RonanRx
PERSONALIZED MEDICINE

CLINICAL MONOGRAPH · ANTIOXIDANT & MITOCHONDRIAL

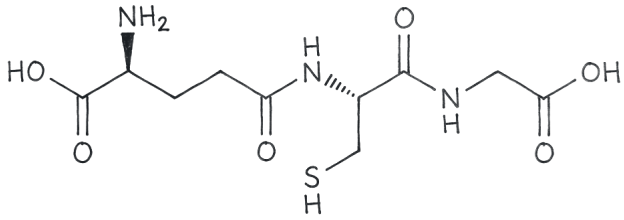
Glutathione

Master endogenous antioxidant in injectable and oral forms

Glutathione is the body's most abundant endogenous antioxidant, a tripeptide of glutamate, cysteine, and glycine present in nearly every cell at millimolar concentrations [sonthalia2018; fda2019glutathione].

Researchers have evaluated supplemental glutathione across several routes (oral, liposomal oral, sublingual, intranasal, intravenous, nebulized, topical) and several research contexts (Parkinson disease, nonalcoholic fatty liver disease, cystic fibrosis, dermatology, healthy-adult biomarker studies) [richie2015; sinha2018; mischley2017]. The cellular biochemistry is well characterized; clinical evidence varies by route and indication and is not uniformly established across uses [bishop2005].

RonanRx-compounded glutathione is dispensed only on a patient-specific prescription written by a licensed doctor for an identified patient. RonanRx does not sell glutathione directly to patients [honda2017; lu2013; wu2004].



EVIDENCE POSTURE | WELL STUDIED | EMERGING | REVIEWED **2026-05-11**

State-licensed 503A | Pharmacist reviewed | Doctor led | Cold-chain ready | Patient choice preserved

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Why Personalized Glutathione

Every glutathione trial in the literature picked one route and lived with the consequences of that choice. Oral capsules above 3 g failed to raise plasma GSH meaningfully in a controlled pharmacokinetic study. IV push peaks within minutes and is cleared by gamma-glutamyl transpeptidase at the renal and hepatic beds inside an hour. Intranasal delivery exploits the olfactory pathway for CNS uptake in Parkinson patients. Nebulized GSH targets airway lining fluid in cystic fibrosis. Liposomal oral, sublingual, and orobuccal formulations exist precisely because intestinal hydrolysis defeats unprotected oral dosing. None of those trials picked their route for your indication, your absorption, your tolerance for injection, or whether you have airway disease, neurodegeneration, or a dermatologic concern.

Matching route to indication is the work a compounding pharmacy does for glutathione specifically. A prescriber who knows the clinical question can choose IV push when speed and systemic load matter, intramuscular for slower release, nebulized when the target is lung lining fluid, intranasal when the target is brain, sublingual or liposomal oral when the patient cannot tolerate needles, and topical when the target is skin. Dose, frequency, preservative profile, and pH all adjust to the patient. The FDA's 2019 alert on endotoxin-related events from poorly sourced injectable GSH makes the second compounding axis explicit: ingredient suitability and sterile preparation are part of the prescription, not assumptions about the supply chain.

This is what pharmacy looked like before mass manufacturing arrived. A doctor wrote the prescription, a pharmacist prepared it for that named patient, and the route was chosen for the body it was going into. Compounded glutathione is that older arrangement, kept honest by modern sterility, ingredient, and traceability discipline.

Quick Facts About Glutathione

Category: Endogenous tripeptide antioxidant

Common aliases: GSH, reduced glutathione, γ-glutamyl-cysteinyl-glycine

Biological role: Intracellular redox balance and Phase II detoxification

Routes studied in humans: Oral, liposomal oral, sublingual, intranasal, intravenous, nebulized, topical

Evidence posture: Mechanism well established; clinical efficacy is indication- and route-specific and remains mixed

FDA-approval status: Not FDA-approved as a drug for any specific indication; N-acetylcysteine (a precursor) is FDA-approved for acetaminophen overdose

Compounded under: 503A, patient-specific prescription only

Important compounding caution: FDA documented endotoxin-related adverse events from certain compounded sterile L-glutathione injections; ingredient quality and sterility are critical

SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY

Glutathione described in this monograph is a 503A compounded preparation. Every dose is made on a prescription, for a named patient, by a licensed pharmacist. It is not a stocked, mass-manufactured product.

- **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
- **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
- **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
- **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
- **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is Glutathione?

Glutathione (γ-glutamyl-cysteinyl-glycine, GSH) is a low-molecular-weight tripeptide synthesized de novo in every nucleated cell [ballatori2009a]. Total body content is several grams, with the highest concentrations in the liver and notable pools in lung lining fluid, kidney, and brain.

Two redox states exist: reduced glutathione (GSH) and oxidized glutathione disulfide (GSSG) [lu2013]. The GSH:GSSG ratio is a primary indicator of cellular redox status across tissues.

Glutathione is structurally distinct from the standard peptide bond by virtue of its γ-glutamyl linkage, a feature that protects it from cleavage by most peptidases except gamma-glutamyl transpeptidase (GGT), which initiates extracellular GSH catabolism [lu2013; wu2004; meister1995a].

How Glutathione Works

Glutathione is the substrate for the glutathione peroxidase family of enzymes, which reduce hydrogen peroxide and lipid hydroperoxides to water and the corresponding alcohols. The oxidized disulfide product is recycled back to GSH by glutathione reductase using NADPH derived from the pentose phosphate pathway.

Glutathione also conjugates electrophilic xenobiotics through glutathione S-transferases (GSTs), enabling Phase II liver detoxification. The resulting glutathione conjugates are exported by multidrug resistance-associated proteins (MRPs) and excreted in bile or urine [lu2013; wu2004; ballatori2009a].

Beyond redox cycling and conjugation, GSH participates in protein S-glutathionylation as a reversible post-translational modification, regulates apoptosis through mitochondrial GSH pools, supports immune cell function, and modulates redox-sensitive signaling pathways [zhang2012; forman2014; forman2016].

Biological Role of Glutathione

Endogenous glutathione concentrations decline with age, oxidative stress, certain chronic disease states, and inadequate sulfur-amino-acid intake [phamhuy2008]. Restoring GSH stores is the therapeutic rationale for both precursor supplementation (NAC) and direct GSH administration.

Disorders of GSH homeostasis, whether acquired (alcoholic and nonalcoholic liver disease, HIV, sepsis, certain cancers) or genetic (rare deficiencies of GCL or glutathione synthetase), are associated with increased susceptibility to oxidative injury and altered drug metabolism [lu2013; wu2004; ballatori2009a].

GSH is also central to cellular handling of heavy metals, electrophilic environmental toxicants, and reactive products of normal lipid and amino-acid metabolism [lushchak2015]. Its breadth of function is why it is sometimes called a 'master antioxidant' in lay literature, though that label oversimplifies a more nuanced biology.

Detailed Mechanism of Glutathione

GSH synthesis occurs in two ATP-dependent steps [lu2013]. Glutamate-cysteine ligase (GCL, the rate-limiting enzyme; previously known as γ-glutamylcysteine synthetase) catalyzes the formation of γ-glutamyl-cysteine from L-glutamate and L-cysteine. Glutathione synthetase then ligates glycine to form the tripeptide. Cysteine availability typically limits the rate of synthesis, which is why N-acetylcysteine (a cell-permeant cysteine precursor) is used clinically when accelerated GSH replenishment is needed [atkuri2007]. Glycine availability is a secondary constraint identified in human metabolic studies; combined glycine + cysteine supplementation restores deficient GSH synthesis in older adults and in

patients with uncontrolled diabetes [sekhar2011elderly, sekhar2011diabetes], a finding that motivated the GlyNAC (glycine + N-acetylcysteine) precursor strategy now in randomized clinical trials [kumar2021, kumar2023] [zhang2012].

The mitochondrial GSH pool is functionally distinct from the cytosolic pool [meister1995b]: GSH is synthesized only in the cytosol but is transported into mitochondria by dedicated carriers, where it serves as the principal antioxidant against the high reactive-oxygen-species flux of oxidative phosphorylation [zhang2012]. Depletion of mitochondrial GSH is a common feature of cellular injury and apoptosis induction [sies2019].

Plasma membrane glutathione transporters and gamma-glutamyl transpeptidase (GGT) together regulate extracellular GSH turnover [ballatori2009b] [zhang2012]. The 'gamma-glutamyl cycle' described by Meister couples extracellular GSH catabolism to amino-acid uptake and intracellular resynthesis [meister1995a]. In vivo, infused GSH has a short plasma half-life, Aebi and colleagues documented rapid post-infusion declines following IV doses with concurrent elevation of plasma and urinary cyst(e)ine, consistent with GGT-mediated catabolism at the renal and hepatic vascular beds [aebi1991].

Intestinal absorption of intact glutathione is limited; in rat small intestine, sodium-independent transport is regulated by alpha-adrenergic input [hagen1991]. In humans, single oral doses up to 3 g did not raise plasma glutathione meaningfully in a controlled PK study [witschi1992], establishing the rationale for liposomal encapsulation [sinha2018], sublingual delivery [schmitt2015], and orobuccal formulations [buonocore2016] that bypass first-pass intestinal hydrolysis [zhang2012].

In the brain, astrocytes synthesize and export GSH precursors used by neurons for their own GSH synthesis [aoyama2021]; this astrocyte-to-neuron support is a recognized neuroprotective mechanism and has motivated interest in glutathione (and its precursors) in neurodegeneration research [zhang2012]. Intranasal delivery exploits the olfactory and trigeminal pathways to bypass first-pass metabolism and the blood-brain barrier; Mischley and colleagues documented CNS uptake of intranasal glutathione in Parkinson disease patients using brain MRS [mischley2016].

Oxidative stress as a unifying concept, defined as a disruption of redox signaling and control, encompasses the biology that connects glutathione status to disease [sies2017] [zhang2012]. The GSH:GSSG ratio remains the most widely used single biomarker of cellular redox state, though its interpretation requires attention to compartmentalization between cytosolic, mitochondrial, and extracellular pools.

Glutathione Research History

Glutathione was isolated and characterized as a sulfur-containing peptide by F. Gowland Hopkins in the early 1920s. Decades of biochemical work characterized its synthesis, redox cycling, conjugation chemistry, and the gamma-glutamyl cycle described by Alton Meister and colleagues from the 1960s onward [meister1995a, meister1995b].

Human pharmacokinetics of exogenous glutathione were first characterized in the early 1990s. Aebi and colleagues reported high-dose intravenous glutathione PK and downstream effects on plasma and urine cyst(e)ine in adult volunteers [aebi1991]. Witschi and colleagues evaluated the systemic availability of oral glutathione, finding that single oral doses up to 3 g did not raise plasma glutathione meaningfully, a key result that motivated subsequent work on liposomal and sublingual formulations [witschi1992]. Mechanistic absorption studies in animals characterized intact-tripeptide transport across the small intestine and the role of alpha-adrenergic regulation [hagen1991].

Clinical investigation as an exogenous therapy began shortly after. A small open-label trial in early Parkinson disease [sechi1996] reported symptomatic improvement on UPDRS following intravenous glutathione, prompting follow-up work. A subsequent placebo-controlled crossover RCT failed its primary endpoint: while both arms improved, the between-group difference was not statistically significant [hauser2009], an important honest negative result. Two intranasal Parkinson trials by Mischley and colleagues (2015 phase I/IIa [mischley2015], 2017 phase IIb [mischley2017]) extended that work to a non-invasive route, with both glutathione and placebo arms improving similarly [aoyama2021]. A separate pharmacologic study confirmed CNS uptake of intranasal glutathione [mischley2016] [wu2004; ballatori2009a].

Outside Parkinson research, an open-label NAFLD pilot evaluated oral glutathione 300 mg daily over four months [honda2017]. Earlier descriptive work documented disturbances in trace elements and oxidative-stress markers in chronic nonalcoholic liver disease [loguercio2001], and pediatric work characterized plasma cysteine/homocysteine elevations correlating with severity in pediatric NAFLD [pastore2014]. Healthy-adult oral supplementation studies have evaluated plasma and lymphocyte GSH biomarkers across capsule [allen2011, richie2015], liposomal [sinha2018], sublingual [schmitt2015], and orobuccal [buonocore2016] forms. Cystic fibrosis trials examined inhaled GSH for pulmonary outcomes [bishop2005, visca2008, roum1999], with a Cochrane review identifying limited high-quality long-term data [tam2013]. A separate nebulized-glutathione trial in mild asthma documented bronchoconstriction in some subjects, an important route-specific safety signal [marrades1997]. The aerosol delivery concept was first explored in idiopathic pulmonary fibrosis a decade earlier [borok1991] [sies2017].

Oncology supportive-care research evaluated intravenous glutathione as a chemoprotectant. A double-blind RCT in women receiving cisplatin for ovarian cancer reported reduced toxicity and improved quality of life [smyth1997], and a placebo-controlled RCT in colorectal cancer reported reduced oxaliplatin-induced peripheral neuropathy [cascinu2002]. Precursor strategies, N-acetylcysteine [atkuri2007], and combined glycine + cysteine supplementation, have been pursued in parallel. Sekhar and colleagues demonstrated that glutathione synthesis is deficient in older adults and in uncontrolled diabetes and can be restored by dietary cysteine + glycine [sekhar2011elderly, sekhar2011diabetes]; the GlyNAC formulation (glycine + N-acetylcysteine) advanced through a pilot [kumar2021] and a randomized clinical trial in older adults [kumar2023] reporting improvements in oxidative-stress and aging-hallmark biomarkers. A small autism trial of oral glutathione [kern2011] reported no significant clinical efficacy [sharma2022; lu2013].

Dermatology research has assessed oral, sublingual, and topical glutathione as a skin-lightening agent [arjinpathana2012; handog2016]. A 2018 critical review specifically addressed IV glutathione marketed for skin lightening, noting absence of high-quality efficacy data and significant safety concerns [sonthalia2018]; a 2025 systematic review came to similar conclusions [sarkar2025] [sonthalia2016]. Indian and Philippine regulatory authorities have explicitly warned against IV glutathione for cosmetic skin-lightening use.

Foundational and contemporary reviews of glutathione's role in cellular redox biology continue to inform the field [sonthalia2016].

FDA issued a 2019 alert describing endotoxin-related adverse events in patients who received compounded sterile injectable L-glutathione that had been prepared from an ingredient that was not suitable for sterile injectable compounding [fda2019glutathione]. The alert is a key reference for distinguishing compound-related literature from compounding-quality risks.

Glutathione Timeline

- **1920s** • Hopkins isolates and characterizes glutathione as a sulfur-containing peptide
- **1960s, 80s** Meister and colleagues characterize the gamma-glutamyl cycle, GSH synthesis enzymes, and mitochondrial GSH biology [meister1995a; meister1995b]
- **1991** • Aebi et al characterize high-dose IV glutathione pharmacokinetics in humans (Eur J Clin Invest); Borok et al publish aerosolized GSH study in idiopathic pulmonary fibrosis [aebi1991; borok1991]
- **1992** • Witschi et al publish 'The systemic availability of oral glutathione', foundational PK finding that single oral doses up to 3 g do not meaningfully raise plasma GSH [witschi1992]
- **1995** • Lomaestro & Malone publish a clinical pharmacotherapy review of glutathione in health and disease [lomaestro1995]
- **1996** • Sechi et al publish small open-label IV glutathione trial in early Parkinson disease [sechi1996]
- **1997** • Smyth et al publish double-blind RCT of IV glutathione as cisplatin chemoprotectant in ovarian cancer; Marrades et al document bronchoconstriction from nebulized GSH in mild asthma [smyth1997; marrades1997]
- **1999** • Roum et al demonstrate that aerosolized GSH suppresses lung epithelial surface oxidant production in cystic fibrosis [roum1999]
- **2002** • Cascinu et al publish randomized placebo-controlled trial of IV glutathione for oxaliplatin neuroprotection in colorectal cancer [cascinu2002]

2004 • Wu et al publish 'Glutathione metabolism and its implications for health' in J Nutrition [wu2004]

2005 • Bishop et al publish pilot study of inhaled buffered reduced glutathione in cystic fibrosis [bishop2005]

2007 • Atkuri et al publish 'N-Acetylcysteine, a safe antidote for cysteine/glutathione deficiency' (Curr Opin Pharmacol) [atkuri2007]

2009 • Hauser et al publish randomized double-blind crossover IV glutathione in Parkinson disease, primary endpoint not met [hauser2009]

2011 • Sekhar et al publish foundational stable-isotope studies showing deficient GSH synthesis in aging and uncontrolled diabetes, restored by cysteine + glycine; Kern et al publish small glutathione RCT in autism (no significant clinical effect) [sekhar2011elderly; sekhar2011diabetes; kern2011]

2012 • Arjinpathana & Asawanonda publish randomized placebo-controlled trial of oral glutathione as a skin-lightening agent in Thai adults [arjinpathana2012]

2009 • Ballatori et al publish reviews of GSH dysregulation in human diseases and plasma membrane GSH transporters [ballatori2009a; ballatori2009b]

2013 • Lu publishes 'Glutathione synthesis' review in Biochim Biophys Acta [lu2013]

2015 • Mischley et al publish phase I/IIa intranasal glutathione study in Parkinson disease [mischley2015]

2015 • Richie et al publish randomized controlled trial of oral glutathione supplementation in healthy adults [richie2015]

2017 • Mischley et al publish phase IIb intranasal glutathione study in Parkinson disease; Honda et al publish open-label oral glutathione pilot in NAFLD [mischley2017; honda2017]

2017 • Sies, Berndt & Jones publish 'Oxidative Stress' in Annual Review of Biochemistry, contemporary unifying framework [sies2017]

2018 • Sonthalia et al publish critical review of IV glutathione for skin lightening, 'a regnant myth or evidence-based verity?' [sonthalia2018]

2019 • FDA issues alert on compounded sterile injectable L-glutathione following endotoxin-related adverse events [fda2019glutathione]

2021 • Aoyama publishes 'Glutathione in the Brain' review in Int J Mol Sci; Kumar et al publish GlyNAC pilot in older adults [aoyama2021; kumar2021]

2023 • Kumar et al publish randomized clinical trial of GlyNAC (glycine + NAC) in older adults, improvements in glutathione status, oxidative stress, mitochondrial function, and aging-hallmark biomarkers [kumar2023]

2025 • Sarkar et al publish systematic review of glutathione as a skin-lightening agent in melasma [sarkar2025]

Clinical Contexts for Glutathione

Parkinson disease EMERGING

Studied in small clinical trials with mixed results; the only placebo-controlled IV RCT failed its primary endpoint.

Open-label and small randomized studies have investigated IV and intranasal glutathione as adjunctive therapy. The original Sechi 1996 open-label trial [sechi1996] reported UPDRS improvement in 9 patients with early Parkinson disease. The Hauser 2009 randomized double-blind crossover [hauser2009] found no statistically significant difference vs placebo on UPDRS, an important honest negative result that tempered enthusiasm. Mischley 2015 phase I/IIa [mischley2015] and 2017 phase IIb [mischley2017] studies of intranasal glutathione both showed improvement in active and placebo arms with no statistically significant between-group difference. A pharmacologic study by the same group documented CNS uptake of intranasal glutathione by MRS [mischley2016], establishing route feasibility independent of efficacy. Effect sizes across the literature are modest and inconsistent; no large pivotal trial has demonstrated efficacy.

Nonalcoholic fatty liver disease EMERGING

Early open-label evidence; no controlled replication.

An open-label single-arm Japanese pilot [honda2017] reported reduction in ALT and other liver markers with daily oral glutathione 300 mg over four months in 29 patients non-responsive to lifestyle intervention. Earlier descriptive work documented disturbances in trace-element and oxidative-stress markers in chronic nonalcoholic liver disease [loguercio2001], and a pediatric NAFLD cohort showed plasma cysteine and homocysteine elevations correlating with severity of liver damage [pastore2014]. Controlled replication of the open-label pilot in a larger sample has not been completed.

Cystic fibrosis (pulmonary) EMERGING

Mixed evidence; Cochrane review identified limited high-quality long-term data.

Inhaled glutathione has been studied in cystic fibrosis based on the observation that lung epithelial lining fluid in CF is glutathione-deficient. Roum and colleagues [roum1999] reported that aerosolized glutathione suppresses oxidant production by lung epithelial surface inflammatory cells in CF. A pilot study by Bishop and colleagues [bishop2005] reported clinical-status improvement with inhaled buffered reduced glutathione, and a follow-up unblinded study by Visca and colleagues [visca2008] observed improvement in selected clinical markers. A 2013 Cochrane review [tam2013] of nebulized and oral thiol derivatives (including glutathione) for cystic fibrosis identified some short-term lung-function effects but limited high-quality long-term data.

Asthma (inhaled GSH safety signal) EMERGING

Safety-relevant route-specific finding outside CF.

Marrades and colleagues [marrades1997] reported that nebulized glutathione induced bronchoconstriction in some patients with mild asthma. This is a route-specific safety signal that does not invalidate inhaled GSH use in CF (where the underlying epithelial physiology differs) but does constrain extrapolation to asthma and other airway populations. The 1991 Borok et al. trial of aerosolized GSH in idiopathic pulmonary fibrosis [borok1991] established earlier pulmonary delivery feasibility.

Oncology supportive care (chemoprotection) EMERGING

Moderate-quality RCT evidence for cisplatin and oxaliplatin toxicity reduction; not standard of care.

Smyth and colleagues [smyth1997] reported a double-blind randomized trial of IV glutathione in women receiving cisplatin for ovarian cancer; glutathione was associated with reduced toxicity and improved quality of life without compromising tumor response. Cascinu and colleagues [cascinu2002] reported a placebo-controlled RCT of IV glutathione in patients receiving oxaliplatin-based therapy for advanced colorectal cancer, demonstrating reduced oxaliplatin-induced peripheral neuropathy. These are among the strongest controlled efficacy signals in the glutathione clinical literature, but the findings have not driven changes in standard oncology practice and theoretical concerns about antioxidant interference with chemotherapy oxidative mechanisms remain.

Glutathione precursor strategies (NAC and GlyNAC) WELL STUDIED

Precursor supplementation raises body GSH in deficiency states; clinical outcomes still under study.

N-acetylcysteine (NAC) is the clinically established cysteine precursor and remains the FDA-approved antidote for acetaminophen overdose [atkuri2007]; its dose-limiting use in acetaminophen toxicity is the mechanistic and regulatory proof that pharmacologic GSH precursor supplementation reaches the liver in clinically meaningful quantities. Sekhar and colleagues identified that glutathione synthesis is also glycine-limited in older adults [sekhar2011elderly] and in patients with uncontrolled diabetes [sekhar2011diabetes], and that combined cysteine + glycine supplementation restored GSH synthesis rates and lowered oxidative-stress biomarkers. Building on that work, Kumar and colleagues advanced the GlyNAC formulation (glycine + NAC) through a pilot [kumar2021] and a randomized clinical trial in older adults [kumar2023], reporting improvements in glutathione status, oxidative-stress markers, mitochondrial function, inflammation, and several aging-related physical-function endpoints.

Autism spectrum disorder EMERGING

Small RCT did not demonstrate clinical efficacy.

Kern and colleagues [kern2011] conducted a small clinical trial of oral and transdermal glutathione supplementation in children with autism spectrum disorder. The trial did not demonstrate statistically significant clinical efficacy on autism-specific outcomes; the study is cited primarily as a documentation of negative findings in this population.

Healthy-adult biomarker studies WELL STUDIED

Route-specific evidence of measurable change in body GSH stores.

Randomized trials in healthy adults have evaluated standard oral capsule [allen2011, richie2015], oral liposomal [sinha2018], sublingual [schmitt2015], and orobuccal [buonocore2016] glutathione. These studies report measurable elevations in plasma, erythrocyte, or lymphocyte GSH and in some markers of oxidative stress, with magnitudes that vary by formulation. The clinical relevance of these biomarker shifts in otherwise healthy adults is not established.

Dermatology (skin lightening) EMERGING

Studied in dermatology research; regulatory and ethical framing requires care.

Oral, sublingual, topical, and (controversially) IV glutathione have been investigated for skin-lightening and melasma effects. A small Thai oral RCT [arjinpathana2012] reported modest skin-lightening effects with 500 mg/day oral glutathione for 4 weeks. An open-label single-arm Filipino study [handog2016] reported similar effects with a novel oral preparation. A pharmacokinetic-focused orobuccal trial [sharma2022] documented enhanced absorption via mucosa. The 2018 critical review of IV glutathione for skin lightening [sonthalia2018] catalogued the regulatory and safety concerns, including FDA enforcement against IV skin-lightening products and Philippine and Indian regulatory warnings, and concluded that the IV-glutathione skin-lightening market is not supported by high-quality efficacy evidence and operates outside appropriate compounded-medication use [sonthalia2016]. A 2025 systematic review [sarkar2025] reached similar conclusions. RonanRx does not support compounded IV glutathione for cosmetic skin lightening.

Oxidative stress and antioxidant support WELL STUDIED

Mechanism well characterized; clinical outcome data limited.

Glutathione's role as the principal cellular antioxidant is established through decades of biochemical research [lu2013; wu2004; lomaestro1995; forman2014]. Whether exogenous administration meaningfully changes clinical outcomes in oxidative-stress conditions remains an active research question, the field has consistent biomarker data but limited patient-relevant outcomes data outside the specific indications listed above [ballatori2009a; sies2017; phamhuy2008; forman2016].

Compounded Glutathione (503A)

Compounded glutathione is dispensed under 503A on a patient-specific prescription. Common preparations include intravenous and intramuscular injectable solutions, intranasal sprays, oral liposomal capsules, sublingual troches or sprays, and nebulized solutions for inhaled use.

RonanRx prescribes glutathione only when the prescribing doctor identifies a patient-specific clinical need that an FDA-approved product is not medically appropriate to address. Compounded glutathione is not promoted as a generic substitute for any FDA-approved drug, and is not sold direct-to-consumer [fda503a].

CHEMISTRY

MOLECULAR FORMULA
$C_{10}H_{17}N_3O_6S$

MOLECULAR WEIGHT
307.32 g/mol

CAS NUMBER
70-18-8

PLASMA HALF-LIFE
~10 min (reduced GSH, IV)

Glutathione Formulations and Routes

Form	Concentration	Description
Injectable solution	200 mg/mL	IV, IM, or SC administration; preservative-free single-dose vials prepared under USP <797> sterile-compounding standards
Intranasal spray	100 mg/mL	Metered-dose nasal spray; route used in published Parkinson disease research
Oral liposomal capsule	500 mg	Phospholipid-encapsulated capsule; designed to reduce gastrointestinal hydrolysis
Sublingual troche / spray	varies	Sublingual administration; evaluated against oral capsule and NAC in head-to-head bioavailability work
Nebulizer solution	200 mg/mL	Sterile solution for use in compatible nebulizers; route used in cystic fibrosis research
Oral capsule (non-liposomal)	250, 1000 mg	Standard oral capsule; route used in healthy-adult RCT

Routes used in published literature: intravenous, intramuscular, subcutaneous, intranasal, oral, sublingual, inhalation, topical.

Glutathione Dosing

Route	Population	Range	Duration	Study type
intravenous	adults (Parkinson trials)	600, 1400 mg per administration	30 days to 3 times weekly for 4 weeks	open-label and small RCT
intranasal	adults (Parkinson trials)	100, 600 mg per day, divided doses	12 weeks	phase I/IIa and phase IIb RCT
oral capsule	healthy adults	250 mg or 1000 mg daily	6 months	randomized double-blind placebo-controlled
oral capsule	adults (NAFLD pilot)	300 mg daily	16 weeks	open-label single-arm
oral liposomal	healthy adults		4 weeks	pilot randomized

Route	Population	Range	Duration	Study type
		500 mg or 1000 mg daily		
sublingual	healthy adults	various; head-to-head vs oral and NAC	weeks	randomized comparison
inhalation (nebulized)	cystic fibrosis	varied per protocol	weeks to months	pilot and unblinded clinical studies

Doses listed reflect published clinical-trial protocols, not RonanRx prescribing recommendations. The prescribing doctor selects route, dose, and frequency based on the patient's clinical context, indication, and goals.

Doses listed should not be presented to patients as instructions. Patient instructions originate from the prescribing physician's prescription, not from this educational page.

Glutathione Safety

Across published clinical trials, exogenous glutathione has been generally well-tolerated at studied doses. Reported adverse effects have been mild and infrequent and include nausea, lightheadedness, and rare hypersensitivity reactions with parenteral administration.

Safety considerations for compounded glutathione fall into two distinct categories that should not be conflated [125]. The first is **compound-related safety**: the literature signal for the molecule itself, summarized below. The second is **compounding-quality safety**: the FDA's 2019 alert documented endotoxin-related adverse events in patients who received compounded sterile injectable L-glutathione prepared from an ingredient that was not suitable for sterile injectable compounding [52]. That alert is a quality-of-preparation signal, not a compound-toxicity signal, and motivates the operational discipline (USP <797> compliance, sterility testing, lot traceability, ingredient suitability) covered in the compounding section below.

Contraindications

Honest gap. No formal contraindications established for compounded glutathione at typical clinical-trial doses. Documented hypersensitivity to any component of a particular preparation is a relative contraindication for parenteral use of that preparation.

Searched: PubMed, DailyMed on 2026-05-07 · terms *glutathione contraindications; intravenous glutathione adverse reactions*.

Drug interactions

Honest gap. No clinically significant drug-drug interactions established. Theoretical concern that high-dose antioxidants may interfere with chemotherapy oxidative mechanisms; oncology patients should consult their treating physician before starting any glutathione preparation.

Searched: PubMed, DailyMed on 2026-05-07 · terms *glutathione drug-drug interactions*.

Adverse events

Across reviewed clinical trials, adverse events were mild and uncommon. Hypersensitivity reactions to IV glutathione have been reported in the broader literature; preservative-free sterile formulations are preferred for parenteral use.

FDA documented endotoxin-related reactions (nausea, vomiting, lightheadedness, chills, body aches, sneezing, hypotension, dyspnea, sudden chills, fever, shaking) in patients who received compounded IV L-glutathione 1400 mg or 2400 mg from preparations later found to contain excessive bacterial endotoxin from an ingredient that was not suitable for sterile injectable compounding [52]. These events are not intrinsic to glutathione; they reflect failures of compounded-sterile-preparation quality control [2].

Monitoring Glutathione Therapy

Routine laboratory monitoring is not required for typical compounded use. The prescribing doctor may monitor disease-specific markers (for example, ALT in liver indications, UPDRS scoring in Parkinson clinical follow-up) at clinically appropriate intervals.

Patients receiving sterile parenteral glutathione should report any signs of acute hypersensitivity (rash, dyspnea, hypotension) or post-infusion symptoms (chills, fever, body aches, lightheadedness) to the prescribing physician promptly, particularly given the FDA-documented endotoxin-related adverse-event pattern [honda2017; fda2019glutathione].

Glutathione Evidence Quality

The biochemistry of glutathione synthesis, redox cycling, conjugation, and inter-tissue handling is well established by decades of biochemical and cell-biology research [lu2013; wu2004; ballatori2009a]. Clinical evidence for exogenous administration is more variable [sonthalia2016]. Most clinical trials are small, often open-label, and use crossover or short-duration designs [schmitt2015]. Larger, well-controlled trials with patient-relevant outcomes are needed before firm efficacy conclusions can be drawn for any clinical indication [buonocore2016].

Evidence by indication: Parkinson disease, the original Sechi 1996 open-label trial [sechi1996] reported UPDRS improvement, but the only placebo-controlled RCT [hauser2009] did not meet its primary

endpoint, and two intranasal trials [mischley2015, mischley2017] showed equivalent improvement in glutathione and placebo arms. Net: no positive pivotal trial. NAFLD, single open-label pilot [honda2017], plus descriptive oxidative-stress/sulfur-amino-acid work [loguercio2001, pastore2014]; no controlled replication. Cystic fibrosis, pilot and open-label studies [bishop2005, visca2008, roum1999] plus a Cochrane review [tam2013] identifying limited high-quality long-term data; a separate trial of nebulized GSH in mild asthma documented bronchoconstriction in some subjects [marrades1997], a route-specific safety signal that is relevant when extrapolating inhaled GSH outside CF populations. Healthy-adult biomarker studies, multiple randomized trials demonstrate route-dependent measurable changes in body GSH stores; clinical relevance of those changes in healthy adults is not established [arjinpathana2012; richie2015; sinha2018]. Oncology supportive care, moderate-quality RCTs report reduced cisplatin toxicity [smyth1997] and reduced oxaliplatin neuropathy [cascinu2002]; these are the strongest controlled efficacy signals in the literature but have not driven changes in standard oncology practice [allen2011]. Precursor strategies, randomized data on glycine + cysteine [sekhar2011elderly, sekhar2011diabetes] and the GlyNAC formulation [kumar2021, kumar2023] show consistent biomarker effects in deficiency states. Autism, a small RCT [kern2011] did not demonstrate clinical efficacy [handog2016; sharma2022]. Dermatology, modest, often transient effects in skin-lightening research; the 2018 critical review [sonthalia2018] and 2025 systematic review [sarkar2025] both conclude the cosmetic compounded-IV market sits outside appropriate use of compounded medications and is not supported by high-quality efficacy evidence [sies2017].

Major Glutathione Clinical Studies

Study	Design	Participants	Duration	Finding
Reduced intravenous glutathione in the treatment of early Parkinson disease	Open-label	9	30 days IV; ~2, 4 month follow-up	Reported symptomatic improvement on UPDRS that persisted after treatment cessation; no control arm [sechi1996].
Randomized, double-blind, pilot evaluation of intravenous glutathione in Parkinson disease	Randomized double-blind crossover	21	4 weeks active; washout	Trend toward improvement on UPDRS that did not reach statistical significance vs placebo [hauser2009].
A randomized, double-blind phase I/IIa study of intranasal glutathione in Parkinson disease	Randomized double-blind	varied	weeks	Established safety and feasibility of intranasal route; preliminary efficacy signal [mischley2015].

Study	Design	Participants	Duration	Finding
Phase IIb intranasal glutathione study in Parkinson disease	Randomized double-blind	45	12 weeks	Both glutathione doses and placebo improved UPDRS-3; no statistically significant between-group difference [mischley2017].
Central nervous system uptake of intranasal glutathione in Parkinson disease	Pharmacologic / imaging	varied	single dose / short term	Demonstrated CNS uptake of intranasal glutathione, supporting feasibility of the delivery route [mischley2016].
Randomized controlled trial of oral glutathione supplementation in healthy adults	Randomized double-blind placebo-controlled	54	6 months	Daily oral glutathione (250 mg or 1000 mg) increased body stores of glutathione (blood, erythrocyte, lymphocyte, buccal cell) vs placebo [richie2015].
Oral supplementation with liposomal glutathione elevates body stores of glutathione and markers of immune function	Pilot randomized	12	4 weeks	Liposomal oral glutathione (500 or 1000 mg/day) elevated GSH markers and selected immune-function markers [sinha2018].
Effects of N-acetylcysteine, oral glutathione, and a novel sublingual form of glutathione on oxidative stress	Comparative human study	varied	weeks	Compared bioavailability and oxidative-stress effects of NAC, oral GSH, and a sublingual GSH formulation; sublingual route showed measurable plasma changes [schmitt2015].
Efficacy of glutathione for the treatment of nonalcoholic fatty liver disease (open-label pilot)	Open-label single-arm	29	4 months	Daily oral glutathione 300 mg associated with reductions in ALT and selected hepatic-fat markers in patients non-responsive to lifestyle intervention [honda2017].
	Pilot	varied	weeks	

Study	Design	Participants	Duration	Finding
Pilot study of inhaled buffered reduced glutathione in cystic fibrosis				Reported clinical-status improvement with inhaled buffered reduced glutathione [bishop2005].
Improvement in clinical markers in CF patients using a reduced glutathione regimen	Unblinded clinical	varied	weeks	Observed improvement in selected clinical markers [visca2008].
Nebulized and oral thiol derivatives for pulmonary disease in cystic fibrosis (Cochrane review)	Systematic review and meta-analysis	pooled	varied	Identified short-term lung-function effects of inhaled glutathione but limited high-quality long-term data; called for adequately powered trials [tam2013].
High-dose intravenous glutathione in man: pharmacokinetics and effects on cyst(e)ine in plasma and urine	Pharmacokinetic study	small healthy-volunteer cohort	single dose with serial sampling	Characterized rapid post-infusion decline in plasma GSH and concurrent elevation of plasma and urinary cyst(e)ine, consistent with GGT-mediated catabolism [aebi1991]. Foundational IV-PK reference.
The systemic availability of oral glutathione	Pharmacokinetic crossover	7 healthy adults	single dose	Single oral doses up to 3 g did not raise plasma glutathione meaningfully [witschi1992]. Key motivating result for liposomal, sublingual, and orobuccal formulation work.
Effects of oral glutathione supplementation on systemic oxidative stress biomarkers in human volunteers	Open-label trial	40	4 weeks	Oral glutathione 500 mg twice daily did not significantly change blood glutathione or oxidative-stress biomarkers in healthy adults, a negative result that complemented the Richie

Study	Design	Participants	Duration	Finding
				2015 RCT and Witschi 1992 PK findings [allen2011].
Bioavailability study of an innovative orobuccal formulation of glutathione	Pharmacokinetic crossover	20	single dose	Orobuccal (mucosal) glutathione formulation achieved measurable plasma elevations relative to standard oral capsule, supporting first-pass-bypass strategies [buonocore2016].
Glutathione reduces toxicity and improves quality of life in cisplatin-treated ovarian cancer (double-blind RCT)	Randomized double-blind placebo-controlled	151	6 cycles of cisplatin	IV glutathione reduced cisplatin toxicity and improved quality of life without compromising tumor response [smyth1997]. Strongest controlled efficacy signal in oncology supportive care literature.
Neuroprotective effect of reduced glutathione on oxaliplatin-based chemotherapy in advanced colorectal cancer	Randomized double-blind placebo-controlled	52	course of oxaliplatin-based chemotherapy	IV glutathione reduced oxaliplatin-induced peripheral neuropathy without compromising chemotherapy efficacy [cascinu2002].
Nebulized glutathione induces bronchoconstriction in patients with mild asthma	Pulmonary challenge study	small mild-asthma cohort	acute challenge	Nebulized GSH caused bronchoconstriction in mild-asthma subjects, a route-specific safety signal relevant when extrapolating inhaled GSH outside CF populations [marrades1997].
Glutathione aerosol suppresses lung epithelial surface inflammatory cell-derived oxidants in cystic fibrosis	Mechanistic clinical study	small CF cohort	acute and short-term	Demonstrated that aerosolized GSH reduces airway oxidant burden in CF, supporting the mechanistic rationale for the Bishop 2005 and Visca 2008 pilot work [roum1999].

Study	Design	Participants	Duration	Finding
Deficient synthesis of glutathione underlies oxidative stress in aging and can be corrected by dietary cysteine and glycine supplementation	Mechanistic clinical study with stable-isotope GSH-synthesis measurement	8 elderly + 8 young controls (initial cohort)	2 weeks supplementation	Older adults had deficient GSH synthesis rates which were normalized by 2 weeks of cysteine + glycine supplementation, with concurrent reduction in oxidative-stress biomarkers [sekhar2011elderly]. Foundational paper for precursor strategies in aging.
Glutathione synthesis is diminished in patients with uncontrolled diabetes and restored by dietary supplementation with cysteine and glycine	Mechanistic clinical study	12 diabetes + 12 controls	2 weeks supplementation	Patients with uncontrolled type 2 diabetes had deficient GSH synthesis which was restored by combined cysteine + glycine supplementation, with parallel reductions in oxidative-stress markers [sekhar2011diabetes].
GlyNAC supplementation in older adults: randomized clinical trial of glycine + N-acetylcysteine on aging hallmarks	Randomized clinical trial	24	16 weeks	GlyNAC supplementation in older adults improved glutathione deficiency, oxidative stress, mitochondrial dysfunction, inflammation, insulin resistance, and physical-function and cognitive measures vs control [kumar2023]. Largest controlled trial of a glutathione-precursor strategy to date.
Glycine and N-acetylcysteine (GlyNAC) pilot in older adults	Open-label pilot	8	24 weeks	Pilot data supporting the GlyNAC randomized trial; documented restoration of GSH synthesis and improvement in multiple oxidative-stress biomarkers and aging hallmarks [kumar2021].

Study	Design	Participants	Duration	Finding
Clinical trial of glutathione supplementation in autism spectrum disorders	Small clinical trial (oral and transdermal arms)	26	8 weeks	Oral and transdermal glutathione increased plasma cysteine and sulfate but did not produce statistically significant improvement on autism-specific clinical outcomes [kern2011].
Glutathione as an oral whitening agent: randomized, double-blind, placebo-controlled study	Randomized double-blind placebo-controlled	60	4 weeks	Oral glutathione 500 mg/day produced modest skin-lightening effects vs placebo in Thai adults; effect size was small and transient [arjinpathana2012]. Frequently cited Thai-population reference in dermatology literature.
Open-label trial of a novel oral glutathione preparation as a skin-lightening agent in Filipino women	Open-label single-arm	30	12 weeks	Reported skin-lightening effects with a novel oral preparation [handog2016]. Cited in the regulatory context that drove Philippine FDA warnings about IV-glutathione skin-lightening products.

Glutathione Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

Intravenous glutathione has a short plasma half-life on the order of 10 minutes due to rapid hydrolysis by gamma-glutamyl transpeptidase at the renal and hepatic vascular beds; the canonical human IV-PK reference is Aebi and colleagues [aebi1991], who documented rapid post-infusion plasma decline with concurrent rise in plasma and urinary cyst(e)ine. Intracellular glutathione has a much longer turnover, hours to days depending on tissue [lu2013].

Standard oral glutathione bioavailability has historically been considered very low because of gastrointestinal hydrolysis and limited intestinal transport of the intact tripeptide. The foundational human PK study [witschi1992] showed that single oral doses up to 3 g did not raise plasma glutathione

meaningfully. Mechanistic absorption work in animals characterized intact-tripeptide transport across the small intestine [hagen1991]. Subsequent randomized trials of standard capsule [allen2011, richie2015], liposomal [sinha2018], sublingual [schmitt2015], and orobuccal [buonocore2016] forms have shown measurable but route-dependent elevations in body GSH stores, with the Allen 2011 trial of unencapsulated oral GSH being a notable negative result that reinforces the Witschi finding.

Intranasal glutathione has been shown to enter the central nervous system in pharmacologic studies in Parkinson disease populations [mischley2016], supporting the feasibility of bypassing first-pass and gut metabolism for CNS-targeted use. Inhaled (nebulized) delivery achieves direct airway exposure and has been studied in cystic fibrosis [roum1999, bishop2005] and historically in pulmonary fibrosis [borok1991]; the asthma bronchoconstriction signal [marrades1997] is a reminder that pulmonary delivery is not pharmacokinetically interchangeable across airway populations.

Pharmacodynamics

Pharmacodynamic effects depend on the target tissue's ability to take up exogenous GSH or its hydrolysis products (cysteine, glutamate, glycine) and resynthesize intracellular glutathione [lu2013, wu2004] [sekhar2011elderly].

Direct GSH supplementation, NAC (cysteine donor), and other precursor strategies all converge on raising tissue GSH but differ in distribution and redox kinetics [atkuri2007]. NAC's clinical proof-of-mechanism is its FDA-approved use as an antidote for acetaminophen toxicity, where it replenishes hepatic GSH stores depleted by NAPQI conjugation. The combined cysteine + glycine (and the GlyNAC formulation) precursor data in older adults and diabetes establish that glycine availability is a secondary rate-limit in some deficiency states [sekhar2011diabetes; kumar2021; kumar2023]. The choice between direct GSH and precursor strategies is route- and indication-dependent and is the prescribing physician's clinical decision.

↓↑ Comparing Glutathione Formulations

Standard oral capsules, liposomal oral capsules, and sublingual forms differ in absorption pathway [sinha2018]. Liposomal encapsulation is designed to protect the tripeptide from gastrointestinal hydrolysis; sublingual delivery aims to bypass first-pass metabolism. Head-to-head studies [schmitt2015] and standalone trials [richie2015] report measurable but route-dependent body-GSH elevations.

Parenteral routes (IV, IM, SC) produce rapid plasma elevations but with short plasma half-life, since gamma-glutamyl transpeptidase efficiently hydrolyzes circulating GSH. Intranasal delivery has been characterized as a CNS-targeted alternative to IV in Parkinson research, with documented CNS uptake in Mischley 2016 [mischley2016].

Inhaled (nebulized) glutathione targets airway lining fluid in pulmonary indications such as cystic fibrosis [bishop2005] [tam2013]. The 2013 Cochrane review concluded that high-quality long-term data remain limited.

RonanRx supports formulations consistent with documented clinical literature and 503A scope [visca2008]. The selection of formulation for a given patient is the prescribing doctor's decision.

Glutathione Storage and Handling

Compounded glutathione injectable solutions are typically stored refrigerated (2, 8 °C) and protected from light to minimize oxidation of the thiol group. Beyond-use dates depend on the formulation and preservative system; refer to the dispensing pharmacy's labeling for the specific preparation received.

Oral capsules and sublingual forms are stored per package labeling, typically at controlled room temperature with desiccant where indicated. Inhalation solutions follow the same refrigeration and light-protection considerations as injectables.

Glutathione Compounding & Operations

503A compounding

RonanRx prepares glutathione under 503A on a patient-specific prescription written by a licensed prescribing physician for an identified patient, consistent with section 503A of the Federal Food, Drug, and Cosmetic Act [fda503a].

Bulk drug substance is sourced from FDA-registered API suppliers, and ingredient suitability for the intended formulation pathway (including suitability for sterile injectable compounding when applicable) is verified before use. Finished sterile preparations follow USP <797> standards. This process is the operational answer to the FDA 2019 glutathione alert: the alert documented harm from preparations whose ingredient was not suitable for sterile injectable compounding, and the appropriate response is rigorous ingredient and sterility control rather than withdrawal from the legitimate 503A scope [fda2019glutathione].

Pharmacist review

Each prescription is reviewed by a licensed pharmacist before dispensing. Review covers prescribed strength, route, formulation suitability, patient-specific contraindications based on the prescription record, ingredient compatibility, and label accuracy.

The pharmacist also confirms the prescribed indication is consistent with 503A use rather than direct-to-consumer or office-stock distribution [fda503a].

Quality and traceability

Every compounded preparation carries a lot number tied to the prescription record. Sterility, potency, and endotoxin testing for sterile preparations follow USP <797> guidelines, with documentation retained per

state board of pharmacy requirements [fda2019glutathione]. Endotoxin testing in particular addresses the failure mode documented in FDA's 2019 alert.

Cold chain

Injectable and inhalation glutathione preparations ship in temperature-controlled packaging with a temperature indicator. Patients are advised to refrigerate immediately on receipt and to contact the pharmacy if temperature integrity is in doubt.

Frequently Asked Questions About Glutathione

Is glutathione FDA-approved?

No. Glutathione itself is not FDA-approved as a drug for any specific indication [fda503a]. The closely related N-acetylcysteine (NAC), a glutathione precursor, has FDA-approved use in acetaminophen overdose. Compounded glutathione is dispensed under 503A on patient-specific prescriptions [atkuri2007]. Compounded drugs are not FDA-approved.

What conditions has glutathione been studied in?

Published clinical investigation has focused on Parkinson disease (small RCTs with mixed results), nonalcoholic fatty liver disease (one open-label pilot), cystic fibrosis (pilot studies and a Cochrane review), healthy-adult biomarker studies (multiple RCTs), and dermatology-related skin-lightening contexts [sechi1996; hauser2009; mischley2017; tam2013]. Trial sizes are mostly small and results are mixed; cellular biochemistry is well characterized [richie2015; sonthalia2018] [honda2017].

How is glutathione administered?

Routes used in clinical trials and 503A compounding include intravenous, intramuscular, subcutaneous, intranasal, oral (standard and liposomal capsules), sublingual, inhaled (nebulized), and topical [richie2015; sinha2018; schmitt2015]. The prescribing doctor selects the route based on clinical context [mischley2017; bishop2005].

Why is oral glutathione thought to be poorly absorbed?

Standard oral glutathione is broken down by gastrointestinal enzymes before absorption, and intestinal transport of the intact tripeptide is limited [allen2011; richie2015; sinha2018]. Liposomal encapsulation and sublingual forms have been studied as workarounds, with measurable but route-dependent body-GSH elevations in randomized trials [schmitt2015].

Is compounded IV glutathione safe?

Compound-related safety has been generally good across clinical trials. However, FDA documented endotoxin-related adverse events in patients who received compounded sterile injectable L-glutathione that had been prepared from an ingredient unsuitable for sterile injectable compounding [fda2019glutathione]. Those events are a quality-of-preparation signal, not a compound-toxicity signal. The appropriate response is rigorous ingredient suitability, sterility, endotoxin testing, and lot traceability, covered in the operations section above.

Does RonanRx sell glutathione directly to patients?

No. Compounded glutathione, if dispensed, requires a patient-specific prescription written by a licensed doctor for an identified patient and a route-specific feasibility review by a pharmacist. RonanRx is not a direct-to-consumer storefront for glutathione or any other compounded substance [fda503a].

What about glutathione precursors, NAC and GlyNAC?

N-acetylcysteine (NAC) is a cysteine precursor with FDA-approved use as an antidote for acetaminophen overdose; that use is mechanistic proof that pharmacologic GSH precursor supplementation reaches the liver in clinically meaningful quantities [atkuri2007]. Sekhar and colleagues identified that combined glycine + cysteine supplementation restores deficient GSH synthesis in older adults and in patients with uncontrolled diabetes [sekhar2011elderly; sekhar2011diabetes]. The GlyNAC formulation (glycine + NAC) has been studied in a pilot and a randomized clinical trial in older adults reporting improvements in glutathione status, oxidative-stress markers, mitochondrial function, and aging-hallmark biomarkers [kumar2021; kumar2023]. Whether to use direct GSH or a precursor strategy is a clinical decision for the prescribing physician.

Has glutathione been studied as a chemoprotectant?

Yes. Two notable randomized double-blind placebo-controlled trials reported reduced toxicity of platinum-based chemotherapy with IV glutathione: Smyth et al (1997) in cisplatin-treated ovarian cancer and Cascinu et al (2002) in oxaliplatin-based therapy for colorectal cancer [smyth1997; cascinu2002]. These remain among the strongest controlled efficacy signals in the glutathione clinical literature, but they have not driven changes in standard oncology practice, and theoretical concerns about antioxidant interference with chemotherapy oxidative mechanisms remain. Oncology patients should consult their treating physician before starting any glutathione preparation.

Is IV glutathione safe for skin lightening?

No high-quality evidence supports IV glutathione for skin lightening, and significant safety concerns exist. The 2018 critical review by Sonthalia and colleagues cataloged regulatory warnings, including FDA enforcement against IV skin-lightening products and Philippine and Indian regulatory warnings, and concluded that the IV-glutathione skin-lightening market is not supported by high-quality efficacy evidence

and operates outside appropriate compounded-medication use. A 2025 systematic review reached similar conclusions. RonanRx does not support compounded IV glutathione for cosmetic skin lightening [sonthalia2018; sarkar2025].

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How to Access Glutathione

Compounded Glutathione is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed Glutathione, sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)