MDMA therapy: supplements and recovery

Research and practice edition | 27 September 2026 | revised rationale edition

What this document is for

The questions behind the supplement list

This guide offers a reasoned shortlist of supplements to consider for reducing harmful biological effects of MDMA and supporting recovery between sessions. The practical goal is to support the conditions for useful therapy now and another beneficial session roughly three months later. Each candidate is judged by what it could help, why that makes biological sense, and the research and practical experience supporting it.

The therapeutic goal

A useful session can involve feeling safer and more open, engaging with difficult emotions or memories, and consolidating new learning afterwards so it carries into ordinary life. These are distinguishable processes whose biology may change at different rates. That is why the guide considers several biological categories. The categories concern general recovery mechanisms; they do not assign supplements to these individual therapeutic processes. [72] [82]

Why recovery is part of the question

MDMA changes the handling of brain messengers and puts the body into an activated state. Experimental exposures can also disrupt energy production, increase damaging chemical reactions and injure nerve endings. The recovery question is which changes persist after the drug wears off, what the brain does to recover, and whether a supplement helps a genuinely limiting process. The sections that follow separate routine restoration of function from repair after demonstrated injury. [79] [5] [81]

The basis for these recommendations

These are recommendations for consideration based on converging biological rationale, human brain findings, MDMA animal studies and adjacent injury research. Direct human evidence that a supplement improves post-MDMA recovery or a later therapy outcome remains missing across the list. That common limitation applies throughout. The detailed entries retain the specific study conditions, doses and remaining questions.

What MDMA does during a session

It changes how brain cells communicate

Serotonin, noradrenaline and dopamine are chemical messengers. MDMA acts through the proteins that normally transport these messengers back into nerve cells. At the serotonin transporter, called SERT, it drives a large release of serotonin into the space between cells and changes its reuptake. That changes which receptors are activated and how connected brain regions respond. SERT is a working transport protein; describing its activity changing is different from showing that the protein or its nerve ending has been physically damaged. [79] [31]

Why those changes can matter for therapy

The resulting state can include greater emotional openness and connection and a reduced sense of threat. This may make difficult material easier to engage with during therapy. The experience also depends on the person, context and therapeutic work. The lasting benefit involves what is learned and retained afterwards, as well as the drug state itself. [82] [72]

It changes physical activation too

MDMA can increase heart rate, blood pressure and temperature, and can produce muscle tension, sweating and reduced appetite. Monitored therapy research documents transient cardiovascular changes and symptoms such as nausea and muscle tightness. These effects create physical recovery needs as well as the emotional work of a session. [82]

Does it use a lot of brain energy?

Brain signalling already requires substantial energy. Cells spend ATP, their immediately usable energy molecule, restoring ion balances, handling calcium and recycling the machinery used to send messages. MDMA changes this workload and can place stress on energy supply. The important issue is whether supply keeps up locally. Human research does not give a reliable extra ATP cost for a therapy session. Brain activity and blood-flow changes alone cannot tell us that its energy reserves have been depleted. [37] [80]

Why mitochondrial findings matter

Mitochondria make much of a cell's ATP. One repeated-dose rat MDMA experiment found about 30% lower ATP in two brain regions; another found mitochondrial DNA damage and changes in components of the energy-producing machinery. This makes energy support a credible research question. It does not establish that a monitored human session causes the same deficit. [14] [5]

Can MDMA damage the brain?

Yes, injury has been demonstrated in animal experiments

Some MDMA exposure regimens damage the fine branches and endings of serotonin-containing nerve cells. Microscopy has shown swollen and fragmented axons, providing evidence of physical injury beyond a low serotonin measurement. The rat experiments used repeated experimental exposure. The amount, route, repeat dosing, temperature and species matter when applying these findings to humans. [81]

How chemical stress can become injury

The processing of released messengers, MDMA metabolites and stressed mitochondria can increase reactive molecules. Cells normally contain and neutralize these molecules. When that balance fails, oxidative stress can damage membrane fats, proteins and mitochondrial material. Heat can intensify toxicity. Studies in which antioxidants protected selected later measures support this pathway; several pathways can operate together. [5] [15] [29]

What human recreational studies show

Some studies of people with a history of recreational MDMA use find lower availability of SERT on brain scans and differences in cognition. These findings raise concern, but exposure histories, other drugs and pre-existing differences complicate interpretation. A lower SERT scan reading measures transporter availability, not a direct count of dead nerve endings. [54] [57]

What this means for a therapy session

Clinical therapy trials measure symptoms, functioning and adverse events under monitored conditions. They do not directly resolve microscopic serotonin-axon damage or quantify ATP recovery after a session. The evidence therefore supports a real potential for injury without establishing that every therapeutic exposure produces nerve damage. Feeling tired, low or mentally different afterwards cannot by itself identify structural injury. [82]

Why this distinction matters for the supplement list

Different problems call for different kinds of support. Changes in messenger handling point toward regulation and replenishment. Mitochondrial strain points toward energy and cellular upkeep. Injured membranes and nerve branches point toward rebuilding materials and growth. These possibilities explain the range of candidates in the comparison.

What recovery would involve

Restoring messenger handling

Cells replenish and redistribute messenger stores and adjust transporter and receptor activity. Animal research shows that transporter function can change temporarily even when stored serotonin is unchanged. Recovery of signalling therefore involves how the machinery works as well as how much serotonin is present. [25] [31]

Restoring energy balance and clearing damaged components

Where energy supply has been disrupted, recovery requires working mitochondria, suitable fuel and appropriate placement of mitochondria near active nerve endings. Cells can repair or remove damaged proteins and mitochondria and replace them. This motivates ENERGY and CELL UPKEEP candidates, although the benefit depends on whether those processes are actually limiting recovery. [37] [46]

Controlling chemical stress and rebuilding structures

Antioxidant systems help keep reactive molecules under control. Where membranes or nerve branches are injured, rebuilding requires energy, membrane ingredients and growth processes. This motivates the ANTIOXIDANT DEFENCES, MEMBRANES and NERVE CONNECTIONS categories. Studies after other nervous-system injuries help identify useful repair mechanisms. [15] [2] [3]

Returning to useful learning and a later session

Energy, communication and functioning nerve connections all support learning. Supporting those processes provides the broader rationale for preparing for a later session. Felt safety, emotional engagement and later consolidation may depend on overlapping biology, which is why recovery deserves consideration across several mechanisms. [26] [48]

Where supplements could contribute

The brain already replenishes messengers, regulates signalling and repairs cellular material. A supplement could help by supplying a useful ingredient, increasing a reserve or supporting one of these processes when it is under strain. The list evaluates how convincing that rationale is for each candidate.

Protection, recovery and the therapy experience

Protection before or during exposure

A preventive treatment is given early enough to be active while MDMA is acting. Some studies give a single injection shortly beforehand; others use repeated pretreatment or simultaneous dosing. Each entry specifies which happened. This is the evidence relevant to reducing harmful effects as they develop.

Treatment soon afterwards

A substance started minutes or hours after exposure may interrupt an ongoing injury process. That is early treatment, and its benefit may depend on a short window. It cannot automatically establish benefit when started days or weeks later.

Recovery and preparation between sessions

Ongoing use may build a reserve, supply ingredients or influence cellular maintenance over time. Creatine is an example of a reserve-building rationale. A reserve built beforehand could remain relevant during the session; an extra dose on the day is a separate question. [1]

Effects on openness, connection and therapeutic work

Protection is useful only in the context of the therapy goal. A supplement that changes MDMA’s action could also change openness, emotional engagement or learning. The entries highlight concrete reasons to expect an interaction, such as resveratrol’s transporter effects and the community discussion around NAC. [22]

How the comparison is judged

Why some candidates appear higher

Placement reflects the combined rationale: relevance to MDMA biology, direct MDMA findings, treatment after injury, human brain effects, practical oral use and functional outcomes. Positive and disappointing findings both count. Higher placement means a stronger overall reason to consider a candidate for this purpose.

What the group labels mean

Strongest overall rationale for considering means several useful reasons come together. Additional candidates with some supporting evidence means there is a credible reason to look further, with bigger gaps. Weaker reasons to prioritize means the relevant support is thinner, concerns a different timing window, or includes disappointing results. Items within a group have no proven order of effectiveness.

Why there are two comparisons

The recovery comparison emphasizes evidence relevant to the weeks between sessions. The protection comparison emphasizes experiments where a supplement was present before, during or soon after MDMA. ALA can therefore appear high for protection and low for ongoing recovery without a contradiction.

Why study timing changes the interpretation

Giving a substance before MDMA tests prevention. Starting it after exposure tests a different question, and treatment minutes afterwards differs from treatment weeks later. The comparison weighs that difference instead of treating every positive animal result as delayed recovery evidence.

Health and medication cautions

Specific health and medication cautions appear beside the relevant supplement. Community reports are attributed separately from research findings. The same biological category can contain several candidates; shared mechanisms help identify overlap when narrowing the list.

Understanding study doses and timing

What the amounts mean

mg/kg means milligrams per kilogram of the animal's body weight. A fixed human amount such as 1,000 mg/day describes what people swallowed in that particular study. A concentration such as 100 micromolar describes the strength of a solution used locally in tissue, not an oral dose.

Why the route matters

Oral means swallowed. Gavage means delivery into an animal's stomach through a tube. Intraperitoneal injection enters the abdominal cavity; intravenous injection enters a vein; subcutaneous injection goes under the skin. Direct brain perfusion delivers a solution into a selected brain area. These routes can produce very different blood and brain exposures.

Is there a human equivalent?

Animal-to-human scaling can produce a numerical estimate, but it does not establish the amount a person should swallow to reproduce protection. Absorption, metabolism, brain entry, repeated dosing and the MDMA exposure all matter. None of the animal protection regimens here has a validated oral human equivalent for MDMA protection or recovery. Human studies give actual human doses for their own outcomes, such as a brain-chemistry change; they do not establish an MDMA regimen.

How incomplete methods are handled

Where the accessible primary report confirms a dose but leaves a route or interval unclear, the entry states exactly what remains unverified. A missing detail is not silently replaced with a forum schedule. Specific examples below are the studies supporting the placement, rather than an exhaustive catalogue of every dose ever tested.

What the tags mean

Tags help you see which biological idea puts a supplement on the list. They describe proposed roles.

SEROTONIN | How brain messages are handled

Serotonin is a chemical messenger. SERT, the serotonin transporter, moves serotonin back into nerve cells after release. Its activity and location can change. This tag concerns messenger handling and its regulation.

ENERGY | Keeping energy available to brain cells

ATP is a molecule cells use to power their work. Mitochondria are parts of cells that produce much of that energy. Cells use it to send messages, maintain themselves and build connections. Creatine helps buffer energy supply when demand changes. NAD and coenzyme Q participate in energy-related chemistry.

CELL UPKEEP | Maintaining and renewing working parts

Cells move, repair and replace their components. Autophagy means breaking down and recycling cellular material; mitophagy is the version involving mitochondria. Mitochondria also need to reach and remain at places where energy is needed. This tag concerns those upkeep processes.

ANTIOXIDANT DEFENCES | Managing potentially damaging chemical stress

Cells produce reactive molecules during normal activity. Too much can cause oxidative stress, which can damage proteins and membranes. Glutathione is one of the cell's protective chemicals. NRF2 helps switch on protective genes. This tag describes a reason to study a supplement's effect on that balance.

Research behind these definitions: These papers explain how brain messengers, transporters and cellular energy are regulated. [31] [32] [33] [34] [35] [37] [38]

What the tags mean

Continued

INFLAMMATION | The response of supporting and immune cells

Astrocytes and microglia help maintain the brain and respond to injury. Their responses can support repair or sustain harmful inflammation. This category concerns the balance of those responses.

MEMBRANES | The material surrounding cells and their connections

Membranes are the thin, fat-containing layers around cells and their working parts. Phospholipids are major building materials in these layers. DHA, choline and uridine-related pathways contribute to their production or properties. This tag concerns supplying or maintaining that material.

NERVE CONNECTIONS | Maintaining and growing nerve branches

An axon is the long branch that carries a nerve cell's signal to other cells. Its endings help pass the message along. Sprouting means growing new branches, sometimes from fibres that survived an injury. Regeneration means regrowing an injured fibre. These are related but different kinds of repair.

LEARNING | Changing connections through experience

Plasticity is the brain's ability to change the strength and structure of its connections. BDNF is a protein involved in this process, acting through a receptor called TrkB. This category concerns biological processes that enable learning and adaptation.

Why location matters too: In nerve cells grown in a laboratory, BDNF caused mitochondria to stop near places where messages are released. Communication improved without an immediate increase in total ATP. Having energy-producing parts in the right place can matter as well as how much energy they make. [46]

Research behind these definitions: These papers explain protective responses, membranes, nerve growth and learning-related changes. [43] [45] [47] [48] [64]

Comparison at a glance

Recovery between sessions | 1-2 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Creatine A naturally occurring compound stored in cells partly as phosphocreatine, a reserve that helps regenerate ATP, the energy cells use. Supplements usually contain creatine monohydrate. **Tags:** ENERGY; NERVE CONNECTIONS	**Strongest overall rationale for considering** Brain cells need energy to communicate, maintain their structures and repair damage. Creatine could help by increasing the reserve available when energy demand rises or supply is strained.	Human: 5 g oral creatine four times daily for four weeks. Mouse injury: 2 g/kg by stomach tube twice daily, up to eight weeks. Full entry separates these studies. [1] [2] [30]	A strong candidate for preparation and recovery because oral use can raise brain creatine, and animal injury research links improved energy availability with nerve regrowth. **Caution:** Kidney disease or abnormal kidney tests: review first. [93]
DHA / omega-3 DHA, docosahexaenoic acid, is an omega-3 fat that forms part of nerve-cell membranes. Oral sources include fish oil and algal oil. Fish oil commonly also contains EPA, another omega-3 fat. **Tags:** MEMBRANES; NERVE CONNECTIONS; LEARNING	**Strongest overall rationale for considering** Maintaining and rebuilding nerve branches and their connections requires membrane material. DHA supplies one of those ingredients. It is worth considering for ongoing recovery support because it contributes to the structures through which brain cells communicate.	Rat: one DHA injection into a vein, 250 nmol/kg, 30 minutes after spinal injury. Treatment at three weeks did not improve recovery. [3] [4]	A strong candidate for membrane and nerve-connection support. In a spinal-injury experiment, DHA encouraged new branching of serotonin-containing fibres, adding a specific repair-related reason to its nutritional role. **Caution:** Review anticoagulants, atrial fibrillation and product-source allergy. [84]

Comparison at a glance

Recovery between sessions | 3-4 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
ALCAR Acetyl-L-carnitine, a form of carnitine involved in cellular metabolism and studied for its effects on mitochondria, the structures that produce much of a cell's energy. **Tags:** ENERGY; ANTIOXIDANT DEFENCES	**Strongest overall rationale for considering** MDMA animal studies identify mitochondrial stress as one possible source of harm. ALCAR could help maintain that energy-producing machinery and reduce associated chemical damage. It preserved mitochondrial and serotonin-related measures in a direct MDMA rat experiment.	Rat MDMA study: ALCAR 100 mg/kg by abdominal injection, 30 minutes before MDMA. A separate injury study began treatment 15 minutes after injury. [5] [6]	A strong candidate for mitochondrial support, with direct MDMA protection findings and separate evidence of improved recovery after spinal injury. **Caution:** Seizure disorder or severe kidney disease: review first. [83]
NAC N-acetylcysteine, a form of the amino acid cysteine. It supplies an ingredient for making glutathione, a major antioxidant inside cells, and also influences regulation of the brain messenger glutamate. **Tags:** ANTIOXIDANT DEFENCES; SEROTONIN	**Strongest overall rationale for considering** Cells use glutathione to manage potentially damaging reactive molecules. Supplying cysteine through NAC could support those defences during recovery. Direct MDMA animal findings and a human brain-glutathione finding give this rationale several lines of support.	Rat: NAC 100 mg/kg by abdominal injection with experimental MDMA; exact lead time unverified. Human: oral 2,400 mg/day for eight weeks, without MDMA. [7] [8] [9]	A strong candidate for antioxidant support between sessions. It also attracts substantial community interest for recovering a diminished MDMA response, with mixed reported results. **Caution:** Review asthma, ulcer history and nitroglycerin use. [87]

Comparison at a glance

Recovery between sessions | 5-6 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Uridine + adequate choline Uridine is a small molecule used in RNA and membrane-building pathways. Choline is an essential nutrient found in foods such as eggs, soy and meat. Adequate choline means sufficient total intake from food and supplements. **Tags:** MEMBRANES; LEARNING	**Additional candidates with some supporting evidence** Uridine, choline and DHA contribute complementary ingredients for making membrane phospholipids. Providing those ingredients could support maintenance and growth of nerve connections, especially where intake or availability is limited.	Human: oral uridine 2 g/day for seven days. Gerbil combination study: dietary uridine/choline plus DHA by stomach tube, daily for four weeks. [10] [4] [52]	A coherent addition to a membrane-support approach. Animal studies found increased membrane fats and synaptic proteins, and a human uridine study found changes in brain membrane precursors. **Caution:** Avoid excessive choline; uridine interaction data are limited. [85]
Sulforaphane A compound formed from substances in broccoli and other cruciferous vegetables. Supplements supply sulforaphane or a precursor with the enzyme needed to produce it. **Tags:** ANTIOXIDANT DEFENCES; INFLAMMATION	**Additional candidates with some supporting evidence** Sulforaphane activates the cell's own defence programmes, including pathways involved in making glutathione. This could help cells handle chemical stress as they recover. A small human study measured increased glutathione in the brain.	Human pilot: oral sulforaphane 100 micromoles/day for seven days, as a standardized extract; nine participants, no placebo group. [11]	Worth considering for ongoing antioxidant-defence support. Its ability to influence a protective brain chemical in people gives the rationale more substance than a laboratory mechanism alone. **Caution:** MDMA co-exposure worsened toxicity in liver cells; human relevance unresolved. [29]

Comparison at a glance

Recovery between sessions | 7-8 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Astaxanthin A red-orange pigment made by certain algae and found in seafood. Supplements generally use an algal source. It is studied for antioxidant and inflammation-related effects. **Tags:** ANTIOXIDANT DEFENCES; INFLAMMATION; NERVE CONNECTIONS	**Additional candidates with some supporting evidence** Limiting oxidative and inflammatory stress could help preserve the conditions needed for nerve repair. Astaxanthin has also been studied after brain and nerve injuries, with findings involving movement, cognition and nerve growth.	Stroke-model mice: astaxanthin 30 mg/kg by stomach tube twice daily for 28 days after surgery. Exact first-dose delay unverified. [16] [17]	An additional recovery candidate with a useful combination of antioxidant biology and treatment-after-injury animal findings.
Nicotinamide riboside (NR) A vitamin-B3-related molecule the body uses to make NAD, a coenzyme involved in energy metabolism and cellular maintenance. **Tags:** ENERGY; CELL UPKEEP	**Additional candidates with some supporting evidence** Energy production and cellular maintenance depend on NAD. NR could support those processes by increasing its availability. A human study found that oral NR increased brain NAD in many participants.	NADPARK human trial: oral NR 1,000 mg/day for 30 days in Parkinson's disease. No MDMA was used. [12]	An additional energy-support candidate with direct evidence that oral use can affect the proposed biochemical pathway in the human brain.

Comparison at a glance

Recovery between sessions | 9-10 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Urolithin A A compound some gut bacteria produce from plant substances found in foods such as pomegranates and walnuts. Supplements provide the compound directly. **Tags:** CELL UPKEEP; LEARNING	**Additional candidates with some supporting evidence** Recovery may require clearing damaged mitochondria and maintaining a healthier population of energy-producing structures. Urolithin A is studied for this cellular upkeep, including mitophagy, the removal of damaged mitochondria.	High-anxiety rodents: dietary urolithin A 25 mg/kg/day, with behaviour monitored during the first four weeks. No MDMA exposure. [13]	An exploratory addition focused on mitochondrial upkeep. Animal brain research connects its effects on mitochondrial pathways with improved nerve-cell function and behaviour.
CoQ10 / ubiquinol Coenzyme Q10 helps move electrons through the machinery that produces mitochondrial energy. Ubiquinol and ubiquinone are two forms of CoQ10 sold as supplements. **Tags:** ENERGY; ANTIOXIDANT DEFENCES	**Additional candidates with some supporting evidence** CoQ10 participates directly in energy production and antioxidant defence. Those roles make it relevant when considering mitochondrial strain after MDMA. An MDMA animal experiment found less serotonin loss when CoQ10 was delivered into the brain.	Rat: ubiquinone solution, 100 micromolar, perfused directly into brain tissue with experimental MDMA exposure. No oral CoQ10 regimen was tested. [14]	An additional candidate for energy and antioxidant support. The direct MDMA finding strengthens the biological reason to consider this pathway. **Caution:** Review warfarin, insulin and cancer treatments. [90]

Comparison at a glance

Recovery between sessions | 11-12 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
ALA Alpha-lipoic acid, a naturally occurring compound involved in energy-producing enzyme reactions and studied for antioxidant effects. Here ALA refers to alpha-lipoic acid. **Tags:** ANTIOXIDANT DEFENCES; ENERGY	**Weaker reasons to prioritize** ALA could help limit oxidative damage while MDMA is active. A direct rat study found that pretreatment preserved later serotonin and transporter measurements, giving it a specific protection rationale.	Rat: ALA 100 mg/kg by abdominal injection twice daily for two days, with pretreatment 30 minutes before MDMA. No delayed-recovery arm. [15]	One of the more compelling candidates for protection around exposure. Its strongest evidence concerns preventing changes as they develop, which explains its lower priority for months-long recovery use. **Caution:** Low blood sugar history or diabetes medicines: review first. [88]
Lion’s mane Hericium erinaceus, an edible mushroom sold as powders and extracts. Researchers also study individual compounds isolated from it. **Tags:** LEARNING; NERVE CONNECTIONS	**Weaker reasons to prioritize** Some lion’s mane compounds influence growth-related signalling and branching in nerve cells. This gives a plausible reason to consider it for maintaining and developing nerve connections during recovery.	Mouse: oral extract 100 or 250 mg/kg, or hericene A 5 mg/kg, over 31 days. Product-specific experiment, without MDMA. [18]	A lower-priority option with interesting nerve-growth and memory findings. Its appeal is the potential to support the biology of nerve connections. **Caution:** Avoid after a prior mushroom-extract allergic reaction. [92]

Comparison at a glance

Recovery between sessions | 13-14 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Citicoline alone CDP-choline, a compound involved in making phosphatidylcholine, a major membrane fat. Oral citicoline provides choline and cytidine-related material. **Tags:** MEMBRANES	**Weaker reasons to prioritize** Supplying membrane-building ingredients could support maintenance of nerve cells and their connections. Citicoline offers one way of supplying material to that pathway.	Human TBI trial: citicoline 2,000 mg/day orally or by feeding tube for 90 days; 1,213 participants. No overall functional/cognitive benefit. [19]	A lower-priority membrane-support option. Its plausible mechanism is tempered by a large human brain-injury trial that found no overall improvement in recovery.
Curcumin A yellow plant compound found in turmeric, sold in concentrated extracts and formulations designed to improve absorption. **Tags:** ANTIOXIDANT DEFENCES; INFLAMMATION	**Weaker reasons to prioritize** Curcumin could help manage inflammation and oxidative stress that interfere with recovery. A rat study involving MDMA found partial improvement in a memory task, giving it a direct reason for inclusion.	Rat: curcumin 200 mg/kg/day for one week; MDMA 15 mg/kg twice daily for four days. Route and exact overlap remain unverified. [66] [20]	A lower-priority recovery candidate with a direct MDMA animal finding. Its potential lies in reducing harmful inflammatory and chemical stress. **Caution:** Avoid medicinal products with current or previous liver problems. [89] [20]

Comparison at a glance

Recovery between sessions | 15-16 of 16

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Spermidine A naturally occurring polyamine, a small molecule involved in cell growth and maintenance. It occurs in foods and concentrated supplements, including wheat-germ extracts. **Tags:** CELL UPKEEP	**Weaker reasons to prioritize** Cells recover partly by clearing and replacing damaged components. Spermidine is studied for effects on autophagy, the recycling process involved in this upkeep.	Human: plant extracts supplying about 1.2 mg/day for three months (pilot), then 0.9 mg/day for 12 months (larger trial). [21] [51]	A lower-priority cellular-upkeep candidate. A promising small memory study was followed by a larger trial with a null primary result, reducing the reason to prioritize it. **Caution:** Check wheat/gluten ingredients; MDMA interaction data are absent. [51]
NMN Nicotinamide mononucleotide, a molecule used in making NAD, which participates in cellular energy production and maintenance. **Tags:** ENERGY; CELL UPKEEP	**Weaker reasons to prioritize** NMN offers another way to supply material for NAD production. This gives it an energy and cellular-maintenance rationale when considering recovery.	No relevant NMN regimen established. The 1,000 mg/day, 30-day human brain study tested NR, not NMN. [12]	A lower-priority NAD-support option. NR currently has the more directly relevant human brain finding in the studies reviewed here.

Comparison at a glance

Protection around the session | 1-2 of 8

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
ALA Alpha-lipoic acid, a naturally occurring compound involved in energy-producing enzyme reactions and studied for antioxidant effects. Here ALA refers to alpha-lipoic acid. **Tags:** ANTIOXIDANT DEFENCES; ENERGY	**Strongest overall rationale for considering** ALA could help limit oxidative damage while MDMA is active. A direct rat study found that pretreatment preserved later serotonin and transporter measurements, giving it a specific protection rationale.	Rat: ALA 100 mg/kg by abdominal injection twice daily for two days; pretreatment 30 minutes before a single MDMA 20 mg/kg injection. ALA alone, not combined with ALCAR. [15]	A leading protection candidate: ALA preserved later serotonin and transporter measures in rats when supplied before MDMA. The proposed benefit is limiting oxidative injury during exposure. **Caution:** Low blood sugar history or diabetes medicines: review first. [88]
ALCAR Acetyl-L-carnitine, a form of carnitine involved in cellular metabolism and studied for its effects on mitochondria, the structures that produce much of a cell’s energy. **Tags:** ENERGY; ANTIOXIDANT DEFENCES	**Strongest overall rationale for considering** MDMA animal studies identify mitochondrial stress as one possible source of harm. ALCAR could help maintain that energy-producing machinery and reduce associated chemical damage. It preserved mitochondrial and serotonin-related measures in a direct MDMA rat experiment.	Rat: ALCAR 100 mg/kg by abdominal injection 30 minutes before four MDMA injections of 10 mg/kg. Outcomes measured two weeks later. [5]	A leading protection candidate for mitochondrial support: pretreatment preserved energy-related machinery and serotonin measures in an MDMA rat experiment. **Caution:** Seizure disorder or severe kidney disease: review first. [83]

Comparison at a glance

Protection around the session | 3-4 of 8

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
NAC N-acetylcysteine, a form of the amino acid cysteine. It supplies an ingredient for making glutathione, a major antioxidant inside cells, and also influences regulation of the brain messenger glutamate. **Tags:** ANTIOXIDANT DEFENCES; SEROTONIN	**Additional candidates with some supporting evidence** Cells use glutathione to manage potentially damaging reactive molecules. Supplying cysteine through NAC could support those defences during recovery. Direct MDMA animal findings and a human brain-glutathione finding give this rationale several lines of support.	Rat: NAC 100 mg/kg by abdominal injection with MDMA exposure. Pretreatment reported; precise lead time and full schedule unverified in accessible primary text. [7] [8]	A plausible protection candidate through glutathione support. Mixed community reports about blunting make use near a session a separate decision from between-session use. **Caution:** Review asthma, ulcer history and nitroglycerin use. [87]
Vitamin C Ascorbic acid, an essential vitamin obtained from food or supplements. It contributes to antioxidant defence and several processes involved in maintaining body tissues. **Tags:** ANTIOXIDANT DEFENCES; ENERGY	**Additional candidates with some supporting evidence** Vitamin C could help limit oxidative stress following MDMA exposure. It has a direct animal finding in which treatment after MDMA preserved selected ATP and serotonin measurements.	Rat: vitamin C 250 mg/kg in separate groups, 30 minutes before, three hours after or five hours after MDMA 20 mg/kg. Route unverified. [23]	A plausible early-recovery candidate. The positive group received it five hours after MDMA, showing that intervention after exposure can still influence developing harm in that model. **Caution:** High-dose use: review iron overload and kidney/stone history. [86]

Comparison at a glance

Protection around the session | 5-6 of 8

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Resveratrol A plant polyphenol found in grapes and some other plants, available as a concentrated supplement. **Tags:** ANTIOXIDANT DEFENCES; SEROTONIN	**Possible effect on the MDMA experience** Resveratrol deserves consideration because a rat MDMA study found better later serotonin-transporter measurements. Its effects at the transporter provide a possible explanation for that protection.	Rat: resveratrol 20 mg/kg into the abdomen together with MDMA 10 mg/kg under the skin, twice daily for four days. Later imaging at two weeks. [22]	A protection candidate with an important session-related question: interacting with SERT could help explain protection while also changing MDMA's serotonin release. **Caution:** Prescription medication review: human enzyme interactions reported. [91]
Glutathione An antioxidant made inside cells from three amino acids: glutamate, cysteine and glycine. Oral supplements supply the molecule itself; NAC supplies one of its building materials. **Tags:** ANTIOXIDANT DEFENCES; INFLAMMATION	**Weaker reasons to prioritize** Glutathione helps neutralize reactive molecules and process some damaging compounds. Supporting that defence could reduce chemical stress during MDMA exposure and early recovery.	Rat: oral glutathione 15 mg/kg with oral MDMA 5 or 15 mg/kg over 56 days. Exact within-day schedule unverified. [24]	A lower-priority protection candidate with a direct oral MDMA animal study showing partial biochemical protection.

Comparison at a glance

Protection around the session | 7-8 of 8

Candidates are ordered by the strength of their combined rationale. Tags show the biological processes each could support.

Supplement and tags	Why consider it	Supporting study	Potential benefit and priority
Creatine A naturally occurring compound stored in cells partly as phosphocreatine, a reserve that helps regenerate ATP, the energy cells use. Supplements usually contain creatine monohydrate. **Tags:** ENERGY; NERVE CONNECTIONS	**Preparation that carries into the session** Brain cells need energy to communicate, maintain their structures and repair damage. Creatine could help by increasing the reserve available when energy demand rises or supply is strained.	Human: 5 g oral creatine four times daily for four weeks. Mouse injury: 2 g/kg by stomach tube twice daily, up to eight weeks. Full entry separates these studies. [1] [2] [30]	Building phosphocreatine reserves beforehand could help buffer energy during the session. The relevant preparation happens over time; the reserve is available when demand rises. **Caution:** Kidney disease or abnormal kidney tests: review first. [93]
DHA / omega-3 DHA, docosahexaenoic acid, is an omega-3 fat that forms part of nerve-cell membranes. Oral sources include fish oil and algal oil. Fish oil commonly also contains EPA, another omega-3 fat. **Tags:** MEMBRANES; NERVE CONNECTIONS; LEARNING	**Preparation that carries into the session** Maintaining and rebuilding nerve branches and their connections requires membrane material. DHA supplies one of those ingredients. It is worth considering for ongoing recovery support because it contributes to the structures through which brain cells communicate.	Rat: one DHA injection into a vein, 250 nmol/kg, 30 minutes after spinal injury. Treatment at three weeks did not improve recovery. [3] [4]	Regular DHA intake supplies material for nerve-cell membranes. The rationale for the next session is ongoing membrane support across the interval. **Caution:** Review anticoagulants, atrial fibrillation and product-source allergy. [84]

Recovery between sessions

Creatine

Strongest overall rationale for considering

Tags: ENERGY; NERVE CONNECTIONS

What it is: A naturally occurring compound stored in cells partly as phosphocreatine, a reserve that helps regenerate ATP, the energy cells use. Supplements usually contain creatine monohydrate.

Why consider it: Brain cells need energy to communicate, maintain their structures and repair damage. Creatine could help by increasing the reserve available when energy demand rises or supply is strained.

My assessment: A strong candidate for preparation and recovery because oral use can raise brain creatine, and animal injury research links improved energy availability with nerve regrowth.

Timing and relevance to the session: The rationale is to build reserves over time so they are available during the session and subsequent recovery. The human brain study used four weeks of supplementation.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Kidney disease or unexplained kidney-test abnormalities call for an individual review before use. Healthy-user safety data do not resolve that situation. [93]

Research details

What researchers found: Dechent et al. measured an average 8.7% increase in total brain creatine in six healthy volunteers. Han et al. found some spinal nerve regrowth with creatine in ordinary mice and additional regrowth in mice with a genetic change that improved mitochondrial movement. Hellem et al. observed increased brain phosphocreatine and improved mood in an uncontrolled stimulant-use sample.

Actual dose, route and timing: Human brain study: creatine monohydrate, 5 g by mouth four times daily for four weeks. Hellem pilot: 5 g/day orally for eight weeks. Mouse spinal-injury experiment: 2 g/kg by stomach tube twice daily, continued for up to eight weeks after injury; the exact delay to the first dose was not confirmed in the accessible methods.

Study limitations and human translation: Actual human doses exist for increasing brain creatine. Neither those doses nor the mouse regimen establish an amount for MDMA protection or recovery.

What would change its place: A study starting creatine after MDMA and measuring improved nerve function or later-session outcomes would strengthen its position.

Research sources: [1] [2] [30]

Recovery between sessions

DHA / omega-3

Strongest overall rationale for considering

Tags: MEMBRANES; NERVE CONNECTIONS; LEARNING

What it is: DHA, docosahexaenoic acid, is an omega-3 fat that forms part of nerve-cell membranes. Oral sources include fish oil and algal oil. Fish oil commonly also contains EPA, another omega-3 fat.

Why consider it: Maintaining and rebuilding nerve branches and their connections requires membrane material. DHA supplies one of those ingredients. It is worth considering for ongoing recovery support because it contributes to the structures through which brain cells communicate.

My assessment: A strong candidate for membrane and nerve-connection support. In a spinal-injury experiment, DHA encouraged new branching of serotonin-containing fibres, adding a specific repair-related reason to its nutritional role.

Timing and relevance to the session: The rationale is regular intake during the interval between sessions, supplying membrane material over time. DHA incorporated into membranes remains relevant during the next session too.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Review use with anticoagulants and a history of atrial fibrillation. Long-term high-dose omega-3 trials found a small increase in atrial fibrillation in cardiovascular-risk populations. Fish allergy requires checking the particular product and its source. [84]

Research details

What researchers found: DHA given soon after spinal injury in rats increased branching from surviving serotonin fibres and their contact with other nerve cells. Separate animal studies used nutrient mixtures containing DHA and found improved injury outcomes.

Actual dose, route and timing: Key rat study: one intravenous dose of DHA, 250 nmol/kg, 30 minutes after spinal-cord hemisection. A separate delayed-treatment arm at three weeks did not improve recovery. The multi-nutrient spinal-injury study used enriched feed for four or nine weeks; it tested a mixture, not an isolated oral DHA dose.

Study limitations and human translation: The strongest serotonin-fibre result used DHA injected into a vein shortly after spinal injury. Oral DHA supplies the same molecule through a different route, with different absorption and timing. The study therefore supports the repair rationale without establishing an effective oral amount or post-MDMA schedule. A fish-oil product's total oil weight differs from its DHA content.

What would change its place: A positive study of oral DHA started after a serotonin-related injury would strengthen the case.

Research sources: [3] [4]

Recovery between sessions

ALCAR

Strongest overall rationale for considering

Tags: ENERGY; ANTIOXIDANT DEFENCES

What it is: Acetyl-L-carnitine, a form of carnitine involved in cellular metabolism and studied for its effects on mitochondria, the structures that produce much of a cell's energy.

Why consider it: MDMA animal studies identify mitochondrial stress as one possible source of harm. ALCAR could help maintain that energy-producing machinery and reduce associated chemical damage. It preserved mitochondrial and serotonin-related measures in a direct MDMA rat experiment.

My assessment: A strong candidate for mitochondrial support, with direct MDMA protection findings and separate evidence of improved recovery after spinal injury.

Timing and relevance to the session: Its clearest MDMA evidence concerns being present before exposure: one injected dose 30 minutes beforehand. Its separate injury study also gives a reason to consider support early in recovery.

Community and practice reports: RollSafe and Psychedelic Support propose oral 500 mg three hours before, one hour after and five hours after MDMA. A Bluelight contributor described preserved MDMA effects while using ALCAR. This is limited subjective reassurance; the sources do not measure subsequent nerve repair or therapy outcomes. [73] [74] [75]

Who should avoid it or seek review: A seizure disorder or severe kidney disease warrants review: broader carnitine safety guidance flags these groups. Those cautions do not quantify ALCAR-specific risk with MDMA. Liver disease requires assessment of the actual diagnosis and product; this MDMA animal study cannot establish suitability. [83]

Research details

What researchers found: Giving ALCAR before MDMA protected mitochondrial and serotonin measures in rats. In a separate spinal-injury experiment, treatment begun soon after injury preserved tissue and improved movement. Together these findings support an energy-related reason to investigate it.

Actual dose, route and timing: MDMA experiment: rats received a single pretreatment of ALCAR 100 mg/kg by abdominal injection 30 minutes before a regimen of four MDMA injections of 10 mg/kg. ALCAR was tested separately from ALA. In the separate spinal-injury study, ALCAR 300 mg/kg was injected into the abdomen at 15 minutes and six hours after injury, then once daily for 21 days in the long-term recovery arm.

Study limitations and human translation: Both key experiments used injected animal doses. Their results do not establish an effective oral human amount or a three-month recovery schedule.

What would change its place: A positive MDMA study in which treatment begins after the drug has cleared would make its recovery placement stronger.

Research sources: [5] [6]

Recovery between sessions

NAC

Strongest overall rationale for considering

Tags: ANTIOXIDANT DEFENCES; SEROTONIN

What it is: N-acetylcysteine, a form of the amino acid cysteine. It supplies an ingredient for making glutathione, a major antioxidant inside cells, and also influences regulation of the brain messenger glutamate.

Why consider it: Cells use glutathione to manage potentially damaging reactive molecules. Supplying cysteine through NAC could support those defences during recovery. Direct MDMA animal findings and a human brain-glutathione finding give this rationale several lines of support.

My assessment: A strong candidate for antioxidant support between sessions. It also attracts substantial community interest for recovering a diminished MDMA response, with mixed reported results.

Timing and relevance to the session: Between-session use and use close to MDMA deserve separate consideration. Reports of weaker effects with nearby dosing make the proposed pause relevant; community suggestions range from roughly 30 hours to two weeks.

Community and practice reports: NAC is repeatedly discussed for recovery and loss of magic. Reports include improvement, no change and blunting. Suggested pauses range from about 30 hours to two weeks, with one to two weeks a recognizable recommendation. A practitioner guide instead proposes NAC shortly before MDMA. This disagreement leaves both the interaction and the correct pause unresolved. [68] [69] [76] [74]

Who should avoid it or seek review: Avoid after an allergic reaction to acetylcysteine. Oral product information calls for caution with asthma and a history of peptic ulcer. Nitroglycerin effects can be enhanced, increasing the risk of low blood pressure and headache. [87]

Research details

What researchers found: NAC reduced selected signs of harm and memory problems in rats receiving MDMA. In a separate study of 40 people with schizophrenia or schizoaffective disorder, eight weeks of NAC increased glutathione in one brain region. That study did not find improved symptoms or thinking skills.

Actual dose, route and timing: 2013 rat study: NAC 100 mg/kg by abdominal injection, with twice-daily MDMA injections of 5, 10 or 20 mg/kg in separate groups. The abstract calls this pretreatment; the exact lead time and total days were not confirmed from the accessible primary text. The 2015 experiment used NAC 100 mg/kg and MDMA 10 mg/kg by abdominal injection; its exact sequence remains unverified. Human brain study: 2,400 mg/day orally for eight weeks.

Study limitations and human translation: The human brain-glutathione study involved schizophrenia-spectrum illness and found no improvement in symptoms or cognition. The MDMA work was in animals. A controlled study has not established whether NAC blunts the human MDMA experience or what pause would prevent that.

What would change its place: A controlled study comparing timing, recovery and later-session effects would directly address its main unanswered questions.

Research sources: [7] [8] [9]

Recovery between sessions

Uridine + adequate choline

Additional candidates with some supporting evidence

Tags: MEMBRANES; LEARNING

What it is: Uridine is a small molecule used in RNA and membrane-building pathways. Choline is an essential nutrient found in foods such as eggs, soy and meat. Adequate choline means sufficient total intake from food and supplements.

Why consider it: Uridine, choline and DHA contribute complementary ingredients for making membrane phospholipids. Providing those ingredients could support maintenance and growth of nerve connections, especially where intake or availability is limited.

My assessment: A coherent addition to a membrane-support approach. Animal studies found increased membrane fats and synaptic proteins, and a human uridine study found changes in brain membrane precursors.

Timing and relevance to the session: This is an ongoing supply-of-materials rationale across the recovery interval. Dietary choline intake helps determine whether an additional choline supplement has a useful role.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Excess choline can cause low blood pressure and other adverse effects, including liver toxicity; the adult upper limit is 3.5 g/day from all sources. Uridine has much less established supplement safety information. Significant liver or kidney disease and medication use need individual assessment. [85]

Research details

What researchers found: Oral combinations increased membrane fats and proteins associated with connections in gerbil brains. A small human study found increased chemicals used to make membranes after oral uridine. Those findings show that the pathway can respond to added material.

Actual dose, route and timing: Human uridine study: 2 g/day orally for seven days in a placebo-controlled study of 17 healthy men. Gerbil work gave dietary uridine monophosphate and choline, with DHA by stomach tube, daily for four weeks. The accessible abstract confirms that schedule but not every ingredient’s concentration; those amounts are not reconstructed from a later paper.

Study limitations and human translation: The seven-day human uridine dose establishes a brain-chemistry experiment, not an MDMA dose. It is also not a validated uridine-DHA-choline combination regimen.

What would change its place: A study showing what uridine adds beyond DHA and adequate nutrition would clarify its value.

Research sources: [10] [4] [52]

Recovery between sessions

Sulforaphane

Additional candidates with some supporting evidence

Tags: ANTIOXIDANT DEFENCES; INFLAMMATION

What it is: A compound formed from substances in broccoli and other cruciferous vegetables. Supplements supply sulforaphane or a precursor with the enzyme needed to produce it.

Why consider it: Sulforaphane activates the cell's own defence programmes, including pathways involved in making glutathione. This could help cells handle chemical stress as they recover. A small human study measured increased glutathione in the brain.

My assessment: Worth considering for ongoing antioxidant-defence support. Its ability to influence a protective brain chemical in people gives the rationale more substance than a laboratory mechanism alone.

Timing and relevance to the session: The human study involved daily use for a week. Around-session use raises a separate concern: sulforaphane worsened MDMA-related toxicity in liver-cell experiments.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: The MDMA liver-cell experiment gives a specific reason to avoid assuming that around-session use is protective. People with liver disease or complex medication regimens need individual review. The cell result is an interaction signal, not a demonstrated human contraindication. [29]

Research details

What researchers found: Nine healthy adults took sulforaphane for a week. Brain scans measuring chemistry found increased glutathione in the thalamus, a brain region involved in relaying information. The study had no placebo group, so it is an early finding.

Actual dose, route and timing: Nine healthy adults received 100 micromoles of sulforaphane orally each day for seven days. The preparation was a standardized broccoli-sprout extract. A label giving only the weight of broccoli powder does not identify the same sulforaphane exposure.

Study limitations and human translation: The pilot supplies an actual human preparation and dose. It does not establish an MDMA-compatible dose, stopping interval or benefit from adding it to NAC.

What would change its place: A larger controlled brain study and evidence of added recovery benefit would strengthen its placement.

Research sources: [11]

Recovery between sessions

Astaxanthin

Additional candidates with some supporting evidence

Tags: ANTIOXIDANT DEFENCES; INFLAMMATION; NERVE CONNECTIONS

What it is: A red-orange pigment made by certain algae and found in seafood. Supplements generally use an algal source. It is studied for antioxidant and inflammation-related effects.

Why consider it: Limiting oxidative and inflammatory stress could help preserve the conditions needed for nerve repair. Astaxanthin has also been studied after brain and nerve injuries, with findings involving movement, cognition and nerve growth.

My assessment: An additional recovery candidate with a useful combination of antioxidant biology and treatment-after-injury animal findings.

Timing and relevance to the session: Its role here is sustained support during recovery. The cited stroke experiment treated animals over 28 days after injury.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Research details

What researchers found: Oral treatment after an experimental stroke in mice improved movement and measures of nerve-branch growth. Other brain-injury experiments reported improvements in memory or proteins associated with nerve connections. These are useful findings from different kinds of injury.

Actual dose, route and timing: Stroke-model mice received astaxanthin 30 mg/kg by stomach tube twice daily for 28 days after the operation. The abstract does not state the exact delay to the first dose. The separate traumatic-brain-injury study also used stomach-tube delivery; its full dose schedule was not verified in the accessible abstract.

Study limitations and human translation: No validated human equivalent exists for the mouse recovery regimen. Commercial supplement doses cannot be treated as equivalent simply because both products contain astaxanthin.

What would change its place: A study of oral astaxanthin started after MDMA, with improved serotonin function or behaviour, would strengthen its position.

Research sources: [16] [17]

Recovery between sessions

Nicotinamide riboside (NR)

Additional candidates with some supporting evidence

Tags: ENERGY; CELL UPKEEP

What it is: A vitamin-B3-related molecule the body uses to make NAD, a coenzyme involved in energy metabolism and cellular maintenance.

Why consider it: Energy production and cellular maintenance depend on NAD. NR could support those processes by increasing its availability. A human study found that oral NR increased brain NAD in many participants.

My assessment: An additional energy-support candidate with direct evidence that oral use can affect the proposed biochemical pathway in the human brain.

Timing and relevance to the session: The brain study used daily supplementation for 30 days. This supports considering NR as an ongoing preparation or recovery candidate.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Research details

What researchers found: The NADPARK trial in people with Parkinson's disease found increased brain NAD after NR, with substantial differences between participants. This supports its biological rationale, while leaving the recovery question open.

Actual dose, route and timing: NADPARK: 30 people with newly diagnosed Parkinson’s disease were assigned to oral NR 1,000 mg/day or placebo for 30 days. Brain NAD responses differed substantially between participants.

Study limitations and human translation: An actual human brain-biomarker dose is available for NR. It is not a validated MDMA regimen and does not transfer to NMN or nicotinamide.

What would change its place: Evidence that NR corrects a measured post-MDMA problem and improves function would move it higher.

Research sources: [12]

Recovery between sessions

Urolithin A

Additional candidates with some supporting evidence

Tags: CELL UPKEEP; LEARNING

What it is: A compound some gut bacteria produce from plant substances found in foods such as pomegranates and walnuts. Supplements provide the compound directly.

Why consider it: Recovery may require clearing damaged mitochondria and maintaining a healthier population of energy-producing structures. Urolithin A is studied for this cellular upkeep, including mitophagy, the removal of damaged mitochondria.

My assessment: An exploratory addition focused on mitochondrial upkeep. Animal brain research connects its effects on mitochondrial pathways with improved nerve-cell function and behaviour.

Timing and relevance to the session: The relevant animal work used repeated intake over weeks, fitting an ongoing recovery rationale.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Research details

What researchers found: In rodent anxiety research, treatment improved selected nerve-connection measures and patterns of gene activity linked to mitochondria. Human mitochondrial research is mainly outside the brain, so the brain-related reason for inclusion remains mostly experimental.

Actual dose, route and timing: In high-anxiety rodents, urolithin A was mixed into food at 25 mg/kg/day. The researchers assessed behaviour during the first four weeks; the full duration of every experimental arm was not verified in the accessible primary text. There was no MDMA exposure.

Study limitations and human translation: There is no established human brain or MDMA dose corresponding to this animal result. Human muscle studies address a different tissue and outcome.

What would change its place: Human brain measurements plus a positive study begun after a relevant injury would strengthen its placement.

Research sources: [13]

Recovery between sessions

CoQ10 / ubiquinol

Additional candidates with some supporting evidence

Tags: ENERGY; ANTIOXIDANT DEFENCES

What it is: Coenzyme Q10 helps move electrons through the machinery that produces mitochondrial energy. Ubiquinol and ubiquinone are two forms of CoQ10 sold as supplements.

Why consider it: CoQ10 participates directly in energy production and antioxidant defence. Those roles make it relevant when considering mitochondrial strain after MDMA. An MDMA animal experiment found less serotonin loss when CoQ10 was delivered into the brain.

My assessment: An additional candidate for energy and antioxidant support. The direct MDMA finding strengthens the biological reason to consider this pathway.

Timing and relevance to the session: The oral supplement rationale concerns supporting energy metabolism over time. Community guides also discuss taking it before a session.

Community and practice reports: RollSafe proposes 200-600 mg of CoQ10 about seven hours before MDMA. This supplies an identifiable practice recommendation, but it does not translate the local brain-perfusion experiment into demonstrated oral protection. The reviewed material supplies little attributable outcome evidence for CoQ10 alone. [73]

Who should avoid it or seek review: Review before use with warfarin, insulin or cancer treatment. CoQ10 can interact with these treatments, and the consequence depends on the drug and clinical situation. [90]

Research details

What researchers found: Locally delivered ubiquinone reduced serotonin depletion in rats exposed to MDMA. This is a direct link between the pathway and MDMA, but the delivery method bypassed the steps a swallowed supplement must take.

Actual dose, route and timing: Darvesh and Gudelsky delivered ubiquinone at 100 micromolar directly into brain tissue by local perfusion while rats underwent systemic MDMA exposure (four abdominal injections of 10 mg/kg). The exact perfusion start and stop times were not verified from the accessible primary abstract.

Study limitations and human translation: The MDMA experiment used local brain perfusion, bypassing oral absorption and entry into the brain. Its 100-micromolar concentration cannot be converted directly into a capsule dose. The practical question is whether oral supplementation achieves enough brain exposure for a useful effect.

What would change its place: An oral study showing a relevant brain effect and recovery benefit would make it more persuasive.

Research sources: [14]

Recovery between sessions

ALA

Weaker reasons to prioritize

Tags: ANTIOXIDANT DEFENCES; ENERGY

What it is: Alpha-lipoic acid, a naturally occurring compound involved in energy-producing enzyme reactions and studied for antioxidant effects. Here ALA refers to alpha-lipoic acid.

Why consider it: ALA could help limit oxidative damage while MDMA is active. A direct rat study found that pretreatment preserved later serotonin and transporter measurements, giving it a specific protection rationale.

My assessment: One of the more compelling candidates for protection around exposure. Its strongest evidence concerns preventing changes as they develop, which explains its lower priority for months-long recovery use.

Timing and relevance to the session: The rat study supplied repeated ALA doses over two days, with pretreatment 30 minutes before MDMA. This makes advance availability central to the rationale.

Community and practice reports: ALA is a recurring choice in RollSafe-linked Reddit discussions and longstanding Bluelight posts. RollSafe proposes oral 300 mg with MDMA and repeated doses every one to two hours. These are circulating schedules, distinct from the injected rat study. Claims of complete protection go beyond what human evidence shows. [73] [75] [77]

Who should avoid it or seek review: Avoid if ALA previously caused insulin autoimmune syndrome or unexplained hypoglycaemia. It can trigger serious low blood sugar in susceptible people; recurrent hypoglycaemia or glucose-lowering treatment warrants review. Liver disease alone cannot be evaluated from the protective rat experiment. [88]

Research details

What researchers found: ALA given around MDMA exposure prevented later losses in serotonin and transporter measurements in rats. That explains its stronger position in the protection comparison.

Actual dose, route and timing: Aguirre et al.: ALA 100 mg/kg by abdominal injection twice daily for two consecutive days, with the reported pretreatment 30 minutes before a single abdominal MDMA injection of 20 mg/kg. Serotonin-related outcomes were measured one week later. ALCAR was not part of this experiment.

Study limitations and human translation: The injected rat regimen has no validated oral human equivalent for MDMA protection. Published practice schedules are extrapolations and are described in the community note below.

What would change its place: A study starting ALA after an established MDMA-related change and showing recovery would raise its position here.

Research sources: [15]

Recovery between sessions

Lion’s mane

Weaker reasons to prioritize

Tags: LEARNING; NERVE CONNECTIONS

What it is: Hericium erinaceus, an edible mushroom sold as powders and extracts. Researchers also study individual compounds isolated from it.

Why consider it: Some lion’s mane compounds influence growth-related signalling and branching in nerve cells. This gives a plausible reason to consider it for maintaining and developing nerve connections during recovery.

My assessment: A lower-priority option with interesting nerve-growth and memory findings. Its appeal is the potential to support the biology of nerve connections.

Timing and relevance to the session: The cited mouse work used oral preparations over 31 days, fitting an ongoing-use rationale.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Avoid if a prior lion’s mane product caused an allergic or respiratory reaction; known mushroom allergy warrants review. A serious respiratory reaction has been reported in a case report. Interaction data for concentrated extracts remain limited. [92]

Research details

What researchers found: Specific extracts and compounds promoted nerve-branch growth in laboratory work, with memory findings in mice. The findings depend on what was actually tested, so they do not apply equally to every product sold as lion's mane.

Actual dose, route and timing: In the cited mouse experiment, oral mushroom extract was tested at 100 or 250 mg/kg and purified hericene A at 5 mg/kg, over 31 days. These were different preparations, tested without MDMA. Cell-culture experiments tested isolated compounds directly on neurons.

Study limitations and human translation: The experiments used particular extracts and isolated compounds. Commercial powders vary in their composition, so the preparation matters. A human post-MDMA amount has not been established.

What would change its place: A consistent oral preparation showing relevant brain effects and recovery would make the case stronger.

Research sources: [18]

Recovery between sessions

Citicoline alone

Weaker reasons to prioritize

Tags: MEMBRANES

What it is: CDP-choline, a compound involved in making phosphatidylcholine, a major membrane fat. Oral citicoline provides choline and cytidine-related material.

Why consider it: Supplying membrane-building ingredients could support maintenance of nerve cells and their connections. Citicoline offers one way of supplying material to that pathway.

My assessment: A lower-priority membrane-support option. Its plausible mechanism is tempered by a large human brain-injury trial that found no overall improvement in recovery.

Timing and relevance to the session: The human injury trial used daily oral or feeding-tube treatment for 90 days, so its relevance is to sustained recovery support.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Research details

What researchers found: The COBRIT trial tested citicoline after traumatic brain injury and found no improvement in its main measures of thinking and function compared with placebo. It is included because a disappointing human result deserves weight alongside an appealing biological explanation.

Actual dose, route and timing: COBRIT randomized 1,213 people with traumatic brain injury to citicoline 2,000 mg/day orally or through a feeding tube, or placebo, for 90 days. Treatment was an injury intervention, not MDMA preparation.

Study limitations and human translation: The human dose and duration were directly tested, with no overall benefit in that injury population. They do not establish a post-MDMA dose.

What would change its place: A convincing positive study in a more relevant setting, or a demonstrated added benefit in a combination, would change its placement.

Research sources: [19]

Recovery between sessions

Curcumin

Weaker reasons to prioritize

Tags: ANTIOXIDANT DEFENCES; INFLAMMATION

What it is: A yellow plant compound found in turmeric, sold in concentrated extracts and formulations designed to improve absorption.

Why consider it: Curcumin could help manage inflammation and oxidative stress that interfere with recovery. A rat study involving MDMA found partial improvement in a memory task, giving it a direct reason for inclusion.

My assessment: A lower-priority recovery candidate with a direct MDMA animal finding. Its potential lies in reducing harmful inflammatory and chemical stress.

Timing and relevance to the session: The MDMA experiment used a week of curcumin treatment. Product formulation and drug-processing interactions matter when considering use near a session.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Avoid medicinal turmeric/curcumin products with current or previous liver problems, consistent with the TGA safety alert. Concentrated and enhanced-absorption products warrant particular attention. The human drug-metabolism study also supports medication review. [89] [20]

Research details

What researchers found: The MDMA study reported better spatial-memory performance and changes in a marker of supporting brain cells. The accessible methods do not make the exact overlap between MDMA and curcumin treatment clear.

Actual dose, route and timing: Nazari et al.: male rats received MDMA 15 mg/kg twice daily for four days; the curcumin group received 200 mg/kg daily for one week. The accessible publisher abstract calls this co-administration but does not specify the exact overlap, first-dose delay or administration route. Those details remain unverified.

Study limitations and human translation: The animal amount has no established oral human equivalent for MDMA recovery. Turmeric powder, curcumin and absorption-enhanced curcumin are not interchangeable preparations.

What would change its place: A clearly timed study using a well-defined preparation would make its recovery rationale easier to judge.

Research sources: [66] [20]

Recovery between sessions

Spermidine

Weaker reasons to prioritize

Tags: CELL UPKEEP

What it is: A naturally occurring polyamine, a small molecule involved in cell growth and maintenance. It occurs in foods and concentrated supplements, including wheat-germ extracts.

Why consider it: Cells recover partly by clearing and replacing damaged components. Spermidine is studied for effects on autophagy, the recycling process involved in this upkeep.

My assessment: A lower-priority cellular-upkeep candidate. A promising small memory study was followed by a larger trial with a null primary result, reducing the reason to prioritize it.

Timing and relevance to the session: Human studies used daily intake over three or twelve months, fitting a sustained cellular-maintenance rationale.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Avoid a wheat-derived product if its ingredients conflict with a wheat allergy; people with coeliac disease need verified gluten-free status. The memory trials do not establish safety in serious illness or with MDMA. [51]

Research details

What researchers found: A small trial reported improved performance on a memory task. The larger SmartAge trial did not improve its main memory outcome. The larger result lowers confidence that the proposed upkeep mechanism reliably produces a useful benefit.

Actual dose, route and timing: The three-month pilot used an oral spermidine-rich plant extract supplying approximately 1.2 mg/day spermidine in 30 older adults. The larger SmartAge trial used 0.9 mg/day in a wheat-germ extract for 12 months in 100 adults with subjective cognitive decline.

Study limitations and human translation: Human doses are available for the tested extracts and memory outcomes. A higher dose is not established as the solution to the larger trial's null result.

What would change its place: A repeatable benefit in a relevant population, linked to a measured brain effect, would strengthen the case.

Research sources: [21] [51]

Recovery between sessions

NMN

Weaker reasons to prioritize

Tags: ENERGY; CELL UPKEEP

What it is: Nicotinamide mononucleotide, a molecule used in making NAD, which participates in cellular energy production and maintenance.

Why consider it: NMN offers another way to supply material for NAD production. This gives it an energy and cellular-maintenance rationale when considering recovery.

My assessment: A lower-priority NAD-support option. NR currently has the more directly relevant human brain finding in the studies reviewed here.

Timing and relevance to the session: Its proposed role is ongoing NAD support during preparation and recovery.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Research details

What researchers found: The human brain-NAD result cited in this guide tested NR. It therefore supports NR directly, while NMN's place here rests on a related biological idea rather than that same brain finding.

Actual dose, route and timing: No NMN regimen supporting human post-MDMA recovery was identified in the reviewed sources. The 1,000 mg/day for 30 days brain-NAD study cited nearby tested NR, a different compound, so its dose is not assigned to NMN.

Study limitations and human translation: There is no established NMN dose for this purpose. The absence of a number here reflects missing relevant evidence, not an omitted recommendation.

What would change its place: Its own human brain-NAD findings and relevant recovery outcomes would raise its priority.

Research sources: [12]

Protection around the session

ALA

Strongest overall rationale for considering

Tags: ANTIOXIDANT DEFENCES; ENERGY

What it is: Alpha-lipoic acid, a naturally occurring compound involved in energy-producing enzyme reactions and studied for antioxidant effects. Here ALA refers to alpha-lipoic acid.

Why consider it: ALA could help limit oxidative damage while MDMA is active. A direct rat study found that pretreatment preserved later serotonin and transporter measurements, giving it a specific protection rationale.

My assessment: One of the more compelling candidates for protection around exposure. Its strongest evidence concerns preventing changes as they develop, which explains its lower priority for months-long recovery use.

Timing and relevance to the session: The rat study supplied repeated ALA doses over two days, with pretreatment 30 minutes before MDMA. This makes advance availability central to the rationale.

Community and practice reports: ALA is a recurring choice in RollSafe-linked Reddit discussions and longstanding Bluelight posts. RollSafe proposes oral 300 mg with MDMA and repeated doses every one to two hours. These are circulating schedules, distinct from the injected rat study. Claims of complete protection go beyond what human evidence shows. [73] [75] [77]

Who should avoid it or seek review: Avoid if ALA previously caused insulin autoimmune syndrome or unexplained hypoglycaemia. It can trigger serious low blood sugar in susceptible people; recurrent hypoglycaemia or glucose-lowering treatment warrants review. Liver disease alone cannot be evaluated from the protective rat experiment. [88]

Research details

What researchers found: Rats given ALA before MDMA avoided later drops in serotonin and transporter measurements. Their body temperature still rose, so the protective finding was not simply explained by preventing overheating.

Actual dose, route and timing: Aguirre et al.: ALA 100 mg/kg by abdominal injection twice daily for two consecutive days, with the reported pretreatment 30 minutes before a single abdominal MDMA injection of 20 mg/kg. Serotonin-related outcomes were measured one week later. ALCAR was not part of this experiment.

Study limitations and human translation: The injected rat regimen has no validated oral human equivalent for MDMA protection. Published practice schedules are extrapolations and are described in the community note below.

What would change its place: A human study measuring both protection and the quality of the session would strengthen the case.

Research sources: [15]

Protection around the session

ALCAR

Strongest overall rationale for considering

Tags: ENERGY; ANTIOXIDANT DEFENCES

What it is: Acetyl-L-carnitine, a form of carnitine involved in cellular metabolism and studied for its effects on mitochondria, the structures that produce much of a cell's energy.

Why consider it: MDMA animal studies identify mitochondrial stress as one possible source of harm. ALCAR could help maintain that energy-producing machinery and reduce associated chemical damage. It preserved mitochondrial and serotonin-related measures in a direct MDMA rat experiment.

My assessment: A strong candidate for mitochondrial support, with direct MDMA protection findings and separate evidence of improved recovery after spinal injury.

Timing and relevance to the session: Its clearest MDMA evidence concerns being present before exposure: one injected dose 30 minutes beforehand. Its separate injury study also gives a reason to consider support early in recovery.

Community and practice reports: RollSafe and Psychedelic Support propose oral 500 mg three hours before, one hour after and five hours after MDMA. A Bluelight contributor described preserved MDMA effects while using ALCAR. This is limited subjective reassurance; the sources do not measure subsequent nerve repair or therapy outcomes. [73] [74] [75]

Who should avoid it or seek review: A seizure disorder or severe kidney disease warrants review: broader carnitine safety guidance flags these groups. Those cautions do not quantify ALCAR-specific risk with MDMA. Liver disease requires assessment of the actual diagnosis and product; this MDMA animal study cannot establish suitability. [83]

Research details

What researchers found: Rats given ALCAR before MDMA had better mitochondrial and serotonin-related measurements afterwards. This connects the energy rationale to an actual MDMA experiment.

Actual dose, route and timing: Rats received a single pretreatment of ALCAR 100 mg/kg by abdominal injection 30 minutes before four MDMA injections of 10 mg/kg. Brain outcomes were assessed two weeks later. This was an ALCAR experiment, not an ALA-plus-ALCAR stack.

Study limitations and human translation: Both key experiments used injected animal doses. Their results do not establish an effective oral human amount or a three-month recovery schedule.

What would change its place: A human study testing protection and session effects together would provide the most useful next evidence.

Research sources: [5]

Protection around the session

NAC

Additional candidates with some supporting evidence

Tags: ANTIOXIDANT DEFENCES; SEROTONIN

What it is: N-acetylcysteine, a form of the amino acid cysteine. It supplies an ingredient for making glutathione, a major antioxidant inside cells, and also influences regulation of the brain messenger glutamate.

Why consider it: Cells use glutathione to manage potentially damaging reactive molecules. Supplying cysteine through NAC could support those defences during recovery. Direct MDMA animal findings and a human brain-glutathione finding give this rationale several lines of support.

My assessment: A strong candidate for antioxidant support between sessions. It also attracts substantial community interest for recovering a diminished MDMA response, with mixed reported results.

Timing and relevance to the session: Between-session use and use close to MDMA deserve separate consideration. Reports of weaker effects with nearby dosing make the proposed pause relevant; community suggestions range from roughly 30 hours to two weeks.

Community and practice reports: NAC is repeatedly discussed for recovery and loss of magic. Reports include improvement, no change and blunting. Suggested pauses range from about 30 hours to two weeks, with one to two weeks a recognizable recommendation. A practitioner guide instead proposes NAC shortly before MDMA. This disagreement leaves both the interaction and the correct pause unresolved. [68] [69] [76] [74]

Who should avoid it or seek review: Avoid after an allergic reaction to acetylcysteine. Oral product information calls for caution with asthma and a history of peptic ulcer. Nitroglycerin effects can be enhanced, increasing the risk of low blood pressure and headache. [87]

Research details

What researchers found: Rat studies reported less memory impairment and fewer signs of cell injury when NAC was used with experimental MDMA exposure.

Actual dose, route and timing: 2013 rat study: NAC 100 mg/kg by abdominal injection, with twice-daily MDMA injections of 5, 10 or 20 mg/kg in separate groups. The abstract calls this pretreatment; the exact lead time and total days were not confirmed from the accessible primary text. The 2015 experiment used NAC 100 mg/kg and MDMA 10 mg/kg by abdominal injection; its exact sequence remains unverified.

Study limitations and human translation: The human brain-glutathione study involved schizophrenia-spectrum illness and found no improvement in symptoms or cognition. The MDMA work was in animals. A controlled study has not established whether NAC blunts the human MDMA experience or what pause would prevent that.

What would change its place: A controlled comparison of different timing windows and session outcomes would resolve the key uncertainty.

Research sources: [7] [8]

Protection around the session

Vitamin C

Additional candidates with some supporting evidence

Tags: ANTIOXIDANT DEFENCES; ENERGY

What it is: Ascorbic acid, an essential vitamin obtained from food or supplements. It contributes to antioxidant defence and several processes involved in maintaining body tissues.

Why consider it: Vitamin C could help limit oxidative stress following MDMA exposure. It has a direct animal finding in which treatment after MDMA preserved selected ATP and serotonin measurements.

My assessment: A plausible early-recovery candidate. The positive group received it five hours after MDMA, showing that intervention after exposure can still influence developing harm in that model.

Timing and relevance to the session: Timing is central to this study: the five-hour treatment helped selected measures, whereas treatment 30 minutes before or three hours after did not produce the same result.

Community and practice reports: Vitamin C appears in common RollSafe-linked supplement schedules. RollSafe proposes 500 mg every three hours beginning three hours before MDMA and continuing through roughly six to seven hours afterwards. This is community advice; it differs from the specific rat timing that protected ATP and serotonin measures. [73] [77]

Who should avoid it or seek review: Avoid high-dose self-supplementation with hereditary haemochromatosis or other iron overload. Kidney disease, hyperoxaluria or a history of relevant kidney stones warrants review before high doses. These concerns differ from ordinary food intake. [86]

Research details

What researchers found: The five-hour-after group preserved selected ATP and serotonin measures. The 30-minute-before and three-hour-after groups did not preserve those same measures. All three timing groups reduced a marker of the brain’s glial response.

Actual dose, route and timing: In the cited rat experiment, vitamin C 250 mg/kg was given in separate groups either 30 minutes before, three hours after, or five hours after MDMA 20 mg/kg. The accessible abstract does not specify the administration routes or repeated-dose details.

Study limitations and human translation: No validated human equivalent exists for the 250 mg/kg animal amount or its five-hour window. The oral schedule in the community note below is a practice proposal.

What would change its place: Human studies checking timing, protection and session effects would clarify its place.

Research sources: [23]

Protection around the session

Resveratrol

Possible effect on the MDMA experience

Tags: ANTIOXIDANT DEFENCES; SEROTONIN

What it is: A plant polyphenol found in grapes and some other plants, available as a concentrated supplement.

Why consider it: Resveratrol deserves consideration because a rat MDMA study found better later serotonin-transporter measurements. Its effects at the transporter provide a possible explanation for that protection.

My assessment: A protection candidate with an important session-related question: interacting with SERT could help explain protection while also changing MDMA's serotonin release.

Timing and relevance to the session: The authors considered the transporter interaction part of a possible protective mechanism. Our inference is that it could also change the acute experience; the direction of that change remains unresolved.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Review use with prescription medicines whose levels depend on drug-metabolizing enzymes. A human study at 1 g/day changed enzyme activity. That raises an interaction concern but does not quantify the effect on MDMA exposure. [91]

Research details

What researchers found: Shih et al. reported less loss of SERT availability in selected brain regions and improvement in a stress-related swimming test. Separate brain scans showed reduced tracer binding, interpreted as resveratrol occupying some serotonin-transporter sites. Occupancy increased with dose.

Actual dose, route and timing: Rats received resveratrol 20 mg/kg by abdominal injection together with MDMA 10 mg/kg injected under the skin, twice daily for four consecutive days. Later brain imaging was performed two weeks after co-treatment. This was repeated co-dosing, not a single oral supplement taken before therapy.

Study limitations and human translation: There is no validated oral human equivalent of the repeated injected regimen. Oral absorption and metabolites would have to be considered, not just body weight.

What would change its place: Research separating protection from changes to the acute MDMA effect would change how it is ranked.

Research sources: [22]

Protection around the session

Glutathione

Weaker reasons to prioritize

Tags: ANTIOXIDANT DEFENCES; INFLAMMATION

What it is: An antioxidant made inside cells from three amino acids: glutamate, cysteine and glycine. Oral supplements supply the molecule itself; NAC supplies one of its building materials.

Why consider it: Glutathione helps neutralize reactive molecules and process some damaging compounds. Supporting that defence could reduce chemical stress during MDMA exposure and early recovery.

My assessment: A lower-priority protection candidate with a direct oral MDMA animal study showing partial biochemical protection.

Timing and relevance to the session: The study used repeated oral co-treatment over 56 days. The research rationale concerns antioxidant support while exposure is occurring.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Research details

What researchers found: A 2026 rat study found partial protection during repeated MDMA exposure. The effect was weaker at higher exposure.

Actual dose, route and timing: Rats received oral glutathione 15 mg/kg alongside oral MDMA at 5 or 15 mg/kg across a 56-day treatment period. The abstract does not specify within-day spacing or enough detail to define a single-session schedule.

Study limitations and human translation: No validated human equivalent exists for this regimen. It also does not establish that liposomal glutathione is superior to other forms or NAC.

What would change its place: Research using a more relevant exposure pattern and showing an effect in the human brain would strengthen its placement.

Research sources: [24]

Protection around the session

Creatine

Preparation that carries into the session

Tags: ENERGY; NERVE CONNECTIONS

What it is: A naturally occurring compound stored in cells partly as phosphocreatine, a reserve that helps regenerate ATP, the energy cells use. Supplements usually contain creatine monohydrate.

Why consider it: Brain cells need energy to communicate, maintain their structures and repair damage. Creatine could help by increasing the reserve available when energy demand rises or supply is strained.

My assessment: A strong candidate for preparation and recovery because oral use can raise brain creatine, and animal injury research links improved energy availability with nerve regrowth.

Timing and relevance to the session: The rationale is to build reserves over time so they are available during the session and subsequent recovery. The human brain study used four weeks of supplementation.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Kidney disease or unexplained kidney-test abnormalities call for an individual review before use. Healthy-user safety data do not resolve that situation. [93]

Research details

What researchers found: Dechent et al. measured an average 8.7% increase in total brain creatine in six healthy volunteers. Han et al. found some spinal nerve regrowth with creatine in ordinary mice and additional regrowth in mice with a genetic change that improved mitochondrial movement. Hellem et al. observed increased brain phosphocreatine and improved mood in an uncontrolled stimulant-use sample.

Actual dose, route and timing: Human brain study: creatine monohydrate, 5 g by mouth four times daily for four weeks. Hellem pilot: 5 g/day orally for eight weeks. Mouse spinal-injury experiment: 2 g/kg by stomach tube twice daily, continued for up to eight weeks after injury; the exact delay to the first dose was not confirmed in the accessible methods.

Study limitations and human translation: Actual human doses exist for increasing brain creatine. Neither those doses nor the mouse regimen establish an amount for MDMA protection or recovery.

What would change its place: A study starting creatine after MDMA and measuring improved nerve function or later-session outcomes would strengthen its position.

Research sources: [1] [2] [30]

Protection around the session

DHA / omega-3

Preparation that carries into the session

Tags: MEMBRANES; NERVE CONNECTIONS; LEARNING

What it is: DHA, docosahexaenoic acid, is an omega-3 fat that forms part of nerve-cell membranes. Oral sources include fish oil and algal oil. Fish oil commonly also contains EPA, another omega-3 fat.

Why consider it: Maintaining and rebuilding nerve branches and their connections requires membrane material. DHA supplies one of those ingredients. It is worth considering for ongoing recovery support because it contributes to the structures through which brain cells communicate.

My assessment: A strong candidate for membrane and nerve-connection support. In a spinal-injury experiment, DHA encouraged new branching of serotonin-containing fibres, adding a specific repair-related reason to its nutritional role.

Timing and relevance to the session: The rationale is regular intake during the interval between sessions, supplying membrane material over time. DHA incorporated into membranes remains relevant during the next session too.

Community and practice reports: No clear supplement-specific pattern emerged from the MDMA community sources reviewed.

Who should avoid it or seek review: Review use with anticoagulants and a history of atrial fibrillation. Long-term high-dose omega-3 trials found a small increase in atrial fibrillation in cardiovascular-risk populations. Fish allergy requires checking the particular product and its source. [84]

Research details

What researchers found: DHA given soon after spinal injury in rats increased branching from surviving serotonin fibres and their contact with other nerve cells. Separate animal studies used nutrient mixtures containing DHA and found improved injury outcomes.

Actual dose, route and timing: Key rat study: one intravenous dose of DHA, 250 nmol/kg, 30 minutes after spinal-cord hemisection. A separate delayed-treatment arm at three weeks did not improve recovery. The multi-nutrient spinal-injury study used enriched feed for four or nine weeks; it tested a mixture, not an isolated oral DHA dose.

Study limitations and human translation: The strongest serotonin-fibre result used DHA injected into a vein shortly after spinal injury. Oral DHA supplies the same molecule through a different route, with different absorption and timing. The study therefore supports the repair rationale without establishing an effective oral amount or post-MDMA schedule. A fish-oil product's total oil weight differs from its DHA content.

What would change its place: A positive study of oral DHA started after a serotonin-related injury would strengthen the case.

Research sources: [3] [4]

Timing and possible interactions

The supplement entries point to these notes where they are relevant.

NAC and the suggested two-week pause

NAC use between sessions and pausing before MDMA recur across the reviewed forums. Reported pauses range from about 30 hours to two weeks. Reports include restored warmth, no improvement and weaker effects; a report in the therapy thread also describes strong effects despite taking NAC shortly beforehand. Clearance from blood alone cannot establish how long any brain effect lasts. The practical issue is an unresolved possible interaction with the experience; 14 days is a community precaution, not a validated threshold. The NAC community entry gives source-specific reports. [68] [69] [7] [9]

Curcumin, turmeric and drug processing

CYP2D6 is an enzyme involved in breaking down some drugs, including MDMA. A small human study found that turmeric changed how a test drug was processed through this pathway. That makes an interaction worth considering, but does not tell us how much a particular turmeric or curcumin product would change MDMA effects. The laboratory concentration-response finding should not be described as a proven dose-response in people. [20]

Sulforaphane and the liver-cell finding

In experiments using human liver-derived cells and rat liver cells, sulforaphane given with MDMA or a related breakdown product worsened reported cell toxicity and glutathione depletion. This is a direct laboratory concern, with the cell setting clearly identified. It does not measure the effect of a supplement in a person having MDMA therapy. The separate small study showing increased brain glutathione answers a different question. [29] [11]

The small EPA study in PTSD

A very small study gave EPA, one type of omega-3 fat, to people with established PTSD without a placebo comparison. Some participants worsened. This is included because it appeared in the supplied research, but its size and design give it little weight. It does not establish that DHA or all fish-oil products have the same effect. [27]

Timing and possible interactions

The supplement entries point to these notes where they are relevant.

NR, NMN and nicotinamide

These compounds are related to NAD but have different evidence. The human brain study used NR. In a separate MDMA rat experiment, nicotinamide worsened serotonin loss. That result cannot simply be assigned to NR or NMN, just as the NR brain finding cannot simply be assigned to NMN. The exact compound and way it was given matter. [12] [14]

Why lithium is outside the ranked list

Lithium appeared in the supplied material, but the evidence does not establish it as a recovery supplement. A published analysis of online reports found seizures among reports combining lithium with classic psychedelics. Those reports concerned other psychedelics and cannot give a reliable MDMA risk percentage. They are retained as a specific reason not to treat the proposed combination as established. [28]

Several supplements may target the same process

A supplement can carry several tags, and several supplements can share a tag. That does not mean their benefits add together. The comparison is meant to help narrow and understand the list; the combined set has not been tested as a recovery regimen.

Practice sources and community experience

This section records what identifiable guides, practitioners and forum contributors say. Amounts and schedules below are attributed reports, not instructions to combine them. Research doses and community doses are deliberately separate. Recurring or popular means visible across the reviewed discussions, not a measured consensus or success rate. The entries retain their research placement regardless of popularity.

Therapy manuals: what they contribute

The MAPS treatment manual describes preparation, the supported drug session and integration, including follow-up the next morning and ongoing contact. It supplies a therapy framework rather than evidence for a supplement stack. Open MDMA discusses ALA, vitamin C and ALCAR as animal antioxidant findings, explicitly says protective oral human amounts are unknown, and discusses pausing supplements on the session day as the author's general precaution. Its antioxidant-recovery time estimates are explicitly AI-generated extrapolations, so they cannot establish a recovery deadline. The supplied MDMA Solo manual suggests a multivitamin after the session and nutrition during it; it does not supply evidence for the candidates' repair rankings. These manuals differ in approach and should not be read as one clinical consensus. [72] [71] [78]

Practice and community reports

Attributed schedules and experiences; kept separate from research rankings

ALA and ALCAR: recurring community protection choices

ALA and ALCAR recur in RollSafe-linked Reddit discussions and longstanding Bluelight posts, making them recognizable community protection choices. RollSafe describes oral ALA 300 mg with MDMA, then every one to two hours through about seven hours, and oral ALCAR 500 mg three hours before, one hour after and five hours after MDMA. Psychedelic Support gives the same ALCAR timing but smaller ALA follow-up amounts of 100-150 mg. These are schedules proposed by those sources, not the injected rat protocols. A Bluelight contributor reported preserved MDMA effects while using ALCAR; other posts ask whether protection blunts the experience. This is limited reassurance about subjective compatibility, not a measured therapeutic result. Repeated links to the same schedule establish popularity more than independent confirmation. [73] [74] [75] [77]

NAC: common recovery discussion, disputed pause

NAC is one of the most repeatedly discussed recovery supplements in the reviewed Reddit and Bluelight material. The Reddit loss-of-magic thread describes roughly 600 mg/day for weeks or months and advises a pause of at least 30 hours. The therapy thread includes 600-1,200 mg/day reports and advice ranging from two or three days to 10 days or one to two weeks. Thus, the two-week pause is a recognizable community precaution, but it is not the only popular position. Contributors report restored effects, partial improvement, no change and occasional blunting. There is also a strong-session report despite taking NAC shortly beforehand, and an unsuccessful two-month trial reported on Bluelight. These reports make a subjective interaction worth investigating; they do not establish its probability or the correct pause. [68] [69] [76]

Practice and community reports

Attributed schedules and experiences; kept separate from research rankings

NAC: practitioner advice actually conflicts with forums

Psychedelic Support proposes 900 mg orally shortly before MDMA and again at the end of the session. That directly conflicts with the forum practice of withholding NAC beforehand. Neither approach has a controlled human comparison measuring protection and the quality of the therapeutic experience. The guide therefore records the disagreement instead of silently choosing one schedule as settled. [74] [68] [69]

Vitamin C and CoQ10: specific schedules circulate

RollSafe proposes oral vitamin C 500 mg every three hours, beginning about three hours before MDMA and ending six to seven hours afterwards. Its minimal schedule includes CoQ10 200-600 mg about seven hours before. Those amounts are community guidance. The selected vitamin C experiment found timing-dependent effects in rats; the CoQ10-related experiment delivered ubiquinone directly into the brain. Neither establishes these oral schedules. The page’s confident wording therefore deserves less weight than its clear documentation of what people recommend. [73] [23] [14]

Practice and community reports

Attributed schedules and experiences; kept separate from research rankings

Candidates with sparse community information

For creatine, DHA, uridine/choline, sulforaphane, astaxanthin, NR, urolithin A, lion’s mane, citicoline, curcumin, spermidine, NMN and glutathione, this review did not establish a comparably recurring, specific MDMA recovery schedule across the selected practice sources. Some appear in generic supplement lists or isolated comments. Their research entries stand on the biological and study-based reasons given there. A product’s popularity for exercise, longevity or general cognition is not itself a community consensus about MDMA recovery.

Research and practice source links

Click a full URL to open the source. Under each reference is a brief explanation of its role. Published titles are retained. Practice and community sources are labelled; their recommendations do not change the research rankings.

[1] Dechent P, Pouwels PJ, Wilken B, Hanefeld F, Frahm J. (1999). Increase of total creatine in human brain after oral supplementation of creatine-monohydrate. The American journal of physiology

https://pubmed.ncbi.nlm.nih.gov/10484486/

Oral creatine increased brain creatine in six healthy volunteers; this shows the supplement can affect its proposed target in the brain.

[2] Han Q, Xie Y, Ordaz JD, Huh AJ, Huang N, Wu W, et al. (2020). Restoring Cellular Energetics Promotes Axonal Regeneration and Functional Recovery after Spinal Cord Injury. Cell metabolism

https://pubmed.ncbi.nlm.nih.gov/32130884/

Mouse spinal-injury research linked energy supply to nerve regrowth; creatine also helped some regrowth in ordinary mice.

[3] Liu ZH, Yip PK, Adams L, Davies M, Lee JW, Michael GJ, et al. (2015). A Single Bolus of Docosahexaenoic Acid Promotes Neuroplastic Changes in the Innervation of Spinal Cord Interneurons and Motor Neurons and Improves Functional Recovery after Spinal Cord Injury. The Journal of neuroscience : the official journal of the Society for Neuroscience

https://pubmed.ncbi.nlm.nih.gov/26377463/

DHA injected soon after spinal injury increased branching of surviving serotonin fibres; starting three weeks later did not work in that experiment.

[4] Pallier PN, Poddighe L, Zbarsky V, Kostusiak M, Choudhury R, Hart T, et al. (2015). A nutrient combination designed to enhance synapse formation and function improves outcome in experimental spinal cord injury. Neurobiology of disease

https://pubmed.ncbi.nlm.nih.gov/26388399/

A nutrient mixture improved outcomes after spinal injury in animals; the result cannot tell us how much each ingredient contributed.

[5] Alves E, Binienda Z, Carvalho F, Alves CJ, Fernandes E, de Lourdes Bastos M, et al. (2009). Acetyl-L-carnitine provides effective in vivo neuroprotection over 3,4-methylenedioximethamphetamine-induced mitochondrial neurotoxicity in the adolescent rat brain. Neuroscience

https://pubmed.ncbi.nlm.nih.gov/19015003/

ALCAR given before MDMA protected energy-related and serotonin measurements in rats; this is the main reason for its protection ranking.

[6] Patel SP, Sullivan PG, Lyttle TS, Magnuson DS, Rabchevsky AG. (2012). Acetyl-L-carnitine treatment following spinal cord injury improves mitochondrial function correlated with remarkable tissue sparing and functional recovery. Neuroscience

https://pubmed.ncbi.nlm.nih.gov/22445934/

ALCAR begun soon after spinal injury preserved energy production and tissue and improved movement in animals; supports an indirect recovery rationale.

[7] Soleimani Asl S, Mousavizadeh K, Pourheydar B, Soleimani M, Rahbar E, Mehdizadeh M. (2013). Protective effects of N-acetylcysteine on 3, 4-methylenedioxymethamphetamine-induced neurotoxicity in male Sprague-Dawley rats. Metabolic brain disease

https://pubmed.ncbi.nlm.nih.gov/23975535/

NAC reduced selected memory problems and signs of brain-cell injury in rats receiving MDMA; supports investigation near exposure.

[8] Soleimani Asl S, Saifi B, Sakhaie A, Zargooshnia S, Mehdizadeh M. (2015). Attenuation of ecstasy-induced neurotoxicity by N-acetylcysteine. Metabolic brain disease

https://pubmed.ncbi.nlm.nih.gov/25096201/

NAC reduced selected memory and cell-injury effects in MDMA-exposed rats; it did not test regrowth of serotonin nerve endings long afterwards.

[9] Yang YS, Maddock RJ, Zhang H, Lee J, Hellemann G, Marder SR, et al. (2022). N-Acetylcysteine effects on glutathione and glutamate in schizophrenia: A preliminary MRS study. Psychiatry research. Neuroimaging

https://pubmed.ncbi.nlm.nih.gov/35839558/

NAC increased a protective chemical, glutathione, in one brain region in a schizophrenia-spectrum study; symptoms and thinking did not improve in this trial.

[10] Wurtman RJ, Ulus IH, Cansev M, Watkins CJ, Wang L, Marzloff G. (2006). Synaptic proteins and phospholipids are increased in gerbil brain by administering uridine plus docosahexaenoic acid orally. Brain research

https://pubmed.ncbi.nlm.nih.gov/16631143/

A combination of oral uridine, DHA and available choline increased membrane fats and connection-related proteins in gerbil brains.

[11] Sedlak TW, Nucifora LG, Koga M, Shaffer LS, Higgs C, Tanaka T, et al. (2018). Sulforaphane Augments Glutathione and Influences Brain Metabolites in Human Subjects: A Clinical Pilot Study. Molecular neuropsychiatry

https://pubmed.ncbi.nlm.nih.gov/29888232/

Nine healthy adults had a measured increase in brain glutathione after a week of sulforaphane; an early study with no placebo group.

[12] Brakedal B, Dölle C, Riemer F, Ma Y, Nido GS, Skeie GO, et al. (2022). The NADPARK study: A randomized phase I trial of nicotinamide riboside supplementation in Parkinson's disease. Cell metabolism

https://pubmed.ncbi.nlm.nih.gov/35235774/

Oral NR increased brain NAD, a molecule used in energy and maintenance, in people with Parkinson's disease; individual responses varied.

[13] Mallet D, Ülgen DH, Grosse J, Zanoletti O, Guillot de Suduiraut I, Monzel AS, et al. (2026). Urolithin A Abolishes High Anxiety and Rescues the Associated Mitochondria-Related Transcriptomic Signatures and Synaptic Function. Biological psychiatry

https://pubmed.ncbi.nlm.nih.gov/40752777/

Urolithin A improved selected anxiety, nerve-connection and mitochondrial-related findings in rodents; the human MDMA recovery link remains untested.

[14] Darvesh AS, Gudelsky GA. (2005). Evidence for a role of energy dysregulation in the MDMA-induced depletion of brain 5-HT. Brain research

https://pubmed.ncbi.nlm.nih.gov/16098955/

Coenzyme Q delivered directly into rat brains reduced MDMA-related serotonin loss, while nicotinamide given systemically worsened it; ingredient and delivery matter.

[15] Aguirre N, Barrionuevo M, Ramírez MJ, Del Río J, Lasheras B. (1999). Alpha-lipoic acid prevents 3,4-methylenedioxy-methamphetamine (MDMA)-induced neurotoxicity. Neuroreport

https://pubmed.ncbi.nlm.nih.gov/10619665/

ALA given around MDMA prevented later serotonin-related deficits in rats even though temperature still rose; supports the protection rationale.

[16] Wang YL, Zhu XL, Sun MH, Dang YK. (2019). Effects of astaxanthin onaxonal regeneration via cAMP/PKA signaling pathway in mice with focal cerebral infarction. European review for medical and pharmacological sciences

https://pubmed.ncbi.nlm.nih.gov/31389584/

Oral astaxanthin after experimental stroke in mice improved movement and nerve-growth measures; a recovery finding from a different injury.

[17] Ji X, Peng D, Zhang Y, Zhang J, Wang Y, Gao Y, et al. (2017). Astaxanthin improves cognitive performance in mice following mild traumatic brain injury. Brain research

https://pubmed.ncbi.nlm.nih.gov/28048972/

Astaxanthin after experimental brain injury improved memory and connection-related measures in mice; supports further recovery research.

[18] Martínez-Mármol R, Chai Y, Conroy JN, Khan Z, Hong SM, Kim SB, et al. (2023). Hericerin derivatives activates a pan-neurotrophic pathway in central hippocampal neurons converging to ERK1/2 signaling enhancing spatial memory. Journal of neurochemistry

https://pubmed.ncbi.nlm.nih.gov/36660878/

Specific lion's mane compounds promoted growth-related changes in nerve cells and memory findings in mice; commercial products may differ.

[19] Zafonte RD, Bagiella E, Ansel BM, Novack TA, Friedewald WT, Hesdorffer DC, et al. (2012). Effect of citicoline on functional and cognitive status among patients with traumatic brain injury: Citicoline Brain Injury Treatment Trial (COBRIT). JAMA

https://pubmed.ncbi.nlm.nih.gov/23168823/

The large COBRIT trial found no improvement in its main thinking or functional outcomes with citicoline after human brain injury.

[20] Al-Jenoobi FI, Al-Thukair AA, Alam MA, Abbas FA, Al-Mohizea AM, Alkharfy KM, et al. (2015). Effect of Curcuma longa on CYP2D6- and CYP3A4-mediated metabolism of dextromethorphan in human liver microsomes and healthy human subjects. European journal of drug metabolism and pharmacokinetics

https://pubmed.ncbi.nlm.nih.gov/24510399/

Turmeric changed how a test drug was processed by CYP2D6, a drug-processing enzyme, in a small human study; it did not measure an MDMA interaction.

[21] Wirth M, Benson G, Schwarz C, Köbe T, Grittner U, Schmitz D, et al. (2018). The effect of spermidine on memory performance in older adults at risk for dementia: A randomized controlled trial. Cortex; a journal devoted to the study of the nervous system and behavior

https://pubmed.ncbi.nlm.nih.gov/30388439/

A small spermidine trial reported improved performance on a memory task; the later, larger trial is important when weighing this result.

[22] Shih JH, Ma KH, Chen CF, Cheng CY, Pao LH, Weng SJ, et al. (2016). Evaluation of brain SERT occupancy by resveratrol against MDMA-induced neurobiological and behavioral changes in rats: A 4-[^{18}F]-ADAM/small-animal PET study. European neuropsychopharmacology : the journal of the European College of Neuropsychopharmacology

https://pubmed.ncbi.nlm.nih.gov/26612383/

Resveratrol given with MDMA preserved selected later rat findings but also occupied SERT, the serotonin transporter, raising a question about changing MDMA's action.

[23] Li SX, Sun AM, Wang X, Li J, Peng ZG, Kuang WH, et al. (2006). [A preliminary study on the mechanism of neurotoxicity of MDMA--oxidative stress harm]. Sichuan da xue xue bao. Yi xue ban = Journal of Sichuan University. Medical science edition

https://pubmed.ncbi.nlm.nih.gov/16608072/

Vitamin C within hours of MDMA protected selected energy and serotonin measurements in rats; evidence concerns early treatment.

[24] Akano OP, Olatinwo G, Hamed MA, Oluwole DT, Jegede AJ, Ajayi AF. (2026). Glutathione as a Potential Neuroprotectant Against MDMA-Induced Oxidative Stress, Neuroinflammation, and Apoptosis in the Rat Brain. Neurochemical research

https://pubmed.ncbi.nlm.nih.gov/42090092/

Glutathione partly protected rats during prolonged repeated MDMA exposure; the result does not establish a single-session product or schedule.

[25] Huff C, Bhide N, Schroering A, Yamamoto BK, Gudelsky GA. (2013). Effect of repeated exposure to MDMA on the function of the 5-HT transporter as assessed by synaptosomal 5-HT uptake. Brain research bulletin

https://pubmed.ncbi.nlm.nih.gov/23318273/

A rat experiment found temporary changes in serotonin uptake without lower stored serotonin, helping distinguish altered function from structural injury.

[26] Heifets BD, Salgado JS, Taylor MD, Hoerbelt P, Cardozo Pinto DF, Steinberg EE, et al. (2019). Distinct neural mechanisms for the prosocial and rewarding properties of MDMA. Science translational medicine

https://pubmed.ncbi.nlm.nih.gov/31826983/

Mouse experiments separated some mechanisms of MDMA's social and rewarding effects; they did not establish why a later human therapy session might feel different.

[27] Zeev K, Michael M, Ram K, Hagit C. (2005). Possible deleterious effects of adjunctive omega-3 fatty acids in post-traumatic stress disorder patients. Neuropsychiatric disease and treatment

https://pubmed.ncbi.nlm.nih.gov/18568065/

A very small EPA study in people with PTSD reported worsening in some participants; it provides a weak, specific observation rather than a conclusion about all omega-3s.

[28] Nayak SM, Gukasyan N, Barrett FS, Erowid E, Erowid F, Griffiths RR. (2021). Classic Psychedelic Coadministration with Lithium, but Not Lamotrigine, is Associated with Seizures: An Analysis of Online Psychedelic Experience Reports. Pharmacopsychiatry

https://pubmed.ncbi.nlm.nih.gov/34348413/

Selected online reports linked lithium plus classic psychedelics with seizures; the study cannot provide a reliable MDMA risk percentage.

[29] Antolino-Lobo I, Meulenbelt J, Molendijk J, Nijmeijer SM, Scherpenisse P, van den Berg M, et al. (2011). Induction of glutathione synthesis and conjugation by 3,4-methylenedioxymethamphetamine (MDMA) and 3,4-dihydroxymethamphetamine (HHMA) in human and rat liver cells, including the protective role of some antioxidants. Toxicology

https://pubmed.ncbi.nlm.nih.gov/21871945/

In human liver-derived cells and rat liver cells, sulforaphane co-exposure worsened reported MDMA-related toxicity and glutathione loss; this is a laboratory liver-cell finding.

[30] Hellem TL, Sung YH, Shi XF, Pett MA, Latendresse G, Morgan J, et al. (2015). Creatine as a Novel Treatment for Depression in Females Using Methamphetamine: A Pilot Study. Journal of dual diagnosis

https://pubmed.ncbi.nlm.nih.gov/26457568/

Small uncontrolled study of creatine in women using methamphetamine measured brain-energy and mood outcomes; a different stimulant population, and the design cannot establish causation.

[31] Baudry A, Pietri M, Launay JM, Kellermann O, Schneider B. (2019). Multifaceted Regulations of the Serotonin Transporter: Impact on Antidepressant Response. Frontiers in neuroscience

https://pubmed.ncbi.nlm.nih.gov/30809118/

Explains how SERT, the protein that transports serotonin, is made, positioned and regulated; background for understanding changes in communication.

[32] Steiner JA, Carneiro AM, Blakely RD. (2008). Going with the flow: trafficking-dependent and -independent regulation of serotonin transport. Traffic (Copenhagen, Denmark)

https://pubmed.ncbi.nlm.nih.gov/18445122/

Explains how serotonin transport can change through movement of the transporter or changes in its activity; these are distinct from losing a nerve fibre.

[33] Bermingham DP, Blakely RD. (2016). Kinase-dependent Regulation of Monoamine Neurotransmitter Transporters. Pharmacological reviews

https://pubmed.ncbi.nlm.nih.gov/27591044/

Describes how regulatory proteins change the activity and location of transporters for serotonin and other brain messengers.

[34] Cooper A, Woulfe D, Kilic F. (2019). Post-translational modifications of serotonin transporter. Pharmacological research

https://pubmed.ncbi.nlm.nih.gov/30394319/

Describes chemical modifications that affect how SERT develops and works; background rather than a supplement-recovery experiment.

[35] Meinke C, Quinlan MA, Paffenroth KC, Harrison FE, Fenollar-Ferrer C, Katamish RM, et al. (2022). Serotonin Transporter Ala276 Mouse: Novel Model to Assess the Neurochemical and Behavioral Impact of Thr276 Phosphorylation In Vivo. Neurochemical research

https://pubmed.ncbi.nlm.nih.gov/33830406/

Mice with a change at one SERT regulatory site showed some behavioural differences despite similar basic serotonin and transporter measurements.

[36] Jayanthi LD, Ramamoorthy S. (2005). Regulation of monoamine transporters: influence of psychostimulants and therapeutic antidepressants. The AAPS journal

https://pubmed.ncbi.nlm.nih.gov/16353949/

Reviews how stimulants and antidepressants affect the regulation of proteins that transport brain messengers.

[37] Raefsky SM, Mattson MP. (2017). Adaptive responses of neuronal mitochondria to bioenergetic challenges: Roles in neuroplasticity and disease resistance. Free radical biology & medicine

https://pubmed.ncbi.nlm.nih.gov/27908782/

Explains how nerve-cell activity and energy demands can trigger adaptation and protective responses; general background for energy and learning.

[38] Kural I, M Mombeek LM, Wilson DM. (2026). Role of mitochondria in neuronal function and survival in the enteric and central nervous systems. Cellular and molecular life sciences : CMLS

https://pubmed.ncbi.nlm.nih.gov/41746390/

Reviews how mitochondria support nerve cells through energy production, calcium handling and other functions; explains why energy is only part of their role.

[39] Steffan D, Pezzini C, Esposito M, Franco-Romero A. (2025). Mitochondrial Aging in the CNS: Unravelling Implications for Neurological Health and Disease. Biomolecules

https://pubmed.ncbi.nlm.nih.gov/41008559/

Reviews aging-related changes in mitochondria and their maintenance; supports general biology rather than an MDMA-specific conclusion.

[40] Ricci V, Martinotti G, Mosca A, Maina G. (2026). Bioenergetic impairment in schizophrenia: role of mitochondrial signaling in synaptic dysfunction - a systematic review. Frontiers in cell and developmental biology

https://pubmed.ncbi.nlm.nih.gov/41836300/

Reviews energy-related brain findings specifically in schizophrenia; a different condition used only as background.

[41] Perez Lao EJ, Fagerli E, Ferrier F, Young JI, Perez-Pinzon MA. (2025). Regulatory Dynamics of Nrf2 With Sirtuins in the Brain: Exploring Cellular Metabolism, Synaptic Plasticity, and Defense Mechanisms. Journal of neurochemistry

https://pubmed.ncbi.nlm.nih.gov/40878635/

Reviews connections between cellular defence programmes and NAD-related maintenance systems; helps explain why supplement mechanisms can overlap.

[42] Gureev AP, Popov VN, Starkov AA. (2020). Crosstalk between the mTOR and Nrf2/ARE signaling pathways as a target in the improvement of long-term potentiation. Experimental neurology

https://pubmed.ncbi.nlm.nih.gov/32165256/

Reviews links between cellular recycling and changes in nerve connections; increasing one process does not automatically improve every outcome.

[43] Navarro E, Esteras N. (2024). Multitarget Effects of Nrf2 Signalling in the Brain: Common and Specific Functions in Different Cell Types. Antioxidants (Basel, Switzerland)

https://pubmed.ncbi.nlm.nih.gov/39765831/

Explains how NRF2, a regulator of protective genes, works across different brain-cell types.

[44] Goel F, Singh P, Rai SN, Yadav DK. (2025). Nrf2/Keap1 Signaling Axis in the Brain: Master Regulator of Oxidative Stress in Neurodegenerative and Psychiatric Disorders. Molecular neurobiology

https://pubmed.ncbi.nlm.nih.gov/41313410/

Reviews NRF2-related protective programmes and the challenges of turning them into treatments; background for antioxidant defences.

[45] Sambra V, Echeverria F, Valenzuela A, Chouinard-Watkins R, Valenzuela R. (2021). Docosahexaenoic and Arachidonic Acids as Neuroprotective Nutrients throughout the Life Cycle. Nutrients

https://pubmed.ncbi.nlm.nih.gov/33803760/

Reviews DHA and arachidonic acid as materials and signals used by brain cells; supports the membrane rationale.

[46] Su B, Ji YS, Sun XL, Liu XH, Chen ZY. (2014). Brain-derived neurotrophic factor (BDNF)-induced mitochondrial motility arrest and presynaptic docking contribute to BDNF-enhanced synaptic transmission. The Journal of biological chemistry

https://pubmed.ncbi.nlm.nih.gov/24302729/

In cultured nerve cells, BDNF caused mitochondria to stop near message-release sites, supporting communication without an immediate rise in total ATP.

[47] K Soman S, Swain M, Dagda RK. (2025). BDNF-TrkB Signaling in Mitochondria: Implications for Neurodegenerative Diseases. Molecular neurobiology

https://pubmed.ncbi.nlm.nih.gov/39030441/

Reviews how the growth-related protein BDNF connects with mitochondrial function and positioning; explains overlap between energy and learning.

[48] Zagrebelsky M, Korte M. (2014). Form follows function: BDNF and its involvement in sculpting the function and structure of synapses. Neuropharmacology

https://pubmed.ncbi.nlm.nih.gov/23752094/

Reviews BDNF in changes to nerve connections; a growth-related signal alone cannot establish useful therapeutic learning.

[49] Yousaf F, Kao S, Ishaq S, Lee SD. (2026). Molecular regulation of exercise training on hippocampal neuroprotection in post-traumatic brain injury: A systematic review. Experimental neurology

https://pubmed.ncbi.nlm.nih.gov/41360177/

Reviews exercise after traumatic brain injury in animals and reports energy, inflammation and learning-related findings; a different recovery setting.

[50] Razazpour F, Golkar A, Heidari M, Seyedi F, Hedayati-Moghadam M, Baghcheghi Y. (2026). Voluntary Exercise and Hippocampal Memory Enhancement: Decoding the Molecular Blueprint. Journal of molecular neuroscience : MN

https://pubmed.ncbi.nlm.nih.gov/42616196/

Reviews exercise and memory-related processes; general context rather than a test of MDMA recovery.

[51] Schwarz C, Benson GS, Horn N, Wurdack K, Grittner U, Schilling R, et al. (2022). Effects of Spermidine Supplementation on Cognition and Biomarkers in Older Adults With Subjective Cognitive Decline: A Randomized Clinical Trial. JAMA network open

https://pubmed.ncbi.nlm.nih.gov/35616942/

The larger SmartAge trial found no benefit on its main memory measure from spermidine; reduces confidence after the smaller positive trial.

[52] Agarwal N, Sung YH, Jensen JE, daCunha G, Harper D, Olson D, et al. (2010). Short-term administration of uridine increases brain membrane phospholipid precursors in healthy adults: a 31-phosphorus magnetic resonance spectroscopy study at 4T. Bipolar disorders

https://pubmed.ncbi.nlm.nih.gov/21176029/

Oral uridine increased brain chemicals used to make membranes in a small human study; it did not directly count new nerve connections.

[53] Svensson JE, Svanborg C, Plavén-Sigray P, Kaldo V, Halldin C, Schain M, et al. (2021). Serotonin transporter availability increases in patients recovering from a depressive episode. Translational psychiatry

https://pubmed.ncbi.nlm.nih.gov/33972499/

SERT scan measurements increased during recovery from depression after CBT; shows that the measure can change in another clinical setting.

[54] van de Blaak FL, Dumont GJH. (2022). Serotonin transporter availability, neurocognitive function and their correlation in abstinent 3,4-methylenedioxymethamphetamine users. Human psychopharmacology

https://pubmed.ncbi.nlm.nih.gov/34506649/

Reviews abstinent MDMA users and finds that transporter scans and thinking skills do not consistently change together.

[55] Tang J, Krushelnycky L, Shaqo A, Cho CE. (2025). A Comprehensive Review of Nutritional Influences on the Serotonergic System. Advances in nutrition (Bethesda, Md.)

https://pubmed.ncbi.nlm.nih.gov/40998119/

Reviews nutrients involved in serotonin biology; supports adequate nutrition without demonstrating a post-MDMA supplement regimen.

[56] Yang D, Gouaux E. (2021). Illumination of serotonin transporter mechanism and role of the allosteric site. Science advances

https://pubmed.ncbi.nlm.nih.gov/34851672/

Examines the structure and operation of SERT; helps explain the transporter rather than showing a repair treatment.

[57] Müller F, Brändle R, Liechti ME, Borgwardt S. (2019). Neuroimaging of chronic MDMA ("ecstasy") effects: A meta-analysis. Neuroscience and biobehavioral reviews

https://pubmed.ncbi.nlm.nih.gov/30439373/

Combines brain-imaging studies of chronic MDMA users; transporter findings and time without use were related, but study limitations complicate interpretation.

[58] Garbarino VR, Gilman TL, Daws LC, Gould GG. (2019). Extreme enhancement or depletion of serotonin transporter function and serotonin availability in autism spectrum disorder. Pharmacological research

https://pubmed.ncbi.nlm.nih.gov/30009933/

Reviews unusually high or low serotonin-transporter function in autism; background from another condition.

[59] Hajisoltani R, Tabatabaei FSA, R Hamblin M, Ramezani F. (2025). Functional Recovery Promotion After Spinal Cord Injury With Astaxanthin Treatment in Preclinical Studies: A Systematic Review and Meta-Analysis. Neurology research international

https://pubmed.ncbi.nlm.nih.gov/40786962/

Combines animal spinal-injury studies reporting astaxanthin benefits; different delivery methods and study quality limit direct application.

[60] Chang MX, Xiong F. (2020). Astaxanthin and its Effects in Inflammatory Responses and Inflammation-Associated Diseases: Recent Advances and Future Directions. Molecules (Basel, Switzerland)

https://pubmed.ncbi.nlm.nih.gov/33207669/

Reviews astaxanthin in inflammation-related conditions; background for why it is considered.

[61] Huang C, Li Z, Qu W, Guo W. (2022). Astaxanthin-folic acid combined treatment potentiates neuronal regeneration and functional recovery after brachial plexus avulsion and reimplantation. Frontiers in neuroscience

https://pubmed.ncbi.nlm.nih.gov/36300168/

Astaxanthin plus folic acid helped recovery in an animal nerve-injury experiment; the combination does not isolate astaxanthin's contribution.

[62] Deo N, Redpath G. (2022). Serotonin Receptor and Transporter Endocytosis Is an Important Factor in the Cellular Basis of Depression and Anxiety. Frontiers in cellular neuroscience

https://pubmed.ncbi.nlm.nih.gov/35280519/

Discusses how serotonin receptors and transporters move inside cells and back to the surface; background for regulation.

[63] Mustafa NS, Bakar NHA, Mohamad N, Adnan LHM, Fauzi NFAM, Thoarlim A, et al. (2020). MDMA and the Brain: A Short Review on the Role of Neurotransmitters in Neurotoxicity. Basic and clinical neuroscience

https://pubmed.ncbi.nlm.nih.gov/33613876/

Reviews brain messengers and possible MDMA injury mechanisms; a starting point for understanding the experimental research.

[64] Egawa N, Lok J, Washida K, Arai K. (2017). Mechanisms of Axonal Damage and Repair after Central Nervous System Injury. Translational stroke research

https://pubmed.ncbi.nlm.nih.gov/27566737/

Reviews how nerve branches are damaged and repaired after brain or spinal injury; explains why growth can depend on several processes.

[65] Young SN. (2007). How to increase serotonin in the human brain without drugs. Journal of psychiatry & neuroscience : JPN

https://pubmed.ncbi.nlm.nih.gov/18043762/

Discusses non-drug influences on serotonin; context rather than proof of repairing MDMA-related injury.

[66] Nazari Z, Bahrehbar K, Sepehri H, Golalipour MJ. (2023). Curcumin can improve ecstasy-induced hippocampal damage in rat. Toxicology and Environmental Health Sciences

https://doi.org/10.1007/s13530-023-00170-z

Rat MDMA study reported memory and brain-tissue findings; accessible methods leave the precise curcumin treatment window unresolved.

[67] Liang JH, Zheng YH, Guo AH, et al. (2024). Astaxanthin promotes nerve repair by regulating the M1/M2 ratio of microglia and promoting angiogenesis. Journal of Functional Foods

https://doi.org/10.1016/j.jff.2024.106424

Astaxanthin promoted repair after a peripheral nerve injury in animals; this differs from injury to serotonin fibres inside the brain.

[68] Reddit community contributors. (2019). N-acetyl cysteine (NAC) and its probable MDMA loss-of-magic effects. Community report

https://www.reddit.com/r/MDMA/comments/ae7lta/

Personal reports and discussion include short suggested NAC pauses; used to document community practice, not prove an effect.

[69] Reddit community contributors. (2022). NAC and MDMA. Community report

https://www.reddit.com/r/mdmatherapy/comments/uvs6fb/

Community discussion includes pauses of one or two weeks and shorter alternatives; no controlled timing comparison was performed.

[70] Sumartha INB, Abdullah ADI, Shukor SA, Majid NA, Ardianto C, Ming LC. (2026). Role of astaxanthin in traumatic brain injury: a systematic review. Discover Neuroscience

https://doi.org/10.1186/s13064-026-00265-6

Reviews animal and laboratory astaxanthin research in traumatic brain injury; adds context to the separate original experiments.

[71] Groeneveld M. (2026). Open MDMA: An Evidence-Based Synthesis, Theory, and Manual for MDMA Therapy Based on Memory Reconsolidation, Complex Systems, and the Defense Cascade. Preprint draft version 6.0, 2 July 2026. Independent practice guide

https://doi.org/10.31234/osf.io/aps5g

Practice source. Sections 4.4-4.6 discuss antioxidants, spacing and interactions; explicitly acknowledges that protective oral human antioxidant doses are unknown. Its AI-generated recovery intervals are not measured human recovery times.

[72] Mithoefer MC and collaborators. (2017). A Manual for MDMA-Assisted Psychotherapy in the Treatment of PTSD, version 8.1. MAPS treatment manual

https://maps.org/wp-content/uploads/2021/10/TreatmentManual_MDMAAssistedPsychotherapyVersion8.1_22Aug2017.pdf

Clinical-practice framework for preparation, sessions and integration. Does not establish a neuroprotective supplement regimen.

[73] RollSafe.org. (accessed 2026). MDMA (Molly/Ecstasy) Supplements. Community harm-reduction guide

https://rollsafe.org/mdma-supplements/

Publishes concrete ALA, ALCAR, vitamin C and CoQ10 schedules that circulate in forums. These are extrapolated community proposals, not tested human protection regimens; the page discloses supplement advertising.

[74] Zelfand E. (accessed 2026). Supplements to Reduce Side Effects of MDMA. Practitioner-authored guidance

https://psychedelic.support/resources/supplements-to-reduce-side-effects-mdma/

Gives suggested supplement doses and timing, including NAC close to MDMA, which conflicts with common forum advice. Its claim that supplements might eliminate risk exceeds the evidence reviewed here.

[75] Bluelight contributors. (2012 onward). Supplements Shown to COMPLETELY BLOCK Serotonin Depletion from MDMA. Community discussion

https://www.bluelight.org/community/threads/supplements-shown-to-completely-block-serotonin-depletion-from-mdma.609672/

Documents longstanding ALA/ALCAR enthusiasm, attempted animal-to-human dose conversion and individual reports of preserved effects. Neither the title nor the numerical conversion establishes human protection.

[76] Bluelight contributors. (2019). MDMA: 3 years experience, 2 months NAC dosage regimen to bring MDMA magic back. Community report

https://www.bluelight.org/community/threads/mdma-3-years-experience-2-months-nac-dosage-regimen-to-bring-mdma-magic-back.863303/

An unsuccessful recovery report after trying a NAC regimen. It also links back to Reddit, illustrating that apparent cross-forum agreement can share one source.

[77] Reddit r/MDMA contributors. (2019). Supplement advice. Community discussion

https://www.reddit.com/r/MDMA/comments/cs0xto/

Describes following the RollSafe ALA/ALCAR/vitamin C schedule. Supports circulation of the recommendation rather than an independent test of efficacy.

[78] The Castalia Foundation; edited by Phoenix Kaspian. (2024). MDMA Solo. Third edition. Independent self-directed practice manual

https://castaliafoundation.com/kali/MDMA-solo.pdf

The supplied manual, printed page 70, suggests nutrition during the session and a standard multivitamin afterwards. Its confident claims about eliminating comedowns are not substantiated by supplement trials in this guide.

[79] Rudnick G, Wall SC (1992). The molecular mechanism of ecstasy: serotonin transporters are targets for MDMA-induced serotonin release. PNAS

https://pubmed.ncbi.nlm.nih.gov/1347426/

Primary transporter experiment explaining how MDMA drives serotonin release. This establishes drug action, not the extent of injury during therapy.

[80] Gamma A, Buck A, Berthold T, Liechti ME, Vollenweider FX (2000). 3,4-Methylenedioxymethamphetamine (MDMA) modulates cortical and limbic brain activity as measured by PET in healthy humans. Neuropsychopharmacology

https://pubmed.ncbi.nlm.nih.gov/10989265/

A single oral MDMA dose changed regional brain blood flow in 16 volunteers. Blood flow is not a measurement of ATP depletion or a whole-brain energy bill.

[81] O'Hearn E, Battaglia G, De Souza EB, Kuhar MJ, Molliver ME (1988). Methylenedioxyamphetamine (MDA) and methylenedioxymethamphetamine (MDMA) cause selective ablation of serotonergic axon terminals in forebrain: immunocytochemical evidence for neurotoxicity. Journal of Neuroscience

https://pubmed.ncbi.nlm.nih.gov/2457659/

Repeated-exposure rat experiments showed swollen, fragmented serotonin axons and loss of terminals. This is structural injury evidence under those experimental conditions.

[82] Mitchell JM, et al. (2023). MDMA-assisted therapy for moderate to severe PTSD: a randomized, placebo-controlled phase 3 trial. Nature Medicine

https://www.nature.com/articles/s41591-023-02565-4

Measured PTSD outcomes and clinical adverse events with monitored therapy. It did not directly measure serotonin-axon integrity, brain ATP recovery or supplement-assisted repair.

[83] NIH Office of Dietary Supplements (accessed 2026). Carnitine: Fact Sheet for Health Professionals. Official health information

https://ods.od.nih.gov/factsheets/Carnitine-HealthProfessional/

Explains carnitine forms and cautions about seizures and uremia. These are broader carnitine cautions, not measured ALCAR-MDMA interaction rates.

[84] NIH Office of Dietary Supplements (accessed 2026). Omega-3 Fatty Acids: Fact Sheet for Health Professionals. Official health information

https://ods.od.nih.gov/factsheets/Omega3FattyAcids-HealthProfessional/

Discusses anticoagulant monitoring and the atrial-fibrillation signal with prolonged high-dose omega-3 use in cardiovascular-risk populations.

[85] NIH Office of Dietary Supplements (accessed 2026). Choline: Fact Sheet for Health Professionals. Official health information

https://ods.od.nih.gov/factsheets/Choline-HealthProfessional/

Defines choline and describes adverse effects of excessive intake, including low blood pressure and liver toxicity; adult upper limit is 3.5 g/day.

[86] NIH Office of Dietary Supplements (accessed 2026). Vitamin C: Fact Sheet for Health Professionals. Official health information

https://ods.od.nih.gov/factsheets/VitaminC-HealthProfessional/

Explains vitamin C, iron absorption and concerns about high intake in iron overload and renal disorders.

[87] Aspire Pharma Ltd (accessed 2026). NACSYS 600 mg Effervescent Tablets: Summary of Product Characteristics. Electronic Medicines Compendium; product information

https://www.medicines.org.uk/emc/product/8576/smpc

Oral acetylcysteine product information identifies hypersensitivity, asthma/bronchospasm and enhancement of nitroglycerin effects.

[88] EFSA Panel on Nutrition, Novel Foods and Food Allergens (2021). Scientific opinion on the relationship between intake of alpha-lipoic acid and the risk of insulin autoimmune syndrome. EFSA Journal

https://doi.org/10.2903/j.efsa.2021.6577

ALA can trigger insulin autoimmune syndrome in susceptible people, causing hypoglycaemia. A threshold dose eliminating this risk could not be established.

[89] Therapeutic Goods Administration (2023). Medicines containing turmeric or curcumin: risk of liver injury. Regulatory safety alert

https://www.tga.gov.au/safety/safety-alerts/medicines-containing-turmeric-or-curcumin-risk-liver-injury

Regulator advises avoiding medicinal turmeric/curcumin products with current or previous liver problems. Normal food use is a different exposure.

[90] National Center for Complementary and Integrative Health (2019). Coenzyme Q10. Official health information

https://www.nccih.nih.gov/health/coenzyme-q10

Identifies potential interactions with warfarin, insulin and some cancer treatments.

[91] Chow HH, et al. (2010). Resveratrol modulates drug- and carcinogen-metabolizing enzymes in a healthy volunteer study. Cancer Prevention Research

https://pubmed.ncbi.nlm.nih.gov/20716633/

One gram daily for four weeks altered drug-metabolizing enzyme activity in healthy volunteers; this supports medication review without predicting a particular MDMA interaction.

[92] Nakatsugawa M, et al. (2003). Hericium erinaceum extract-induced acute respiratory distress syndrome monitored by serum surfactant proteins. Internal Medicine

https://pubmed.ncbi.nlm.nih.gov/14714963/

A case report of a serious respiratory reaction after lion's mane extract supports attention to prior allergy or reactions; it cannot establish frequency.

[93] NIH Office of Dietary Supplements (accessed 2026). Dietary Supplements for Exercise and Athletic Performance: Fact Sheet for Health Professionals. Official health information

https://ods.od.nih.gov/factsheets/ExerciseAndAthleticPerformance-HealthProfessional/

Describes creatine as an energy-buffering compound and summarizes safety evidence mainly in healthy users. It does not establish an MDMA regimen or suitability in kidney disease.