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Haemoglobin becomes ferritin-dependent only at low iron stores in infancy: an age-adjusted breakpoint analysis

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Abstract

Background. Ferritin thresholds defining iron deficiency in infancy remain debated, and guidelines have moved the diagnostic threshold upward. We examined the ferritin concentration below which haemoglobin (Hb) becomes measurably related to iron status.

Methods. We retrospectively analysed children aged 0–24 months from a Bulgarian telemedicine cohort. Each child contributed the first interpretable ferritin measurement before iron therapy and its paired Hb. The Hb–ferritin relationship was modelled by age-adjusted segmented regression with bootstrap confidence intervals; models were compared by Akaike information criterion. Sensitivity analyses restricted the cohort to age ≥ 6 months.

Results. Of 939 consultations, 489 children met criteria and 450 had paired diagnostic Hb and ferritin. A single breakpoint was identified at 11.0 µg/L (95% CI 8–23); below it Hb increased with ferritin, whereas above it the relationship was minimal. A second breakpoint did not improve fit, and the estimate remained 11.0 µg/L at age ≥ 6 months. Among children with ferritin < 30 µg/L and follow-up Hb (n = 132), Hb increased by 1.53 g/dL when baseline Hb was < 11.0 g/dL but by 0.23 g/dL when baseline Hb was ≥ 11.0 g/dL; the latter did not differ from a ferritin ≥ 30 µg/L reference group.

Conclusion. The Hb–ferritin breakpoint in infancy was approximately 11 µg/L. This is not a diagnostic threshold for iron deficiency; it identifies the range below which declining ferritin becomes measurably associated with Hb. Iron depletion may therefore precede the point at which Hb becomes ferritin-dependent.

What is Known

- • Ferritin identifies iron depletion before anaemia develops, but the ferritin concentration at which haemoglobin becomes measurably iron-dependent in infancy is uncertain.
- • Recent ASH and AAP guidance recommends a ferritin threshold of ≤ 20 µg/L for diagnosing iron deficiency in young children, higher than the historical WHO threshold of < 12 µg/L.

What is New:

- • In 450 children, the age-adjusted haemoglobin–ferritin relationship showed a single breakpoint at approximately 11 µg/L, robust to restriction to age ≥ 6 months.
- • The findings distinguish the diagnostic threshold for iron deficiency from the lower ferritin range at which haemoglobin itself becomes ferritin-dependent.

1. Introduction

Iron deficiency is the most common micronutrient disorder of early childhood and the leading nutritional cause of anaemia. Its importance in infancy derives not only from anaemia, but also from the

vulnerability of the developing brain: iron is required for myelination, monoamine synthesis and hippocampal energy metabolism, and deficiency in the first two years of life has been associated with adverse neurodevelopmental outcomes.[1–5] The clinically relevant problem therefore extends into the pre-anaemic, iron-depleted state.

Identifying that state depends largely on serum ferritin, yet the ferritin concentration that should define deficiency in infancy remains debated. The 2020 WHO guideline uses < 12 µg/L for apparently healthy children aged 0–23 months.[6] More recent guidance has moved this threshold upward: the 2026 American Society of Hematology guideline suggests ferritin ≤ 20 µg/L for children aged 9 months to 4 years, and the 2026 American Academy of Pediatrics clinical report likewise considers ferritin ≤ 20 µg/L consistent with iron deficiency in young children.[24,25] These recommendations address the diagnosis of iron deficiency, but not necessarily the same biological question as the ferritin concentration below which Hb synthesis becomes measurably iron-dependent. The latter can be examined directly from paired ferritin and Hb measurements.

A further source of uncertainty is the interpretation of the Hb response to oral iron. A rise after supplementation is often taken as evidence that a child was functionally iron-deficient, including when baseline Hb was within the usual clinical range. Such reasoning is vulnerable to normal age-related changes in Hb during infancy and to regression to the mean, whereby children selected for a relatively low initial value tend to have a higher repeat value irrespective of treatment.[11]

Telemedicine data offer an opportunity to examine these questions at scale, but require restraint in interpretation. Clinical information is elicited because of the presenting problem; dietary and perinatal details are often documented more completely once deficiency is suspected, so apparent associations based on the frequency of recorded exposures may reflect informed presence rather than biology.[12,13] We therefore treated cohort characteristics descriptively and confined inference to two quantitative questions: the Hb–ferritin breakpoint and the Hb change after oral iron. The aim was to locate the ferritin concentration below which Hb becomes measurably related to ferritin in infancy and to determine whether post-treatment Hb change provides information beyond baseline Hb.

2. Methods

2.1 Data source and setting

Data were drawn from a structured export of a Bulgarian paediatric telemedicine platform covering consultations from late 2020 to mid-2026. Consultations concerned infants and toddlers aged 0–24 months whose families sought advice about iron status, anaemia or a related complaint. Each record contained the free-text dialogue and laboratory results shared by the family. Tests had been performed in routine external laboratories selected by the families; assay manufacturer and analytical platform were not systematically available. Values were harmonised to common units, but cross-laboratory calibration was not possible. Clinical variables — feeding, perinatal history, presenting complaint and treatment —

were coded from the meaning of the whole consultation rather than from isolated keywords; a double-coded sample showed high agreement (κ ≥ 0.95 for breastfeeding, prior iron therapy and maternal anaemia).

2.2 Cohort definition

The selection funnel is shown in Fig. 3. Of 939 consultations, 269 were excluded because no valid child ferritin result was available (n = 254) or because the record did not concern a child aged 0–24 months (n = 15). A further 181 were excluded because ferritin had been measured during iron therapy (n = 100), the first ferritin measurement could not be identified unambiguously (n = 71), or twin attribution remained unresolved (n = 10). The main analysis cohort therefore comprised 489 children, each contributing a single interpretable first ferritin measurement obtained before iron treatment. Reporting follows the STROBE principles for observational studies.

2.3 One observation per child

Because many children had several laboratory reports over time, the diagnostic analyses used one observation per child to avoid giving repeatedly tested children disproportionate weight. For the Hb–ferritin model this was the first interpretable ferritin before iron therapy and its paired Hb, available for 450 children. Ferritin categories (< 15, 15–30 and ≥ 30 µg/L) were prespecified for descriptive purposes only and were not used to estimate the breakpoint. Hb is reported in g/dL throughout.

2.4 Statistical analysis

The Hb–ferritin relationship was modelled by segmented (piecewise-linear) regression with age in months as a covariate. The breakpoint was located by grid search as the minimum residual sum of squares, and its confidence interval was obtained by a 1000-replicate child-level bootstrap.[14] Linear, single-breakpoint and two-breakpoint models were compared by Akaike information criterion (AIC) and by an F-test for change in slope. Sensitivity analyses restricted the cohort to children aged ≥ 6 months and excluded ferritin values > 100 µg/L. For the longitudinal analysis, children with baseline ferritin < 30 µg/L and a later Hb measurement (n = 132) were stratified pragmatically by baseline Hb < 11.0 versus ≥ 11.0 g/dL and compared with children with baseline ferritin ≥ 30 µg/L and follow-up over a comparable interval. The 11.0 g/dL split was used as a clinical stratifier rather than as an age-specific definition of anaemia across the entire 0–24-month age range. A regression model related Hb change to baseline Hb, change in age and baseline ferritin, and the analysis was repeated after restriction to children aged ≥ 6 months at baseline. Oral iron was prescribed as part of routine care; formulation, dose and adherence were not sufficiently standardised or completely documented for comparative efficacy analysis. Weight-for-age z-scores were computed against the WHO Child Growth Standards. Analyses were performed in Python 3.13 (statsmodels, scipy).

2.5 Ethics

The study used retrospectively collected, de-identified consultation data and was conducted in accordance with the Declaration of Helsinki. The protocol was approved by the Ethics Committee of the

Specialised Hospital for Active Treatment of Haematological Diseases (SBALHZ), Sofia, Bulgaria (approval No. 19/21.08.2026), with waiver of individual consent for secondary analysis of anonymised records. National identifiers or dates of birth visible on uploaded reports were used only within the secure platform environment to establish age or record attribution; they were not exported or retained in the analytical dataset.

3. Results

3.1 Cohort characteristics

The 489 children had a median age of 9.3 months (IQR 7.0–13.0); sex was ascertainable for 283 (58%), of whom 165 (58%) were boys. Median ferritin was 17.9 µg/L (IQR 10.6–31.1). By the prespecified descriptive groups, 199 children (41%) had ferritin < 15 µg/L, 159 (33%) 15–30 µg/L and 131 (27%) ≥ 30 µg/L (Table 1). Among the 184 children with ferritin < 15 µg/L and a paired Hb, 97 (53%) had Hb ≥ 11.0 g/dL. Weight-for-age was close to the WHO median (mean z + 0.12, SD 1.00, n = 239; z < − 2 in 2 children and > + 2 in 6).

Table 1
Characteristics of the main analysis cohort (n = 489).

Characteristic	Value
Age at ferritin, months — median (IQR)	9.3 (7.0–13.0)
Sex male — n/ascertained (%)	165/283 (58%)
Serum ferritin, µg/L — median (IQR)	17.9 (10.6–31.1)
Ferritin < 15 µg/L — n (%)	199 (41%)
Ferritin 15–30 µg/L — n (%)	159 (33%)
Ferritin ≥ 30 µg/L — n (%)	131 (27%)
Hb ≥ 11.0 g/dL among ferritin < 15 µg/L — n/paired (%)□	97/184 (53%)
Weight-for-age z (WHO) — mean (SD)	+ 0.12 (1.00), n = 239
Breastfed at consultation — n (%)	319 (65%)
Infant formula — n (%)	100 (20%)
Regular meat intake (≥ 3–4×/week) — n (%)	61 (12%)
Disturbed sleep as presenting complaint — n (%)	336 (69%)
□ Haemoglobin ≥ 11.0 g/dL, among children with a paired haemoglobin.	

3.2 The haemoglobin–ferritin relationship

In the 450 children with paired diagnostic Hb and ferritin, the age-adjusted segmented model identified a single breakpoint at ferritin 11.0 µg/L (95% CI 8–23) (Fig. 1). Below the breakpoint, Hb increased with ferritin (slope + 0.14 g/dL per µg/L, p < 0.001). Above it, the slope was small (− 0.005 g/dL per µg/L) and became non-significant after exclusion of ferritin values > 100 µg/L (p = 0.11). Age was independently associated with Hb (+ 0.05 g/dL per month, p < 0.001). The single-breakpoint model fitted substantially better than a straight line (ΔAIC − 41; F = 45, p < 0.001), whereas adding a second breakpoint did not improve fit (ΔAIC + 0.9; middle-segment slope p = 0.54). The breakpoint remained 11.0 µg/L when the cohort was restricted to children aged ≥ 6 months and when ferritin values > 100 µg/L were excluded (Table 2). Median Hb was 11.0 g/dL (IQR 10.3–11.8) in the ferritin < 15 µg/L group, 11.5 g/dL (10.9–12.0) at 15–30 µg/L and 11.4 g/dL (10.7–11.9) at ≥ 30 µg/L.

Table 2
Haemoglobin–ferritin segmented model: breakpoint and slopes (one observation per child).

Analysis set	n	Breakpoint, µg/L (95% CI)	Slope below (g/dL per µg/L)	Slope above (g/dL per µg/L)
Full set (age-adjusted)	450	11.0 (8–23)	+ 0.14 (p < 0.001)	−0.005 (p < 0.001)
Age ≥ 6 months	434	11.0	+ 0.14 (p < 0.001)	−0.005 (p < 0.001)
Age ≥ 6 months, ferritin ≤ 100 µg/L	423	11.0	+ 0.14 (p < 0.001)	−0.005 (p = 0.11)
Single-breakpoint vs linear model: ΔAIC − 41, F = 45, p < 0.001. Two-breakpoint vs single-breakpoint: ΔAIC + 0.9 (no improvement).				

3.3 Haemoglobin response to oral iron

Among children with baseline ferritin < 30 µg/L, 132 had a follow-up Hb after oral iron (median interval 4.3 months). In those starting with Hb < 11.0 g/dL (n = 56), Hb rose from a mean of 10.1 to 11.7 g/dL (mean change + 1.53 g/dL). In those starting with Hb ≥ 11.0 g/dL (n = 76), it changed from 11.9 to 12.2 g/dL (mean change + 0.23 g/dL, 95% CI − 0.07 to + 0.52) (Fig. 2). The ferritin ≥ 30 µg/L reference group with follow-up (n = 26) changed by + 0.18 g/dL over a comparable interval; the difference from the baseline-Hb ≥ 11.0 g/dL group was 0.05 g/dL (p = 0.82). In the regression model, Hb change was associated predominantly with baseline Hb (coefficient − 0.76 g/dL per g/dL, p < 0.001; R^2=0.48), with no independent contribution from change in age (p = 0.21) or baseline ferritin (p = 0.70). Restriction to children aged ≥ 6 months at baseline (n = 127) produced essentially the same result: Hb increased by + 1.53 g/dL when baseline Hb was < 11.0 g/dL and by + 0.20 g/dL (95% CI − 0.11 to + 0.50) when baseline Hb was ≥ 11.0 g/dL; the latter again did not differ from the ferritin ≥ 30 µg/L reference group of the same age range (difference 0.03 g/dL, p = 0.90).

3.4 Cohort groups (descriptive)

At the diagnostic consultation 319 children (65%) were being breastfed and 100 (20%) received infant formula; regular meat intake (≥ 3–4 times/week) was documented in 61 (12%). Maternal iron-deficiency anaemia was recorded in 80 of the 116 children for whom maternal status was documented, prematurity

in 18 (4%), and disturbed sleep was the presenting complaint in 336 (69%). These frequencies describe the recorded cohort only and, in the absence of a structured comparison group, were not analysed as risk factors.

4. Discussion

In this retrospective telemedicine cohort of children aged 0–24 months, we examined the relationship between serum ferritin and Hb using one diagnostic observation per child, with adjustment for age and formal comparison of alternative segmented models. The analysis identified a single Hb–ferritin breakpoint at approximately 11 µg/L. The estimate was unchanged when the analysis was restricted to children aged ≥ 6 months and when ferritin values > 100 µg/L were excluded, suggesting that the finding was not driven by early-infancy physiology or by a small number of high ferritin values.

The estimated breakpoint lies close to the historical WHO ferritin cut-off of < 12 µg/L for apparently healthy children aged 0–23 months, but its interpretation changes in light of the 2026 ASH and AAP recommendations. Both now use a ferritin threshold of ≤ 20 µg/L to identify iron deficiency in young children.[24,25] Our 11 µg/L estimate should therefore not be interpreted as a competing diagnostic threshold for iron deficiency. It identifies a different point: the ferritin range below which declining iron stores become measurably associated with Hb. The distinction is clinically relevant because iron depletion may already be present at ferritin concentrations between approximately 12 and 20 µg/L while Hb remains largely insensitive to that depletion.

Above the breakpoint, the relationship between ferritin and Hb was minimal. Hb values were similar across the higher ferritin ranges, indicating that further differences in iron stores were poorly reflected by Hb. This is compatible with the newer diagnostic framework rather than contradictory to it: a higher ferritin threshold may identify depleted stores before erythropoiesis is sufficiently restricted to affect Hb. More than half of the children with ferritin < 15 µg/L and paired Hb still had Hb ≥ 11.0 g/dL, illustrating directly that low iron stores can precede a fall in Hb. The present study does not define a storage threshold, nor does it establish 20 or 30 µg/L as physiological breakpoints; it shows that Hb cannot be used to distinguish reliably between these higher ferritin states.

The longitudinal analysis supports the same interpretation. Children starting with Hb < 11.0 g/dL showed the expected substantial rise after oral iron, whereas those starting at Hb ≥ 11.0 g/dL had only a small mean increase (+ 0.23 g/dL), with a confidence interval that included zero and a change almost identical to that observed in the ferritin ≥ 30 µg/L reference group. Baseline Hb explained most of the observed variation in Hb change, while baseline ferritin and change in age did not contribute independently. Importantly, restricting the analysis to children aged ≥ 6 months produced virtually identical results. This makes normal post-nadir Hb maturation an unlikely explanation for the pattern and indicates that a small post-treatment Hb rise, by itself, should not be regarded as functional proof of latent iron deficiency in a child whose baseline Hb is already ≥ 11.0 g/dL.

The clinical implication is therefore not that a single Hb-derived ferritin breakpoint should replace current diagnostic guidance. Rather, Hb and ferritin describe different stages of iron depletion. Ferritin can identify low stores at concentrations for which Hb remains largely uninformative, while Hb becomes measurably ferritin-dependent only at a lower range. This provides a physiological explanation for why the newer ASH and AAP diagnostic thresholds are higher than the breakpoint observed in our data, and why a haemoglobin-first strategy will miss a proportion of children with iron deficiency.

The descriptive features of the cohort — predominant breastfeeding, infrequent documented regular meat intake and frequent disturbed sleep as a presenting complaint — are clinically recognisable, but should not be interpreted as risk estimates from these data. In telemedicine and electronic-record datasets the completeness of a recorded exposure depends on why the consultation occurred: dietary and perinatal histories are elicited more thoroughly once deficiency is suspected.[12,13] Quantifying these associations requires prospective data collection with a structured questionnaire and a defined comparison group.

Several limitations remain. Laboratory results came from multiple routine laboratories, and assay platform was not systematically available; values were harmonised by unit but could not be analytically recalibrated across laboratories. C-reactive protein was not consistently available, so ferritin could not be adjusted for inflammation. Mean cell volume and red-cell distribution width were also incomplete, and thalassaemia trait or other causes of microcytosis could not be excluded individually. The cohort was self-selected by families seeking paediatric-haematology advice and therefore cannot provide population prevalence estimates. The Hb 11.0 g/dL split in the treatment analysis was a pragmatic clinical stratifier, not an age-specific definition of anaemia throughout infancy. Treatment formulation, dose and adherence were not standardised, and follow-up was available only in a subset. Finally, the breakpoint confidence interval was broad (8–23 µg/L); the point estimate should therefore be interpreted as the centre of an uncertain range rather than as a new precise diagnostic cut-off.

The strengths of the study are the exact cohort definition, the use of a single diagnostic observation per child, explicit participant flow, age adjustment, child-level bootstrap estimation, formal comparison of alternative breakpoint models, sensitivity analyses addressing both age and high ferritin values, and an internal higher-ferritin reference group for the longitudinal Hb analysis.

In conclusion, the age-adjusted Hb–ferritin relationship in this telemedicine cohort had a single breakpoint at approximately 11 µg/L. This value should not be interpreted as the diagnostic threshold for iron deficiency. In the context of recent ASH and AAP recommendations using ferritin ≤ 20 µg/L in young children, our findings suggest that depletion of iron stores precedes the point at which Hb becomes measurably ferritin-dependent. Above the breakpoint, Hb carried little information about ferritin status, and a small Hb rise after oral iron in children starting with Hb ≥ 11.0 g/dL was no greater than in the higher-ferritin reference group. Ferritin therefore remains the more informative marker for identifying pre-anaemic iron deficiency in infancy.

Declarations

Ethics approval.

The protocol was approved by the Ethics Committee of the Specialised Hospital for Active Treatment of Haematological Diseases (SBALHZ), Sofia, Bulgaria (No. 19/21.08.2026), with waiver of individual consent for secondary analysis of anonymised retrospective records. The study was conducted in accordance with the Declaration of Helsinki.

Consent to participate.

Individual consent was waived by the institutional ethics committee for the secondary analysis of anonymised retrospective records.

Consent for publication.

Not applicable; no identifiable individual data are reported.

Competing interests.

A.B. provides paediatric-haematology consultations on the telemedicine platform from which the anonymised data originate. He received no payment related to this study and had no role in, or financial benefit from, families' decisions to consult. G.T. declares no competing interests.

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Author Contribution

A.B. conceived the study, curated and coded the data, performed the analysis, interpreted the results and drafted the manuscript. G.T. contributed to clinical interpretation and critical revision. Both authors reviewed and approved the final manuscript.

Data Availability

De-identified aggregate data supporting the findings are available from the corresponding author on reasonable request, subject to platform data-governance approval.

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Figures

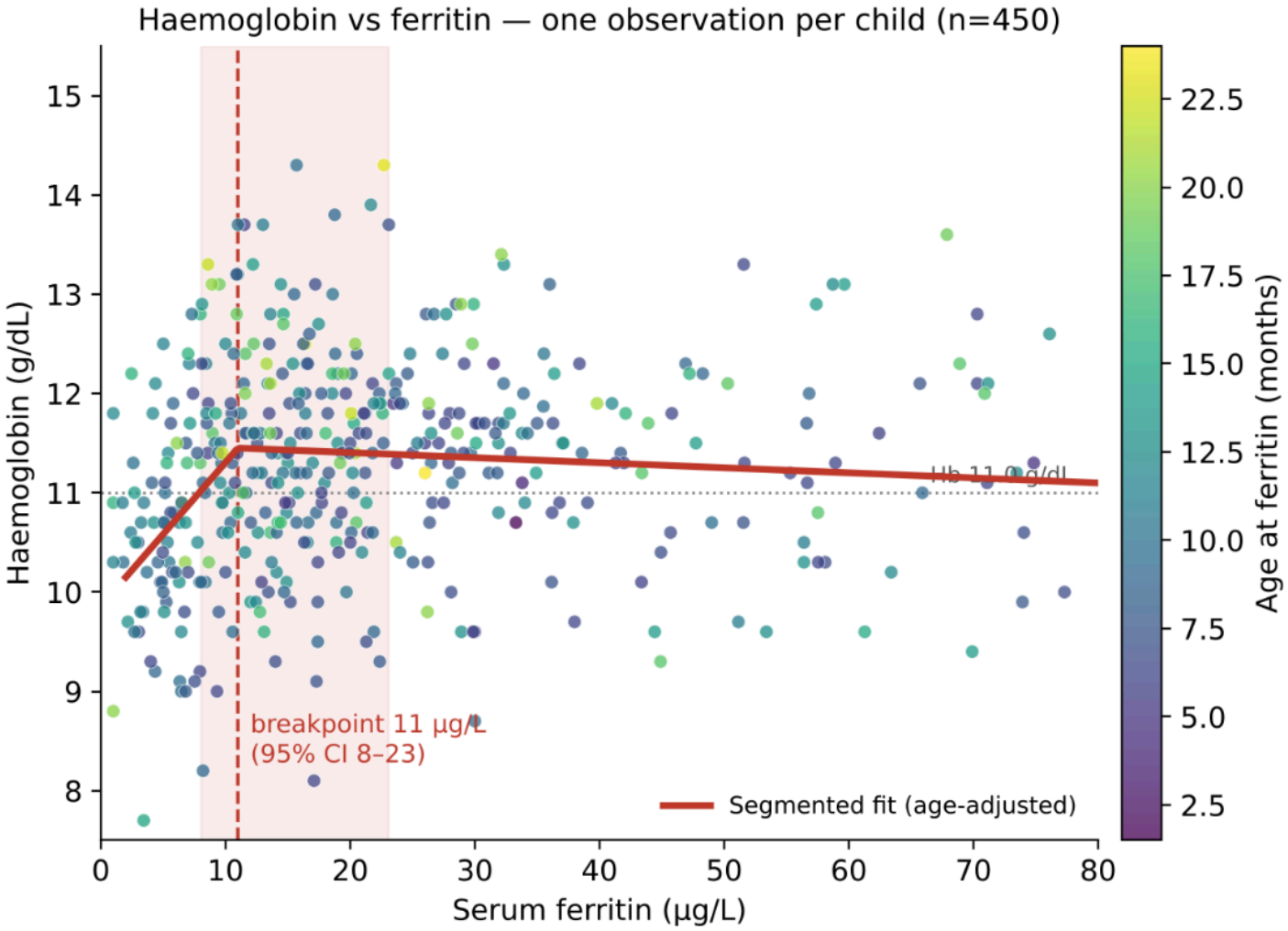

Figure 1

Haemoglobin versus serum ferritin, one diagnostic observation per child (n=450). Points are coloured by age; the red line shows the age-adjusted segmented fit, with the breakpoint at 11.0 µg/L (95% CI 8–23, shaded). Below the breakpoint Hb increases with ferritin; above it the relationship is minimal.

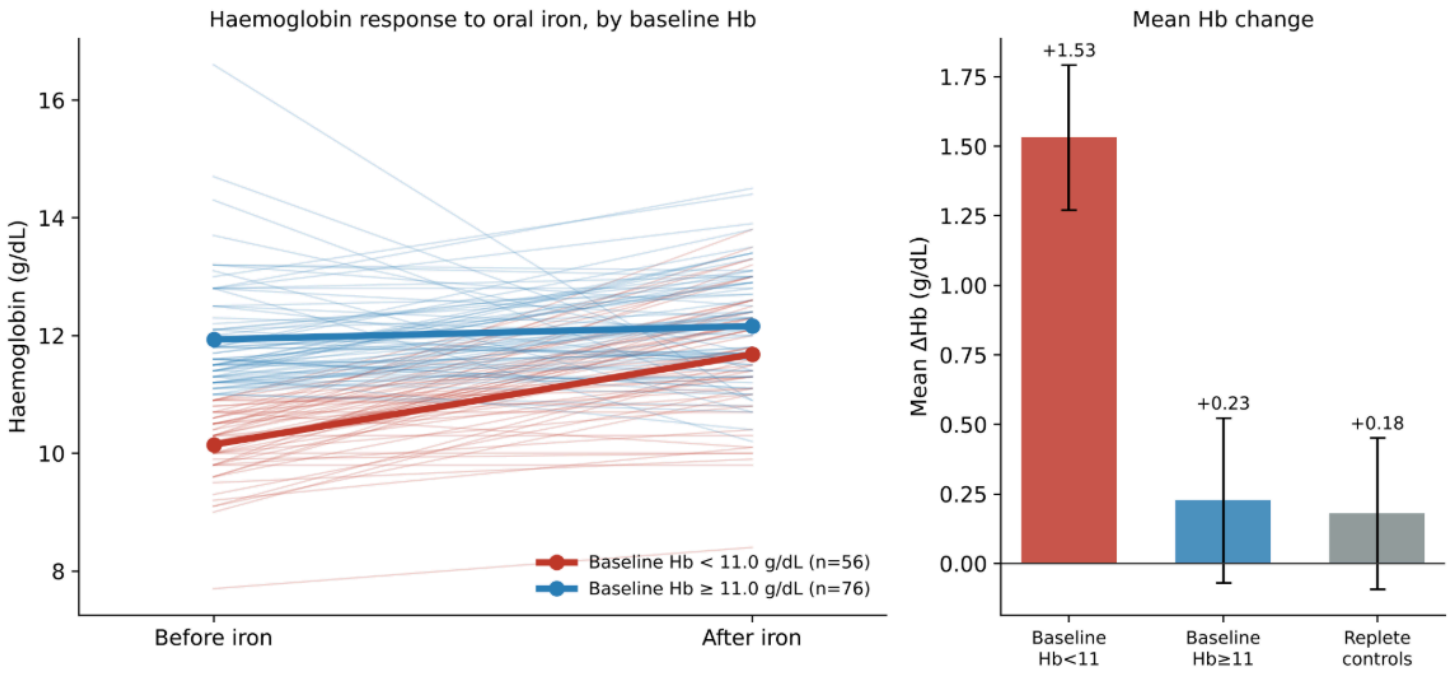

Figure 2

Haemoglobin change after oral iron among children with baseline ferritin <30 µg/L and follow-up Hb (n=132), stratified by baseline Hb. Left: individual and mean trajectories. Right: mean Hb change (95% CI) for baseline Hb <11.0 g/dL, baseline Hb ≥11.0 g/dL, and a ferritin ≥30 µg/L reference group. The small change in the baseline-Hb ≥11.0 g/dL group was similar to the reference group.

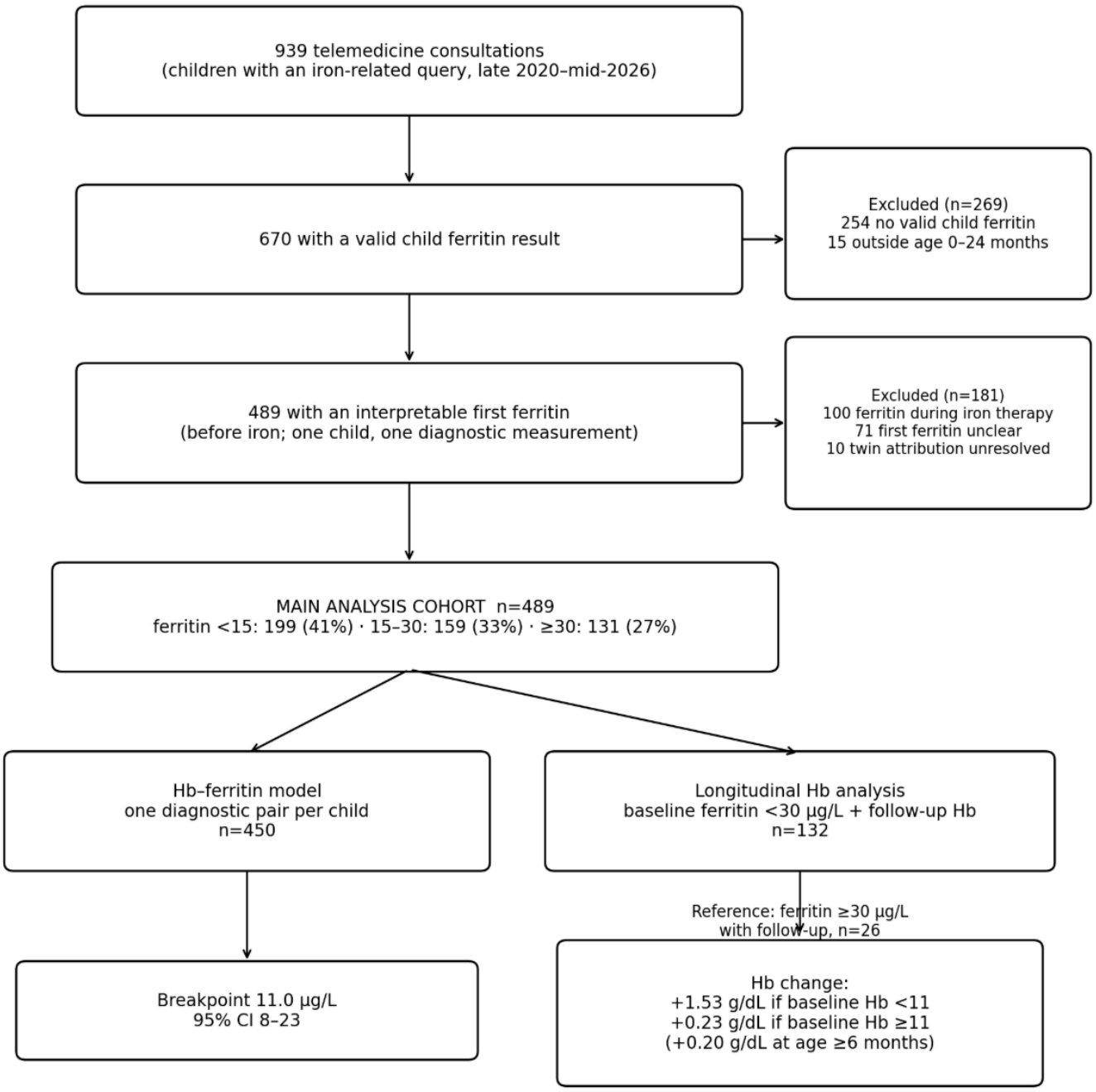

Figure 3

STROBE-style participant flow from 939 telemedicine consultations to the 489-child main analysis cohort and the principal analytic subsets.