

Glutathione Available for Research Use Only

SC 200–400 mg daily OR 300–600 mg every other day

Glutathione (GSH) is a tripeptide made from glutamate, cysteine, and glycine. It is an endogenous antioxidant with oral and IV clinical evidence. Some oral regimens increase glutathione stores. Common IV clinical and wellness-spa use is discussed for skin-lightening and liver health, but findings are limited. Routine subcutaneous (SC) use for wellness has no established validated dose. [3–11]

 SC plausible safe based on IV findings. [5,9,13,14]

 SC benefits are plausible based on IV findings.

Caution: Cancer or anticancer treatment warrants review with the treating oncology team. [3,15]

 Inconclusive for SC wellness use; evidence from other routes is summarized below. [4–11,13]

Disclaimer: These notes are not authoritative and should not be interpreted as definitive scientific guidance. They are summaries, compilations and plausible extrapolations drawn from the most relevant research I was able to locate, and may omit nuances, conflicting data, or emerging evidence. The material reflects an attempt to organize and understand complex topics, not to provide clinical recommendations or expert interpretation.

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Safety: Small oral and IV studies provide some short-term tolerability data, but the long-term safety of repeated SC injections remains uncertain. The body's normal use of glutathione does not establish the safety of an injected product. [5,9,13,14]

Energy, recovery, and immune support: Exercise-related fatigue reduction is plausible for SC use, based on indirect oral and animal findings. Immune-cell biomarker changes have been reported with oral dosing. [4,11]

Liver support: Improvement in liver-injury markers is plausible. The principal NAFLD pilot used oral glutathione and reported lower ALT, an enzyme marker of liver injury; its uncontrolled design does not establish a treatment effect or general “liver detoxification.” [8]

Skin lightening: Oral studies suggest a modest effect in some participants, with inconsistent results across sites and studies. These findings provide indirect support for a plausible SC effect. [9,10]

Dosage & Patterns

The following speculative doses and patterns are commonly discussed in peptide communities, but there is no validated clinical doses. Published pharmacy schedules vary. [17]

- **200–400 mg SC once daily**

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- **300–600 mg SC every other day**

Community and pharmacy context: As one documented practice example, Strive Pharmacy lists 200 mg SC one to two times weekly or 100 mg SC every other day. These are lower schedules than often discussed in peptide communities. [17].

Mechanistic rationale: An SC depot could spread absorption over time and lower peak exposure relative to an IV bolus. Repeated administration could replenish circulating glutathione or its metabolites, but the interval depends on PK unknowns: absorption, degradation, and tissue handling. [6,7]

Human study regimens for comparison:

- **Oral: 250–1,000 mg/day** in studies of glutathione stores, liver markers, skin pigmentation, or exercise-related fatigue. Treatment durations and populations differed. [2,4,8–11]
- **IV: 1,400 mg three times weekly for four weeks** in a small Parkinson disease trial; it did not show a significant symptomatic advantage over placebo. [5]

IV-to-SC extrapolation — illustrative, not validated: 1,400 mg IV three times weekly totals 4,200 mg/week, averaging 600 mg/day. [5]

Benefits

- **Increases measured glutathione stores with some oral regimens.** A six-month randomized trial in 54 healthy adults found increased glutathione in several measured compartments and improved whole-blood redox balance. A separate four-week trial found no significant benefit, and a single-dose study found no significant plasma increase. **Response depends on the regimen and endpoint measured.** [1,2,4]
- **Plausible improvement in liver-injury markers in NAFLD.** In an uncontrolled pilot, 29 completers with nonalcoholic fatty liver disease (NAFLD) had lower ALT after **300 mg/day orally for four months.** [8]
- **Plausible support for glutathione-dependent conjugation when glutathione availability is limiting.** Endogenous GSH participates in enzyme-mediated conjugation of electrophilic compounds. Supplemental benefit depends on whether it increases GSH where needed; this physiological role alone does not establish faster toxin removal or improved alcohol clearance. [3]
- **Plausible modest skin lightening with oral supplementation.** One 60-person trial of **500 mg/day orally for four weeks** found a greater reduction in melanin index than placebo at two of six sites. In another trial using 250 mg/day for 12 weeks, overall pigmentation comparisons were not significant, although a subgroup result favored reduced GSH. A reliable whole-body effect or lasting pigment change is not established. [9,10]

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- **Likely reduction in exercise-related fatigue with oral use.** Animal findings are supported by an eight-man crossover trial using **1 g/day orally for two weeks**, which reported lower exercise lactate and fatigue-related psychological scores. General recovery or treatment of chronic fatigue cannot be inferred from this small exercise study. [11]
- **Increased natural-killer-cell activity in one oral trial.** The high-dose arm of the six-month oral trial showed increased natural-killer-cell cytotoxicity at three months in a tested subset. This laboratory finding supports a plausible immune effect; it is not evidence of reduced infections. [4]

Duration for Functional Benefits

- **Plasma exposure:** IV concentrations fall rapidly. The duration of SC symptom benefits cannot be estimated from these IV measurements. [6,7]
- **Glutathione stores:** In the six-month oral trial, levels returned toward baseline after a one-month washout. This is a biomarker finding, not a defined duration of symptom relief. [4]
- **Skin effects:** The cited oral studies measured changes over four to twelve weeks. Their treatment periods do not establish how long any effect persists after stopping. [9,10]

Studied Uses

Studied uses include skin pigmentation, exercise-related fatigue, and liver markers in NAFLD. [1,2,4,8–11]. (No glutathione drug product is FDA-approved in the United States. [14])

Contraindications and Cautions

- **Cancer and anticancer therapy:** Effects on tumor redox biology and treatment response are context-dependent. Glutathione has also been studied under oncology supervision to reduce chemotherapy toxicity. Use alongside cancer treatment should be assessed by the treating oncology team for the specific regimen. [3,15]

Side Effects

- **Reported symptoms:** Nausea, dizziness, headache. [13,14]
- **Bronchospasm with nebulized glutathione:** A study in people with mild asthma demonstrated bronchoconstriction after inhalation. This is an inhalation-route finding. [12]
- **Hypersensitivity, including anaphylaxis:** Serious reactions have been reported with IV administration. Breathing difficulty, fainting, or signs of shock after injection require emergency care. [13,14]

Drivers of Diminished Response and Mitigation Strategies

Receptor desensitization is not the principal expected limitation for an antioxidant substrate.

Feedback regulation of synthesis and substrate availability can affect glutathione stores. [3,16]

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Biological Mechanism

Glutathione (GSH) is γ -glutamyl-cysteinyl-glycine, a major intracellular antioxidant and redox buffer. It supplies reducing equivalents to **glutathione peroxidases**, which reduce hydrogen peroxide to water and organic hydroperoxides to their corresponding alcohols, producing oxidized glutathione (**GSSG**). Glutathione reductase uses NADPH to recycle GSSG to GSH.

Glutathione S-transferases use GSH in conjugation reactions; glutathione also participates in protein-thiol redox regulation. [3] Cells synthesize GSH from its amino acid precursors, with feedback control at glutamate-cysteine ligase. [16] **IV administration raises plasma GSH transiently** and changes circulating thiol metabolites. This does not imply uniform uptake of intact glutathione into every tissue, including the brain, or equivalent exposure from SC dosing. [6,7] Clinical outcomes therefore depend on route, formulation, tissue delivery, and the condition being treated.

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