



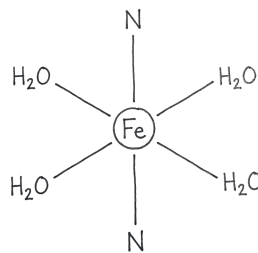
CLINICAL MONOGRAPH · ENERGY & NUTRITIONAL

Iron (Compounded)

Iron preparations for deficiency repletion

Iron is the mineral your body uses to make hemoglobin, the protein in red blood cells that carries oxygen. When iron runs low, you can develop iron-deficiency anemia, which causes fatigue, breathlessness on exertion, headache, and reduced exercise capacity [camaschella2015]. Iron deficiency is the most common nutritional deficiency in the world; women of reproductive age, pregnant patients, and people with chronic blood loss or inflammatory disease are most affected [lopez2016; pasricha2021].

Iron can be replaced by mouth (cheap, widely available, but slow and often poorly tolerated because of nausea, constipation, and a metallic taste) or by IV infusion (faster, more complete repletion, useful when oral iron does not work or cannot be tolerated). FDA-approved IV options include iron sucrose (Venofer), ferric carboxymaltose (Injectafer), ferric derisomaltose (Monoferric), ferumoxytol (Feraheme), and low-molecular-weight iron dextran (INFeD) [fda_label_venofer; fda_label_injectafer; fda_label_monoferric]. Each has its own dosing schedule, infusion time, and side-effect pattern [fda_label_feraheme].



EVIDENCE POSTURE

FDA APPROVED

WELL STUDIED

REVIEWED 2026-05-11





State-licensed
503A



Pharmacist
reviewed



Doctor
led



Cold-chain
ready



Patient choice
preserved



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FOR CLINICIANS

Iron deficiency, with or without anemia, is the most common nutritional deficiency globally and affects roughly a quarter of the world's population [kassebaum2014, lopez2016]. First-line repletion in most non-pregnant adults with uncomplicated iron-deficiency anemia (IDA) is oral elemental iron 40, 100 mg per dose [camaschella2015]. Daily dosing increases serum hepcidin within 24 hours and suppresses subsequent enteral iron absorption, so alternate-day single morning dosing absorbs more total iron per administered dose and is now supported by direct stable-isotope evidence [moretti2015, stoffel2017, stoffel2020]. Gastrointestinal adverse events with ferrous sulfate are substantial, nausea, abdominal pain, constipation, and dark stool occur in roughly a third of users in pooled randomized data [tolkien2015]. Concomitant proton pump inhibitors, calcium, and tea reduce non-heme iron absorption [hutchinson2007].

Intravenous iron is indicated when oral iron fails, is not tolerated, is contraindicated (severe IBD activity, recent GI surgery), or when rapid repletion is required (late pregnancy, pre-operative anemia, dialysis-dependent CKD, decompensated heart failure with iron deficiency) [pasricha2021]. FDA-approved IV products include iron sucrose (Venofer), ferric carboxymaltose (FCM; Injectafer), ferric derisomaltose (FDI; Monoferric), ferumoxytol (Feraheme), and low-molecular-weight iron dextran (INFeD). Modern non-dextran colloidal iron preparations have a much lower hypersensitivity-reaction rate than the historic high-molecular-weight iron dextrans; pooled safety analyses [avni2015, auerbach2017_history, auerbach2024_consensus] characterize life-threatening reactions as rare (~0.04, 0.1 per 100 administrations). Total-dose-infusion regimens of FCM (typically 750, 1000 mg per infusion to a cumulative 1500 mg) and FDI (up to 20 mg/kg in a single infusion) have largely supplanted multi-dose iron sucrose where access permits [auerbach2024_consensus].

Indication-specific evidence is robust. Heart failure with iron deficiency: FAIR-HF [anker2009], CONFIRM-HF [ponikowski2015], AFFIRM-AHF [ponikowski2020], and IRONMAN [kalra2022] support IV ferric carboxymaltose or derisomaltose for symptomatic chronic HFrEF and post-discharge acute HF; IV iron sucrose in FERRIC-HF [okonko2008] showed improved exercise tolerance, and oral iron in IRONOUT-HF [lewis2017] did not improve peak VO₂ in HFrEF [pasricha2021]. CKD: DRIVE [coyne2007], FIND-CKD [macdougall2014], REPAIR-IDA [onken2014], FERWON-NEPHRO [bhandari2021], and PIVOTAL [macdougall2019] support IV iron in non-dialysis and dialysis CKD. Inflammatory bowel disease: FERGICor [evstatiev2011] established FCM superiority over iron sucrose. Pregnancy: UK BSH guidelines [pavord2020] recommend oral iron first-line with IV iron from second trimester onward when warranted, supported by the maternal-mortality signal in severe antenatal anemia [daru2018]. Heavy menstrual bleeding: clinical-guideline review [mansour2021_hmb] frames IV iron as appropriate where oral iron fails. Restless legs syndrome: IRLSSG consensus [allen2018_rls] recommends IV iron when ferritin <75 ng/mL and oral iron has failed. Pre-operative anemia: international consensus [munoz2017] supports IV iron repletion; PREVENTT [richards2020] found no reduction in transfusion or postoperative complications but a signal for fewer readmissions and improved post-discharge hemoglobin.

Comparative IV iron safety considerations are dominated by hypophosphatemia (FCM > FDI [wolf2020, schaefer2022]), hypersensitivity (historic iron dextrans > modern non-dextrans [auerbach2017_history]), and free-iron-mediated infusion reactions controllable by appropriate dilution and infusion rate [adkinson2018, hetzel2014, fda_label_injectafer]. FCM-induced hypophosphatemia can be severe and prolonged (>2 weeks in a substantial minority), can present with osteomalacia after repeated dosing, and is a published reason to prefer FDI in patients requiring repeated IV iron [wolf2020, schaefer2022]. RonanRx role under 503A is narrow: custom oral concentrations for pediatric or dysphagic patients (e.g., flavored ferrous sulfate liquid at alternate-day dosing), and IM iron at non-



commercial strengths only when documented. RonanRx does not compound parenteral iron into multi-ingredient mixes [pasricha2021].

↪ Why Personalized Iron (Compounded)

FDA-approved iron products were dose-calibrated for a generic adult with iron-deficiency anemia and a tolerable GI tract. The label does not account for the things that actually drive iron repletion in a real patient: how deep the deficit is, whether a proton pump inhibitor or tea or calcium is blocking absorption, whether ferrous sulfate triggers nausea and constipation severe enough to stop the regimen, whether the patient is a child who cannot swallow an adult capsule, whether they are pregnant in the second trimester, and whether daily dosing is suppressing absorption through the hepcidin response that alternate-day stable-isotope studies have now documented. None of those individual factors fit the population-average tablet.

Compounding fills that gap inside a narrow lane. A 503A pharmacy can prepare a flavored liquid ferrous salt at a child-appropriate concentration, an alternate-day morning dose split that takes advantage of the hepcidin-window evidence, a capsule strength between commercial breakpoints for a patient titrating up from intolerance, or a preservative-free preparation for a patient with a documented excipient sensitivity. The elemental iron is the same iron the FDA reviewed; the dose, the vehicle, the flavor, and the schedule are written for one chart. RonanRx does not compound parenteral iron into multi-ingredient infusions, that is the lane where commercial iron sucrose, carboxymaltose, and derisomaltose belong.

This is the older arrangement: a prescriber who knows the patient, a pharmacist who prepares the medicine, and a label with one name on it. Modern state inspection and 503A oversight keep that arrangement honest.

↪ Quick Facts About Iron (Compounded)

Category: Essential trace mineral, erythropoietic substrate and mitochondrial cofactor

Active ingredient: Elemental iron, supplied as oral ferrous salts (sulfate, fumarate, gluconate, bisglycinate), oral ferric maltol, or as parenteral colloidal iron-carbohydrate complexes (iron sucrose, ferric carboxymaltose, ferric derisomaltose, ferumoxytol, low-molecular-weight iron dextran)

FDA-approved branded forms: Venofer (iron sucrose, 2000), Injectafer (ferric carboxymaltose, 2013), Monoferric (ferric derisomaltose, 2020), Feraheme (ferumoxytol, 2009), INFeD (low-molecular-weight



iron dextran, 1991), Triferic (ferric pyrophosphate citrate for HD dialysate, 2015), plus numerous OTC and prescription oral iron preparations

Routes: Oral, intravenous, intramuscular (rarely)

Evidence posture: Iron repletion in iron-deficiency anemia is one of the longest-standing evidence bases in medicine. Randomized trials support IV iron in heart failure with iron deficiency (FAIR-HF, CONFIRM-HF, AFFIRM-AHF, IRONMAN), in non-dialysis and dialysis CKD (DRIVE, FIND-CKD, REPAIR-IDA, PIVOTAL), in inflammatory bowel disease (FERGIcor), in heavy menstrual bleeding, and in pregnancy.

FDA-approval status: Multiple FDA-approved manufactured oral and IV iron products are available. Compounded iron preparations are not FDA-approved.

Compounded under: 503A, patient-specific prescription only. Compounded role is narrow: custom oral concentrations and flavors for pediatric or dysphagic patients, alternate-day liquid pediatric formulations, IM iron at custom doses where commercial product is unsuitable. IV iron is typically dispensed as the manufactured product.

Important compounding caution: Iron salts are physically and chemically incompatible with many infusion components. Adding parenteral iron to compounded amino-acid, B-vitamin, or 'Myers cocktail'-type mixes is generally discouraged because of precipitation, oxidative interactions with ascorbate, and loss of dose accuracy. Each parenteral iron product has a manufacturer-specified diluent and infusion protocol that should be followed.

SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY

Iron (Compounded) 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 Iron (Compounded)?

Iron is a transition metal and essential trace element. Roughly 70% of total body iron is in hemoglobin within circulating erythrocytes, 10, 15% in muscle myoglobin, and the remainder distributed across heme-containing and non-heme iron-sulfur-cluster proteins (cytochromes, ribonucleotide reductase, mitochondrial aconitase, ferritin storage). The adult human contains 3, 4 g of iron, with daily losses of approximately 1 mg in men and non-menstruating women, and 1.5, 3 mg per day on average in menstruating women across the cycle [fda_label_venofer]. Iron-deficiency anemia develops when intake or absorption fail to keep up with losses over weeks to months [camaschella2015].

Pharmaceutical iron is supplied either as oral salts (predominantly ferrous sulfate, fumarate, gluconate, or bisglycinate; or as the newer oral ferric maltol) or as parenteral colloidal complexes in which an iron(III) oxyhydroxide core is shielded by a carbohydrate shell (sucrose, carboxymaltose, isomaltoside/derisomaltose, polyglucose-sorbitol-carboxymethyl-ether, or dextran) [fda_label_venofer] [fda_label_infed]. The carbohydrate shell determines stability, infusion-rate ceiling, single-dose limit, and the kinetics of iron release to transferrin. These differences underlie meaningful clinical distinctions in administration time, total-dose-infusion capability, and adverse-event profile [auerbach2017_history, auerbach2024_consensus].

FDA-approved parenteral iron products in the United States are iron sucrose (Venofer, approved 2000), sodium ferric gluconate complex (Ferrlecit, approved 1999), low- and high-molecular-weight iron dextrans (INFeD; the older Dextran was withdrawn 2014), ferumoxytol (Feraheme, approved 2009), ferric carboxymaltose (Injectafer, approved 2013), ferric derisomaltose (Monoferric, approved 2020, the same molecule previously marketed in Europe as iron isomaltoside 1000/Monoferric), and ferric pyrophosphate citrate for hemodialysate addition (Triferic, approved 2015) [fda_label_injectafer; fda_label_monoferric; fda_label_feraheme].

⚙️ How Iron (Compounded) Works

Iron is required for oxygen transport (hemoglobin, myoglobin), electron transport (cytochromes), DNA synthesis (ribonucleotide reductase), and dozens of mitochondrial and cytosolic enzymes. Without adequate iron, erythropoiesis is rate-limited at the heme-synthesis step, red cells produced are microcytic and hypochromic, oxygen-carrying capacity falls, and tissue oxygen delivery is compromised [auerbach2024_consensus].



Systemic iron is regulated almost entirely at the level of absorption and macrophage iron recycling, mediated by the hepatic hormone hepcidin acting on the cellular iron exporter ferroportin. When body iron stores are adequate, hepcidin rises and ferroportin is internalized and degraded, blocking duodenal absorption and macrophage iron release. When iron stores are low or erythropoietic drive is high, hepcidin falls and absorption rises [auerbach2024_consensus].

Pharmacologically, oral iron supplies elemental iron to duodenal enterocytes for absorption via DMT1 after reduction at the apical brush border. Intravenous iron bypasses the gut entirely: the colloidal iron-carbohydrate complex is taken up by macrophages of the reticuloendothelial system (predominantly liver and spleen), and iron is gradually released to transferrin for delivery to the bone marrow over hours to days. Single large doses of stable modern IV iron formulations therefore physiologically simulate weeks of effective oral absorption [auerbach2017_history] [auerbach2024_consensus].

Ⓜ Biological Role of Iron (Compounded)

Iron is essential to oxygen transport, mitochondrial respiration, and DNA synthesis. It is the catalytic metal at the active site of every cytochrome in the electron transport chain, of ribonucleotide reductase that supplies deoxyribonucleotides for DNA replication, and of dozens of dehydrogenases, hydroxylases, and oxygenases including prolyl hydroxylase (the oxygen sensor that controls HIF- α) and aromatic amino-acid hydroxylases that generate dopamine and serotonin. Brain iron deficiency in particular impairs dopaminergic signaling, which underlies the well-documented restless-legs-syndrome phenotype in patients with low brain iron stores even at normal hemoglobin [allen2018_rls].

Body iron is partitioned: hemoglobin in circulating erythrocytes (~2.5 g in an adult), storage as ferritin and hemosiderin in liver, spleen, and bone marrow macrophages (~1 g), myoglobin in muscle (~0.3 g), and the remainder in enzymes. Iron is conserved aggressively, there is no regulated excretory pathway, and net losses are limited to desquamated cells, sweat, urinary tubular cells, and blood loss. This evolutionary economy is also why iron overload, when it occurs (transfusion-dependent, HFE hemochromatosis, repeated parenteral iron in low-iron-loss patients), produces hepatic, cardiac, and endocrine end-organ damage.

Ⓜ Detailed Mechanism of Iron (Compounded)

Oral iron is absorbed predominantly in the duodenum and proximal jejunum. Non-heme iron (the form in supplements and most plant foods) is reduced from Fe^{3+} to Fe^{2+} at the apical enterocyte surface by duodenal cytochrome b and then transported across the apical membrane by DMT1. Heme iron is absorbed intact through HCP1. Within the enterocyte, iron is either stored in ferritin and lost when the enterocyte sloughs, or exported across the basolateral membrane by ferroportin and oxidized to Fe^{3+} by hephaestin for binding to transferrin in the circulation. Hepcidin, secreted by hepatocytes in response to iron,



inflammation, and BMP6 signaling, binds ferroportin and triggers its internalization and degradation, which terminates further iron egress from the enterocyte and from macrophages.

A single oral iron dose increases serum hepcidin within 24 hours, blunting absorption of further iron given within that window. The Moretti, Stoffel, and follow-on studies in iron-deficient women established that single morning doses on alternate days absorb more total iron per administered dose than the same daily dose split twice daily, and that 60, 120 mg every other morning produces fractional absorption of 20, 35% on day 1 falling to 5, 10% on day 2 [moretti2015, stoffel2017, stoffel2020]. This is the mechanistic rationale for alternate-day single-morning oral iron dosing, which also reduces GI symptom burden by halving the daily mucosal exposure [tolkien2015]. Proton pump inhibitors, H2 blockers, calcium, and polyphenol-rich tea all reduce non-heme iron absorption substantially [hutchinson2007].

Parenteral iron preparations are colloids in which an iron(III) oxyhydroxide core is stabilized by a carbohydrate shell. The shell determines several clinically important properties: the maximum single dose that can be safely administered (iron sucrose 200, 300 mg per dose vs FCM 750, 1000 mg per dose vs FDI up to 20 mg/kg single infusion vs ferumoxytol 510 mg per dose), the infusion duration required, the stability of free iron release in plasma (less stable formulations such as iron sucrose require multiple small doses; very stable formulations such as FCM and FDI permit total-dose infusion), and the immunogenicity (high-molecular-weight iron dextran historically caused most reported anaphylaxis; modern non-dextran preparations have rates 1, 2 orders of magnitude lower) [auerbach2017_history, auerbach2024_consensus, avni2015].

Iron from the parenteral colloid is delivered to reticuloendothelial macrophages, where it is sequestered in ferritin and then exported via ferroportin to transferrin in the plasma over hours to days. Free, non-transferrin-bound iron is potentially redox-active and can mediate oxidative stress at the infusion site or systemically, this is the basis of the 'Fishbane reaction' (transient flushing, chest tightness, back/joint pain) seen with too-rapid administration, and partly of FCM-associated hypophosphatemia. FCM specifically induces FGF23 cleavage products, causing renal phosphate wasting and frequently profound hypophosphatemia that may persist for weeks; this signal is markedly less pronounced with FDI and absent with iron sucrose, ferumoxytol, and iron dextran [wolf2020, schaefer2022].

🕒 Iron (Compounded) Research History

Iron has been used therapeutically since antiquity, and ferrous sulfate as the modern oral iron salt has been in continuous use since the nineteenth century. The first injectable iron, iron-saccharated colloid, was introduced in the 1930s, followed in the 1950s by high-molecular-weight iron dextran (Imferon), which became dominant for parenteral iron until the late 1990s but accumulated a substantial signal for anaphylactoid reactions that limited its uptake [auerbach2017_history]. The Global Burden of Disease analyses [kassebaum2014] confirmed iron-deficiency anemia as the leading cause of years lived with disability among nutritional conditions worldwide.



Iron sucrose (Venofer) entered the US market in 2000 and was rapidly adopted in the dialysis-CKD setting, where the DRIVE trial [coyne2007] demonstrated that IV iron in hyperferritinemic, low-TSAT hemodialysis patients on erythropoietin produced meaningful hemoglobin response, challenging the prior ferritin-based ceiling on IV iron use. PIVOTAL [macdougall2019] subsequently showed that a proactive, high-dose IV iron strategy in incident hemodialysis was non-inferior to a reactive low-dose strategy on cardiovascular events and reduced erythropoietin requirements. Non-dialysis CKD evidence came from FIND-CKD [macdougall2014] (FCM superior to oral iron) and REPAIR-IDA [onken2014] (FCM versus iron sucrose).

Ferric carboxymaltose was FDA-approved as Injectafer in 2013, ferumoxytol as Feraheme in 2009 (originally also developed as an MR contrast agent), and ferric derisomaltose as Monoferric in January 2020 in the US. These modern non-dextran colloidal preparations support single-dose infusion of 500, 1000 mg or, for FDI, a single 20-mg/kg infusion to complete repletion [bhandari2021, auerbach2019_ferwon_ida, auerbach2024_consensus].

The heart-failure indication for IV iron originated with FERRIC-HF [okonko2008], in which iron sucrose improved exercise tolerance in iron-deficient HFrEF. FAIR-HF [anker2009] established symptom and quality-of-life benefit with ferric carboxymaltose regardless of anemia status. CONFIRM-HF [ponikowski2015] extended benefit to 52 weeks, AFFIRM-AHF [ponikowski2020] addressed the post-acute setting at hospital discharge, and IRONMAN [kalra2022] tested ferric derisomaltose in UK chronic HF. IRONOUT-HF [lewis2017] separately showed that oral iron polysaccharide was not adequate in HFrEF, supporting the IV route specifically. Inflammatory bowel disease evidence came from FERGICor [evstatiev2011]. Heavy-menstrual-bleeding and pregnancy evidence developed through UK and international guideline syntheses [pavord2020, mansour2021_hmb, daru2018].

📅 Iron (Compounded) Timeline

- 1832** • Pierre Blaud popularizes the 'Blaud pill' of ferrous sulfate and potassium carbonate as a treatment for chlorosis, the first systematic oral iron pharmacotherapy
- 1950s** • High-molecular-weight iron dextran (Imferon) introduced as the first widely used parenteral iron, eventually withdrawn for hypersensitivity-reaction burden [auerbach2017_history]
- 1991** • FDA approves low-molecular-weight iron dextran (INFeD) for treatment of iron-deficiency anemia in patients unable to take oral iron [fda_label_infed]
- 2000** • FDA approves iron sucrose (Venofer) for iron-deficiency anemia in CKD [fda_label_venofer]
- 2007** • DRIVE trial (Coyne et al., J Am Soc Nephrol) demonstrates that IV ferric gluconate produces hemoglobin response in hyperferritinemic, low-TSAT hemodialysis patients on erythropoietin [coyne2007]



- 2008 • FERRIC-HF (Okonko et al., JACC), IV iron sucrose improves exercise tolerance in iron-deficient symptomatic HFrEF [okonko2008]

- 2009 • FDA approves ferumoxytol (Feraheme) for iron-deficiency anemia in CKD; FAIR-HF (Anker et al., NEJM) demonstrates symptom and quality-of-life benefit of ferric carboxymaltose in chronic HFrEF with iron deficiency, regardless of anemia status [fda_label_feraheme; anker2009]

- 2011 • FERGIcor (Evstatiev et al., Gastroenterology), ferric carboxymaltose superior to iron sucrose for IDA in inflammatory bowel disease [evstatiev2011]

- 2013 • FDA approves ferric carboxymaltose as Injectafer for iron-deficiency anemia in adults intolerant of, or with unsatisfactory response to, oral iron [fda_label_injectafer]

- 2014 • Kassebaum et al [hetzel2014]. (Blood) Global Burden of Disease analysis, iron-deficiency anemia is the leading cause of YLD among nutritional conditions; FIND-CKD (Macdougall et al., NDT), high-ferritin-target IV FCM superior to oral iron in non-dialysis CKD; REPAIR-IDA (Onken et al., NDT), FCM vs iron sucrose in IDA with impaired renal function; Hetzel et al [kassebaum2014; macdougall2014; onken2014]. (Am J Hematol), ferumoxytol non-inferior to iron sucrose

- 2015 • CONFIRM-HF (Ponikowski et al., Eur Heart J), sustained benefit of IV ferric carboxymaltose over 52 weeks in chronic HFrEF; Camaschella IDA review in NEJM; Lopez et al [ponikowski2015; camaschella2015; lopez2016]. IDA seminar in Lancet 2016; Moretti et al. (Blood) describe oral iron-induced hepcidin rise that blunts absorption of subsequent doses; Tolkien et al [moretti2015]. (PLoS One) systematic review confirms substantial GI AE rate with ferrous sulfate; Avni et al [tolkien2015; avni2015]. (Mayo Clin Proc) meta-analysis of IV iron safety

- 2017 • Stoffel et al. (Lancet Haematol), alternate-day single-morning oral iron absorbs more per dose than daily or twice-daily dosing; Munoz et al [stoffel2017; munoz2017; lewis2017]. international consensus on perioperative anaemia management; IRONOUT-HF (Lewis et al., JAMA), oral iron does not improve peak VO₂ in HFrEF

- 2018 • Adkinson et al. (Am J Hematol), head-to-head comparative safety of ferumoxytol vs FCM in IDA; IRLSSG/Allen et al [adkinson2018]. (Sleep Med) consensus on iron treatment of restless legs syndrome including IV iron when ferritin <75 ng/mL; Daru et al [allen2018_rls; daru2018]. (Lancet Glob Health), severe maternal anaemia and mortality

- 2019 • PIVOTAL trial (Macdougall et al., NEJM), proactive high-dose IV iron non-inferior to reactive low-dose in incident hemodialysis on cardiovascular endpoint; FERWON-IDA (Auerbach et al., Am J Hematol), ferric derisomaltose vs iron sucrose for IDA [macdougall2019; auerbach2019_ferwon_ida]



- 2020 • FDA approves ferric derisomaltose (Monoferric) for non-CKD IDA in adults intolerant of oral iron; AFFIRM-AHF (Ponikowski et al., Lancet), IV ferric carboxymaltose at discharge after acute heart failure; UK BSH iron-in-pregnancy guideline (Pavord et al., BJH); Wolf et al [fda_label_monoferric; ponikowski2020; pavord2020; wolf2020; richards2020; means2020]. (JAMA) randomized comparison of hypophosphatemia after FCM vs FDI; PREVENTT (Richards et al., Lancet), preoperative IV iron in abdominal surgery; Means iron-deficiency review (Nutrients)

- 2021 • Pasricha et al. (Lancet) iron-deficiency seminar; FERWON-NEPHRO (Bhandari et al., NDT), FDI vs iron sucrose in non-dialysis CKD; Mansour et al [pasricha2021; bhandari2021; mansour2021_hmb]. clinical-guideline review of HMB management

- 2022 • IRONMAN (Kalra et al., Lancet), ferric derisomaltose in UK chronic heart failure with iron deficiency; Schaefer et al [kalra2022; schaefer2022]. (JCEM) on persistent and severe hypophosphatemia after FCM

- 2024 • Auerbach et al [auerbach2024_consensus]. (Am J Hematol) expert consensus on intravenous iron uses, formulations, administration, and reaction management

- 2025 • Auerbach et al [auerbach2025_jama]. (JAMA) state-of-the-art review of iron deficiency in adults

📖 Clinical Contexts for Iron (Compounded)

Iron-deficiency anemia in adults intolerant of, or with unsatisfactory response to, oral iron, non-dialysis CKD or non-CKD FDA APPROVED

FDA-approved indications across multiple manufactured IV iron products.

Manufactured IV iron products are labeled for IDA in adults who cannot tolerate or do not respond to oral iron [fda_label_feraheme; fda_label_infed]. FCM (Injectafer) and FDI (Monoferric) are labeled in adults without restriction to CKD; iron sucrose (Venofer) is labeled in CKD (HD, PD, and non-dialysis); ferumoxytol (Feraheme) is labeled for IDA in adults intolerant of or with unsatisfactory response to oral iron and in CKD [fda_label_venofer; fda_label_injectafer; fda_label_monoferric]. Phase 3 evidence supporting these labels includes FIND-CKD [macdougall2014], REPAIR-IDA [onken2014], FERWON-IDA [auerbach2019_ferwon_ida], FERWON-NEPHRO [bhandari2021], the Hetzel ferumoxytol vs iron sucrose trial [hetzel2014], and the Adkinson FCM vs ferumoxytol head-to-head [adkinson2018].

Branded product: Venofer (iron sucrose), Injectafer (ferric carboxymaltose), Monoferric (ferric derisomaltose), Feraheme (ferumoxytol), INFED (iron dextran)



Iron-deficiency anemia in adults with chronic kidney disease on hemodialysis

FDA APPROVED

FDA-approved indication for iron sucrose (Venofer) and ferric pyrophosphate citrate (Triferic); broad use evidence base.

Iron sucrose is labeled for IDA in adults with CKD including those on hemodialysis [fda_label_venofer]. Evidence supporting IV iron in dialysis CKD includes DRIVE [coyne2007] (response in hyperferritinemic, low-TSAT HD patients) and PIVOTAL [macdougall2019] (proactive high-dose IV iron non-inferior to reactive low-dose on cardiovascular endpoint with reduced erythropoietin requirement). Triferic is added to the hemodialysate to replace ongoing iron losses on dialysis.

Branded product: Venofer (iron sucrose); Triferic (ferric pyrophosphate citrate dialysate)

Symptomatic chronic heart failure with reduced ejection fraction and iron deficiency

WELL STUDIED

Studied in dedicated randomized phase 3 trials; ESC heart failure guidelines recommend IV iron in this population. Not a separately labeled US indication.

FAIR-HF [anker2009] randomized 459 adults with NYHA II, III HFrEF and iron deficiency (ferritin <100 ng/mL, or 100, 299 with TSAT <20%) to IV FCM or saline placebo; the primary patient-global-assessment and 6-minute-walk endpoints favored FCM. CONFIRM-HF [ponikowski2015] confirmed sustained benefit over 52 weeks. AFFIRM-AHF [ponikowski2020] tested IV FCM at discharge after acute HF, the primary composite (recurrent HF hospitalization + CV death) showed a numerical reduction that did not reach statistical significance under the prespecified COVID-19-adjusted analysis. IRONMAN [kalra2022] tested ferric derisomaltose in UK chronic HF and reported a reduction in HF hospitalization and CV death in the COVID-19-adjusted analysis. FERRIC-HF [okonko2008] with iron sucrose preceded these and showed exercise-tolerance benefit. Oral iron polysaccharide was negative on peak VO₂ in IRONOUT-HF [lewis2017], supporting the IV route specifically.

Iron-deficiency anemia in inflammatory bowel disease WELL STUDIED

Studied in FERGICor and supported by IBD society guidelines.

FERGICor [evstatiev2011] randomized 485 adults with IBD and IDA to ferric carboxymaltose or iron sucrose. FCM was superior to iron sucrose on the primary hemoglobin response endpoint at 12 weeks, with fewer infusions required. Oral iron in active IBD is often poorly tolerated because of mucosal exposure and can exacerbate inflammation in some patients; IV iron is the preferred route in moderate-to-severe active disease.



Iron deficiency in pregnancy WELL STUDIED

Oral iron first-line per UK BSH guidelines; IV iron from second trimester onward when oral iron fails, is not tolerated, or rapid repletion is required.

UK BSH 2019 guidelines on iron in pregnancy [pavord2020] frame oral iron as first-line for IDA and prophylaxis where indicated, with IV iron (FCM, FDI, or iron sucrose) reserved for second-trimester-onward use in oral-iron failure, intolerance, severe anemia near term, or specific obstetric scenarios. The maternal-mortality signal in severe antenatal anemia [daru2018] underlies the urgency to correct anemia before delivery.

Iron-deficiency anemia secondary to heavy menstrual bleeding WELL STUDIED

Routine indication; IV iron is recommended where oral iron fails or rapid repletion is required.

Clinical-guideline review [mansour2021_hmb] frames IV iron (FCM, FDI, or iron sucrose) as appropriate in HMB-related IDA where oral iron has failed, has produced unacceptable GI side effects, or is insufficient to keep up with menstrual losses. Treatment of the underlying HMB (hormonal, surgical) is the separate question.

Restless legs syndrome / Willis, Ekbom disease in adults with low iron stores

WELL STUDIED

IRLSSG consensus recommends IV iron when ferritin <75 ng/mL and oral iron has failed.

Allen et al. IRLSSG consensus [allen2018_rls] recommends a trial of oral iron when peripheral ferritin is below 75 ng/mL and considers IV iron (FCM, ferumoxytol, or iron sucrose; iron dextrans are not generally recommended) when oral iron has failed, is not tolerated, or rapid response is desired. Brain iron, not peripheral hemoglobin, is the relevant pool; many RLS patients are not anemic.

Pre-operative anemia before major elective surgery WELL STUDIED

International consensus supports IV iron repletion; PREVENTT trial findings nuanced.

International consensus [munoz2017] recommends correction of pre-operative anemia, including IV iron where indicated, to reduce transfusion need and improve postoperative outcomes. PREVENTT [richards2020] randomized 487 anaemic adults undergoing major abdominal surgery to IV FCM or placebo 10, 42 days pre-op; the co-primary endpoints of death/transfusion and number of transfusions were not met, but pre-specified secondary analyses suggested fewer readmissions and improved 8-week post-discharge hemoglobin.



Ⓢ Off-Label Uses of Iron (Compounded)

Iron deficiency without anemia in fatigue or athletic performance EMERGING

Off-label; mixed evidence. Oral iron may benefit fatigue in non-anemic iron-deficient women in some trials but the effect size is modest.

Some randomized data suggest fatigue improvement with iron repletion in non-anemic women with low ferritin, but trial quality and effect sizes vary. The Auerbach 2024 expert consensus and the 2025 JAMA review recommend evaluating ferritin and considering iron repletion in non-anemic iron-deficient adults with fatigue, while cautioning against routine IV iron for fatigue alone without documented deficiency [auerbach2024_consensus, auerbach2025_jama].

✓ FDA-Approved Uses of Iron (Compounded)

Brand	Indication	Year	Route
Venofer (iron sucrose)	Iron-deficiency anemia in adults and pediatric patients ≥2 years with chronic kidney disease (HD, PD, or non-dialysis)	2000	Intravenous
Injectafer (ferric carboxymaltose)	Iron-deficiency anemia in adults intolerant of or with unsatisfactory response to oral iron; in adults with non-dialysis-dependent CKD; iron deficiency in adults with NYHA II/III heart failure (added)	2013	Intravenous
Monoferric (ferric derisomaltose)	Iron-deficiency anemia in adults intolerant of oral iron or with non-hemodialysis-dependent CKD	2020	Intravenous
Feraheme (ferumoxytol)	Iron-deficiency anemia in adults intolerant of or with unsatisfactory response to oral iron, including those with CKD	2009	Intravenous
INFeD (low-molecular-weight iron dextran)	Iron-deficiency anemia in adults in whom oral iron is unsatisfactory or impossible	1991	Intravenous or intramuscular

Multiple manufactured iron products are FDA-approved. Oral iron is available as numerous OTC and prescription ferrous sulfate, ferrous fumarate, ferrous gluconate, ferrous bisglycinate, polysaccharide-iron complex, and ferric maltol preparations. Parenteral iron is available as iron sucrose (Venofer), ferric carboxymaltose (Injectafer), ferric derisomaltose (Monoferric), ferumoxytol (Feraheme), low-molecular-weight iron dextran (INFeD), and ferric pyrophosphate citrate for hemodialysate addition (Triferic) [fda_label_venofer; fda_label_monoferric; fda_label_feraheme].



Each parenteral iron product carries product-specific labeling on hypersensitivity warnings, infusion duration, single-dose limits, and post-infusion monitoring [fda_label_venofer]. Injectafer and Feraheme labels include warnings regarding serious hypersensitivity reactions; Injectafer carries a specific warning on symptomatic hypophosphatemia [fda_label_injectafer]. INFED historically carried a Boxed Warning for anaphylactic-type reactions including a test-dose requirement [fda_label_infed].

⚠ Compounded Iron (Compounded) (503A)

The 503A compounded role for iron is narrow. The IV iron products listed above are manufactured, FDA-approved, and widely available; compounding parenteral iron from bulk in a 503A pharmacy is rarely clinically necessary and is not a routine RonanRx service. Adding parenteral iron to multi-ingredient infusions (compounded amino-acid mixes, B-complex bags, 'Myers cocktail'-style preparations) is generally discouraged because iron is physically and chemically incompatible with many of those components: iron precipitates with phosphate; oxidizes ascorbate; reacts with thiols (glutathione, N-acetylcysteine); and is destabilized at pH extremes and with calcium gluconate. Each manufactured IV iron product specifies a permitted diluent (normal saline, often only in a defined volume) and an infusion rate ceiling; deviations from labeled administration are a documented source of free-iron reactions [fda_label_injectafer, fda_label_venofer, auerbach2024_consensus].

Legitimate 503A iron compounding scenarios include: (1) custom-strength oral liquid iron for pediatric patients or for adults with dysphagia, especially when a clinically appropriate alternate-day dosing schedule [stoffel2017] is desired and no commercial product matches the prescriber's mg-per-mL and flavor specifications; (2) excipient-free or specific-excipient oral preparations for patients with documented sensitivity to a component of commercial iron tablets or liquids; (3) IM iron dextran in non-standard volumes for patients in whom IV access is impossible and IM administration is appropriate, when no manufactured product matches the prescribed regimen. In all cases the compound is dispensed only on a patient-specific prescription with a documented clinical reason that the manufactured product cannot meet [fda503a, fda_essentially_a_copy].

RonanRx does not compound parenteral iron into multi-ingredient infusion bags. RonanRx does not compound or dispense 'iron infusions' as a routine wellness service. Manufactured Venofer, Injectafer, Monoferric, and Feraheme are the standard of care for IV iron; the compounded oral and (rarely) IM iron preparations described above are the only iron preparations RonanRx produces and only on documented patient-specific need.



Iron (Compounded) Formulations and Routes

Form	Concentration	Description
Compounded oral liquid (503A)	Custom mg elemental iron per mL, typical pediatric strengths include ferrous sulfate 75 mg/mL (15 mg elemental iron/mL) or lower, flavored to improve adherence	Patient-specific oral liquid prepared under USP <795>. Used when manufactured pediatric liquid iron is not available in the prescribed strength, when a specific flavoring or excipient profile is required, or to support alternate-day single-morning dosing in pediatric or dysphagic adults.
Compounded oral capsule (503A)	Custom elemental iron strength, typically 25, 65 mg per capsule	Patient-specific oral solid prepared under USP <795> with documented potency. Used when a manufactured product at the prescribed strength is unavailable or when a specific excipient profile is required.
Manufactured oral preparations (reference products)	Ferrous sulfate 325 mg (65 mg elemental), ferrous fumarate 324 mg (106 mg elemental), ferrous gluconate 324 mg (38 mg elemental), polysaccharide-iron 150 mg elemental, ferric maltol 30 mg elemental	Standard OTC and prescription oral iron preparations. Ferrous salts are absorbed similarly per elemental iron content; differences in GI tolerability are modest and dose-dependent [tolkien2015].
Manufactured IV iron sucrose (Venofer; reference product)	20 mg elemental iron per mL; 5 mL or 10 mL single-dose vials	Slow IV push of 100 mg over 2, 5 min or infusion of 200, 300 mg over ≥15 min, typically 5, 10 doses per repletion course. CKD-labeled.
Manufactured IV ferric carboxymaltose (Injectafer; reference product)	50 mg elemental iron per mL; 15 mL single-dose vials	Two 750 mg doses at least 7 days apart (cumulative 1500 mg) or 15 mg/kg up to 750 mg infused over ≥15 min. Single-dose label option in non-US markets allows up to 1000 mg in one infusion.
Manufactured IV ferric derisomaltose (Monoferric; reference product)	100 mg elemental iron per mL; 1 mL or 10 mL single-dose vials	1000 mg over ≥20 min as a single dose for patients with weight ≥50 kg; 20 mg/kg up to a maximum 1000 mg total dose otherwise. Designed for total-dose infusion in one or two visits.
Manufactured IV ferumoxytol	30 mg elemental iron per mL; 17 mL single-dose vials (510 mg per vial)	



Form	Concentration	Description
(Feraheme; reference product)		Two 510 mg doses 3, 8 days apart (cumulative 1020 mg) infused over ≥15 min. Originally also developed as an MR contrast agent.
Manufactured IV/IM low-molecular-weight iron dextran (INFeD; reference product)	50 mg elemental iron per mL	Total-dose infusion possible but requires test dose and longer infusion than non-dextran products. Largely supplanted in clinical practice by FCM, FDI, and ferumoxytol where available.

Routes used in published literature: oral, intravenous, intramuscular.

📖 Iron (Compounded) Dosing

Route	Population	Range	Duration	Study type
Oral	Adults with iron-deficiency anemia (non-pregnant)	40, 100 mg elemental iron per dose, given as a single morning dose every other day or daily for a planned course of 8, 12 weeks beyond hemoglobin normalization to replete stores	8, 12 weeks beyond Hb normalization, then reassess ferritin	Mechanistic stable-isotope absorption studies and clinical-guideline consensus
Oral	Adults with iron deficiency in pregnancy	Per UK BSH 2019: 40, 80 mg elemental iron daily for prophylaxis where indicated; 100, 200 mg elemental iron daily for treatment of established IDA	Continued through pregnancy and at least 3 months postpartum	UK BSH 2019 guideline
Intravenous	Adults with IDA (iron sucrose / Venofer)	100, 200 mg per dose over 15, 30 min; typical cumulative repletion 1000 mg over 5, 10 doses	Course over 2, 3 weeks	FDA-approved labeled regimen
Intravenous	Adults with IDA (ferric carboxymaltose / Injectafer)	Two doses of 750 mg ≥7 days apart (cumulative 1500 mg) infused over ≥15 min; or 15 mg/kg up to 750 mg single dose	Single or two-dose course	FDA-approved labeled regimen
Intravenous				



Route	Population	Range	Duration	Study type
	Adults with IDA (ferric derisomaltose / Monoferric)	1000 mg single infusion over ≥20 min (weight ≥50 kg); 20 mg/kg up to a maximum 1000 mg single infusion otherwise; repeat as required to a calculated total iron deficit	Single-dose total-dose-infusion regimen	FDA-approved labeled regimen
Intravenous	Adults with IDA (ferumoxytol / Feraheme)	Two 510 mg doses 3, 8 days apart (cumulative 1020 mg) infused over ≥15 min	Two-dose course	FDA-approved labeled regimen
Intravenous	Adults with HFrEF and iron deficiency (off-label US, guideline-recommended internationally)	Ferric carboxymaltose dosed by weight and Hb per FAIR-HF / CONFIRM-HF schedule (typical cumulative 500, 2000 mg over multiple visits); IRONMAN used ferric derisomaltose dosed by weight every 4 months as required	Indefinite while iron-deficient and clinically beneficial	Phase 3 RCT regimens
Intramuscular	Adults unable to take oral or IV iron (rare)	Iron dextran 100 mg per injection at separate gluteal sites with Z-track technique; total dose calculated by weight and Hb deficit	Multi-injection course	FDA-approved INFeD label; rarely used in modern practice

Oral iron is first-line for most non-pregnant adults with uncomplicated IDA. Single-morning alternate-day dosing of 40, 100 mg elemental iron is now supported by direct stable-isotope absorption data [moretti2015, stoffel2017, stoffel2020] and produces less GI symptom burden than daily or twice-daily dosing [tolkien2015]. A typical adult repletion course is 8, 12 weeks beyond hemoglobin normalization to refill ferritin stores. Patients should be counseled to take oral iron away from calcium, proton pump inhibitors, and tea, all of which materially reduce non-heme iron absorption [hutchinson2007].

Intravenous iron should be used when oral iron has failed, is not tolerated, is contraindicated, or when rapid repletion is needed. Choice of IV iron product is driven by access, single-dose-infusion capability, and patient-specific safety considerations. FCM is widely available and convenient but carries the highest risk of clinically meaningful hypophosphatemia, particularly in patients with normal kidney function and ongoing causes of urinary iron-FGF23-axis stimulation [wolf2020, schaefer2022]. FDI is a total-dose-infusion option with markedly less hypophosphatemia. Ferumoxytol and iron sucrose are appropriate alternatives. Modern non-dextran preparations have a much lower hypersensitivity-reaction profile than the historic iron dextrans [auerbach2017_history, auerbach2024_consensus].



☑ Iron (Compounded) Safety

Oral iron safety is dominated by gastrointestinal adverse events. The Tolkien et al. systematic review and meta-analysis³² reported nausea, abdominal pain, constipation, diarrhea, and dark stool occurring substantially more often with ferrous sulfate than placebo, with an overall GI AE odds ratio of approximately 2.3. AE rate scales with dose; alternate-day single-morning dosing reduces both daily exposure and aggregate GI symptom burden²⁸. Oral iron should be taken away from PPIs, calcium, and tea to preserve absorption³³. Acute oral iron overdose in children is a clinically important poisoning syndrome and is the basis for the FDA-mandated child-resistant packaging on oral iron products containing ≥ 30 mg elemental iron per dosage unit.

Intravenous iron safety is dominated by infusion reactions and, with FCM specifically, hypophosphatemia⁴. Pooled safety analyses of modern non-dextran IV iron preparations³¹³⁴³⁵ report a serious-hypersensitivity-reaction rate of approximately 0.04, 0.1 per 100 administrations, much lower than the historic 0.6, 0.7 per 100 reported with high-molecular-weight iron dextran. The Adkinson head-to-head trial¹⁵ reported broadly comparable composite hypersensitivity rates between FCM and ferumoxytol with each product showing a distinct subtype distribution. Mild 'Fishbane reaction' (transient flushing, chest tightness, back/joint pain without true anaphylaxis) is rate-related and resolves with infusion-rate reduction.

FCM-induced hypophosphatemia is now a well-characterized adverse event. Wolf et al.¹⁶ randomized adults with IDA to FCM or FDI; incident hypophosphatemia (phosphate < 2.0 mg/dL) occurred in 75% on FCM vs 8% on FDI by day 14, and severe hypophosphatemia (< 1.3 mg/dL) in 12% on FCM vs $< 1\%$ on FDI. Schaefer et al.¹⁷ characterized persistent (> 2 -week) and severe hypophosphatemia following FCM, with risk factors including lower baseline phosphate, normal kidney function, and higher cumulative FCM dose. Clinical sequelae include weakness, bone pain, and, with repeated dosing, osteomalacia and stress fractures; routine baseline phosphate measurement and phosphate monitoring after FCM, and preferential use of FDI in patients requiring repeated IV iron, are now recommended³⁵. Iron sucrose, ferumoxytol, and iron dextran do not produce clinically meaningful hypophosphatemia.

Cardiovascular safety of IV iron in heart failure with iron deficiency is supported by FAIR-HF, CONFIRM-HF, AFFIRM-AHF, and IRONMAN; in maintenance hemodialysis the PIVOTAL trial¹⁰ reported non-inferiority of a proactive high-dose IV iron strategy versus a reactive low-dose strategy on the cardiovascular composite¹². Pre-specified meta-analyses of IV iron safety³¹ did not find an excess of serious infections with IV iron. Iron-overload safety (transferrin saturation persistently $> 50\%$, ferritin $> 500, 800$ ng/mL in non-inflammatory states) is the relevant concern with repeated dosing in low-iron-loss populations and is monitored by serial ferritin and TSAT³⁵.



Contraindications

Iron is contraindicated in: known hypersensitivity to the specific iron preparation or to any of its excipients; anemia not caused by iron deficiency (the indication is iron deficiency, not anemia of other cause); and iron overload syndromes including HFE hemochromatosis with elevated ferritin, transfusional iron overload, and other primary iron-loading disorders. Parenteral iron is additionally contraindicated in patients with a history of serious hypersensitivity reaction to any IV iron product ³⁸.

Caution is warranted in active systemic infection, where IV iron is typically deferred until the infection is controlled because of theoretical concerns about iron and bacterial virulence (the supporting evidence on real-world infection risk is mixed and pooled safety analyses have not consistently shown a clear excess). Caution is also warranted in patients with multiple drug allergies and asthma due to higher reported rates of mild infusion reactions ^{3539 384041}.

Drug interactions

Oral iron absorption is reduced by concomitant proton pump inhibitors, H2 blockers, calcium-containing supplements and dairy, antacids, levothyroxine, fluoroquinolone and tetracycline antibiotics, bisphosphonates, levodopa, mycophenolate, and tea polyphenols. Where coadministration is necessary, oral iron should be spaced by at least 2 hours from the interacting agent ³³. Ascorbic acid 200 mg coadministered with oral iron increases fractional absorption modestly; the clinical importance is debated and the addition is not universally required.

Intravenous iron has limited pharmacokinetic drug-drug interactions beyond temporary interference with serum iron and transferrin saturation measurements (which should be interpreted with caution within 1, 2 weeks of an IV iron dose). Concomitant erythropoiesis-stimulating agents are commonly co-administered in CKD and are not contraindicated; in fact, IV iron substantially reduces the ESA dose required in many patients ⁷¹⁰.

Adverse events

Most common adverse events with oral iron are gastrointestinal: nausea, abdominal pain, constipation, diarrhea, and dark stool. Tolkien et al. ³² reported pooled OR ~2.3 for any GI AE with ferrous sulfate vs placebo and ~1.5 for any GI AE with ferrous sulfate vs other oral iron preparations; the absolute event rates remain substantial across all oral iron salts.

Most common adverse events with IV iron are mild infusion-site and Fishbane-type reactions (flushing, chest tightness, back/joint pain, usually rate-related and self-limited), transient muscle aches, and transient post-infusion headache. Serious hypersensitivity reactions are rare with modern non-dextran preparations (~0.04, 0.1 per 100 administrations) ³¹³⁵. Hypophosphatemia is frequent with FCM (75% by day 14 in randomized data) and infrequent with FDI, iron sucrose, ferumoxytol, and iron dextran ¹⁶¹⁷. The Adkinson head-to-head ¹⁵ reported broadly comparable composite hypersensitivity rates between FCM and



ferumoxytol. Hetzel et al. ¹⁴ reported non-inferiority of ferumoxytol vs iron sucrose. Iron-overload risk with repeated IV iron is monitored by ferritin and TSAT.

↗ Monitoring Iron (Compounded) Therapy

Baseline assessment for any patient being started on iron therapy: complete blood count with indices, ferritin, transferrin saturation (iron and TIBC), and reticulocyte count if anemia is present; identification of the underlying cause of deficiency (menstrual loss, GI loss, malabsorption, dietary inadequacy, increased demand) is mandatory before committing to a repletion course [auerbach2024_consensus].

On-therapy monitoring: hemoglobin and ferritin at 4, 8 weeks of oral iron to confirm response (expected Hb rise ≥ 1 g/dL by 4 weeks); ferritin and TSAT at completion of an IV iron course to confirm repletion and at intervals during ongoing dosing to avoid iron overload (target ferritin generally < 500 ng/mL in non-CKD, with TSAT $< 50\%$). After FCM specifically, baseline and post-infusion serum phosphate are recommended given the documented hypophosphatemia signal [wolf2020, schaefer2022]. Patients on chronic IV iron should be re-evaluated periodically to ensure continued indication [auerbach2024_consensus].

☺ Iron (Compounded) in Special Populations

⊕ Iron (Compounded) Evidence Quality

The evidence base for iron repletion is one of the largest and most mature in clinical pharmacotherapy. Iron-deficiency anemia epidemiology is well characterized [kassebaum2014, lopez2016]. Oral iron pharmacokinetics, hepcidin biology, and dosing optimization have a strong recent mechanistic and clinical literature [moretti2015; stoffel2017; stoffel2020]. Indication-specific phase 3 trials support IV iron in CKD (DRIVE [coyne2007], FIND-CKD [macdougall2014], REPAIR-IDA [onken2014], FERWON-NEPHRO [bhandari2021], PIVOTAL [macdougall2019]), in chronic and acute heart failure with iron deficiency (FAIR-HF [anker2009], CONFIRM-HF [ponikowski2015], AFFIRM-AHF [ponikowski2020], IRONMAN [kalra2022], FERRIC-HF [okonko2008]; oral iron negative in IRONOUT-HF [lewis2017]), in inflammatory bowel disease (FERGIcor [evstatiev2011]), and in iron-deficiency anemia broadly (FERWON-IDA [auerbach2019_ferwon_ida], Hetzel et al. [hetzel2014], Adkinson et al. [adkinson2018]). Pregnancy and HMB indications are framed by guideline consensus [pavord2020, mansour2021_hmb] supported by epidemiologic mortality data [daru2018]. RLS is framed by IRLSSG consensus [allen2018_rls]. Perioperative anemia is framed by international consensus [munoz2017] and PREVENTT [richards2020]. Comparative IV iron safety, particularly hypophosphatemia after FCM, is supported by direct randomized comparisons [wolf2020; schaefer2022; tolkien2015].



Evidence specifically supporting compounded iron preparations is limited because the FDA-approved manufactured products dominate clinical use. The 503A role for iron is therefore narrow, custom oral concentrations and flavors for pediatric or dysphagic patients, alternate-day liquid pediatric formulations, and IM iron at non-commercial strengths in the rare patient who cannot use IV [auerbach2024_consensus]. RonanRx does not compound parenteral iron into multi-ingredient infusion bags.

📄 Major Iron (Compounded) Clinical Studies

Study	Design	Participants	Duration	Finding
FAIR-HF (Anker et al., NEJM 2009)	Phase III randomized, double-blind, placebo-controlled trial of IV ferric carboxymaltose in chronic HFrEF with iron deficiency (ferritin <100 ng/mL, or 100, 299 with TSAT <20%)	459	24 weeks	FCM significantly improved Patient Global Assessment and NYHA class vs placebo; benefit observed regardless of anemia status [anker2009]
CONFIRM-HF (Ponikowski et al., Eur Heart J 2015)	Phase III randomized, double-blind, placebo-controlled, multicentre trial of IV ferric carboxymaltose in chronic HFrEF with iron deficiency	304	52 weeks	Sustained improvement in 6-minute walk distance, NYHA functional class, and quality of life at 52 weeks; reduced first heart failure hospitalization [ponikowski2015]
AFFIRM-AHF (Ponikowski et al., Lancet 2020)	Multicentre randomized double-blind placebo-controlled trial of IV ferric carboxymaltose at discharge after acute heart failure with iron deficiency	1132	52 weeks	Numerical reduction in composite of recurrent HF hospitalization and CV death; primary endpoint not statistically significant under prespecified COVID-19-adjusted analysis (RR 0.79, 95% CI 0.62, 1.01) [ponikowski2020]
IRONMAN (Kalra et al., Lancet 2022)	UK investigator-initiated, prospective, randomized, open-label, blinded-endpoint trial of IV	1137	Median 2.7 years	Reduction in composite of HF hospitalizations and cardiovascular death in the COVID-19-adjusted analysis (RR



Study	Design	Participants	Duration	Finding
	ferric derisomaltose in chronic heart failure with iron deficiency			0.76, 95% CI 0.58, 1.00 [kalra2022]
FERRIC-HF (Okonko et al., JACC 2008)	Randomized observer-blind trial of IV iron sucrose vs no iron in iron-deficient symptomatic CHF, anemic and non-anemic strata	35	18 weeks	Improved exercise tolerance, NYHA class, and symptom score with IV iron sucrose; preceded FAIR-HF [okonko2008]
IRONOUT-HF (Lewis et al., JAMA 2017)	Randomized double-blind placebo-controlled trial of oral iron polysaccharide complex vs placebo in HFrEF with iron deficiency	225	16 weeks	Oral iron did not improve peak VO ₂ , supports the IV route specifically in HFrEF [lewis2017]
DRIVE (Coyne et al., J Am Soc Nephrol 2007)	Multicentre randomized open-label trial of IV ferric gluconate vs no iron in anemic hemodialysis patients on erythropoietin with high ferritin (500, 1200 ng/mL) and low TSAT (≤25%)	134	6 weeks	IV ferric gluconate produced meaningful hemoglobin response; challenged ferritin-based ceiling on IV iron in HD [coyne2007]
FIND-CKD (Macdougall et al., NDT 2014)	Randomized open-label trial of IV ferric carboxymaltose targeting two ferritin thresholds vs oral iron in non-dialysis CKD with IDA	626	56 weeks	High-ferritin-target IV FCM superior to oral iron and to low-ferritin-target IV FCM on the composite Hb response endpoint [macdougall2014]
REPAIR-IDA (Onken et al., NDT 2014)	Two identical phase 3 randomized open-label trials of IV ferric carboxymaltose vs iron sucrose in adults	2584	56 days	FCM non-inferior to iron sucrose on mean Hb change; modestly higher transient blood pressure elevation post-FCM [onken2014]



Study	Design	Participants	Duration	Finding
	with IDA and non-dialysis CKD			
PIVOTAL (Macdougall et al., NEJM 2019)	UK multicentre randomized open-label trial of proactive high-dose vs reactive low-dose IV iron sucrose in incident hemodialysis patients	2141	Median 2.1 years	Proactive high-dose IV iron non-inferior to reactive low-dose on composite of non-fatal MI, stroke, HF hospitalization, or death; reduced erythropoietin dose and transfusion requirement [macdougall2019]
FERGIcor (Evstatiev et al., Gastroenterology 2011)	Multicentre randomized open-label trial of IV ferric carboxymaltose vs iron sucrose in adults with IBD and IDA	485	12 weeks	FCM superior to iron sucrose on Hb response and required fewer infusions; established FCM as preferred IV iron in IBD [evstatiev2011]
FERWON-IDA (Auerbach et al., Am J Hematol 2019)	Multicentre randomized open-label trial of IV ferric derisomaltose vs iron sucrose in adults with IDA from various causes	1512	8 weeks	FDI non-inferior to iron sucrose on Hb response and superior on time-to-response; supported FDA approval of Monoferric [auerbach2019_ferwon_ida]
FERWON-NEPHRO (Bhandari et al., NDT 2021)	Multicentre randomized open-label trial of IV ferric derisomaltose vs iron sucrose in adults with non-dialysis CKD and IDA	1538	8 weeks	FDI non-inferior to iron sucrose on Hb change with fewer infusions and comparable safety [bhandari2021]
Hetzel et al. (Am J Hematol 2014)	Phase III multicentre randomized open-label trial of IV ferumoxytol vs iron sucrose in adults with IDA	605	5 weeks	Ferumoxytol non-inferior to iron sucrose on Hb response; comparable safety profile [hetzel2014]
Adkinson et al. (Am J Hematol 2018)	Multicentre randomized double-blind head-to-head	2014	Single-course follow-up	Composite moderate-to-severe hypersensitivity reactions and moderate-to-severe hypotension



Study	Design	Participants	Duration	Finding
	trial of IV ferumoxytol vs ferric carboxymaltose in adults with IDA, comparative hypersensitivity safety			broadly comparable between ferumoxytol and FCM; product-specific differences in AE subtypes [adkinson2018]
Wolf et al. (JAMA 2020)	Two parallel phase 3 randomized open-label trials of IV ferric carboxymaltose vs ferric derisomaltose for hypophosphatemia as the primary outcome in adults with IDA	245	5 weeks	Incident hypophosphatemia (<2.0 mg/dL) by day 14: 75% on FCM vs 8% on FDI in trial 1; similar magnitudes in trial 2; severe hypophosphatemia (<1.3 mg/dL) 11.3% on FCM vs 0% on FDI in trial 1 [wolf2020]
Schaefer et al. (JCEM 2022)	Pooled analysis of randomized FCM trials for risk factors for persistent (>2-week) and severe hypophosphatemia	—	Pooled trial-level analysis	Persistent hypophosphatemia common after FCM; risk factors include lower baseline phosphate, preserved renal function, and higher cumulative FCM dose; framework for routine post-FCM phosphate monitoring [schaefer2022]
PREVENTT (Richards et al., Lancet 2020)	UK multicentre randomized double-blind placebo-controlled trial of preoperative IV ferric carboxymaltose 10, 42 days before major elective abdominal surgery in anaemic adults	487	8 weeks post-randomization plus surgical follow-up	Co-primary endpoints (death or blood transfusion, number of transfusions) not met; pre-specified secondary analyses suggested fewer hospital readmissions and improved 8-week post-discharge Hb with IV iron [richards2020]
Stoffel et al. (Lancet Haematol 2017)	Stable-isotope ⁵⁷ Fe/ ⁵⁸ Fe oral iron absorption study in iron-depleted young women	40	Single-course measurement	Single morning doses on alternate days absorb more iron per dose and produce less hepcidin suppression of subsequent absorption than the



Study	Design	Participants	Duration	Finding
				same daily total split into twice-daily doses [stoffel2017]
Moretti et al. (Blood 2015)	Stable-isotope oral iron absorption study in iron-depleted young women, mechanistic hepcidin biology	54	Single-course measurement	Oral iron doses ≥ 60 mg significantly increase plasma hepcidin and reduce fractional iron absorption of subsequent doses within 24, 48 hours; mechanistic foundation for alternate-day dosing [moretti2015]
Stoffel et al. (Haematologica 2020)	Stable-isotope absorption study of alternate-day vs consecutive-day oral iron in iron-deficient anemic women	40	14-day course	Cumulative iron absorption higher with alternate-day single-morning dosing than with consecutive-day dosing at matched daily dose; confirms hepcidin-mediated absorption ceiling [stoffel2020]
Tolkien et al. (PLoS One 2015)	Systematic review and meta-analysis of GI adverse events with ferrous sulfate vs placebo and vs other oral iron preparations	—	Pooled trial-level analysis	GI AE OR ≈ 2.32 vs placebo, ≈ 1.99 vs IV iron, and ≈ 1.45 vs other oral iron preparations; quantifies the GI-tolerability problem with daily ferrous sulfate [tolkien2015]
Avni et al. (Mayo Clin Proc 2015)	Systematic review and meta-analysis of 103 RCTs of IV iron safety vs oral iron, IM iron, no iron, or placebo	—	Pooled trial-level analysis	IV iron not associated with increased serious adverse events overall; severe hypersensitivity rare; established baseline IV iron safety frame [avni2015]
Kassebaum et al. (Blood 2014)	Global Burden of Disease 2010 systematic analysis of anemia burden by cause, age, sex, year, region	—	1990, 2010 GBD database	Iron-deficiency anemia is the leading nutritional cause of years lived with disability worldwide; ~ 1.93 billion prevalent cases of anemia in 2010, with IDA the dominant subtype [kassebaum2014]
Daru et al. (Lancet Glob Health 2018)	Multilevel analysis of WHO Multicountry Survey on Maternal	312281	Cross-sectional with outcome ascertainment	Severe maternal anaemia significantly associated with increased risk of maternal



Study	Design	Participants	Duration	Finding
	and Newborn Health linking severe maternal anaemia to mortality			mortality (adjusted OR ~1.86); underlies urgency to correct severe antenatal IDA [daru2018]
Hutchinson et al. (Gut 2007)	Controlled study of dietary non-heme iron absorption with and without omeprazole in hereditary haemochromatosis	—	—	Proton pump inhibitor substantially reduced dietary non-heme iron absorption; mechanistic foundation for the well-documented PPI-iron interaction in repletion-failure cases [hutchinson2007]
Auerbach et al. (Am J Hematol 2024), Expert consensus	Multidisciplinary expert consensus guideline on intravenous iron uses, formulations, administration, and management of reactions	—	—	Practical guidance on product selection, infusion protocols, hypersensitivity-reaction management, and post-FCM phosphate monitoring; framework adopted into current US practice [auerbach2024_consensus]
Auerbach et al. (JAMA 2025), State-of-the-art review	Narrative review of diagnosis and management of iron deficiency and IDA in adults	—	—	Synthesizes diagnostic ferritin thresholds (often higher than the historic <15 ng/mL given inflammation), alternate-day oral iron dosing, and modern IV iron product selection [auerbach2025_jama]

Iron (Compounded) Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

Oral iron: only a small fraction (5, 20%) of an oral iron dose is absorbed in iron-replete adults; fractional absorption rises in iron deficiency. A single dose increases plasma hepcidin within 24 hours and reduces absorption of further doses given within that window; alternate-day single-morning dosing therefore absorbs more iron per administered dose than divided daily dosing [moretti2015, stoffel2017, stoffel2020] [auerbach2017_history]. Absorption is reduced by PPI, calcium, antacids, and tea polyphenols [hutchinson2007].



IV iron: each parenteral iron product has a distinctive PK profile determined by its carbohydrate shell. Iron sucrose is least stable, requiring multiple small doses; FCM and FDI are highly stable, supporting single-dose total-dose infusion of 750, 1000 mg; ferumoxytol supports 510 mg single doses. After infusion, iron is taken up by reticuloendothelial macrophages (liver, spleen, bone marrow), incorporated into ferritin storage, then exported to transferrin over hours to days for delivery to erythroid precursors. Serum iron and TSAT measurements within 1, 2 weeks of an IV iron dose reflect the colloid rather than the physiologically utilizable pool and should be interpreted with caution [auerbach2017_history; auerbach2024_consensus].

Pharmacodynamics

Pharmacodynamic effect of iron repletion is reflected in: reticulocytosis within 5, 10 days; rise in hemoglobin of ≥ 1 g/dL by 2, 4 weeks of effective oral repletion (faster after a complete IV dose); normalization of ferritin and TSAT once stores are filled. Reversal of fatigue, exercise intolerance, and (in RLS) sensory-motor symptoms tracks repletion of the relevant iron pool, which for RLS appears to be brain-specific rather than systemic [allen2018_rls].

Negative pharmacodynamic effects: oral iron-induced hepcidin elevation transiently blunts intestinal iron absorption [moretti2015]; FCM-induced FGF23 cleavage products drive renal phosphate wasting and hypophosphatemia for days to weeks [wolf2020, schaefer2022] [allen2018_rls].

↕↑ Comparing Iron (Compounded) Formulations

Oral iron preparations differ modestly in tolerability at matched elemental iron dose [auerbach2024_consensus]. Ferrous sulfate is the most widely studied and the cheapest; ferrous fumarate and ferrous gluconate are alternatives. Ferrous bisglycinate is often promoted as better tolerated; the evidence is mixed. Ferric maltol (Accrufer) is a newer oral preparation with a distinct GI tolerability profile but limited head-to-head data. Polysaccharide-iron complex products were negative on the primary VO₂ endpoint of IRONOUT-HF in HFrEF [lewis2017].

IV iron preparations differ materially. Iron sucrose (Venofer) is the long-standing CKD-labeled option, dosed at 100, 300 mg per administration and typically requiring multiple visits [auerbach2024_consensus; fda_label_venofer]. Ferric carboxymaltose (Injectafer) and ferric derisomaltose (Monoferric) are total-dose-infusion-capable [fda_label_injectafer; fda_label_monoferric]. Ferumoxytol (Feraheme) is dosed in two 510 mg infusions [fda_label_feraheme]. Low-molecular-weight iron dextran (INFeD) can deliver total-dose infusion but is now uncommon [fda_label_infed]. The dominant comparative-safety axis between FCM and FDI is hypophosphatemia, which is far more frequent and severe with FCM [wolf2020, schaefer2022]; the FCM-vs-ferumoxytol axis [adkinson2018] is broadly balanced on composite hypersensitivity outcomes with distinct AE subtypes.



🔑 Iron (Compounded) Storage and Handling

Manufactured oral iron preparations are stored at controlled room temperature in light-resistant child-resistant containers. Compounded oral liquid iron is stored per the pharmacy's stability data and beyond-use date assignment under USP <795>; refrigerated storage may be required for some flavored preparations to maintain palatability and chemical stability [usp_795].

Manufactured IV iron products are stored at controlled room temperature in their original carton; once diluted in normal saline they have a manufacturer-specified short in-use window [usp_795]. IV iron is not a cold-chain product. Each product has a specific permitted diluent and final-concentration window; deviation is a documented source of free-iron adverse reactions [fda_label_injectafer, fda_label_venofer, auerbach2024_consensus].

🏢 Iron (Compounded) Compounding & Operations

503A compounding

Compounded iron preparations are prepared under 503A on patient-specific prescriptions in state-licensed compounding pharmacies [fda503a]. Nonsterile oral preparations (capsules, liquids) are governed by USP General Chapter <795>, with documented active ingredient sourcing, potency verification, and beyond-use date assignment per the pharmacy's quality-management system [usp_795]. The compounded role for iron is narrow because the FDA-approved manufactured oral and IV products are widely available and address most clinical needs.

Legitimate 503A iron-compounding scenarios at RonanRx include custom-strength oral liquid iron for pediatric or dysphagic adults (with alternate-day single-morning dosing per current absorption evidence [stoffel2017]) and custom-strength oral capsules where no manufactured product matches the prescribed regimen [fda503a]. RonanRx does not compound parenteral iron into multi-ingredient infusion bags and does not dispense 'iron infusions' as a routine wellness service: the manufactured IV iron products are the standard of care, and the physical-chemical incompatibilities of iron with amino-acid, B-vitamin, ascorbate, and thiol components of common 'cocktail' infusions are well documented [auerbach2024_consensus, fda_label_injectafer].

Pharmacist review

Each prescription for compounded iron undergoes pharmacist review prior to dispensing [fda_essentially_a_copy]. The review confirms: a documented patient-specific clinical reason that the manufactured iron product is not appropriate (custom strength not available, excipient sensitivity, dysphagia, pediatric dose not matched by a commercial liquid); absence of iron-overload contraindications



(HFE hemochromatosis with elevated ferritin, transfusional overload); a clinical workup for the underlying cause of iron deficiency (menstrual loss, GI loss, malabsorption, dietary inadequacy); and a prescribed regimen consistent with current evidence on alternate-day oral iron dosing [stoffel2017, stoffel2020] where applicable.

RonanRx does not fill prescriptions that read as routine substitution of compounded for manufactured iron without documented clinical rationale, consistent with FDA guidance on compounded copies of commercially available drugs [fda_essentially_a_copy]. RonanRx does not compound parenteral iron into multi-ingredient infusion bags under any circumstances [auerbach2024_consensus].

Quality and traceability

Active pharmaceutical ingredients for compounded iron preparations are sourced from FDA-registered facilities with documented certificates of analysis. Each compounded batch is recorded with lot numbers traceable to API source, compounding date, beyond-use date, potency verification, and dispensing pharmacist of record. Finished product lot records are retained per state board of pharmacy retention requirements.

Cold chain

Oral iron, manufactured or compounded, is not a cold-chain product and is stored at controlled room temperature. Compounded flavored liquid iron preparations may require refrigeration to maintain palatability and chemical stability per the pharmacy's stability data [usp_795]. Manufactured IV iron products (iron sucrose, FCM, FDI, ferumoxytol, iron dextran) are also stored at controlled room temperature in original carton and are not cold-chain products [fda_label_injectafer; fda_label_venofer].

🗨 Frequently Asked Questions About Iron (Compounded)

What is the best oral iron to take?

For most non-pregnant adults with iron-deficiency anemia, ferrous sulfate at 40, 100 mg elemental iron taken as a single morning dose every other day is well supported by recent absorption studies [stoffel2017; stoffel2020; tolkien2015]. Other ferrous salts (fumarate, gluconate, bisglycinate) and oral ferric maltol are reasonable alternatives if ferrous sulfate is not tolerated. Take oral iron away from coffee, tea, calcium, antacids, and proton pump inhibitors [moretti2015; hutchinson2007].

When is IV iron preferred over oral iron?

When oral iron has failed, is not tolerated, is contraindicated (active inflammatory bowel disease, post-bariatric surgery), or when rapid repletion is needed (late pregnancy with severe IDA, pre-operative anemia, dialysis CKD, decompensated heart failure with iron deficiency). The choice between IV iron products is driven by access, single-dose-infusion capability, and product-specific safety considerations



such as the higher hypophosphatemia rate with ferric carboxymaltose [auerbach2024_consensus; wolf2020].

Are IV iron infusions dangerous?

Modern non-dextran IV iron preparations (iron sucrose, ferric carboxymaltose, ferric derisomaltose, ferumoxytol) have a serious-hypersensitivity-reaction rate of approximately 0.04, 0.1 per 100 administrations, much lower than the historic high-molecular-weight iron dextrans [avni2015; adkinson2018]. Mild infusion reactions (transient flushing, chest tightness, back/joint pain) occur with too-rapid administration and resolve with infusion-rate reduction. IV iron should be administered in a setting equipped to manage hypersensitivity reactions [auerbach2017_history; auerbach2024_consensus].

Why does ferric carboxymaltose cause low phosphate?

FCM increases circulating intact FGF23 cleavage products, which drive renal phosphate wasting. In randomized comparison with ferric derisomaltose, FCM produced hypophosphatemia (<2.0 mg/dL) in 75% of patients by day 14 vs 8% on FDI [wolf2020; schaefer2022]. The effect can be severe and prolonged, can cause weakness and bone pain, and, with repeated dosing, has been associated with osteomalacia and stress fractures. Routine pre- and post-FCM phosphate monitoring is recommended, and ferric derisomaltose is preferred when repeated IV iron is anticipated [auerbach2024_consensus].

Does RonanRx compound 'iron drips' or IV iron cocktails?

No. RonanRx does not compound parenteral iron into multi-ingredient infusion bags and does not dispense IV iron as a routine wellness service. Iron is physically and chemically incompatible with many components of common 'cocktail' infusions, and the FDA-approved manufactured IV iron products (Venofer, Injectafer, Monoferic, Feraheme, INFeD) are the standard of care [auerbach2024_consensus; fda_label_injectafer; fda_label_venofer].

What is the 503A role for compounded iron?

Custom-strength oral liquid iron for pediatric or dysphagic patients where no manufactured product matches the prescribed regimen (including alternate-day single-morning dosing schedules), excipient-free or specific-excipient oral preparations for patients with documented sensitivity, and (rarely) IM iron at non-commercial strengths [stoffel2017]. All are dispensed only on a patient-specific prescription with documented clinical rationale [fda503a; fda_essentially_a_copy].

Should I take iron if I have low ferritin but normal hemoglobin?

Iron deficiency without anemia can cause fatigue and (with low brain iron) restless legs syndrome [allen2018_rls]. Current expert reviews support evaluating ferritin and considering iron repletion in non-anemic iron-deficient adults symptomatic of deficiency, but caution against routine IV iron for fatigue alone



without documented deficiency and underlying cause workup [auerbach2024_consensus; auerbach2025_jama]. Talk to your clinician about your ferritin number in context.

Who should not take iron?

Patients with iron overload, HFE hemochromatosis with elevated ferritin, transfusional overload, other primary iron-loading disorders, and patients with a history of serious hypersensitivity reaction to a specific IV iron product. Anemia not caused by iron deficiency is not an indication for iron therapy. Caution in active systemic infection, where IV iron is typically deferred until the infection is controlled [auerbach2024_consensus; fda_label_venofer].

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🔗 How to Access Iron (Compounded)

Compounded Iron (Compounded) 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 Iron (Compounded), 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)

