BioChemMed

Technium BioChemMed Vol. 13, pp.154-163 (2025)
ISSN: 2734-7990
www.techniumscience.com

Use of Ketamine as an Anesthetic, Antidepressant, and Treatment for Irritability Associated with ASD

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Abstract

This essay explores how ketamine operates as an anesthetic and as an antidepressant through separate but linked mechanisms. Drug repurposing is the application of a drug for a new medical use different from its original purpose, as indicated by the FDA. By comparison of peer-reviewed research on PubMed, NCBI, and clinical trials of ketamine's pharmacology, we find that the mechanism of its use in depression and irritability with Autism Spectrum Disorder (ASD) is derived from blocking the NMDA receptor. As an anesthetic, ketamine rapidly binds to the NMDA receptor and blocks signals that cause excitement and induces dissociation. As an antidepressant, ketamine slowly blocks NMDA receptors. This has the effect of creating the mTOR pathway, which causes synaptogenesis and neural regeneration. In either case, ketamine is beneficial, but must be watched as it has some side effects and is addictive. Future research is extremely vital in determining long-term side effects and realizing its full medical potential.

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ISSN: 2734-7990
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Keywords

Ketamine antidepressant mechanism; NMDA receptor ketamine; Ketamine anesthesia pharmacology; Drug repurposing ketamine

Introduction

Drug repurposing is a practice of using drugs for a new therapeutic purpose outside of its primary FDA approved indication. The cost-effective and time-efficient process emerged in mainstream around 2004 as an alternative for the former orthodox method that costs $1-2 billion and takes 10-15 years to create new drugs. The reduction of unidentified risks of side effects due to the existence of safety and pharmacokinetic data also contributed to its popularity. Based on the biomechanics data of a certain drug, active research on shared pathways or receptor targets through diverse methods such as gene expression database research, AI modeling, and observational studies are bursting all around the world.

One of the relatively recent examples of a successful drug repurposing was the research on ketamine, a drug primarily used as anesthetic, and its potential target on both treatment-resistant major depressive disorder, especially with treating suicidal impulse, and irritability associated with ASD. Treatment-resistant depression is a condition where antidepressants and depression therapies do not show significant improvement on the symptoms of the patient. Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder that impacts how people interact with others, communicate, learn, and behave. Irritability and other disruptive side effects are common in individuals with ASD. Researched since the 21st century, ketamine finally got FDA approval in 2019, although with firm restricted use and boxed warning due to its potential of abuse and severe adverse effects. As researchers studied depression-present brains, they noticed abnormality within the glutamatergic system. Ketamine was addressed during the process of this research, with the goal to target the glutamatergic system. This was an unusual process of drug development, for this discovery was the result of basic neuroscience research unlike many other discoveries which were found unexpectedly.

Ketamine, or ketamine hydrochloride, is a molecule that has a formula of C13H16ClNO. The functional groups that build ketamine are carbonyl group (ketone), benzene ring, amine group, and methyl group. As a synthetic drug, ketamine was first synthesized by Calvin Stevens in Parke Davis Laboratories, Belgium in 1963. It was first made for veterinary purposes, but later found to have an abundance of usages other than only for animals. Soon after, the United State Food and Drug Administration (US FDA) approved ketamine as an anesthetic in the 1970s. The route of administration is suggested to be intravascular (IV) or intramuscular (IM), yet it is possible to be administered intranasal, sublingual, oral, and suppository. It is still commonly used throughout the world as popular anesthesia for brief medical procedures or pre-anesthetic processes. Anesthesia for children is also done frequently with ketamine, for it has less side effects compared to other anesthetics used for adults. Another FDA-approved usage of ketamine is enhancing effects of low-potency substances. Low-potency refers to substances that need to be administered in a relatively greater amount of dosage for proper effectiveness. Off label usages include pain management in lower dosage, treating treatment-resistant depression, managing suicidal ideation, treating refractory status epilepticus, and Rapid Sequence Intubation. This paper focuses on ketamine usage as anesthetics and antidepressants.

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Although ketamine is a polypharmacological drug, meaning it has multiple targets, both its anesthetic and antidepressant effects derive from the characteristic of being an N-methyl-D-aspartate (NMDA) receptor antagonist. NMDA receptors are ligand-gated protein channels in the membrane of the brain that allow ions that generate electrical signals for the neurons such as sodium, calcium, and potassium to pass through. Glutamate, a neurotransmitter, acts as the ligand for NMDA receptors by binding on the surface of the receptor and opening the channel path. On the other hand, ketamine blocks this gateway of ions through binding with NMDA receptors. It approaches an open channel then blocks the passageway, preventing ions to pass through and send signals through the brain. This causes reduction of brain signals, creating a distinct state where consciousness is unclear. Greater dosage of ketamine intensifies the separation of consciousness, eventually causing anesthesia. There are two isomers of ketamine: R-ketamine and S-ketamine. Esketamine binds more strongly to NMDA receptors and is the FDA-approved version used in the nasal spray Spravato for TRD, as declared in 2019. Although the exact mechanism is unknown, esketamine is believed to increase glutamate level through its blockage of NMDA receptors. Glutamate not only acts as a trigger to opening the NMDA receptor channel, it also regulates the learning and memory actions and production of another neurotransmitter, gamma-aminobutyric acid (GABA), in the human brain as an excitatory neurotransmitter. The role of GABA in our brain is to slow down the actions of the human brain, granting calmness. Therefore, esketamine indirectly stimulates calmness and slow thinking while also accelerating ability to learn and think, potentially treating the treatment-resistant depression. Targeting irritability of ASD patients involves the same mechanism as that of targeting depression involving NMDA receptors, yet specifically located in the hippocampus, cerebellum, and prefrontal cortex. These are the regions of the human brain which triggers this symptom. Although further research is needed, the medication is well-tolerated by patients with ASD, indicating promising results. Exploring the two fields and ketamine as the junction of two, this paper suggests an answer to the question–how is ketamine used as both an anesthetic and antidepressant medication?

Methods

A search for literature was conducted from databases such as PubMed, Google Scholar, and NCBI Bookshelf. Keywords such as "ketamine antidepressant mechanism," "NMDA receptor ketamine," "ketamine anesthesia pharmacology," and "drug repurposing ketamine" were used. All of the selected papers were peer-reviewed studies, RCTs, and meta-analyses that examined the bimodal action of ketamine as an anesthetic and antidepressant with some studying irritability with ASD from the year 1990 to 2025.

Inclusion Criteria

- Human studies on ketamine as anesthetic and/or antidepressant
- Clinical trial, peer-reviewed articles, pharmacology papers published between 1990 and 2025

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Exclusion Criteria

- Recreational use or abuse-focused studies
- Animal-only studies
- Opinion pieces

After reviewing literature, five core studies were selected for rigorous methodology and direct correlation to the impacts of ketamine used as an anesthetic or antidepressant, with some sources focusing on irritability associated with ASD.

Results and Discussion

Original Use: Anesthetic

The origin of anesthesia is presumed to be the herbal of prehistoric ages. Ethanol, known as the first general anesthetic, was recorded as being used in ancient Mesopotamia, where the opium poppy was believed to have been cultivated in 3400 BC. Other naturally-occurred anesthesia used in ancient times include mandrake fruit extract, and was recorded to use sponge soaked in opium and mandragora being used during surgery around 1200 to relieve pain.

In 1799, British chemist Humphrey Davy discovered anesthetic effects of nitrous oxide while researching treatments for asthma and tuberculosis. He was the first to document that gas analgesic effects and benefits for suppressing pain during surgery. In 1804, Japanese physician Seishu Hanaoka performed the world's first successful mastectomy under general anesthesia using a complex herbal preparation called Tongxianshan. General anesthesia involves temporarily blocking the central nervous system to anesthetize the entire body. It reduces your consciousness, senses, and body functions to a state of domesticity.

General anesthesia is a medically induced state of temporary, reversible unconsciousness that allows pain-free surgical procedures. The state of general anesthesia includes memory loss, unconsciousness, and suppression of pain. This action is mediated by inhibition of different neural pathways and different receptors.

Anesthetics such as propofol and halothane block the connection between the thalamus and cerebral cortex, which transmit and process sensory information, prevent the recognition of pain or perceive external stimuli.

Opiate anesthetics, such as isoflurane and fentanyl, inhibit the pathways between the spinal cord that transmits pain stimulus information and the brain , preventing pain signals from being transmitted, making the body functions unable to feel or perceive pain.

Dissociative anesthetics such as ketamine and nitrous oxide block the exchange of information among the thalamus, cortex, and the limbic system that is responsible for interpreting and processing sensory stimulation, making the brain lose contact with reality. These anesthetics disassociate the brain from the body and senses.

Ketamine was first synthesized in 1962 by British chemist Calvin Stevens. It began human clinical trials in 1964 and was licensed for human use in 1970 by the United States FDA and used on soldiers as a

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battlefield anesthetic during the Vietnam War. In the United States, it is listed as a Schedule III drug. The scheduling system classifies drugs based on their legitimate medical use and potential for abuse and dependence. Schedule III drugs are rated as having minimal physical and psychological dependence potential.

Ketamine is a cyclohexanone derivative with analgesic and hallucinogenic activity, primarily as a non-competitive N-methyl-D-aspartate (NMDA) receptor antagonist. It is employed as an anesthetic and also has therapeutic applications in anesthesia induction, treatment-resistant depression, and pain therapy. Also, ketamine stops the action of glutamate at NMDA receptors, a glutamate-activated receptor, to dampen the central nervous system and eliminate pain. This silences sensory perception by blocking the sensory relay between the cerebral cortex and thalamus, causing dissociative anesthesia. The eyes of patients may be open, but their responses to outside stimuli are lost and they are unresponsive. Ketamine is administered via multiple modes of administration, like intravenous and intramuscular injection, and nasal spray, which enter the brain quickly through the bloodstream. Ketamine is a non-competitive NMDA receptor antagonist, the receptors that facilitate neural excitation following glutamate. Ketamine prevents the action of glutamate and thereby inhibits excess excitatory neurotransmission in the brain. When the anesthetic effect is explained, unlike with general anesthetics, ketamine induces dissociable anesthesia, which is the state of dissociation in which one appears to be awake but displays a cataleptic state without consciousness or memory. When under the blockade of sensory transmission, the patient may open their eyes but not respond to external stimuli and become disengaged from reality.

Because ketamine has bronchodilation effect, or widening of bronchial passages, it is the drug of choice for patients with bronchospasm. It is also used in trauma patients with massive bleeding or patients requiring blood pressure and heart rate maintenance due to the fact that it activates the cardiovascular system to increase heart rate, blood pressure, and cardiac output.

Vlisides et al. (2018): Two different ketamine administrations, 0.5 mg/kg for 40 minutes and 1.5 mg/kg under anesthesia, to 10 healthy adults were given, with 8 mg of ondansetron to avoid vomiting and nausea. EEG monitoring under anesthesia showed an enormous rise in theta band power (4.25 ± 1.9 dB) from baseline (0.64 ± 0.28 dB) after administration of anesthetics. Moreover, gamma power increased considerably, whereas alpha power gradually decreased (0.49 ± 0.22 dB). The increase in theta and gamma power was restricted to frontal and occipital channels, without anteriorization of alpha power.

The suppression of the alpha wave, which is associated with sensory processing, and the emergence of gamma and theta waves show that the thalamus-to-cerebral cortex passage of sensory information was suppressed, actually blocking the perception of the external stimulus.

Drug Repurposed: Antidepressant and ASD

Ketamine has rapid and dramatic antidepressant effects, especially in treatment-resistant depressed (TRD) patients and suicidal ideation. Unlike regular antidepressants, whose onset of action may be weeks, ketamine may be effective within hours. Research now suggests encouraging but preliminary evidence for ketamine's use as a therapeutic agent for irritability in ASD, including aggression, depression, and social withdrawal.

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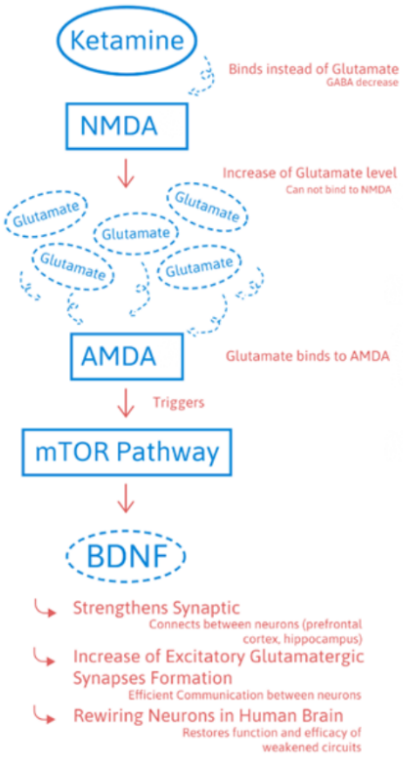
Mechanistically, ketamine works by binding to N-methyl-D-aspartate (NMDA) receptors, molecules that normally repress activity of neurons by binding to glutamate. When ketamine noncompetitively inhibits NMDA receptors, there is heightened brain activity due to the reduction of gamma-aminobutyric acid (GABA). By releasing inhibitory control on excitatory neurons, AMPA receptors bind with increased levels of glutamate, causing the protein-building signal cascade known as the mTOR pathway. The final product of the mTOR pathway is a protein called Brain-Derived Neurotrophic Factor (BDNF), which helps brain cells to survive, grow, and become incorporated by making synapses between neurons stronger, especially in the case of the prefrontal cortex and hippocampus, two brain regions linked to mood regulation, memory, and decision making. Excitatory glutamatergic synapses are induced to form, which increases neuron-to-neuron communication using glutamate as a neurotransmitter (Figure 1). Weakened circuits caused by mental illness like depression regain their functioning and effectiveness with rebalancing of the brain. Unlike regular antidepressants, which take weeks to work, ketamine works in just hours. Thus, the drug has become necessary for treating people with risky or treatment-resistant depression and suicide ideation..

Figure 1. Molecular Mechanism of Ketamine (Original Diagram)



Zarate et al.: A single IV ketamine infusion (0.5mg/kg over 40 minutes) in 18 adults with TRD was followed by a 70% response rate on the MADRS scale, compared to 0% in the control group. 35-45% of patients had lasting ketamine effects through 72 hours.

Grunebaum et al.: In 80 adults with MDD (54% of whom were taking antidepressants), a single IV infusion of ketamine (0.5mg/kg over 40 minutes) was noted to reduce suicidal ideation within 24 hours

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compared to a midazolam control. The ketamine group reduced 4.96 points more on the SSI scale than the control, with the effects lasting up to six weeks.

Wink et al.: Ketamine was administered (30mg and 50mg) compared to placebo in a study of 21 individuals with ASD aged 14-19. Ketamine was found to be well tolerated with minimal side effects and no notable changes in behavior. Nevertheless, this suggests that future research with larger populations should address effect on aggression and depression in ASD, in which glutamate dysregulation and BDNF deficits also exist.

These studies confirm the unique mechanism of action of ketamine based on the glutamate-dependent mechanism. These cascades of events make it a hopeful therapeutic option for those who are finding standard methods of treatment ineffective. In addition, there are new emerging uses of ketamine in the management of neurodevelopmental disorders such as ASD. Although no significant alteration in behavior could be observed, the drug itself was well tolerated, with encouraging implications in the future

Mechanism

Irrespective of whether it is used as anesthesia or antidepressant medication, ketamine targets the same pathway–blocking NMDA receptors. When used as anesthesia, a larger dose of ketamine is administered so that instantaneous blocking results in dissociation. When used as an antidepressant, a smaller dose is administered, triggering mTOR, hence creating BDNF which helps the brain regain balance and form new synapses.

Drug Repurposing

Ketamine has completely revolutionized depression treatment, especially for suicidal ideation. With its profile of acting at the BDNF receptors, this medication has opened other avenues for positive benefit like in ASD, PTSD, and other psychiatric related illnesses. Like with many medications extremely beneficial in so many ways, careful administration has to be made mandatory because hallucination and addiction are possible. Long-term safety is not established at all and hence careful monitoring is the way to go.

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Limits and Future Steps

Though helpful in so many other therapeutic applications, ketamine's antidepressant effect is short-lived and holds for only seven days after a single dose. There is also potential for abuse because the drug acts extremely rapidly and produces instant relief. It is also known that not all patients respond and its certain genetic determinants for the same are still unknown. There are several areas to be optimized and researched further to advance these problems. Implementing maintenance guidelines, another avenue of delivery for extended release, and further investigation on children and neurodiverse populations are needed in order to unlock ketamine's full therapeutic potential.

Conclusion

Ketamine employs a single molecule that can act in multiple ways, and it is beneficial both as an anesthetic, resulting in rapid sedation, and as an antidepressant, resulting in the creation of new synapses. It represents the promise and power of drug repurposing, proving the success of the process. However, caution should be exercised, and more research must be carried out to observe long-term effects and unveil its full potential.

Acknowledgements

We would also like to thank Professor Sello for supervising our efforts thoroughly and providing helpful guidance throughout this project. Sarahsihu Kim, Jiwoo Won, and Dayeon Lee all contributed equally to this project.

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