



IRON SUCROSE VERSUS FERRIC CARBOXYMALTOSE IN PREGNANCY-ASSOCIATED IRON DEFICIENCY ANAEMIA

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1. INTRODUCTION

Anaemia in pregnancy is common, and iron deficiency is its leading cause. The World Health Organization (WHO) estimated that roughly four in ten pregnant women worldwide were anaemic in 2011, with the highest burden in South Asia and sub-Saharan Africa [1, 2]. Haemoglobin (Hb) below 110 g/L in the second and third trimesters is the usual diagnostic threshold, and below 70 g/L counts as severe [3]. The consequences are not trivial. Severe anaemia has been linked to higher maternal mortality, and iron deficiency has been associated with preterm birth, low birth weight and poorer maternal wellbeing [4, 5]. Pregnancy raises iron needs sharply

Abstract

Iron deficiency anaemia affects a large share of pregnant women worldwide, and intravenous iron is increasingly used when oral therapy fails, is not tolerated or cannot work quickly enough. This review, which reports no new data, compares iron sucrose and ferric carboxymaltose as they are described in the published literature. It covers the chemistry and pharmacokinetics of the two complexes, dosing schedules, haemoglobin and ferritin responses, hypersensitivity, hypophosphataemia and other adverse effects, and practical points for the pharmacist and obstetric team. Both agents correct anaemia faster than oral iron, and ferric carboxymaltose allows fewer visits, but head-to-head pregnancy trials are small and short, and evidence on maternal and neonatal outcomes remains limited.

because the red cell mass expands and the fetus and placenta take their share, so a woman who starts pregnancy with small stores can be depleted by the late second trimester. Treatment has to be chosen quickly and with an eye on the weeks remaining until delivery.

Oral ferrous salts are the first-line option for most women, yet they often disappoint. Gastrointestinal side effects lead many to stop, absorption is limited by hepcidin, and the response in advanced pregnancy may be too slow to correct anaemia before delivery [6, 7]. Intravenous (IV) preparations bypass the gut altogether. Two of the most widely used in obstetric practice are iron sucrose, a long-established product given in repeated small doses, and ferric carboxymaltose (FCM), a newer complex that permits a large dose in one short infusion [8, 9]. Clinicians and pharmacists often have to choose between them, usually guided by local protocols and drug budgets rather than head-to-head evidence. This review sets out what published studies say about the two agents in pregnancy, with attention to chemistry, efficacy, safety and practical use. It draws on reviews, randomised trials and observational reports and presents no new patient data.

Why does the comparison matter now? Intravenous iron was once reserved for women who could not tolerate tablets or who were close to term with severe anaemia, partly because older dextran products carried a real risk of serious reactions. Newer carbohydrate complexes changed that picture. Auerbach and Macdougall argued that fear of serious reactions had outrun the evidence for modern products, and that withholding treatment from women who need it carries its own harm [10]. Recommendations from the United Kingdom and from European expert groups now describe IV iron as an option from the second trimester when oral therapy has failed, is not tolerated or when anaemia is found late [6, 11]. As use grows, differences between individual products in dosing, visits, cost and rare adverse effects begin to matter more than the general question of whether iron should be given by vein.

2. DIAGNOSIS AND ORAL IRON

Diagnosis is less straightforward than it sounds. Haemoglobin alone cannot separate iron deficiency from other causes of anaemia such as thalassaemia trait, folate or vitamin B12 deficiency, or chronic inflammation, and it falls normally in pregnancy through haemodilution. Serum ferritin is the most

useful single test, although it is an acute-phase protein and can read falsely reassuring when infection is present. A systematic review of thresholds suggested that a ferritin cut-off higher than the conventional 15 µg/L may suit pregnancy better, and UK guidance uses 30 µg/L as a practical threshold for treatment [12, 13]. Transferrin saturation and red cell indices add supporting information. Getting the diagnosis right before choosing a product has a practical payoff: women with a haemoglobinopathy or a non-iron cause gain nothing from an infusion, and treatment without a confirmed deficit exposes them to avoidable risk.

Oral therapy has a respectable evidence base. Cochrane reviews found that daily iron supplementation reduces anaemia and iron deficiency at term, though the effect on clinical outcomes for mother and baby is less certain [7, 14]. Absorption is limited because a dose raises hepcidin, which then blunts uptake from the next dose for about a day. Stoffel and colleagues showed in iron-depleted women that giving iron on alternate days or as a single morning dose improved fractional absorption compared with consecutive-day or twice-daily dosing [15]. The finding has made low-dose and alternate-day schedules popular, and these may also reduce side effects. For women with mild anaemia found early, oral iron is still a reasonable first step. Its limit is time: even with good adherence, a meaningful Hb rise within a few weeks cannot be guaranteed when the delivery date is near [6, 11].

The case for IV iron is strongest in particular situations. These include intolerance or failure of oral therapy after a trial of a few weeks, malabsorption, moderate or severe anaemia diagnosed late in the second or third trimester, and conditions in which a rapid rise is wanted before delivery, such as a woman at high risk of postpartum haemorrhage [6, 11]. Most guidelines avoid the first trimester because safety data there are limited and organogenesis is under way [6, 12]. Meta-analyses of randomised trials report that IV iron raises Hb and ferritin more than oral iron and causes fewer gastrointestinal complaints, though the trials were mostly small and of uneven quality [16, 17, 18].

Whether that translates into fewer transfusions, fewer preterm births or better neonatal outcomes is still not established. The decision therefore rests mainly on practical grounds, speed and tolerability, rather than on proof of better clinical endpoints.

Monitoring after treatment is simple but often neglected. Haemoglobin is usually rechecked two to four weeks after an infusion, and a rise of about 10 g/L within that window is taken as a satisfactory response in several guidelines. Ferritin should be interpreted cautiously for some weeks, because it is transiently raised by the iron just given [6, 19]. A poor response should prompt a search for ongoing blood loss, haemoglobinopathy, inflammation or an incorrect diagnosis, not an automatic repeat dose. The same checks apply after delivery, when women who were treated late in pregnancy may still be short of iron and at risk of fatigue in the early weeks with a newborn [11]. A clear plan for who orders the repeat blood test, and who acts on it, is part of the prescription.

3. PHARMACOLOGY OF THE TWO COMPLEXES

Iron sucrose is a polynuclear iron(III)-hydroxide core surrounded by sucrose molecules, with an average molecular weight in the region of 34–60 kDa. The complex is relatively weak, so a measurable fraction of iron is released in labile form after injection, and for that reason single doses are restricted. In most labels and obstetric protocols the dose is 100–200 mg per infusion, repeated over several days or weeks until the calculated deficit is replaced [8, 20]. Iron is taken up by macrophages of the reticuloendothelial system, released to transferrin and delivered to erythroid precursors. The plasma half-life is short, a matter of hours, but the iron persists in tissue stores [8, 20]. The total dose is commonly calculated with the Ganzoni formula, which multiplies body weight by the Hb deficit and adds an allowance for stores. Regimens for pregnant women therefore tend to mean several visits.

Ferric carboxymaltose has a ferric hydroxide core stabilised by a carbohydrate shell derived from maltodextrin, forming a larger

and more stable complex of about 150 kDa [9, 21]. Because it releases little labile iron, a much bigger dose can be given at once; the licensed single dose is up to 1000 mg, infused over at least 15 minutes, with a ceiling set by body weight [9, 21]. Iron is again handled by the reticuloendothelial system, and radiolabelled studies in iron-deficient patients showed rapid and extensive red cell iron utilisation [9]. For many women the whole deficit can be replaced in one or two visits. The drug is also linked to a rise in fibroblast growth factor 23 (FGF23), which is the starting point for the phosphate effects discussed later [22, 23]. Pharmacists should check which weight-based dose table their local label follows.

Hepcidin sits behind both clinical observations and practical limits. Oral iron has to cross the enterocyte under hepcidin control, whereas IV iron enters the reticuloendothelial system directly and is then released to transferrin according to need, which is why a single infusion can overcome a block that defeats tablets [19, 20]. The route does not remove the problem entirely. In women with infection or inflammation, raised hepcidin traps iron in macrophages and can blunt the erythropoietic response even when stores are full, so a poor Hb rise after an infusion does not always mean the dose was too small [19]. Serum ferritin and transferrin saturation both rise within days of an infusion and can mislead if sampled too early. For this reason, laboratory follow-up is timed for weeks after treatment, not days.

Geisser and Burckhardt grouped IV iron products by the stability of the complex. Robust complexes, which include iron dextran and FCM, release little iron to the plasma and tolerate large single doses; semi-robust complexes such as iron sucrose release more and need smaller, repeated doses; labile complexes release the most and are given in the smallest amounts [20]. Physicochemical comparisons of newer products point the same way, linking core size, carbohydrate shell and stability constant to the maximum dose that can be given safely [21]. The classification is a neat teaching tool, although it does not predict everything. Phosphate effects, for instance, do not follow

complex stability in any simple manner, and hypersensitivity depends on more than free iron. It is best treated as a guide to dosing rather than a full account of tolerability.

4. COMPARATIVE EFFICACY

Direct comparisons in pregnancy are few. Christoph and colleagues compared high-dose FCM with iron sucrose in a small group of pregnant women and reported comparable correction of anaemia, with the single-dose regimen needing fewer attendances [24]. Jose and co-workers carried out a randomised trial in Indian women in the second and third trimesters with iron deficiency anaemia; Hb and ferritin rose in both arms, with faster or larger improvements reported for the carboxymaltose group and similar tolerability [25]. The same research group had earlier compared the two drugs for anaemia due to abnormal uterine bleeding in non-pregnant women, with results pointing the same way [26]. Observational series in Australia support the safety and efficacy of FCM in pregnancy, though without a comparator [27, 28]. Overall the direction of effect favours carboxymaltose for speed and convenience, but sample sizes are small.

Both agents have also been tested against oral iron. Iron sucrose has been compared with tablets in randomised trials in pregnancy [29, 30]. A Turkish trial found higher Hb and ferritin with the IV route [29], while a large multicentre Indian trial examined women with moderate to severe anaemia, a group in whom oral therapy is slow [30]. The FER-ASAP trial compared FCM with oral ferrous sulphate in women with iron deficiency anaemia in the late second and third trimesters and reported a higher Hb response with better tolerability [31]. These results fit the pooled analyses, which conclude that IV iron raises Hb faster, with the size of the gain varying with baseline severity and the interval between treatment and measurement [16, 17, 18]. A weakness of this literature is that most trials ran for weeks only and measured laboratory endpoints. Few were large enough to examine rarer events or neonatal outcomes.

The main comparative findings are collected in table 1, which also records dose schedules

and the type of evidence behind each entry. Entries are grouped by theme in fig. 1: pharmacology, efficacy, safety and practice. The grouping makes plain where the literature is thick and where it is thin. Efficacy and practice entries are the most numerous, because most trials report Hb response and visit numbers, whereas direct comparisons in pregnancy are still few [24, 25]. Pharmacological entries explain the dosing difference, and practice entries capture operational consequences, such as fewer visits and the need for monitored infusion areas. Read across the table, FCM reduces the number of visits, iron sucrose has the longer record and a gentler phosphate profile, and neither has convincingly been shown to improve neonatal outcomes [17, 22, 32].

5. SAFETY AND TOLERABILITY

Hypersensitivity is the reaction clinicians fear most. Modern carbohydrate complexes carry a much lower risk of true anaphylaxis than high-molecular-weight iron dextran, and a large cohort analysis of insured patients in the United States found anaphylaxis to be uncommon with all the main products, including iron sucrose and FCM [33]. Many reactions that look like anaphylaxis are infusion reactions, with flushing, chest tightness and back pain that settle on slowing or pausing the infusion. They are generally attributed to complement activation-related pseudoallergy and not to immunoglobulin E (IgE) mediated allergy [34, 35]. Rampton and colleagues recommended that centres give IV iron only where trained staff and resuscitation equipment are available, with observation after the infusion [34]. In pregnancy, guidance applies the same precautions as in other patients, with extra attention to maternal hypotension and fetal monitoring if a reaction occurs [6, 10].

Hypophosphataemia has emerged as the most distinctive difference between products. In two randomised trials in iron-deficient adults, FCM caused a far higher incidence of low serum phosphate than iron isomaltoside, and the effect lasted weeks in many patients [32]. Mechanistic work showed a rise in intact FGF23, which promotes renal phosphate loss and reduces vitamin D activation [22]. Iron sucrose appears to cause the effect less often,

although it is not entirely free of it [23]. Most affected patients have no symptoms, but severe or prolonged cases with osteomalacia and fractures have been described after repeated carboxymaltose doses [23]. In pregnancy the data are limited to small series and secondary analyses, and the clinical meaning of a temporary dip in phosphate for mother and fetus is unclear. A reasonable approach is to avoid repeated high doses in women with risk factors, and to check phosphate in anyone with symptoms [23].

Table 1: Iron sucrose and ferric carboxymaltose in pregnancy: summary of published findings

Topic	Reported finding	Ref.
Complex structure iron sucrose	Semi-robust complex of about 34–60 kDa; more labile iron, so single doses are limited	[8, 20]
Complex structure ferric carboxymaltose	Larger, robust complex of about 150 kDa; little labile iron, permitting large single doses	[9, 21]
Usual dose schedule	Iron sucrose 100–200 mg per infusion, repeated; FCM up to 1000 mg in one infusion	[9, 20]
Head-to-head trials in pregnancy	Comparable or better Hb response with FCM, usually with fewer visits	[24, 25]
Versus oral iron	Both IV products raise Hb faster than tablets, with less gastrointestinal upset	[29, 30, 31]
Pooled analyses	IV iron improves Hb; evidence on maternal and neonatal outcomes remains inconclusive	[16, 17, 18]
Hypersensitivity	Serious reactions rare with both; usually infusion reactions rather than IgE-mediated allergy	[33, 34, 35]
Hypophosphataemia	More frequent and longer-lasting after FCM than after iron isomaltoside; iron sucrose appears lower	[22, 23, 32]
Monitoring	Recheck Hb after two to four weeks; ferritin is transiently raised by the dose	[6, 19]
Timing in pregnancy	Second and third trimesters; first trimester generally avoided	[6, 11, 12]

Other adverse effects are mostly minor. Headache, nausea, dizziness, transient

hypotension, injection-site reactions and a metallic taste are the commonest complaints, and extravasation can leave long-lasting brown skin staining, a problem avoided by secure cannulation and an observed infusion [10, 27]. In the Australian series of FCM in pregnancy, no serious maternal or neonatal adverse events attributable to the drug were reported, although the samples were modest in size [27, 28]. Because iron sucrose requires repeated visits, a woman given it faces more cannulations and more chances for such problems, while a single large FCM dose concentrates exposure into one sitting. Neither pattern is clearly safer on current evidence. Oxidative stress and a higher infection risk with iron have been argued in theory, but there is no convincing signal for either product in pregnancy [10, 19].

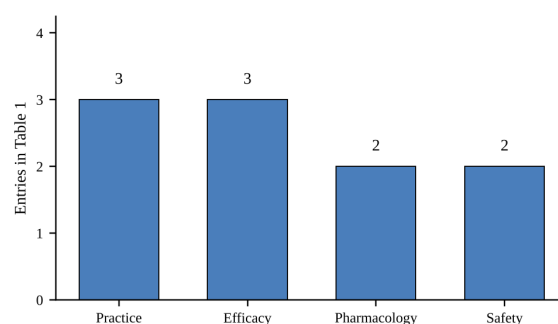


Fig. 1: Number of entries in table 1 by theme

6. PRACTICE AND LIMITATIONS

Choosing between the two in practice involves more than efficacy. Iron sucrose suits settings where short infusions of 200 mg can be given on an outpatient basis and where phosphate effects are a concern; FCM suits a woman who lives far from the hospital, works, has other children, or is close to term and needs the deficit corrected fast [11, 24]. Acquisition cost differs, with the newer product usually the more expensive per milligram, but cost analyses vary by health system and savings from fewer visits can offset part of the price difference. Infusion capacity and staffing matter, as does training of nurses to recognise infusion reactions. Local formularies often settle the question. Where both are available, shared decision-making that explains the number of visits, the small risk of reactions and the phosphate issue seems the most defensible approach [6, 34].

Dosing needs care. Total iron requirement is usually calculated from weight, target Hb and iron stores using a Ganzoni-type formula or a simplified dose table, and the result is rounded to a practical dose [11, 19]. Labels limit FCM to 1000 mg in a single infusion with a weekly ceiling tied to body weight, and iron sucrose to 200 mg per dose, with the cumulative maximum set by the product information [9, 20]. Iron is stored but not excreted, so repeat dosing without checking ferritin is discouraged [10, 19]. Women at the extremes of body weight, with multiple pregnancy or with ongoing bleeding may need adjustment and earlier review. Pharmacist verification of the calculation and of the product matters, since the two have different maximum doses and infusion times, and mixing up the schedules is the kind of slip that dose banding in busy units is meant to prevent.

Preparation and administration details are set out in the product information and deserve to be checked at the bedside. Both products are diluted in sodium chloride 0.9% solution and not in glucose, the infusion rate depends on the dose given, and FCM in particular has minimum infusion times for larger doses [9, 20]. The patient should be seated or lying down and observed for a period after the infusion ends, with blood pressure and pulse recorded. Dilution errors and rate errors are the commonest preventable problems in clinical areas where IV iron is given only occasionally. A one-page protocol, stored with the drug, listing the dose table, diluent, rate and observation period is a simple safeguard that many units adopt.

The evidence has several gaps. Head-to-head trials in pregnancy are few, small and short, and often run in single centres with open-label designs, which raises the risk of bias in subjective outcomes [24, 25]. Haemoglobin and ferritin are surrogate endpoints, and the trials rarely follow women through delivery and beyond to examine postpartum haemorrhage, transfusion, breastfeeding, infant iron status or neurodevelopment. Phosphate was seldom measured in older pregnancy studies, so the risk of hypophosphataemia in pregnant women is poorly characterised [23, 32].

Populations are also unrepresentative: much of the data comes from India and Western Europe, with little on women with sickle cell disease, malaria, human immunodeficiency virus (HIV) infection or obesity, groups with a high anaemia burden [2, 30]. Future studies should be multicentre, include clinical endpoints, report phosphate, and use registry follow-up to detect rare reactions [17, 35].

7. CONCLUSION

Iron sucrose and FCM both correct iron deficiency anaemia in pregnancy more quickly than oral iron and with fewer gastrointestinal complaints [16, 29, 31]. The main practical difference is dose: repeated infusions of up to 200 mg for iron sucrose against a single large infusion for FCM, which cuts visits and may suit women late in pregnancy (table 1) [9, 24, 25]. The main safety difference is hypophosphataemia, more frequent after FCM, while serious hypersensitivity is rare with both products [22, 32, 33]. Neither choice is wrong. The better choice is the one that fits the woman's gestation, travel, bleeding risk and local resources.

Neither product has been shown to improve neonatal outcomes, and pregnancy-specific trials remain small [17, 18]. In day-to-day practice, pharmacists can add value by checking the diagnosis, calculating and verifying doses, counselling on infusion reactions and arranging follow-up of haemoglobin [6, 19]. Larger trials with clinical and phosphate endpoints are needed before either product can be declared superior in this population, and until they exist, decisions should be framed openly as a balance of convenience, cost and uncertain long-term effects [23, 25]. Until then, the choice remains a matter of judgement.

8. ABBREVIATIONS

FCM-Ferric carboxymaltose, FGF23-Fibroblast growth factor 23, Hb-Haemoglobin, HIV-Human immunodeficiency virus, IgE-Immunoglobulin E, IV-Intravenous, kDa-Kilodalton, WHO-World Health Organization

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10. AUTHORS CONTRIBUTIONS

All the authors have contributed equally.

11. CONFLICT OF INTERESTS

Declared none

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