the 1960s.[132] In the early 1960s the use of LSD and other hallucinogens was advocated by new proponents of consciousness expansion such as Leary, Huxley, Alan Watts and Arthur Koestler,[133][134] and according to L. R. Veysey they profoundly influenced the thinking of the new generation of youth.[135]

On October 24, 1968, possession of LSD was made illegal in the United States.[136] The last FDA approved study of LSD in patients ended in 1980, while a study in healthy volunteers was made in the late 1980s. Legally approved and regulated psychiatric use of LSD continued in Switzerland until 1993.[137]

In November 2014, Oregon became the first US state to decriminalize possession of small amounts of LSD after voters approved Ballot Measure 110.[138]

IV.CONCLUSIONS

By the mid-1960s, the youth countercultures in California, particularly in San Francisco, had adopted the use of hallucinogenic drugs, with the first major underground LSD factory established by Owsley Stanley.[139] From 1964, the Merry Pranksters, a loose group that developed around novelist Ken Kesey, sponsored the Acid Tests, a series of events primarily staged in or near San Francisco, involving the taking of LSD (supplied by Stanley), accompanied by light shows, film projection and discordant, improvised music known as the psychedelic symphony.[140][141] The Pranksters helped popularize LSD use, through their road trips across America in a psychedelically decorated converted school bus, which involved distributing the drug and meeting with major figures of the beat movement, and through publications about their activities such as Tom Wolfe's The Electric Kool-Aid Acid Test (1968).[142]

In San Francisco's Haight-Ashbury neighborhood, brothers Ron and Jay Thelin opened the Psychedelic Shop in January 1966.[143] The Thelins opened the store to promote safe use of LSD, which was then still legal in California. The Psychedelic Shop helped to further popularize LSD in the Haight and to make the neighborhood the unofficial capital of the hippie counterculture in the United States. Ron Thelin was also involved in organizing the Love Pageant rally, a protest held in Golden Gate park to protest California's newly adopted ban on LSD in October 1966. At the rally, hundreds of

attendees took acid in unison. Although the Psychedelic Shop closed after barely a year-and-a-half in business, its role in popularizing LSD was considerable.[144]

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