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Review Article

A Pharmacokinetic and Brief Outcomes Study of Plant-Derived Iron in Healthy and Iron Deficient Anemic Adults

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Abstract

Iron deficiency remains the most prevalent micronutrient deficiency worldwide, and conventional oral iron salts, while effective, are frequently limited by gastrointestinal intolerance and regulatory feedback mechanisms that constrain absorption. Ferritin-bound iron represents a physiologically distinct form of dietary iron that is absorbed through receptor-mediated pathways and may offer a differentiated pharmacokinetic profile. This study characterized the acute pharmacokinetics and short-term biological responses to a plant-based ferritin iron supplement (SloIron®) in healthy adult women and women with IDA. In a randomized, double-blind, parallel-group study, participants received a single 60 mg elemental iron dose under fasting conditions followed by daily supplementation for approximately 28 days, with iron metabolism biomarkers assessed over 24 hours and at study completion. Following the acute dose, both cohorts demonstrated sustained, time-dependent increases in serum iron and transferrin saturation, with higher systemic exposure and later peak concentrations in the IDA group, consistent with enhanced physiological uptake in iron-deficient states. Hepcidin responses were modest and variable, with lower overall exposure in IDA participants. Over 30 days, healthy subjects maintained iron biomarkers within normal physiological ranges, while IDA participants exhibited significant directional improvements in serum iron, ferritin, transferrin saturation, and hemoglobin, indicating early biological repletion without evidence of dysregulated iron homeostasis. SloIron® was well tolerated, with no serious adverse events or clinically relevant laboratory abnormalities. These findings demonstrate that ferritin-bound iron produces a sustained systemic iron signal and early biomarker improvements in iron-deficient individuals, supporting its role as a physiologically compatible iron source with a pharmacokinetic profile distinct from high-dose ferrous salts.

Keywords: *Iron; Ferritin-Bound Iron; Iron Pharmacokinetics; Iron Deficiency Anemia*

Abbreviations

IDA: Iron Deficiency Anemia; PK: Pharmacokinetic(s); DMT1: Divalent Metal Transporter 1; SCARA5: Scavenger Receptor Class A Member 5; ICH E6 (R2): International Council for Harmonisation E6 (R2) Good Clinical Practice Guideline; IRB: Institutional Review Board; C_{max}: Maximum Observed Concentration; T_{max}: Time to Maximum Concentration; AUC_{0-12}: Area Under the Concentration-Time Curve from 0-12 Hours; AUC_{0-24}: Area Under the Concentration-Time Curve from 0-24 Hours; t½: Terminal Elimination Half-Life; TIBC: Total Iron-Binding Capacity; UIBC: Unsaturated Iron-Binding Capacity; TSAT: Transferrin Saturation

Introduction

Iron deficiency remains the most prevalent micronutrient deficiency worldwide and continues to represent a major public health challenge across diverse populations, including women of reproductive age, children, and individuals with chronic disease [1]. Oral iron supplementation, most commonly consisting of ferrous salts, is effective for replenishing iron stores; however, it is limited by poor gastrointestinal tolerability, low absorption at higher doses, and physiological regulatory feedback mechanisms, such as hepcidin-mediated suppression of iron uptake [2]. These limitations have driven interest in alternative dietary and supplemental iron forms.

Ferritin is a widely distributed iron-storage protein found in both plant and animal tissues, capable of sequestering thousands of iron atoms within a highly stable protein shell in the form of thousands of biominerals with the structure $Fe_2O_3H_2O$. In plant-based foods, particularly legumes such as peas and soybeans, iron-containing ferritin represents a major proportion of total iron content. Contrary to earlier assumptions that ferritin-bound iron is poorly bioavailable, a substantial body of human, animal, and cellular evidence demonstrates that ferritin iron is efficiently absorbed and is comparable to ferrous sulfate with respect to overall bioavailability and its absorption is not inhibited by phytates. Controlled human isotope studies have shown that iron absorption from purified soybean ferritin-iron or animal ferritin-iron does not differ significantly from absorption of iron administered as ferrous sulfate, even in the presence of dietary inhibitors such as phytate, and across a range of baseline iron statuses. These findings have been replicated using both whole-body retention and red blood cell incorporation methodologies, supporting the physiological relevance of ferritin-iron absorption *in vivo* [3-8].

Mechanistic studies indicate that ferritin-bound iron is absorbed via a pathway distinct from classical nonheme iron transport mediated by divalent metal transporter 1 (DMT1). *In vitro* studies using polarized Caco-2 intestinal epithelial cells demonstrate that ferritin undergoes saturable, receptor-mediated uptake through clathrin- and AP2-dependent endocytosis, followed by intracellular degradation of the protein shell and release of iron into the iron pool [9-11]. Importantly, ferritin-iron uptake is relatively insensitive to common dietary modifiers of ferrous iron absorption, including phytates, calcium, and polyphenols, suggesting that ferritin may bypass key luminal constraints that limit conventional iron salts. Complementary evidence from animal models and human absorption studies further supports the concept that ferritin-iron is absorbed as an intact protein-iron complex rather than dissociating fully into ionic iron before uptake [4,6,7].

Beyond intestinal absorption, emerging data also identify specific ferritin receptors involved in non-transferrin iron delivery in peripheral tissues, including scavenger receptor class A member 5 (SCARA5), underscoring the physiological relevance of ferritin as a distinct iron trafficking entity [12]. While these findings provide strong support for ferritin as a bioavailable dietary iron source, comparatively little is known about the acute pharmacokinetics (PK) of ferritin-bound iron following oral ingestion in humans, including its temporal appearance in circulation, peak serum iron responses, and relationship to regulatory biomarkers such as hepcidin.

Pharmacokinetic characterization of iron sources is increasingly recognized as critical for optimizing dosing strategies, minimizing adverse effects, and improving efficacy. Studies of ferrous sulfate demonstrate rapid increases in serum iron accompanied by acute hepcidin induction, which suppresses iron absorption from subsequent doses [13]. In contrast, limited PK data suggest that ferritin-bound iron may exhibit a slower, more regulated release of iron into systemic circulation, potentially resulting in a differentiated serum iron and hepcidin response [14]. However, these observations have been questioned.

Purpose of the Study

The purpose of this study was to characterize the pharmacokinetic profile of orally administered plant-based ferritin-bound iron in healthy subjects and in iron deficient but otherwise healthy subjects.

Materials and Methods

Study design

This is a secondary analysis of a prospective, randomized, double-blind, parallel-group pharmacokinetic/pharmacodynamic study conducted in accordance with ICH E6 (R2) Good Clinical Practice and the Declaration of Helsinki. This study was reviewed and approved by Medstar IRB, protocol no: CP-24-873 on April 3, 2025. The present analysis focuses exclusively on participants randomized to receive SloIron® (plant-based ferritin iron) and evaluates within-group responses over time in two groups: (1) healthy adult female subjects and (2) adult female subjects with iron deficiency anemia (IDA).

Eligible participants were females aged 18 - 55 years. Healthy subjects were required to have normal hemoglobin concentrations at screening (≥ 12 g/dL), while the IDA cohort included participants with hemoglobin values < 12 g/dL. Key exclusion criteria included chronic disease, recent iron supplementation, pregnancy, or abnormal clinical laboratory findings unrelated to iron status.

Intervention

Both groups receiving SloIron® received an acute, in-unit oral bolus of 60 mg elemental iron administered as three 400 mg capsules (each providing 20 mg iron as ferritin-bound iron) under fasting conditions on Day 1 (PK day). Following completion of the in-unit PK assessments, participants were discharged and instructed to continue daily at-home dosing for approximately 28 days (short-term pharmacodynamics). Healthy subjects received 20 mg elemental iron once daily, whereas IDA subjects received 60 mg elemental iron daily, administered one hour before meals in accordance with the study protocol.

Pharmacokinetic assessments

Venous blood samples were collected pre-dose and at 0.5, 1, 2, 3, 6, 12, and 24 hours post-dose on the PK day. Serum iron and biomarker concentrations were used for noncompartmental pharmacokinetic analysis. The following PK parameters were derived using Phoenix® WinNonlin® (version 8.4 or higher): maximum observed concentration (C_{max}), time to maximum concentration (T_{max}), area under the concentration-time curve from 0 - 12 hours (AUC_{0-12}), and area under the concentration-time curve from 0 - 24 hours (AUC_{0-24}). Terminal elimination half-life (t½) was not included in the present analysis.

Iron metabolism biomarkers

Biomarkers of iron metabolism-including hemoglobin, serum iron, ferritin, hepcidin, transferrin, total iron-binding capacity (TIBC), and transferrin saturation-were measured at baseline (pre-dose), over the first 24 hours and at the end of the approximately 30-day supplementation period. All assays were performed by certified clinical laboratories using validated analytical methods as specified in the protocol.

Statistical analysis

Analyses were conducted on a within-group basis for the SloIron® cohorts only. Continuous variables are summarized as mean ± standard deviation unless otherwise noted. Within-group changes from baseline to end of study were assessed using paired t-tests or nonparametric equivalents where appropriate. PK parameters are presented descriptively. A two-sided p-value <0.05 was considered statistically significant. No between-group comparisons or multiplicity adjustments were performed for this focused analysis.

Safety

SloIron® was well tolerated in both healthy and IDA participants. No serious adverse events were reported, and no participants discontinued due to gastrointestinal intolerance or laboratory abnormalities. Clinical safety parameters remained within acceptable limits throughout the study.

Results

Study population

A total of 30 participants received SloIron®, 15 healthy subjects and 15 subjects with IDA. All participants completed the in-unit PK assessment, and with at-home dosing. No protocol deviations affecting PK or biomarker interpretation were identified.

Acute pharmacokinetics of SloIron®

Following administration of a single 60-mg oral dose of ferritin-bound iron (SloIron®), measurable 24-hour pharmacokinetic responses were observed in serum iron and across additional biomarkers of iron metabolism in both healthy participants and those with iron deficiency anemia (IDA) (Table 1).

In healthy participants, mean serum iron reached a C_{max} of 95.1 µg/dL with corresponding AUC_{0-12} and AUC_{0-24} values of 630.6 and 1098.2 µg·h/dL, respectively. Median T_{max} occurred at 3 hours (range 0-24). In the IDA cohort, higher systemic exposure was observed, with a mean C_{max} of 116.0 µg/dL, AUC_{0-12} of 806.2 µg·h/dL and AUC_{0-24} of 1350.3 µg·h/dL, and a later median T_{max} of 6 hours (range 2-24).

Transferrin saturation (TSAT) showed a parallel pattern. In healthy participants, mean C_{max} was 28.0%, with AUC_{0-12} and AUC_{0-24} values of 181.5 and 312.9%·h, respectively, and a median T_{max} of 6 hours. IDA participants demonstrated higher overall exposure, with a TSAT C_{max} of 32.5% and corresponding AUC_{0-12} and AUC_{0-24} values of 229.1 and 378.5%·h, respectively. Median T_{max} was again 6 hours.

Unsaturated iron-binding capacity (UIBC) declined transiently following dosing, consistent with increased iron availability. In healthy participants, mean UIBC C_{max} (lowest value) was 389.8 µg/dL with AUC_{0-12} and AUC_{0-24} values of 3977.2 and 8191.6 µg·h/dL, respectively; IDA participants demonstrated similar exposure magnitudes (C_{max} 412.2 µg/dL; AUC_{0-24} 7901.3 µg·h/dL), with median T_{max} at 1 hour in both groups.

Total iron-binding capacity (TIBC) showed modest variability over the 24-hour interval. In healthy subjects, mean C_{max} was 423.5 µg/dL and AUC_{0-24} was 9286.9 µg·h/dL; in IDA participants, mean C_{max} was 448.0 µg/dL with AUC_{0-24} of 9251.4 µg·h/dL. Median T_{max} occurred at 1 hour for both cohorts.

Serum ferritin concentrations demonstrated small-magnitude fluctuations over 24 hours. In healthy participants, mean C_{max} was 26.2 ng/mL with AUC_{0-24} of 425.6 ng·h/mL and a median T_{max} of 1 hour. IDA subjects exhibited slightly higher exposure (C_{max} 28.0 ng/mL; AUC_{0-24} 536.9 ng·h/mL), with a later median T_{max} of 24 hours.

Serum transferrin concentrations remained within expected physiological ranges. In healthy participants, mean C_{max} was 290.2 mg/dL with AUC_{0-24} of 4802.6 mg·h/dL and a median T_{max} of 2 hours. In IDA subjects, mean C_{max} was 317.2 mg/dL with higher cumulative exposure (AUC_{0-24} 6511.4 mg·h/dL) and a later median T_{max} of 6 hours.

Hepcidin exhibited modest and variable changes across the 24-hour window. In healthy participants, mean C_{max} was 11.45 ng/mL with AUC_{0-24} of 229.1 ng·h/mL and median T_{max} of 6 hours. In the IDA cohort, both peak concentration (8.61 ng/mL) and overall exposure (AUC_{0-24} 116.9 ng·h/mL) were lower, with a median T_{max} of 1 hour.

PK Parameter	C_{max}	AUC 0-12 hr	AUC 0 - 24 hr	T_{max}
Test Parameter - Iron				
Healthy SloIron	95.11	630.568	1098.201	3.00 (0.00-24.00)
IDA SloIron	116	806.182	1350.262	6.00 (2.00-24.00)
PK Parameter	**C_{max}**	**AUC 0-12 hr**	**AUC 0 - 24 hr**	**T_{max}**
Test Parameter - UIBC				
Healthy SloIron	389.8	3977.19	8191.561	1.00 (0.00 - 24.00)
IDA SloIron	412.2	3834.362	7901.322	1.00 (0.00 - 24.00)
PK Parameter	**C_{max}**	**AUC 0-12 hr**	**AUC 0 - 24 hr**	**T_{max}**
Test Parameter - Ferritin				
Healthy SloIron	26.18	213.654	425.614	1.00 (0.00 - 24.00)
IDA SloIron	28.03	261.518	536.918	24.00 (0.00 - 24.00)
PK Parameter	**C_{max}**	**AUC 0-12 hr**	**AUC 0 - 24 hr**	**T_{max}**
Test Parameter TIBC				
Healthy SloIron	423.5	4604.874	9286.879	1.00 (0.00-24.00)
IDA SloIron	448	4640.343	9251.383	1.00 (0.00-24.00)
PK Parameter	**C_{max}**	**AUC 0-12 hr**	**AUC 0 - 24 hr**	**T_{max}**
Test Parameter - Transferrin Saturation %				
Healthy SloIron	27.98	181.52	312.914	6.00 (0.00 - 24.00)
IDA SloIron	32.48	229.13	378.482	6.00 (2.00 - 24.00)
PK Parameter	**C_{max}**	**AUC 0-12 hr**	**AUC 0 - 24 hr**	**T_{max}**
Test Parameter - Transferrin				
Healthy SloIron	290.2	2703.871	4802.642	2.00 (0.00 - 24.00)
IDA SloIron	317.2	3214.357	6511.437	6.00 (0.00 - 24.00)
PK Parameter	**C_{max}**	**AUC 0-12 hr**	**AUC 0 - 24 hr**	**T_{max}**
Test Parameter - Hepcidin				
Healthy SloIron	11.45	113.874	229.063	6.00 (1.00 - 24.00)
IDA SloIron	8.607	58.281	116.881	1.00 (0.00 - 24.00)

Table 1: *Acute pharmacokinetics of SloIron®.*

Changes in iron metabolism biomarkers over 30 days

In healthy subjects receiving SloIron®, mean serum iron increased from 48.7 ± 26.0 µg/dL at baseline to 55.1 ± 28.5 µg/dL at end of study, representing a modest, non-statistically significant within-group change. Ferritin concentrations and hemoglobin values remained within the normal range, with no evidence of iron overload or dysregulated iron homeostasis. See table 2.

In contrast, subjects with IDA demonstrated larger directional improvements across multiple biomarkers of iron metabolism. Mean serum iron increased from 42.7 ± 19.5 µg/dL at baseline to 75.6 ± 30.4 µg/dL at end of study. Ferritin concentrations increased over the 30-day period, and hemoglobin values showed early upward trends consistent with initial iron repletion. While the study was not designed to detect full correction of iron deficiency within 30 days, these changes provide an early signal of biological response.

Across both cohorts, hepcidin concentrations did not demonstrate disproportionate acute or chronic elevations, suggesting that ferritin-bound iron delivery did not elicit excessive regulatory suppression of iron absorption over the dosing period.

Group	Baseline	End of Study	Change from Baseline	P-Value
Test Parameter - Iron				
Healthy SloIron	48.7400	55.1308	9.9538	0.3759
IDA SloIron	42.6600	75.6357	33.9786	0.0016
Test Parameter - UIBC				
Healthy SloIron	351.2000	350.6000	-7.9538	0.7413
IDA SloIron	352.3000	339.8000	-17.8643	0.2731
Test Parameter - Ferritin				
Healthy SloIron	20.9733	37.0308	15.2385	0.0179
IDA SloIron	21.9333	45.3429	25.4357	0.0036
Test Parameter - TIBC				
Healthy SloIron	400.0000	405.7000	2.0000	0.9245
IDA SloIron	394.9000	415.5000	16.1143	0.2463
Test Parameter - Transferrin Saturation %				
Healthy SloIron	13.1860	14.0846	1.8200	0.5146
IDA SloIron	11.5620	18.7429	7.5693	0.0018
Test Parameter - Transferrin				
Healthy SloIron	220.2000	289.9000	78.6462	0.0076
IDA SloIron	263.7000	309.9000	52.1500	0.0230
Test Parameter - Hepcidin				
Healthy SloIron	8.4820	4.1323	-4.3777	0.1391
IDA SloIron	5.5793	5.6629	-0.0693	0.9775

Table 2: *Changes in iron metabolism biomarkers.*

Discussion

This study provides a focused characterization of the acute (0 - 24h) and short-term (30-day) biological responses to ferritin-bound iron administered as SloIron® in healthy adult women and women with iron deficiency anemia (IDA). This data allows for an understanding of a unique iron supplement and how it impacts iron health and metabolism acutely and over the short-term in healthy and iron deficient female adults. In examining the outcomes data, it is apparent that this form of iron is well tolerated and appears to offer the same characteristics towards iron health as other iron therapies typically used.

Following a single 60 mg oral dose of ferritin-bound iron, both healthy and IDA cohorts demonstrated measurable, time-dependent increases in serum iron over the first 24 hours, characterized by moderate C_{max} values and sustained exposure reflected by AUC_{0-12} and AUC_{0-24}. The observed T_{max} values-occurring several hours post-dose and extending up to 24 hours in some participants-suggest a gradual appearance of iron in circulation rather than a rapid peak-and-decline pattern.

This PK profile is consistent with prior mechanistic and human absorption studies demonstrating that ferritin-iron is absorbed via receptor-mediated endocytosis of the intact protein nanocage, followed by intracellular processing and controlled release of iron into the labile iron pool [6,10-12]. Unlike ionic iron salts, which rely on highly acidic gastric acid pH, luminal solubilization and divalent metal transporter 1 (DMT1)-mediated uptake, ferritin-bound iron bypasses key rate-limiting steps associated with nonheme iron absorption, including the disassembly of the biomineral $Fe_2O_3H_2O$ [15,16]. The extended T_{max} and sustained AUC observed in the present study support the concept that ferritin-iron acts as a slow-release iron source at the systemic level.

Importantly, the higher systemic exposure observed in the IDA cohort relative to healthy subjects aligns with established physiological regulation of iron absorption, wherein iron-deficient states enhance uptake efficiency [5]. This finding indicates that ferritin-bound iron remains responsive to homeostatic cues and is not absorbed indiscriminately, a key consideration for both safety and efficacy.

High-dose ferrous salts are known to produce rapid increases in serum iron, often accompanied by supraphysiologic peaks that acutely stimulate hepcidin production. This hepcidin response can suppress iron absorption from subsequent doses for 24 hours or longer, thereby reducing fractional absorption and complicating dosing strategies [13]. In contrast, the PK behavior observed with SloIron®-characterized by moderated C_{max} values and prolonged exposure-suggests a fundamentally different absorption profile.

Notably, ferritin-bound iron delivery did not appear to provoke disproportionate acute increases in hepcidin over the PK interval, consistent with prior evidence that ferritin-iron is absorbed through pathways distinct from those engaged by soluble ferrous salts [4,6]. This differentiated PK behavior may help explain why ferritin-iron has demonstrated comparable bioavailability to ferrous sulfate in human studies while potentially avoiding the sharp regulatory feedback associated with high-dose ionic iron [3].

Over the approximately 30-day supplementation period, changes in biomarkers of iron metabolism differed by baseline iron status, as expected. In healthy subjects, iron-related biomarkers remained within physiological ranges, with only modest fluctuations, indicating maintenance of iron homeostasis and no evidence of iron excess. These findings are consistent with prior studies showing that ferritin-iron absorption is appropriately downregulated in iron-replete individuals [3,5].

In contrast, participants with IDA demonstrated larger directional improvements in serum iron, ferritin, and hemoglobin over the same period. Although the study duration was intentionally limited and not designed to achieve full iron repletion-which typically requires three months or longer-these early changes provide a biologically meaningful signal that ferritin-bound iron contributes to restoration of iron stores and erythropoietic support [7]. The absence of exaggerated hepcidin responses over the 30-day period further supports the notion that ferritin iron delivery integrates with endogenous regulatory systems rather than overwhelming them.

The present findings are highly concordant with a substantial body of literature demonstrating that dietary and supplemental ferritin-iron is efficiently absorbed in humans and is comparable to ferrous sulfate in terms of bioavailability. Human isotope studies have shown that ferritin-iron absorption is similar to that of ferrous salts across a range of iron statuses, including in women with low iron stores and in clinical populations predisposed to increased iron absorption [3,14]. Cellular and animal studies further support receptor-mediated uptake, intracellular ferritin processing, and regulated iron release [10,12].

By extending this literature with PK endpoints and short-term biomarker responses, the current study adds to the temporal understanding of ferritin-iron handling *in vivo*. Specifically, it demonstrates that ferritin-bound iron produces a sustained systemic iron signal over 24 hours and an early biomarker response over one month, without evidence of dysregulated absorption.

Limitation of the Study

This study has several limitations. The 30-day duration, while appropriate for detecting early biological signals, is insufficient to evaluate full iron repletion or long-term clinical outcomes. Additionally, the analysis was limited to within-group changes and did not

include formal statistical comparisons with other iron forms. Future studies of longer duration, incorporating clinical endpoints and repeated PK assessments, would further elucidate the implications of ferritin-bound iron supplementation.

Conclusion

In summary, plant-based ferritin-iron administered as SloIron® demonstrates a distinct pharmacokinetic and biological profile characterized by moderated peak exposure, sustained systemic iron availability over 24 hours, and early improvements in iron metabolism biomarkers over one month in iron-deficient individuals. These findings align with established ferritin absorption mechanisms and differentiate ferritin-iron from high-dose ferrous salts that are associated with rapid serum iron spikes and endogenous regulatory suppression. Collectively, the results support plant-based ferritin-iron as a physiologically compatible iron source with potential advantages in dosing efficiency and regulatory integration.

Conflicts of Interest

Not applicable.

Author Contribution Statement

S.H., A.M.A. and D.K. contributed to the study design, study oversight, data interpretation and the manuscript. The study was executed by a contract research organization, Notrox Research Bangalore, India.

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