



A Prospective Review Article on Lysergic Acid Diethylamide

S. A. Surale Patil¹, P.V. Pakale² and Daksh Bhora³

¹Department of Pharmacology, Krishna Institute of Medical Sciences, Krishna Institute of Medical Sciences, Krishna Vishwa Vidyapeeth "Deemed To Be University", Karad.

²Department of Medicine, Krishna Institute of Medical Sciences Krishna Institute of Medical Sciences, Krishna Vishwa Vidyapeeth "Deemed To Be University", Karad.

ABSTRACT

Research has shown that LSD is a habit-forming hallucinogenic chemical that has intriguing possibilities for its therapeutic use. Studies have further shown that there is a suggestion that administering large amounts of psychedelic substances, such as 100–200 µg of LSD or 35 mg of psilocybin, might be beneficial in treating mood and anxiety problems. Additionally, various other studies have shown that there are several anecdotal accounts suggesting that regular usage of modest doses of LSD (e.g., 10–15 µg) might enhance mood, cognitive function, and overall well-being. Hence, in our review, we have discussed history, pharmacokinetics, adverse effects, mechanisms, contraindications, use, and preventive measures for LSD.

Key words: History, Pharmacokinetics, Adverse Effects, Mechanisms, Contraindications, Use, Preventive Measures, LSD.

Received 28.09.2023

Revised 20.10.2023

Accepted 30.11. 2023

INTRODUCTION

Numberous studies have shown that , LSD is also known as Lysergsäure-Diethylamid, is a potent psychedelic drug that is commonly referred to as acid or lucy due to its slang names.[1] Addition to this, studies have also shown that , the symptoms generally involve increased cognitive abilities, emotional responses, and sensitivity.[2] Furthermore, various studies have shown that , in larger doses, LSD tends to induce auditory and visual hallucinations.[3,4] Studies also concluded that , common symptoms may include dilated pupils, elevated blood pressure, and an increased core body temperature.[5] In addition, studies also revealed that ,the effects usually begin to fade after about 30 minutes and can last for up to 20 hours (on average, experiences last 8–12 hours).[5] Additionally, studies revealed that , LSD can lead to mystical experiences and a breakdown of the ego.[6] Henceforth, studies says that ,many individuals choose to use this drug for either recreational or religious reasons.[5] Furthermore, studies have concluded that, LSD was widely recognized as the most scientifically and culturally significant psychedelic, solidifying its position as the archetype and one of the "classical" psychedelics.[1] Studies also proved that, LSD is a chemical that is commonly produced as a powder or crystalline substance.[1] Additionally, studies revealed that, to create a solution, the solid LSD is dissolved in a liquid solvent such as ethanol or distilled water.[2]Various other studies also concluded that, the liquid serves as a medium for the LSD, enabling precise dosing and application to the tablets.[2] Usually, researches have concluded that, LSD is consumed by placing it under the tongue or ingesting it.[2] Therefore, in our analysis, we aimed to assess and deliberate on LSD.

HISTORY

According to numerous studies, "Albert Hoffman was credited for the initial synthesis of LSD in 1938. He accidentally discovered the drug while experimenting with ergots to develop circulatory and respiratory stimulants. The author successfully created a model for psychosis and studied the short-term psychotic episodes that were induced in animals because they had a big effect on them. He displayed remarkable recall and gained a deeper understanding during his psychotic episode. He acknowledged both of these things".[7,8,9]

MECHANISM

Studies have concluded that, LSD produces its effects by stimulating serotonin receptors, namely the 5HT_{2A} receptors (sometimes referred to as the 5hydroxytryptamine 2A receptor, 5-HT_{2AR}), and causing changes in the 5HT_{2C} and 5HT_{1A} receptors. Nevertheless, the intricate connections between the activation of receptors, cognitive decline, and the production of hallucinations are still not well understood.[10] One study found that LSD activates 5-HT_{2AR}, which disrupts the inhibitory processes of the hippocampus and prefrontal cortex. Evidence demonstrates that it reduces activity in certain brain areas, such as the left superior frontal gyrus, postcentral gyrus, cerebellum, right middle temporal gyrus, superior/middle/inferior frontal gyrus, and anterior cingulate cortex. Induced visual images have been associated with the activation of the right hemisphere, alterations in thalamic functioning, and heightened activity in the paralimbic regions and frontal cortex.[11,12,13]

ADVERSE EFFECT[10]

1. Hallucination
2. Sight
3. Sensation
4. Increased awareness owing to mind expansion
5. Euphoria
6. Anxiety
7. Panic
8. Fear
9. Depression
10. Despair & disappointment
11. Flashback due to stress or fatigue or using other drugs

CONTRAINDICATION [10]

1. CVS patients
2. Pregnant lady
3. Epilepsy patient
4. Paranoid personality
5. Overt Psychosis
6. Organic-toxic cerebral disorder

USE

Studies have confirmed that, after LSD was synthesized in 1938, human testing began.[10] In another study, authors gave 49 doses of LSD (20–130 micrograms) to 22 schizophrenic and healthy volunteers. They found that, the mental state of the individuals was mostly euphoric.[14] The authors also explored that the use of LSD as a research tool in psychiatry. The effects were similar to those of mescaline, which caused hallucinations.[14] Furthermore, studies revealed that the low dosages of LSD may aid psychotherapy by facilitating the release of repressed information into awareness.[14] In another study, authors, started offering LSD to doctors and research organizations worldwide for free as an experimental drug with the commercial name of "Delysid".[15] The pharmaceutical prospectus showed its potential in analytical psychotherapy and psychosis research.[15] Furthermore studies have shown that, the researchers first used this later indication of LSD as a foundation for studying mental disorders like schizophrenia. The notion of "model psychosis" suggests that LSD might provide valuable insights into the nature of psychosis by imitating the psychotic state, which is referred to as the "psychotomimetic effect" of LSD. Sandoz suggested that psychiatrists undergo LSD administration to get firsthand insight into their patients' experiences. It was anticipated that this encounter would enable them to get a more profound comprehension of their patient's psychological condition. Furthermore, there was a theoretical proposition that the administration of LSD to those without psychosis would induce a condition resembling schizophrenia. The intention was to provide a framework for investigating this ailment, with the prospect of uncovering novel and enhanced therapies for schizophrenia and its associated psychotic diseases. The inception of the model psychosis hypothesis predates the use of LSD. Before the production of LSD, Beringer proposed that mescaline may be used to enhance psychiatrists' comprehension of the psychotic encounters of their patients.[16]

PHARMACOKINETIC PROFILE [17,18,19]

Healthy Subjects	Oral Dose	C _{max}	t _{max}	t _{1/2}
12 males, 11 females	5 µg	0.151 ng / mL	66 min.	180 min.
12 males, 11 females	10 µg	0.279 ng / mL	60 min.	198 min.
12 males, 11 females	20 µg	0.5 ng / mL	66 min.	216 min.
12 males, 12 females	100 µg	1.3 ng / mL	84 min.	156 min.
13 males	160 µg	1.8 - 8.8 ng / mL	40-130 min.	-
8 males, 8 females	200 µg	4.3 ng / mL	90 min.	216 min.

C_{max}: estimated maximum plasma concentration, t_{max}: estimated time to reach C_{max},
t_{1/2} estimated half-life plasma elimination

PREVENTIVE MEASURE

Research has shown that when a healthcare professional encounters a patient using LSD, it is recommended to refer them to a drug rehab facility. LSD is devoid of any medical benefits and is linked to numerous adverse psychiatric effects. Typically, individuals who use LSD also tend to use other illegal substances, making it advisable to undergo a urine screen. Regrettably, LSD addiction can be quite powerful, and even with various therapies, the success rates of treatments tend to be low. Regardless, it is important for primary care providers to strongly advise the patient to stop using LSD and/or sharing needles and to seek psychological assistance and treatment.[10] Studies have concluded that prevention programs encompass various aspects of the ecological environment, including schools, communities, parents, and skills. Internet-based prevention programs have become an essential part of modern life and are highly effective. With the affordability of Internet connectivity, a larger number of people will have the opportunity to access and assess online prevention programs. By prioritizing the individual's growth in areas such as stress management, emotion control, and emotional expression, we can effectively decrease the consumption of illegal substances like LSD. The techniques employed to enhance individuals' pain tolerance and emotional acuity in this context are of utmost importance.[20]

PHARAMACODYNAMIC

Studies have concluded that LSD was put on the most restrictive drug control schedule in 1966, but there has been no human research done on the effects that LSD has on the human brain. This is because there are no human studies. Before 1966, a great deal of in vitro and in vivo research was carried out, but the techniques used were far more primitive.[22]

1. BRAIN TISSUE

Synder et al.,(1966) conducted original research, where researchers discovered that squirrel monkeys (*Saimiri sciurus*; dosages varied from 0.5–2 mg/kg when given intravenously; n = 4) studied the regional distribution of LSD in the brains of these animals. After receiving an injection of LSD, these animals were subjected to flames thirty minutes later. They found that LSD was unevenly distributed throughout the major areas of the brain. The concentrations found in the pituitary and pineal glands were seven to eight times higher than those found in the cortex. These glands had the highest levels of the chemical overall in the body. The structures of the limbic system, which include the hippocampus, amygdala, fornix, and septal area, contained around two to three times as much LSD as the structures of the cortical system. Thus, they come to the conclusion that the visual and auditory areas, the hypothalamus, the extrapyramidal system, and the thalamus all contained two to five times the concentration of LSD compared to the cortical regions. The brain stem, much like the cortex, contained concentrations of LSD. The LSD was equally distributed across the white matter and the gray matter.[23]

Arnold et al., (1985) conducted original research, where researchers discovered that [14C]-LSD is dispersed in the brain. Extremely high doses of the drug (8.12 mg/kg intraperitoneally) were administered to the mice that were studied. They found that cellular structures had far greater levels of LSD than any other form of brain tissue. In addition to this ratio of decreasing concentration, the hippocampus was found to have the highest concentration, followed by the basal ganglia, periventricular gray matter, and the frontoparietal cortex.[24]

2. EFFECT ON CEREBRAL CIRCULATION

Sokoloff et al., (1957) conducted original research to evaluate and assess the circulation and metabolism of the brain in humans. At the peak of LSD's effects (n = 13, 120 g i.v.), there were no significant changes in the brain's overall blood flow (measured with nitrous oxide), vascular resistance, oxygen or glucose use, or the amount of glucose used. They found that it is possible that LSD causes changes in brain

circulation or metabolism, but only in areas that make up such a small part of the whole brain that the effects can't be seen when measuring the brain as a whole. Thus, they come to the conclusion that it may be that many of those pieces are functionally inversely or reciprocally connected in a heterogeneous organ like the brain, and so changes in the net metabolic rate of the brain stay unaltered" (p. 475).[25]

3. NEUROPHYSIOLOGICAL ACTIONS

LSD's major neurological effects include dose-dependent hyperreflexia and mild ataxia, according to Forrer and Goldner [26] and Hertle et al. [27]. Studies have been showing since ages that EEG shows the effects of LSD are quite mild and cannot be differentiated from background noise. The observation that there has been an increase in the mean frequency is, by a significant margin, the one that is made the most often. [28,29,30] Other investigations have shown that after taking LSD, there is a gradual dissynchronization that happens as a result of a quantitative reduction in the slow component.[31,32] This has been reported by studies to occur as a consequence of a change in the frequency of the slow component. It was found that this was the case because the size of the component that moved slowly was decreased. This dissynchronization is brought on by the fact that men experience a decline in the slow component, which in turn brings about a decrease in the slow component. As reported by Goldstein et al. [33] there was a 33% reduction in the amount of EEG variability after taking LSD (0.3–1.0 g/kg p.o.). In their research, Goldstein et al. reached this conclusion. When the subjects were given LSD, the typical pattern of lateralization was inverted for the majority of the people. Goldstein and Stoltzfus [34] studied human EEG amplitude levels in the right and left occipital lobes and found this to be the case. The fact that the subjects were provided with the psychoactive drug contributed to the formation of this conclusion. They arrived at this conclusion as a result of the results of their inquiry.[22]

4. NEUROMETABOLIC EFFECTS

Studies have shown that LSD has effects on the neurometabolic system, but no studies have yet come up with results that can be considered conclusive. On the other hand, research on the neurometabolic effects of similar hallucinogens like psilocybin, [35,36] dimethyltryptamine (DMT) [37], and mescaline [38] has been published. The hypotheses that were developed to explain the neurofunctional abnormalities that were brought on by the effects of hallucinogens are less plausible as a result of the many discrepancies that were discovered in the findings of the numerous studies [39]. The major hallucinogens seem to stimulate the right hemisphere of the brain, change the function of the thalamus, and increase metabolism in paralimbic structures as well as the frontal cortex. It is not easy to distinguish which alterations are primary substance-induced and which are due to secondary (compensatory) psychophysical processes induced by general psychosocial stress during hallucinogen intoxication under experimental conditions. This is due to the fact that the majority of these metabolic changes are also found in people when they are under psychological stress. [40,41] Because of this, it is difficult to determine which alterations are caused by the primary substance and which are caused by secondary (compensatory) psychophysical processes. This is because it is difficult to differentiate which changes are caused by the main substance-induced processes and which are attributable to secondary (compensatory) psychophysical processes induced by general stress. This is because it is difficult to establish which alterations are caused by primary substance-induced alterations. When researchers looked at the metabolism of the whole brain, they came to two different conclusions: some found an enhanced metabolism [42,43], while others found no change.[44,45] Furthermore, studies revealed that since the bulk of these metabolic changes are also found in humans when they are under psychological stress.[40,46] circumstances due to this. Some researchers found an increased metabolism when they looked at the metabolism of the total brain [36,38], whereas other researchers found no change.[35,45]

CONCLUSION

This analysis presents current information on LSD. Additional research is necessary to explore the mechanism of action of LSD in the fields of psychiatry and neuroscience due to the existing uncertainty. Psychologists and psychiatrists can gain valuable understanding of the origins of certain mental health conditions through the examination of LSD's neurobiological mechanisms and pathophysiology. In recent times, psychiatrists have made significant contributions to the advancement of novel and highly efficient medications, aiming to enhance the quality of patient care. There have been advancements in identifying new medications to address mental health issues, but unfortunately, existing treatments still have limitations. Research has shown that LSD may have potential for treating depression, anxiety, and other addictions. However, there has been a decrease in interest regarding its therapeutic benefits. Major pharmaceutical companies may utilize LSD as a research tool and a source of inspiration for developing novel mental medications. Regarding this matter, there is still much to be discovered about the potential effects of LSD.

REFERENCES

1. Nichols, D. E. (2016). Psychedelics. *Pharmacological reviews*, 68(2), 264-355.
2. Hardaway, R., Schweitzer, J., & Suzuki, J. (2016). Hallucinogen use disorders. *Child and Adolescent Psychiatric Clinics*, 25(3), 489-496.
3. Leptourgos, P., Fortier-Davy, M., Carhart-Harris, R., Corlett, P. R., Dupuis, D., Halberstadt, A. L., ... & Jardri, R. (2020). Hallucinations under psychedelics and in the schizophrenia spectrum: an interdisciplinary and multiscale comparison. *Schizophrenia Bulletin*, 46(6), 1396-1408.
4. Holze, F., Vizeli, P., Ley, L., Müller, F., Dolder, P., Stocker, M., ... & Liechti, M. E. (2021). Acute dose-dependent effects of lysergic acid diethylamide in a double-blind placebo-controlled study in healthy subjects. *Neuropsychopharmacology*, 46(3), 537-544.
5. Nichols, D. E. (2018). Dark classics in chemical neuroscience: lysergic acid diethylamide (LSD). *ACS chemical neuroscience*, 9(10), 2331-2343.
6. Coney, L. D., Maier, L. J., Ferris, J. A., Winstock, A. R., & Barratt, M. J. (2017). Genie in a blotter: A comparative study of LSD and LSD analogues' effects and user profile. *Human Psychopharmacology: Clinical and Experimental*, 32(3), e2599.
7. Liechti, M. E., Dolder, P. C., & Schmid, Y. (2017). Alterations of consciousness and mystical-type experiences after acute LSD in humans. *Psychopharmacology*, 234, 1499-1510.
8. Preuss, C. V., Kalava, A., & King, K. C. (2019). Prescription of controlled substances: benefits and risks.
9. Preller, K. H., Razi, A., Zeidman, P., Stämpfli, P., Friston, K. J., & Vollenweider, F. X. (2019). Effective connectivity changes in LSD-induced altered states of consciousness in humans. *Proceedings of the National Academy of Sciences*, 116(7), 2743-2748.
10. Johnson, M. W., Hendricks, P. S., Barrett, F. S., & Griffiths, R. R. (2019). Classic psychedelics: An integrative review of epidemiology, therapeutics, mystical experience, and brain network function. *Pharmacology & therapeutics*, 197, 83-102.
11. Hwang, K. A. J., & Saadabadi, A. (2018). Lysergic acid diethylamide (LSD).
12. Kvam, T. M., Stewart, L. H., & Andreassen, O. A. (2018). Psychedelic drugs in the treatment of anxiety, depression and addiction. *Tidsskrift for Den norske legeforening*.
13. Jalal, B. (2018). The neuropharmacology of sleep paralysis hallucinations: serotonin 2A activation and a novel therapeutic drug. *Psychopharmacology*, 235, 3083-3091.
14. Cameron, L. P., & Olson, D. E. (2018). Dark classics in chemical neuroscience: N, N-Dimethyltryptamine (DMT). *ACS chemical neuroscience*, 9(10), 2344-2357.
15. Stoll, W. A. (1947). Lysergsäure-diethylamid, ein Phantastikum aus der Mutterkornggruppe. Royal Society of Medicine, Microfilm unit.
16. Hofmann, A., & Child, L. M. P. (2009). Reflections on Sacred Drugs, Mysticism, and Science, MAPS, Multidisciplinary Association for Psychedelic Studies.
17. Beringer, K. (1927). *Der Meskalinrausch, seine Geschichte und Erscheinungsweise*.
18. Aghajanian, G. K., & Bing, O. H. (1964). Persistence of lysergic acid diethylamide in the plasma of human subjects. *Clinical Pharmacology & Therapeutics*, 5(5), 611-614.
19. Dolder, P. C., Schmid, Y., Steuer, A. E., Kraemer, T., Rentsch, K. M., Hammann, F., & Liechti, M. E. (2017). Pharmacokinetics and pharmacodynamics of lysergic acid diethylamide in healthy subjects. *Clinical pharmacokinetics*, 56, 1219-1230.
20. Holze, F., Duthaler, U., Vizeli, P., Müller, F., Borgwardt, S., & Liechti, M. E. (2019). Pharmacokinetics and subjective effects of a novel oral LSD formulation in healthy subjects. *British journal of clinical pharmacology*, 85(7), 1474-1483.
21. Linehan, M. M., Schmidt, H., Dimeff, L. A., Craft, J. C., Kanter, J., & Comtois, K. A. (1999). Dialectical behavior therapy for patients with borderline personality disorder and drug-dependence. *American Journal on Addictions*, 8(4), 279-292.
22. Passie, T., Halpern, J. H., Stichtenoth, D. O., Emrich, H. M., & Hintzen, A. (2008). The pharmacology of lysergic acid diethylamide: a review. *CNS neuroscience & therapeutics*, 14(4), 295-314.
23. Snyder, S. H., & Reivich, M. (1966). Regional localization of lysergic acid diethylamide in monkey brain. *Nature*, 209(5028), 1093-1095.
24. Arnold, O. H., Hofmann, G., & Leupold-Lowenthal, H. (1958). Untersuchungen zum Schizophrenieproblem IV Mitteilung: Die Verteilung des C14-radioaktiven Lysergsäurediethylamid (C14-LSD-25) im tierischen Organismus. *Wien Z Nervenheilk*, 15, 15-27.
25. Sokoloff, L., Perlin, S., Kornetsky, C., & Kety, S. S. (1957). The effects of d-lysergic acid diethylamide on cerebral circulation and over-all metabolism. *Annals of the New York Academy of Sciences*, 66(3), 468-477.
26. Forrer, G. R., & Goldner, R. D. (1951). Experimental physiological studies with lysergic acid diethylamide (LSD-25). *AMA Archives of Neurology & Psychiatry*, 65(5), 581-588.
27. Hertle, F., Zipf, K. E., & Broghammer, H. (1961). Beobachtungen über Stoffwechselverhalten, Muskeltonus und einige Kreislaufgrößen beim Menschen unter LSD-25-Einwirkung. *Archiv für Psychiatrie und Nervenkrankheiten*, 202, 569-591.
28. Anderson, E. W., & Rawnsley, K. (1954). Clinical studies of lysergic acid diethylamide. *Monatsschrift für Psychiatrie und Neurologie*, 128(1-2), 38-55.
29. Bradley, P. B., Elkes, C., & Elkes, J. (1953). On some effects of lysergic acid diethylamide (LSD 25) in normal volunteers. *The Journal of physiology*, 121(2), 50P-51P.

30. Elkes, J., Elkes, C., & Bradley, P. B. (1954). The effect of some drugs on the electrical activity of the brain, and on behaviour. *Journal of Mental Science*, 100(418), 125-128.
31. TM, I. (1969). Quantitative EEG and behavior changes after LSD and Ditran. *Neurophysiological and behavioral aspects of psychotropic drugs*, 67-72.
32. PF, W. (1964). *Electroencephalographic Effects Of Lsd, And Some Psychiatric Implications*. *Journal of Neuropsychiatry*, 5, 516-524.
33. Goldstein, L., Murphree, H. B., Sugeran, A. A., Pfeiffer, C. C., & Jenney, E. H. (1963). Quantitative electroencephalographic analysis of naturally occurring (schizophrenic) and drug-induced psychotic states in human males. *Clinical Pharmacology & Therapeutics*, 4(1), 10-21.
34. Goldstein, L., & Stoltzfus, N. W. (1973). Psychoactive drug-induced changes of interhemispheric EEG amplitude relationships. *Agents and actions*, 3(2), 124-132.
35. Gouzoulis-Mayfrank, E., Schreckenberger, M., Sabri, O., Arning, C., Thelen, B., Spitzer, M., ... & Sass, H. (1999). Neurometabolic effects of psilocybin, 3, 4-methylenedioxyethylamphetamine (MDE) and d-methamphetamine in healthy volunteers: a double-blind, placebo-controlled PET study with [18F] FDG. *Neuropsychopharmacology*, 20(6), 565-581.
36. Vollenweider, F. X., Leenders, K. L., Scharfetter, C., Maguire, P., Stadelmann, O., & Angst, J. (1997). Positron emission tomography and fluorodeoxyglucose studies of metabolic hyperfrontality and psychopathology in the psilocybin model of psychosis. *Neuropsychopharmacology*, 16(5), 357-372.
37. Riba, J., Valle, M., Urbano, G., Yritia, M., Morte, A., & Barbanoj, M. J. (2003). Human pharmacology of ayahuasca: subjective and cardiovascular effects, monoamine metabolite excretion, and pharmacokinetics. *Journal of Pharmacology and Experimental Therapeutics*, 306(1), 73-83.
38. Hermle, L., Fünfgeld, M., Oepen, G., Botsch, H., Borchardt, D., Gouzoulis, E., ... & Spitzer, M. (1992). Mescaline-induced psychopathological, neuropsychological, and neurometabolic effects in normal subjects: experimental psychosis as a tool for psychiatric research. *Biological psychiatry*, 32(11), 976-991.
39. Vollenweider, F. X., & Geyer, M. A. (2001). A systems model of altered consciousness: integrating natural and drug-induced psychoses. *Brain research bulletin*, 56(5), 495-507.
40. Wang, J., Rao, H., Wetmore, G. S., Furlan, P. M., Korczykowski, M., Dinges, D. F., & Detre, J. A. (2005). Perfusion functional MRI reveals cerebral blood flow pattern under psychological stress. *Proceedings of the National Academy of Sciences*, 102(49), 17804-17809.
41. Sinha, R., Lacadie, C., Skudlarski, P., & Wexler, B. E. (2004). Neural circuits underlying emotional distress in humans. *Annals of the New York Academy of Sciences*, 1032(1), 254-257.
42. Vollenweider, F. X., Leenders, K. L., Scharfetter, C., Maguire, P., Stadelmann, O., & Angst, J. (1997). Positron emission tomography and fluorodeoxyglucose studies of metabolic hyperfrontality and psychopathology in the psilocybin model of psychosis. *Neuropsychopharmacology*, 16(5), 357-372.
43. Hermle, L., Fünfgeld, M., Oepen, G., Botsch, H., Borchardt, D., Gouzoulis, E., ... & Spitzer, M. (1992). Mescaline-induced psychopathological, neuropsychological, and neurometabolic effects in normal subjects: experimental psychosis as a tool for psychiatric research. *Biological psychiatry*, 32(11), 976-991.
44. Gouzoulis-Mayfrank, E., Schreckenberger, M., Sabri, O., Arning, C., Thelen, B., Spitzer, M., ... & Sass, H. (1999). Neurometabolic effects of psilocybin, 3, 4-methylenedioxyethylamphetamine (MDE) and d-methamphetamine in healthy volunteers: a double-blind, placebo-controlled PET study with [18F] FDG. *Neuropsychopharmacology*, 20(6), 565-581.
45. Riba, J., Romero, S., Grasa, E., Mena, E., Carrió, I., & Barbanoj, M. J. (2006). Increased frontal and paralimbic activation following ayahuasca, the pan-Amazonian inebriant. *Psychopharmacology*, 186, 93-98.
46. Sinha, R., Lacadie, C., Skudlarski, P., & Wexler, B. E. (2004). Neural circuits underlying emotional distress in humans. *Annals of the New York Academy of Sciences*, 1032(1), 254-257.

CITATION OF THIS ARTICLE

S. A. Surale Patil, P.V. Pakale, Daksh Bhora. A Prospective Review Article on Lysergic Acid Diethylamide.. *Bull. Env. Pharmacol. Life Sci., Spl Issue [2]: 2023: 347-352.*