Cognitive Vitality Reports® are reports written by neuroscientists at the Alzheimer's Drug Discovery Foundation (ADDF). These scientific reports include analysis of drugs, drugs-in-development, drug targets, supplements, nutraceuticals, food/drink, non-pharmacologic interventions, and risk factors. Neuroscientists evaluate the potential benefit (or harm) for brain health, as well as for age-related health concerns that can affect brain health (e.g., cardiovascular diseases, cancers, diabetes/metabolic syndrome). In addition, these reports include evaluation of safety data, from clinical trials if available, and from preclinical models.

Psilocybin

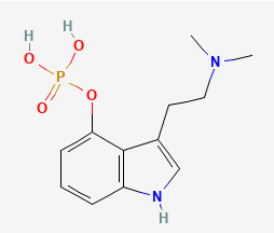
Evidence Summary

A single dose of psilocybin has shown lasting benefits for depression. Preclinical studies have shown neuroprotective benefits, but they have not been confirmed in people with neurodegenerative diseases.

Neuroprotective Benefit: Cognitive effects are mixed, and likely depend on many factors, including the dose, mood, and health condition. Preclinical studies have shown increased neuroplasticity, neurogenesis, and synapse density while decreasing inflammation.

Aging and related health concerns: Significant and sustained reduction in anxiety and depression are seen in life-threatening cancer patients. A case series reported significant pain relief in a few patients with chronic neuropathic pain.

Safety: High doses may cause panic reactions and psychosis. Common adverse events include hallucinations, headache, increased blood pressure, increased breathing rate, confusion, and sleep problems. The risk of abuse appears to be low.

Availability: not available; It is a Schedule I drug. It is being tested in clinical trials.	**Dose**: Most clinical studies used single oral doses ranging from 10 to 30 mg.	**Chemical formula:** $C_{12}H_{17}N_2O_4P$ **MW**: 284.25  Source: PubChem
Half-life: 3 hours	**BBB**: penetrant	
Clinical trials: The largest placebo-controlled trial investigating cognitive function included 89 healthy adults.	**Observational studies**: none available	

What is it? Psilocybin is a chemical compound present in over 180 species of hallucinogenic mushrooms (dried or fresh) found in Mexico, South America, and parts of the US (Drugs.com). Dried "magic mushrooms" contain about 0.2-0.4% psilocybin. Psilocybin can also be produced in the laboratory.

Psilocybin is quickly converted by the body to psilocin, which acts as an agonist for serotonin receptors, 5HT-2A, 5HT-2C, and 5HT-1A (Erkizia-Santamaria et al., 2022). Psychedelics acting on 5HT-2A and other serotonin receptors are drawing increasing interest recently as potential treatments for neurodegenerative diseases (Saeger et al., 2022; Kozlowska et al., 2022; vann Jones and O'Kelly, 2020). This is stemming from recent studies showing that these compounds can exert persisting anxiolytic and antidepressant effects lasting several months after a single administration and have shown potential for a wide range of brain disorder including depression, post-traumatic stress disorder, and substance use disorder. Psilocybin has received FDA Breakthrough Therapy designation for the treatment of drug-resistant depression (2018) and major depressive disorder (2019)(Scientific American).

Neuroprotective Benefit: Cognitive effects are mixed, and likely depend on many factors, including the dose, mood, and health condition. Preclinical studies have shown increased neuroplasticity, neurogenesis, and synapse density while decreasing inflammation.

Types of evidence:

- 8 placebo-controlled trials in healthy adults
- 1 open-label clinical study in healthy adults
- 1 open-label clinical trial in major depressive disorder
- 4 review articles on the use of psychedelics for neurodegenerative disorders
- Several laboratory studies

Human research to suggest prevention of dementia, prevention of decline, or improved cognitive function:

Overall, the effects of psilocybin on cognitive function have been mixed, with some studies showing a lack of change, a few showing a trend for impairment, and a few showing potential benefits. Effects on cognition may depend on many factors, including the dose, expectation of the drug effect, mood, and underlying health conditions. High dose psilocybin can impair cognitive function including attention due to increased awareness of sensory stimuli that are usually filtered out (vann Jones and O'Kelly, 2020). Recent studies are exploring low-dose or microdose psilocybin treatments that do not produce psychedelic or negative cognitive effects. There is rapid desensitization of 5HT-2A receptors by psilocybin, so single or infrequent dosing is likely better than daily dosing.

In a phase 1 double-blind randomized controlled trial of 89 healthy men and women, a single oral dose of psilocybin (10 or 25 mg) was well-tolerated compared to placebo, with no detrimental short- or long-term effect on cognitive functioning or emotional processing (Rucker et al., 2022). There were no significant differences across psilocybin and placebo groups in global cognition (measured by the CANTAB global composite score), measures of cognitive domains, or emotional processing scale scores. For the global composite, there was a trend of better performance for the 10 and 25 mg psilocybin treatment on day 29 compared to baseline, but no difference was observed when compared with placebo. A measure of sustained attention (RVP-A') showed trends of better performance on day 29 compared to baseline with 10 or 25 mg psilocybin, but no differences were observed when compared with placebo. A measure of working memory (SWM-BE) and a measure of executive function and planning (SWM-S) also showed trends of better performance with 25 mg psilocybin by day 29 compared to baseline, but no difference was observed when compared with placebo. A measure of episodic

memory (PAL-TEA) showed no significant changes from baseline for any of the groups and there were no differences across groups.

In a double-blind placebo-controlled study of 34 healthy adults, a single microdose of dried mushroom (0.5 g) resulted in a few small changes towards cognitive impairment (Cavanna et al., 2022). Specifically, decreased performance on the attentional blink task, increased time required in the Stroop task, and increased time required for the Trail-making test (part A) were significant, but only when not corrected for multiple comparisons. Psilocybin administration also reduced power of EEG theta oscillations, reflecting reduced vigilance.

In a blinded placebo-controlled study of 15 healthy adults, oral administration of psilocybin (10 mg/70 kg body weight) induced widespread dysregulation of cortical activity, including significantly decreasing the amplitude of low frequency fluctuations as well as the variance of blood-oxygenation level-dependent (BOLD) signal in the left and right claustrum that highly expresses 5HT-2A receptors (Barrett et al., 2020). Psilocybin administration also significantly altered both left and right claustrum connectivity with brain networks that support sensory and cognitive processes [decreased functional connectivity of the right claustrum with auditory and default mode networks (DMN), increased right claustrum connectivity with the fronto-parietal task control network (FPTC), and decreased left claustrum connectivity with the FPTC].

In a double-blind placebo-controlled clinical study of 11 healthy hallucinogen users, cognitive effects of psilocybin (10, 20, or 30 mg/70kg) and dextromethorphan (400 mg/70 kg) were compared with placebo (Barrett et al., 2018). Psilocybin did not result in global cognitive impairment, as measured by the MMSE, or delirium, during peak drug effects. However, dose-dependent effects of psilocybin were observed on psychomotor performance, working memory, episodic memory, associative learning, and visual perception. Both psilocybin and dextromethorphan decreased the free recall of words and caused impairments in associative learning (substitution recall of the DSST). Psilocybin administration also showed a dose-dependent impairment on discriminability and increased response time for correct responses (during the 2-back condition in the N-back task)

In a double-blind placebo-controlled crossover trial of 20 healthy adults, a single oral dose of psilocybin (0.26 mg/kg, manufactured according to GMP standards from THC_Pharm GmbH, Frankfurt, Germany) induced robust psychedelic effects and psychotic symptoms while decreasing event-related potentials at the P300 amplitude, which reflects higher order cognitive processing, and decreasing N100 amplitude,

which reflects sensory-related processing (Bravermanova et al., 2018). However, preattentive cognitive processing (mismatch negativity, or MMN amplitude) was not affected by psilocybin.

In a double-blind placebo-controlled study of 8 healthy adults, an acute oral dose of psilocybin (215 µg/kg) significantly reduced attentional tracking ability but had no significant effect on spatial working memory (Carter et al, 2005). Pretreatment with ketanserin, a 5HT-2A antagonist, did not attenuate the effect of psilocybin on attentional performance, suggesting a primary involvement of the 5-HT1A receptor in the observed deficit. The authors speculate that this impaired attentional performance may reflect a reduced ability to suppress or ignore distracting stimuli rather than reduced attentional capacity.

In a double-blind placebo-controlled study of 32 healthy adults, acute effects of psilocybin (0.2 mg/kg, 15 mg max), the entactogen 3,4-methylenedioxyethyl-amphetamine (MDE; 2 mg/kg), and the stimulant d-methamphetamine (0.2-0.4 mg/kg) (all obtained from the Pharmaceutical Institute of the University of Tubingen) were compared with placebo (Gouzoulis-Mayfrank et al., 1999). Psilocybin administration increased regional magnetic resonance glutamate levels of the right hemisphere frontotemporal cortical regions (particularly in the anterior cingulate) and decreased it in the thalamus. However, psilocybin administration led to significantly fewer words in an association task compared to placebo administration, which was accompanied by diminished activation of frontal regions. No significant differences across four treatment conditions were seen for global cerebral metabolism. Cognitive activation-related increases in left frontocortical regions were dampened after administration of the three psychoactive drugs.

In an open-label study of 10 healthy adults, a single dose of 15 or 20 mg psilocybin produced a global increase in cerebral metabolic rate of glucose (CMRglu) with the greatest increases in the frontomedial and frontolateral cortex (by 24.3%), anterior cingulate (by 24.9%), and temporomedial cortex (by 25.3%)(Vollenweider et al., 1997). The CMRglu increases in the prefrontal cortex, anterior cingulate, temporomedial cortex, and putamen positively correlated with psychotic symptoms (hallucinatory ego disintegration).

In an open-label study of 24 patients with major depressive disorder, a single dose of moderately high (20 mg/70 kg body weight) or a high dose (30 mg/70 kg body weight) of psilocybin reduced symptoms of depression while increasing cognitive flexibility, an effect seen for at least 4 weeks post-treatment (Doss et al., 2021). However, cognitive tasks measuring response inhibition, selective attention, and abstract reasoning were not affected. One week after psilocybin administration, glutamate and N-

acetylaspartate concentrations were decreased in the anterior cingulate cortex (ACC), and dynamics of functional connectivity (dFC) was increased between the ACC and the posterior cingulate cortex (PCC). But greater increases in dFC between the ACC and PCC were associated with less improvement in cognitive flexibility after psilocybin therapy. The authors speculate that subpopulations of patients, for example, those with lower baseline neural flexibility, may be more likely to benefit from psilocybin therapy. Because this study was an open-label study and not placebo-controlled, these results could be attributed to expectancy, practice, or exposure effects.

Human research to suggest benefits to patients with dementia:
None available.

Mechanisms of action for neuroprotection identified from laboratory and clinical research:
Psychedelic compounds acting on 5HT-2A and other serotonin receptors are drawing increasing interest recently as potential treatments for neurodegenerative diseases (Saeger et al., 2022; Kozlowska et al., 2022; vann Jones and O'Kelly, 2020). In Alzheimer's disease and related disorders, 5HT-2A receptor density is significantly reduced and this loss is correlated with cognitive decline (Franceschi et al., 2005; Lai et al., 2005). Psychedelic compounds activating the 5HT-2A receptor produces hallucinogenic effects while also playing other roles such as promoting cortical neuron growth, activating neuronal survival mechanisms, and modulating the immune system (Saeger et al., 2022; Kozlowska et al., 2022; vann Jones and O'Kelly, 2020).

In a double-blind randomized controlled crossover trial of 28 healthy participants, acute effects of psilocybin (15 and 30 mg), LSD (100 and 200 µg), and placebo were compared (Holze et al., 2022). Both psilocybin and LSD significantly increased plasma cortisol, prolactin, and oxytocin levels, but neither significantly elevated plasma BDNF levels.

In pig brain, a single dose of psilocybin (0.08 mg/kg, i.v.) increased hippocampal synaptic density by 4.42% (measured by SV2A, a synaptic protein), while lowering 5HT-2A receptor density by 15.21-50.19% in the hippocampus and prefrontal cortex (Raval et al., 2021). Seven days after psilocybin treatment, the increase in synaptic density was greater—by 9.24% in the hippocampus and by 6.10% in the prefrontal cortex. However, there were no longer any differences in 5HT-2A receptor density, suggesting that psilocybin effects on 5HT-2A density is acute and temporary, while changes in synaptic density are longer lasting.

In rats, an acute low dose pretreatment with psilocybin (0.05-0.1 mg/kg, s.c.) improved attentional accuracy and a measure of impulsive action exclusively in low performers, but no overall effects were seen when data from all rats were combined (Higgins et al., 2021).

In rats, a single administration of psilocybin (0.5-20 mg/kg) increased the expression of c-Fos, an immediate early gene implicated in neuroplasticity, in the prefrontal cortex, but not the hippocampus (Jefsen et al., 2021).

In rats, a single dose of psilocin (1 or 4 mg/kg; s.c.; synthesized at Pharmaceutical Faculty of Charles University in Prague) significantly impaired the acquisition of the Carousel maze (Rambousek et al., 2014). In the Morris water maze, the 4 mg/kg dose of psilocin disrupted reinforced retrieval, but the lower dose had no significant effect. When psilocin was injected during the post-training period, memory consolidation in the water maze was unaffected, suggesting that psilocin does not interfere with memory consolidation.

In mice, a single dose of psilocybin (1 mg/kg, i.p.; Usona Institute) led to a ~10% increase in dendritic spine size and density, driven by an increase in spine formation rate, in the medial frontal cortex (Shao et al., 2021). This neuronal structural remodeling occurred within 24 hours and 34-37% of the spines that were formed remained persistent 1 month later. These changes were accompanied by reduced stress-related behavioral deficit and increased excitatory neurotransmission.

In a different study in mice, a 1 mg/kg dose (i.p.) of psilocybin significantly decreased the number of newborn neurons in the hippocampus, while lower doses (0.1 or 0.5 mg/kg, i.p.) showed a trend toward an increase in hippocampal neurogenesis compared to vehicle treatment (Catlow et al., 2013).

In microglia culture, treatment with psilocin upregulated the neuroprotective TREM2 receptor important for phagocytosis and synaptic refinement, while reducing proinflammatory markers (TLR4, p65, CD80 proteins)(Kozlowska et al., 2021). There is also evidence that psilocybin has anti-inflammatory effects in peripheral tissues (Kozlowska et al., 2022).

APOE4 interactions: Unknown.

Aging and related health concerns: Significant and sustained reduction in anxiety and depression are seen in life-threatening cancer patients. A case series reported significant pain relief in a few patients with chronic neuropathic pain.

Types of evidence:

- 1 double-blind randomized controlled trial on sleep measures
- 1 clinical study in cancer patients
- 1 case series of chronic pain

Cancer: REDUCED DEPRESSION AND ANXIETY, SUSTAINED FOR MONTHS/YEARS
In a long-term follow-up of 15 patients with life-threatening cancer who received psilocybin-assisted psychotherapy, significant reductions in anxiety, depression, hopelessness, demoralization, and death anxiety in 60-80% of the participants, 4.5 years after the therapy (Agin-Liebes et al., 2020).

Sleep: MIXED; MAY SUPPRESS SLOW WAVE ACTIVITY
In a double-blind randomized controlled crossover clinical trial of 20 healthy adults, a single dose of psilocybin (0.26 mg/kg; range of 15-22 mg) prolonged REM sleep patency and caused a trend toward a decrease in overall REM sleep duration (Dudysova et al., 2020). No significant differences in sleep latency, total sleep time, sleep efficiency, NREM sleep, or number of sleep cycles were observed between psilocybin and placebo conditions. Psilocybin did not affect EEG power spectra in NREM or REM sleep, but it did suppress slow wave activity (measured by absolute delta power) during slow wave sleep in the first sleep cycle.

Neuropathic pain: INCONCLUSIVE; PAIN RELIEF
In a case series of 3 patients with chronic neuropathic pain, low-dose psilocybin-containing mushroom self-administration (varying preparations) achieved robust pain relief with decreased reliance on analgesic medications (Lyes et al., 2022). The exact contents of the dried mushrooms and doses of psilocybin cannot be determined but can roughly be estimated at 1.25 mg-10 mg across the 3 subjects (the dried *Psilocybe cubensis* mushrooms contain roughly 0.5-1.0% psilocybin by mass). Analgesic effects for all 3 patients occurred at doses without a psychedelic effect with minimal cognitive or somatic adverse events.

- The first patient is a 37-year-old man with a history of quadriplegia due to a vehicle accident that resulted in a cervical spinal cord injury and lower extremity neuropathic pain. He was prescribed tramadol and diazepam, but they were ineffective and produced cognitive side effect. His first exposure was a 5 g dose of dried ground psilocybin-containing mushroom powder that produced a

psychedelic experience, accompanied by near total relief from his lower extremity neuropathic pain that lasted for 8-10 hours. Subsequently, he tried lower doses in the 250 mg range and the analgesic effects persisted with a similar magnitude of relief, but for a shorter duration of 6-8 hours. In addition to pain relief, he experienced stimulating muscle spasms that were soothing and therapeutic to the paralyzed muscles. He then lowered the dose further to 50 mg daily with no muscle spasms and 90-95% pain relief lasting 6-8 hours. He has continued to use this dose daily for 6 months and he has stopped using tramadol, valium, and cannabis, as the analgesic effects from psilocybin exceeded the marginal benefit from these other medications. The patient denied rebound pain or withdrawal symptoms, but the pain returns to baseline on days when he does not self-administer psilocybin.

- The second patient is a 69-year-old female with a history of complex regional pain syndrome secondary to lower extremity trauma, the injuries of which were non-operative. The debilitating pain has made it impossible for her to perform activities of daily living without assistance, and she has tried numerous interventions including physical therapy, acupuncture, yoga, biofeedback, desensitization therapy, nerve blocks, steroid injections, and stem cell injections without success. She has tried NSAIDs and opioid agonists (oxycodone, fentanyl patches and buprenorphine patches), gabapentinoids, and antidepressants (duloxetine and amitriptyline), with limited efficacy and untoward side effects. Her first experience with psilocybin was with 2 g of ground, dried psilocybin-containing mushroom powder and within an hour, her pain level reduced to zero, though this was accompanied by significant psychotropic effects including altered mood, changes in visual/auditory perception as well in thought patterns and cognition that continued for 6 hours. The analgesia lasted 18-20 hours. She experimented with microdosing to reduce side effects, and she typically uses a 500 mg/day dose for 7-10 days (producing 80% pain relief for 3-4 hours, gradually returning to baseline after 12 hours) followed by a rest period of 2-3 days to prevent gastrointestinal side effects. She denies rebound pain or withdrawal effects when she skips doses, and she has been microdosing for over a year and has not noticed any tolerance or loss of efficacy.
- The third patient is a 40-year-old female with a history of radiculopathy secondary to degenerative disc disease, with pain that decreased over time such that it interferes with activities of daily living. The pain was unresponsive to medications, physical therapy, and epidural steroid injections. She began experimenting with psilocybin-containing mushrooms to reduce opioid use, starting at 1000 mg of dried ground mushroom incorporated into a chocolate bar. Within one hour, her bilateral radicular pain and focal low back pain was completely resolved. She experiences a sensation of improved flexibility and relaxation in areas that are usually stiff and painful. After a single dose coupled with exercise, analgesic effects persist for two weeks. Pain slowly returns to baseline over the subsequent 2-4-week period; thus, she spaces out psilocybin administration by 6-8 weeks. She

has found the longevity of analgesic effects are far more sustained when used as an adjunct to physical therapy exercises. She has experienced an overall improvement in baseline pain with each psilocybin session. Musculoskeletal pain has gone from a pain score of 9 (out of 10) to a 7. Neuropathic pain relief has been profound, with a complete resolution after the third session, and has not recurred. The patient denies noticeable somatic, cognitive, or behavioral side effects, and her mood has been unaffected. She has not noticed tolerance to pain relieving effects, rebound pain, or withdrawal effects.

Findings from this study may not be generalizable to other chronic neuropathic pain patients, as these patients sought psilocybin on their own and experienced positive effects (Lyes et al., 2022). As this was not a placebo-controlled study, the role of placebo effect cannot be determined. Also, no objective measures of function or efficacy were used. Randomized, double-blind, placebo-controlled trials are needed, with formal pain assessment and psychiatric evaluation to minimize the impact of confounding factors.

Safety: High doses may cause panic reactions and psychosis. Common adverse events include hallucinations, headache, increased blood pressure, increased breathing rate, confusion, and sleep problems. The risk of abuse appears to be low.

Types of evidence:

- 3 double-blind controlled clinical trials

Psilocybin use can cause visual and auditory hallucinations, an inability to discern fantasy from reality, and at high doses, may also cause panic reactions and psychosis (Drugs.com; WebMD.com). Other common effects of psilocybin (and other hallucinogens) include intensified feelings/sensations, changes in sense of time, confusion, headache, increased blood pressure, increased breathing rate, increased body temperature, loss of appetite, dry mouth, sleep problems, spiritual experiences, uncoordinated movements, lowered inhibition, excessive sweating, and paranoia. Despite being a Schedule I controlled substance, the risk of abuse with psilocybin appears to be low, and withdrawal symptoms and physical dependence do not appear to be a major concern (WebMD.com).

In a phase 1 double-blind randomized controlled trial of 89 healthy men and women, a single oral dose of psilocybin (10 or 25 mg) was well-tolerated compared to placebo, with no detrimental short- or long-term effect on cognitive functioning or emotional processing, and no clinically significant findings in vital

signs (Rucker et al., 2022). A total of 511 treatment-emergent adverse events (TEAEs) were reported, with a median duration of one day; 67% of all TEAEs started and resolved on the day of administration. Of these, 208, 188 and 77 were deemed by the investigator to be potentially related to study treatment in the 25 mg psilocybin, 10 mg psilocybin, and placebo arms, respectively. There were no serious TEAEs. Four participants reported anxiety on the day of study drug administration (2 people with 25 mg psilocybin, 1 person with 10 mg psilocybin, and 1 person with placebo). There were 57 adverse events related to "altered mood", which included negative mood, introspection, reflection, and sense of oneness. There were 86 reports of hallucination, 56 reports of illusion, 15 reports of euphoric mood, and 11 reports of altered time perception. There was one participant in the 10 mg psilocybin arm who reported suicidal thoughts, which started and resolved on day 19, was mild in severity and was deemed by the investigator to be possibly related to study drug. But there was also one participant in the placebo arm who reported two TEAEs of suicidal ideation (one started on day 4 and lasted for 5 days; one started on day 18 and lasted for 17 days) and two TEAEs of suicidal thoughts (one started and resolved on day 79; one started and resolved on day 91). All four events were moderate in severity and considered possibly related to study drug.

In a double-blind randomized controlled crossover trial of 28 healthy participants where acute effects of psilocybin (15 and 30 mg), LSD (100 and 200 µg), and placebo were compared, both psilocybin and LSD significantly increased diastolic and systolic blood pressure, body temperature, and pupil size compared with placebo (Holze et al., 2022). Psilocybin at a dose of 30 mg, but not 15 mg, moderately increased heart rate compared with placebo. Psilocybin at a dose of 30 mg significantly impaired normal light-induced pupil constriction compared with placebo. Other adverse effects included headaches (4 subjects after psilocybin), nosebleed (1 subject after psilocybin), low mood (2 subjects after psilocybin), nausea (2 subjects after psilocybin), nightmares (1 subject after psilocybin), restlessness (1 subject after psilocybin), insomnia (1 subject after psilocybin), and flashback episodes (4 episodes after psilocybin). No severe adverse events were observed.

In a double-blind placebo-controlled crossover trial of 20 healthy adults, a single oral dose of psilocybin (0.26 mg/kg, manufactured according to GMP standards from THC_Pharm GmbH, Frankfurt, Germany) led to a significant increase in systolic and diastolic blood pressure by about 10-20 mmHg and heart rate by about 10 beats per minute during peak intoxication (Bravermanova et al., 2018). No psychotic symptoms or other psychopathology were observed in any of the subjects.

Because the studies to date have been limited to small short-term studies, more studies with a larger sample size and longer follow-up are needed to understand the full spectrum of adverse events associated with psilocybin.

Drug interactions: Psilocybin may increase serotonin levels, and therefore, taking psilocybin with other medications that increase serotonin might increase serotonin too much, causing serious side effects including heart problems, seizures, and vomiting (WebMD.com). Also taking psilocybin with stimulants (e.g., amphetamines, cocaine) may cause serious problems including increased heart rate and high blood pressure.

Sources and dosing: Psilocybin is a Schedule I controlled substance and is not available. However, in several US states, psilocybin is accessible to people who need them clinically (e.g., Oregon, Washington DC)(Drugs.com). Dosage has not been established for any condition. In clinical trials, single oral doses ranging from 10 to 30 mg have often been used. Because there is rapid desensitization of 5HT-2A receptors by psilocybin, a single or infrequent dosing is likely better than daily dosing (vann Jones and O'Kelly, 2020).

Research underway: Based on ClinicalTrials.gov, there are 80 ongoing clinical studies that are testing psilocybin (ClinicalTrials.gov). Most trials are in people with major depressive disorder, anxiety, alcohol use disorder, anorexia nervosa, obsessive-compulsive disorder, bipolar disorder, and other conditions. There is one ongoing study testing psilocybin for depression in people with mild cognitive impairment or early Alzheimer's disease (NCT04123314). The estimated study completion is in December 2023. Another trial is testing psilocybin for depression and anxiety in Parkinson's disease (NCT04932434). It is scheduled to be completed in December 2022.

Search terms:

Pubmed, Google: psilocybin

- + Alzheimer, + meta-analysis, + cognitive

Websites visited for psilocybin:

- Clinicaltrials.gov
- NIH RePORTER
- Examine.com
- DrugAge (0)
- Geroprotectors (0)
- Drugs.com
- WebMD.com
- PubChem
- DrugBank.ca
- Cafepharma
- Pharmapro.com (0)