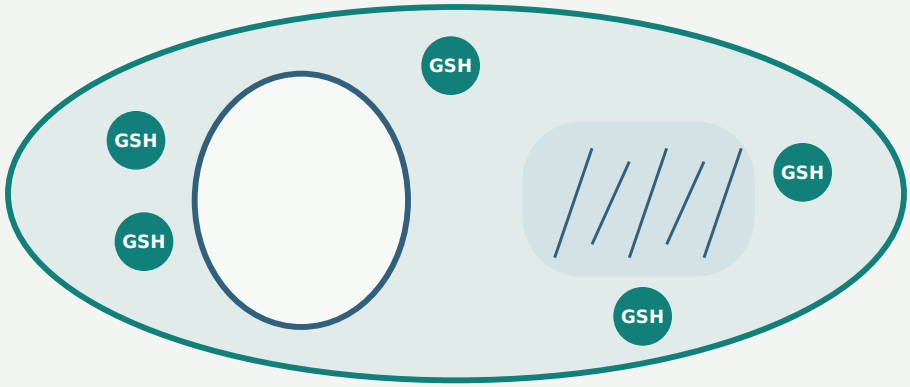
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The Glutathione Question

A central player in cellular redox biology. Decades of experiments. A surprising gap between biochemical mechanisms and clinical proof.

INSIDE A LIVING CELL



Conceptual cell illustration; not a microscopy image or a claim of injected-tissue delivery.

Membranes, organelles and local antioxidant systems depend on their own chemistry.

Glutathione | An exploration of its cellular biology, published human trials, animal experiments, surprising findings and unanswered questions.

Prepared by Vanguard Compound Research. Literature cut-off: 26 October 2026. This is a general educational review of the scientific literature, not evidence for any particular commercial product or formulation. It is not a peer-reviewed publication, medical advice or a clinical protocol. No human dosing or administration guidance is provided. This publication has not been reviewed or approved by Health Canada.

BEFORE THE FIRST STUDY

A familiar molecule. A difficult scientific question.

Every cell must manage a contradiction: oxygen helps it produce energy, yet the chemistry of life also creates reactive molecules capable of damaging proteins, membranes and DNA. Glutathione sits within one of the systems that help make this balancing act possible. [1]

That makes glutathione an unusually compelling subject. It is made by the body, constantly recycled, and indispensable to biochemical defense. Researchers have therefore asked an attractive question: if glutathione is important when cells are under stress, might giving extra glutathione protect tissues during disease or injury?

The answer turns out to be much more interesting than a simple yes or no. When investigators infused glutathione into healthy volunteers, blood concentrations rose sharply but faded surprisingly quickly. In some chemotherapy trials, patients experienced less treatment-related toxicity. In another, larger trial, the hoped-for protection did not appear. Parkinson disease research produced striking early observations followed by inconclusive controlled results. Animal studies revealed mechanisms that clinical experiments have not yet proved in people. [2-11,28]

> The most important distinction in this story is between what glutathione **does inside a cell** and what administering glutathione **can accomplish in a person**.

This review follows those discoveries in the order that makes them understandable. First we examine the chemistry, then the challenge of delivering a molecule to its target, and then the human experiments in which researchers placed their hypotheses at risk. Along the way, the science will remain clear about uncertainty: intriguing findings will be treated as intriguing, and negative findings will receive as much attention as positive ones.

HOW TO READ THIS PAPER

Studies are cited in brackets and linked in the source library at the end. A human placebo-controlled trial gives a different kind of evidence from an animal experiment, and a blood measurement gives a different kind of answer from a change in symptoms. Figures marked “conceptual” explain mechanisms; graphs based on studies state the specific outcome represented.

CHAPTER 01 / THE EVIDENCE STORY

The quiet chemistry that keeps cells alive

Before discussing injections, we need to understand why biologists became interested in glutathione in the first place.

Imagine a small workshop that never closes. It is constantly generating energy, building new components and dismantling worn-out ones. Some of that activity creates reactive chemical by-products. A living cell resembles this workshop: it needs regulated oxidation and reduction reactions, but excessive or misplaced oxidation can become destructive. Glutathione is one of the molecules that help keep these reactions in balance.

Chemically, glutathione is a tripeptide assembled from glutamate, cysteine and glycine. The sulfur-containing thiol group of cysteine is especially important because it can participate in electron-transfer reactions. In its reduced state it is called GSH; when two GSH molecules become linked by a disulfide bond, the product is oxidized glutathione, GSSG. Cells maintain this dynamic system using enzymes and the reducing power of NADPH. [1]

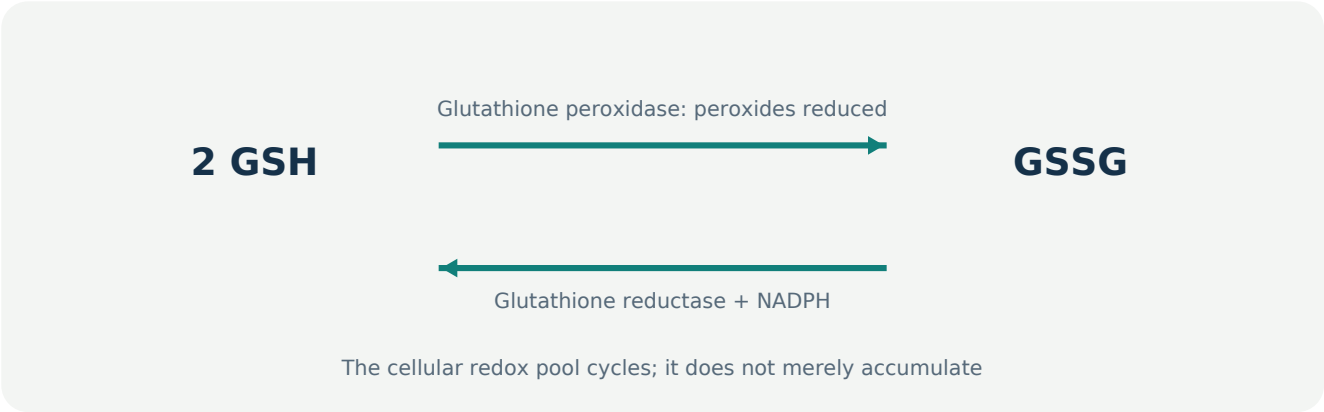


FIGURE 01. The GSH-GSSG cycle is a simplified view of a living, enzyme-controlled network. It is not a treatment pathway.

Why does this matter? Consider lipid membranes. Their delicate fatty components can undergo oxidative chain reactions. An enzyme known as glutathione peroxidase 4, or GPX4, helps prevent harmful accumulation of oxidized membrane lipids. In a landmark experimental paper, Yang and colleagues linked loss of glutathione-dependent defense and GPX4 activity to ferroptosis, a regulated form of cell death associated with iron and lipid oxidation. Their work established a mechanism with implications across disease biology, but did not test glutathione injections in people. [1]

The discovery makes glutathione biologically exciting because it is not merely a substance that neutralizes reactive chemicals in a test tube. It participates in pathways affecting whether a stressed cell survives. Yet this is also the point where interpretation must be careful. Cells require a balance, and the same defenses that help normal tissue resist injury may help some cancer cells survive stress. “More antioxidant” is not automatically better in every biological context. [1]

A USEFUL ANALOGY

A fire sprinkler can limit damage inside a building, but adding water somewhere outside the building is not the same as delivering it to the room where a fire has started. The location, timing and downstream effect matter. Likewise, extra glutathione in plasma is not identical to restoring a deficient pool inside a particular organelle.

CHAPTER 02 / THE EVIDENCE STORY

The injection paradox

Intravenous administration can overcome a delivery barrier - but leaves another scientific problem unsolved.

The natural next thought is straightforward: if swallowing glutathione is limited by digestive and metabolic processes, perhaps delivering it directly into blood makes more sense. For studying systemic exposure, the intravenous route indeed offers an important advantage. But an IV infusion answers the first question - what reaches the bloodstream - more clearly than it answers the later question of what benefits an organ or patient. [2,3,17]

In 1991, Aebi and colleagues infused glutathione into healthy volunteers and followed its plasma kinetics. Concentrations increased substantially during administration. Then came the surprising result: the mean elimination half-life was about 14 minutes, with considerable variability. A 2005 study in seven volunteers similarly described rapid disappearance and changes in related sulfur-containing metabolites. Those studies did not measure whether patients felt better; they measured the movement and processing of a molecule. [2,3]

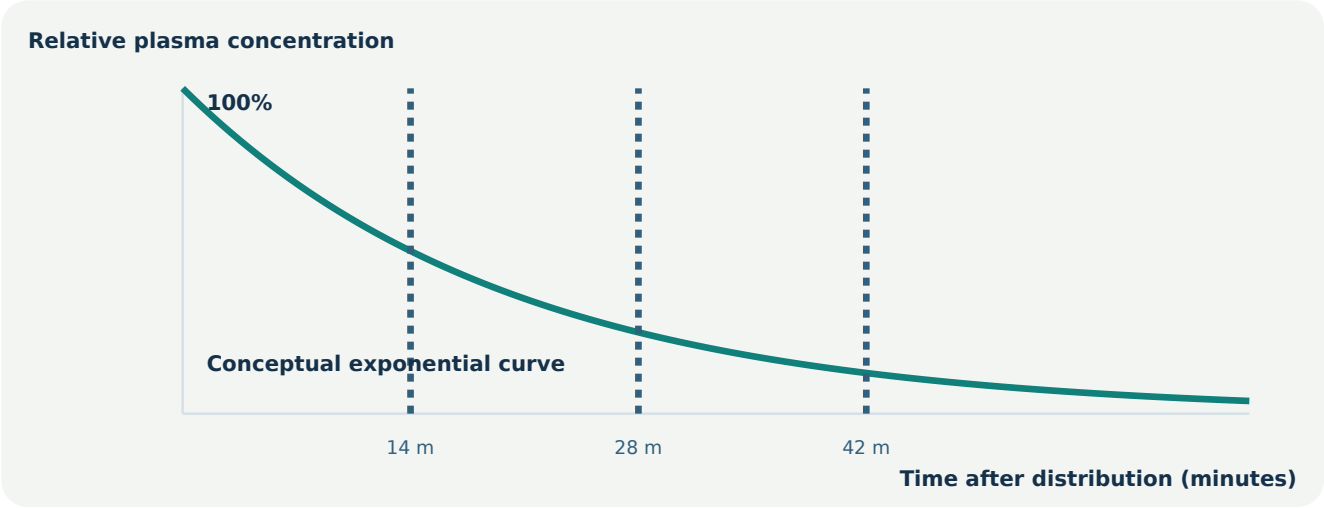


FIGURE 02. Mathematical illustration of a 14.1-minute half-life, normalized to an arbitrary starting concentration. It is not patient-level data, an injection schedule or evidence of how long any clinical effect lasts.

This short-lived plasma signal is not proof that glutathione cannot work. Biochemical effects may outlast the presence of a parent compound, and metabolites can participate in later chemistry. Equally, the fact that a molecule briefly enters circulation is not proof that it crosses into the required cells, corrects a tissue deficit or changes disease. The two pharmacokinetic papers make a powerful point: a successful delivery route is only the beginning of an efficacy argument. [2,3]

> Blood exposure is a measurable event. A clinical benefit is a separate proposition that must be tested.
>
> **Interpretation of human pharmacokinetic research [2,3]**

The distinction becomes clearer when comparing compartments. An erythrocyte measurement, a plasma measurement and a measurement within the substantia nigra of the brain represent different biological spaces. A transient rise in circulating GSH does not, by itself, establish delivery to neurons or mitochondria. Nor can results from oral, intranasal or inhaled preparations be translated into an IV, intramuscular or subcutaneous claim by simple arithmetic. [2-4,14-17,21,22]

These exposure findings concern the particular materials and supervised research conditions studied. They do not establish a validated dose, route, formulation, schedule or exposure target for a commercially available preparation. No administration protocol can be derived from this review.

CHAPTER 03 / THE EVIDENCE STORY

When the first clinical signals looked promising

Oncology researchers tested whether glutathione might help healthy nerves tolerate toxic chemotherapy.

The chemotherapy story is one of the strongest illustrations of how scientific enthusiasm should evolve. Cisplatin and related drugs can damage normal tissues while treating cancer. Peripheral nerve damage is particularly important because numbness, weakness and discomfort can interrupt therapy and affect daily life. Researchers reasoned that a molecule involved in tissue defense might reduce some of this collateral injury. Importantly, this was a supportive-care question - not a proposal that glutathione itself treats cancer. [5-8]

In 1995, Cascinu and colleagues studied 50 people with advanced gastric cancer receiving cisplatin-based treatment. Participants were randomly assigned to glutathione or placebo in a double-blind trial. The investigational regimen included both IV and intramuscular glutathione. By week 15, 4 of 24 assessable participants in the glutathione arm had clinically evident neurotoxicity compared with 16 of 18 in the placebo arm. The reported difference was striking. Yet the trial was small, had unequal assessable groups by that time point and used a specific historical chemotherapy regimen. [6]

Two years later, a randomized trial in 151 women receiving cisplatin for ovarian cancer produced a different but also meaningful signal. Smyth and colleagues asked whether patients could complete six chemotherapy courses. Fifty-eight percent of the glutathione group met the reported completion endpoint, versus 39 percent of controls. Several quality-of-life measures also favored glutathione. Completion of treatment is closer to a real clinical concern than an isolated redox measurement; it still does not establish better survival or a universal protective effect. [5]

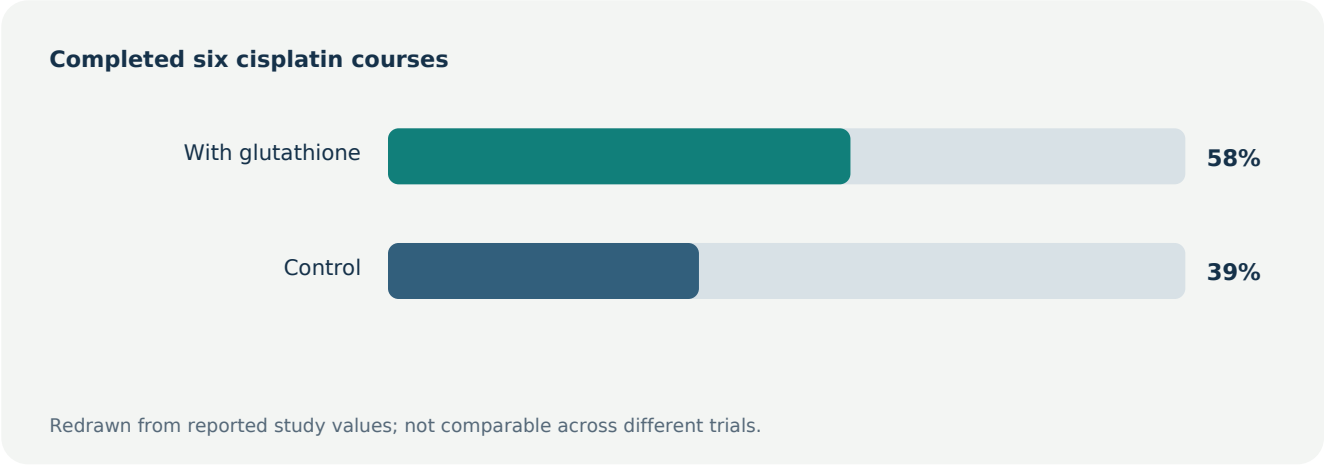


FIGURE 03. Proportion meeting the six-course cisplatin treatment-completion endpoint in Smyth et al. [5]. The bars do not show cancer response or survival.

The sequence continued in 2002. In a double-blind study involving 52 people receiving oxaliplatin-based therapy for colorectal cancer, Cascinu and colleagues reported fewer moderate-to-severe neuropathy cases in the glutathione arm - 2 compared with 11 in the placebo arm at a specified assessment. Nerve-conduction findings also supported a possible protective effect. Again, the question was highly specific: prevention of neurological toxicity during that particular therapy. [7]

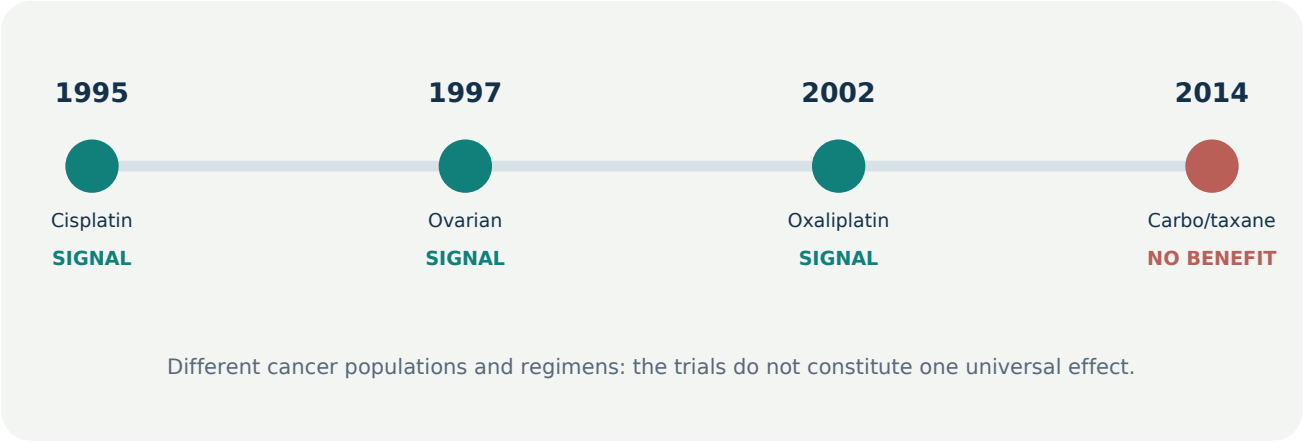


FIGURE 04. A brief chronology of randomized oncology trials. The later negative study involved different chemotherapy, so it narrows general claims without mechanically cancelling the earlier results.

CHAPTER 04 / THE EVIDENCE STORY

The experiment that changed the conversation

If a treatment looks promising in small studies, the next test is whether the signal survives a larger, stricter experiment.

By 2014 there were understandable reasons to investigate glutathione more seriously. Several earlier trials had found signals of reduced toxicity. But clinical research advances by asking whether those signals recur under new, well-controlled conditions. The North Central Cancer Treatment Group / Alliance N08CA study was one such test, and its findings were not what an enthusiast might have expected.

Leal and colleagues enrolled 185 people receiving paclitaxel plus carboplatin, randomly assigning them to glutathione or placebo before chemotherapy. Researchers assessed neuropathy using a validated patient-reported instrument and clinical toxicity scales. On the principal sensory outcome, the groups did not differ significantly ($p=0.21$). Nor was there a significant reduction in grade 2 or greater neuropathy incidence. Some other comparisons, including time to grade 2 neuropathy and pain with weekly paclitaxel, favored placebo. The authors concluded that glutathione was not supported for prevention of neuropathy with this regimen. [8]

> A result that does not confirm the hypothesis is still a discovery. It tells researchers where a promising explanation may be incomplete.

It would be a mistake to say that the N08CA result proves every cisplatin or oxaliplatin study wrong. Paclitaxel and carboplatin differ from cisplatin and oxaliplatin in their patterns of injury, and the participating populations and measured outcomes also differed. It would be an equally serious mistake to ignore the larger negative study and claim that glutathione protects nerves during chemotherapy in general. The accurate interpretation is narrower and more interesting: if protective effects exist, they may depend strongly on drug, disease context, timing and endpoint. [5-8]

That is the real scientific value of placing the results together. A positive small trial can point toward a mechanism worth pursuing; a larger null trial puts boundaries around what may

responsibly be claimed. This is why the strongest research discussions contain uncomfortable results alongside encouraging ones.

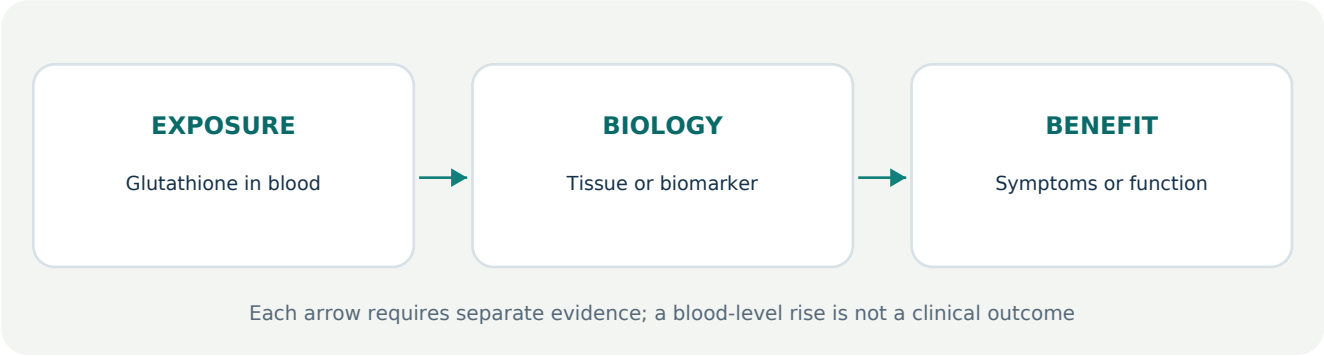


FIGURE 05. Why a convincing mechanism must still pass through exposure, biological change and direct patient benefit.

THE HUMAN APPLICATION WORTH INVESTIGATING

Could a carefully defined subgroup receiving a particular neurotoxic chemotherapy regimen obtain a measurable supportive-care benefit? Answering that requires adequately powered, regimen-specific, modern trials, with both neuropathy outcomes and cancer safety carefully evaluated. These historical results do not establish a general injectable wellness benefit.

CHAPTER 05 / THE EVIDENCE STORY

Could glutathione protect the brain?

The Parkinson disease story begins with a biological observation, grows through an encouraging small study, and meets a difficult controlled test.

The brain is an especially tempting place to investigate oxidative biology. Dopamine-producing neurons depend on complex metabolism and can be vulnerable to cellular stress. Early research described lower glutathione in the substantia nigra of people with Parkinson disease, creating a compelling hypothesis: perhaps strengthening the glutathione system could alter symptoms or vulnerability. But a measured deficit can be a consequence of disease, a contributing process, or both. Changing it is not automatically the same as reversing the disease. [4,28]

A 1996 paper by Sechi and colleagues helped generate excitement. Nine people with early, untreated Parkinson disease received IV glutathione in an open-label study, and the authors reported improvements in disability measures, including a 42% decline in the reported disability score. The finding was memorable, but the design had no randomized comparison group. Placebo responses, measurement variation, natural changes and observer expectations therefore could not be separated reliably from a treatment effect. [28]

In 2009, Hauser and colleagues conducted a randomized, placebo-controlled, double-blind pilot. Twenty-one people with Parkinson disease were assigned to IV glutathione or placebo. Glutathione appeared tolerable during the four-week study, with similar reported adverse events between groups. But there was no statistically significant difference in UPDRS change. During active treatment, the combined activities-of-daily-living and motor score improved by a numerical 2.8 points more than placebo (p=0.32), followed by numerical worsening relative to placebo over the subsequent eight-week observation period (p=0.54). Those comparisons do not establish benefit. [4]

9	21	No
Early open-label cases	**Randomized IV pilot**	**Significant UPDRS result**
1996; no control group [28]	2009; placebo comparison [4]	Not an efficacy demonstration

This does not mean Parkinson research should stop. It identifies the questions that remain: Does administered GSH engage the relevant brain tissue? Is a particular subgroup likely to respond? Does any change persist? Could a larger study detect a clinically meaningful effect? A later phase IIb trial used intranasal rather than IV administration in 45 patients; neither active group was superior to placebo, further illustrating the challenge of moving from a compelling rationale to confirmed benefit. [21]

The important distinction is between improving a laboratory measure, changing a symptom score for a few weeks, and altering the long-term course of a degenerative disorder. Each would be valuable, but each requires its own direct evidence. Nothing in these studies proves disease modification, neuronal regeneration or an injectable benefit in healthy people.

WHAT MAKES THIS EXCITING - AND UNCERTAIN

The biology gives researchers a reasonable target. The early case series supplied a hypothesis. The controlled trials have not yet delivered reliable IV efficacy. An honest research story can hold all three statements at once.

CHAPTER 06 / THE EVIDENCE STORY

A vascular clue, then a striking animal experiment

Two distinct lines of research asked whether glutathione could help tissues facing poor blood flow and oxidative injury.

Not all useful research signals come from neurology or cancer. In 2002, Arosio and colleagues studied 40 people with Fontaine stage II peripheral arterial disease, a condition in which reduced blood flow can produce leg pain during walking. Their randomized, double-blind infusion study evaluated both blood-flow measurements and pain-free walking distance. In the glutathione group, the reported mean distance increased from approximately 143 to 196 metres, alongside changes in selected circulatory measurements. [9]

Walking is a meaningful human function. That makes the finding more interesting than a change in an antioxidant marker alone. But this was one small, short trial, not proof of restored arteries, long-term prevention of cardiovascular events or routine clinical benefit. The observation needs replication and longer follow-up before it could support a treatment claim. [9]

40	143 m	196 m
Participants	**Starting distance**	**Later distance**
Controlled PAD study [9]	Pain-free walking	Reported in GSH group

Animal experiments opened another window on the same broad issue: what happens when circulation returns to an injured tissue? After a period of ischemia, the return of oxygen can itself contribute to reactive chemistry, inflammatory activation and injury. This process is called ischemia-reperfusion injury. It is particularly relevant in transplant and surgical research, where investigators can define the timing of oxygen deprivation and restoration. [10,11]

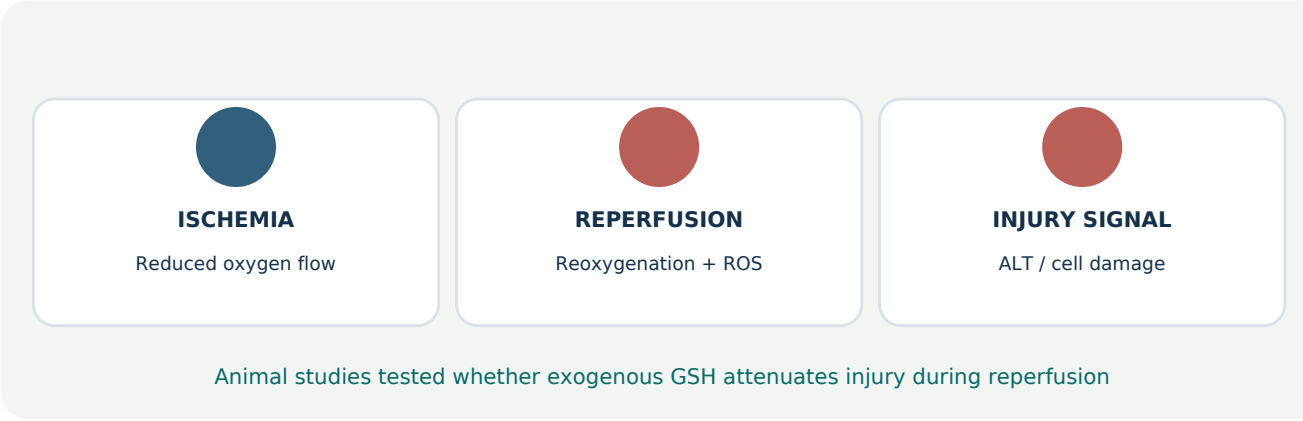


FIGURE 06. Conceptual illustration of hepatic ischemia followed by reperfusion. The experiment examines a specific acute injury process, not generalized liver “detox.”

In 2004, Schauer and colleagues reported that postischemic IV glutathione reduced liver enzyme measures of injury by roughly 50-60% in a rat model of prolonged warm ischemia. Importantly, the investigators proposed that improved extracellular antioxidant capacity, rather than simply filling up cells with glutathione, was part of the protective explanation. A related rat transplantation experiment reported lower ALT after glutathione infusion during reperfusion and less visible injury to sinusoidal endothelial cells and other tissue structures. [10,11]

This is the kind of experiment that can make a laboratory scientist lean forward: it links a predicted mechanism to measured tissue consequences. Yet the level of evidence has to travel with the claim. Rat ischemia models can test timing and tissue sampling that are difficult to reproduce in people, but they are not human transplant trials. A dramatic animal ALT reduction cannot be translated into a percentage benefit for human cirrhosis, fatty liver, longevity or everyday wellness. [10,11]

> An animal experiment can reveal **how something might work**. It cannot, on its own, tell us **that it works in people**.

CHAPTER 07 / THE EVIDENCE STORY

The boundaries of possible human applications

Where else has the science looked, and which observations actually apply to an injectable formulation?

Glutathione biology touches almost every organ, so it is not surprising that research has branched into liver health, metabolic stress, exercise, skin pigmentation and immune markers. The temptation is to collect every encouraging observation under the word “benefits.” Scientifically, these are different hypotheses and need to be discussed separately. Most of the studies in these areas used oral supplementation or animals, not human injections. [12-20,26,27]

The liver: a sensible hypothesis, a limited human test

In rats exposed to a toxic acetaminophen challenge, Sugimura and Yamamoto found that orally administered reduced or oxidized glutathione helped restore liver GSH and limited some injury measures. The experiment reinforces the biochemical role of the glutathione system under a defined toxic stress. But it did not test human overdoses, and it does not replace established emergency treatments. [12]

A 2017 pilot moved into a very different human problem: nonalcoholic fatty liver disease, now often discussed under updated metabolic liver-disease terminology. Honda and colleagues gave oral glutathione after a lifestyle intervention and observed reductions in ALT among completers. The study was open-label and single-arm, without a concurrent placebo control. It raised a hypothesis, but could not establish reversal of fat accumulation, inflammation, fibrosis or a long-term clinical benefit - much less an injectable one. [18]

Diabetes and exercise: measurements with a long journey to clinical meaning

In diabetic rats, a diet containing glutathione was associated with more favorable urinary markers and selected kidney and sensory measures. In another research program, Aoi and colleagues combined mouse metabolic experiments with a crossover cycling study involving only eight men, reporting short-term changes in lactate and fatigue-related responses. These are legitimate scientific observations, but kidney biomarkers in experimental diabetes and fatigue questionnaires after a brief protocol are not proof of prevention of human kidney failure, recovery from neuropathy, enhanced athletic performance or weight loss. [13,19]

Skin pigmentation: what route was actually tested?

Small human skin studies investigated oral glutathione and reported changes in selected pigmentation measurements. A later experiment in guinea pigs used intraperitoneal glutathione following induced UVB pigmentation and observed reduced pigmentation under some experimental conditions. The animal route is not a surrogate for human IV, IM or subcutaneous administration, and the experiment was too short to establish human cosmetic safety. An animal pigmentation finding therefore cannot be presented as an evidence-based promise of “skin whitening injections.” [20,26,27]

Even successful delivery can fail to improve health

One of the clearest lessons came from outside injection research. In a six-month randomized study of 153 patients with cystic fibrosis, inhaled glutathione measurably reached the airways, yet primary lung-function outcomes were not significantly improved. Exacerbations and quality of life did not meaningfully improve either. The contrast is almost a perfect experiment in scientific humility: the molecule arrived, but the hoped-for clinical effect did not. [22]

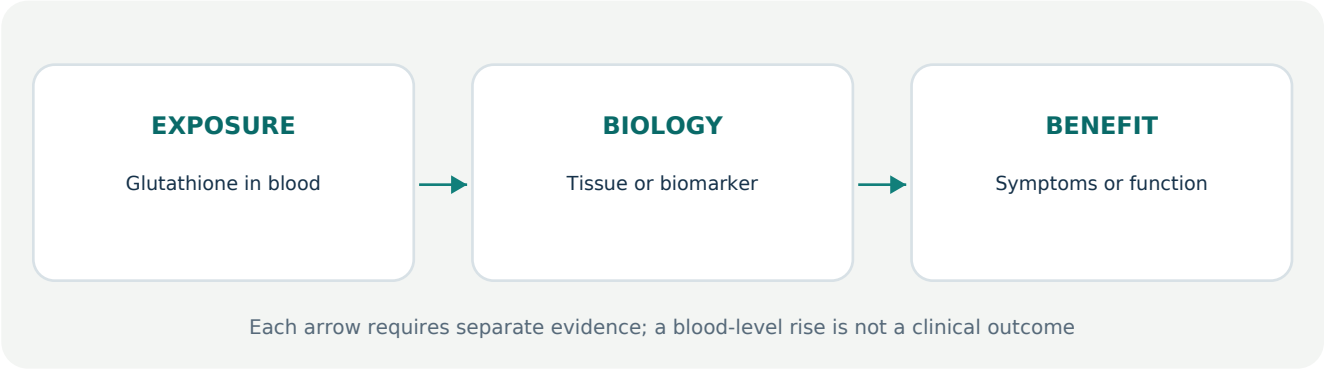


FIGURE 07. A biological effect is not automatically a patient benefit. The cystic fibrosis trial demonstrates why each link in the evidence chain must be tested.

Repeated oral supplementation can raise glutathione stores in some compartments, as demonstrated in a six-month controlled trial, while another shorter controlled trial showed no significant change in targeted oxidative-stress measurements. A small uncontrolled liposomal study also reported favorable biomarker changes. Such results are worth understanding, but do not define a conversion ratio to injectable exposure or prove fewer infections, better metabolism or clinical rejuvenation. [14-17]

CHAPTER 08 / THE EVIDENCE STORY

The reality that changes how we read the studies

A biologically familiar molecule can still become dangerous when its injectable formulation is unsuitable.

There is a tendency to think that a substance naturally present in the body must be harmless when given from the outside. The 2026 glutathione safety investigation showed why that intuition is unreliable. A compound can be chemically recognizable yet prepared from materials unsuitable for injection. And injection moves substances past protective barriers that usually stand between the outside world and the bloodstream. [23-25]

In August 2026, the U.S. FDA warned about adverse events involving compounded injectable glutathione made using dietary-supplement-grade starting materials. Reports included fever, chills, dizziness and shock-like or sepsis-like illness consistent with the possibility of excess endotoxin exposure. The FDA reported at least 30 affected patients in its August communication. [23]

CDC subsequently described illness among more than 25 recipients across at least four U.S. states, including hospitalizations and a critically ill patient. These overlapping agency reports should not be added together as though they were independent case groups. In October, a separate FDA-posted recall notice described certain compounded products with potential elevated endotoxin and emergency-care reports after a product formulated for subcutaneous use was administered intravenously. The details illustrate why the route and formulation must match their intended use. [24,25]

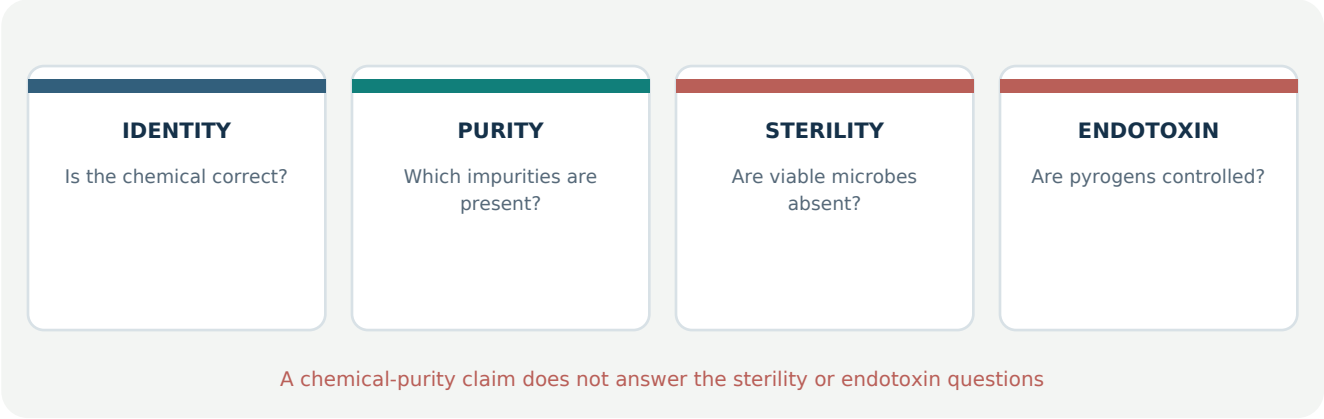


FIGURE 08. Chemical identity, chemical purity, sterility and endotoxin control are separate attributes. A single HPLC percentage does not demonstrate injectable suitability.

The main lesson is not that the glutathione molecule caused every reported reaction. Instead, it is that an injectable product must be evaluated as a complete, route-specific preparation. Chemical identity, assay content, impurities, sterility, bacterial endotoxins, particulates, stability and container integrity answer different questions. A chemical-purity claim or a certificate showing only HPLC results does not answer them all. [23-25]

The clinical studies reviewed earlier also cannot serve as safety tests for any particular commercial glutathione product. Their participants, formulations, routes, supervision, screening and follow-up were specific to those experiments. A few weeks of acceptable tolerability in a small monitored study cannot exclude uncommon reactions or risks with long-term or unsupervised use.

SCOPE AND SAFETY BOUNDARY

This is scientific education, not an injection or reconstitution guide. It does not establish safe human use, dosage, route, schedule, product quality or Canadian market authorization for any specific glutathione preparation. No product sold by Vanguard Compound Research was evaluated in the cited trials. For research use only; not for human use.

CHAPTER 09 / THE EVIDENCE STORY

What the research has actually earned

The most compelling conclusion is precise, not exaggerated: biology is strong, clinical evidence is selective, and several questions remain open.

It is reasonable to be excited about glutathione as a scientific subject. Its chemistry is essential to normal cells; GPX4 links it to regulated cell survival; pharmacokinetic experiments show measurable exposure after IV administration; several small, disease-specific trials report signals worth investigating; and carefully designed animal models reveal potential protection in defined injuries. [1-11]

It is not reasonable, on the available evidence, to turn those findings into broad promises of better immunity, detoxification, skin lightening, athletic recovery, brain protection or general longevity from injections. The most reliable conclusion depends on the exact question. A study about chemotherapy neuropathy does not validate an exercise claim; a rat liver experiment does not validate an IV wellness intervention; a measurable blood increase is not the same as a clinical outcome. [4-27]

A concise evidence map

Research question	Best current reading
Does injected glutathione reach blood?	Yes. Human IV pharmacokinetic studies directly establish brief systemic exposure. [2,3]
Does it protect nerves during chemotherapy?	Some older regimen-specific trials were promising; a larger paclitaxel/carboplatin trial was negative. No universal claim. [5-8]
Does IV glutathione treat Parkinson disease?	Open-label observations were interesting; the placebo-controlled pilot did not demonstrate efficacy. [4,28]
Does it improve walking in vascular disease?	An early 40-person controlled infusion study reported a functional signal; replication is limited. [9]
Can IV glutathione reduce experimental liver injury?	Yes in selected rat ischemia-reperfusion models; no demonstrated equivalent human outcome. [10,11]
Does it improve general wellness or pigmentation by injection?	No adequately established route-specific human clinical benefit. [14-22,26,27]

A better next trial would begin with a defined condition and a reason to expect glutathione biology to matter there. It would use a verified, route-specific clinical formulation, enroll the right population, establish tissue or pathway engagement where possible, and measure an outcome patients truly notice. A randomized blinded comparison, adequate size, careful safety follow-up and independent replication would determine whether the story becomes a therapy. [4-11,23-25]

> Good science is allowed to be fascinating before it is conclusive. It is not allowed to call a hypothesis a fact.

That is the most useful way to read glutathione today: as a molecule with remarkable biological responsibilities and a clinical research record that contains genuine leads, meaningful limits and much work still to do.

HOW THIS REVIEW WAS PREPARED

Sources, interpretation and limits

This is an editorial narrative review of selected primary biomedical studies and official safety communications available through 26 October 2026. It emphasizes studies involving human intravenous glutathione and injected glutathione in animal models; oral, inhaled and intranasal findings are included only where they help explain the biological question or its boundaries. It is not a systematic review, a meta-analysis or an independently peer-reviewed manuscript.

Trial figures and counts were taken from study reports or indexed abstracts where full text was not readily available. No new patient data, laboratory experiments, clinical product assays or meta-analytic effect estimates were generated. Study-specific limitations, including small samples, historical regimens, nonrandomized designs, route differences and absence of long-term outcomes, are described in the text. The selection of literature is purposeful rather than exhaustive, and unpublished or newly indexed results may have been missed.

Interpretations about possible human applications are explicitly hypotheses when confirmatory clinical evidence is absent. In particular, mechanistic studies do not authorize broad medical claims or administration advice for a commercially listed research product.

Canadian publication context

Health Canada distinguishes scientific information from advertising by considering the entire message and where and how it is distributed, including links to sales material. A general literature review placed next to a product listing may therefore still be assessed as promotional content. This publication is sponsor-prepared, not an authorization, endorsement or assessment by Health Canada. It makes no product-specific therapeutic, safety or dosing claim. The authorization and legal sale status of any particular product require separate regulatory assessment; a “research use only” statement does not itself resolve that question. [29,30]

PRIMARY LITERATURE AND OFFICIAL SAFETY SOURCES

Source library

The references below are linked to the original journal record or official public-health publication. They are grouped in a single numbered sequence so readers can move from a claim in the text to the study behind it.

[1] Yang WS et al. (2014). Regulation of ferroptotic cancer cell death by GPX4. Cell 156:317-331. **READ STUDY**

[2] Aebi S, Assereto R, Lauterburg BH (1991). High-dose intravenous glutathione in man: pharmacokinetics and effects on cyst(e)ine. Eur J Clin Invest 21:103-110. **READ STUDY**

[3] Hong SY et al. (2005). Pharmacokinetics of glutathione and its metabolites in normal subjects. J Korean Med Sci 20:721-726. **READ STUDY**

[4] Hauser RA et al. (2009). Randomized, double-blind, pilot evaluation of intravenous glutathione in Parkinson disease. Mov Disord 24:979-983. **READ STUDY**

[5] Smyth JF et al. (1997). Glutathione reduces toxicity and improves quality of life of women with ovarian cancer receiving cisplatin. Ann Oncol 8:569-573. **READ STUDY**

[6] Cascinu S et al. (1995). Neuroprotective effect of reduced glutathione on cisplatin-based chemotherapy in advanced gastric cancer. J Clin Oncol 13:26-32. **READ STUDY**

[7] Cascinu S et al. (2002). Neuroprotective effect of reduced glutathione on oxaliplatin-based chemotherapy in advanced colorectal cancer. J Clin Oncol 20:3478-3483. **READ STUDY**

[8] Leal AD et al. (2014). N08CA: glutathione for prevention of paclitaxel/carboplatin-induced peripheral neuropathy. Cancer 120:1890-1897. **READ STUDY**

[9] Arosio E et al. (2002). Effect of glutathione infusion on leg circulation, microcirculation and pain-free walking distance in peripheral arterial disease. Mayo Clin Proc 77:754-759. **READ STUDY**

[10] Schauer RJ et al. (2004). Glutathione protects the rat liver against reperfusion injury after prolonged warm ischemia. Ann Surg 239:220-231. **READ STUDY**

[11] Schauer RJ et al. (2004). Intravenous glutathione protects liver cells against reperfusion injury after rat liver transplantation. World J Gastroenterol 10:864-870. **READ STUDY**

[12] Sugimura Y, Yamamoto K (1998). Oral reduced/oxidized glutathione against acetaminophen-induced rat liver injury. J Nutr Sci Vitaminol 44:613-624. **READ STUDY**

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