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# Iron Given, Iron Denied: Neonatal Hematologic Alterations in Obese Pregnancies Despite Universal Supplementation

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***ABSTRACT***

***Background**: Maternal obesity, a pro-inflammatory state now highly prevalent among women of reproductive age in Indonesia, may undermine the effectiveness of antenatal iron supplementation. Neonatal iron deficiency remains a public health concern, with maternal inflammation potentially disrupting fetal iron transfer. **Objective**: To investigate the association between maternal third-trimester Body Mass Index (BMI) and neonatal hematologic and iron parameters, within the context of standardized antenatal iron supplementation. **Methods**: This hospital-based cross-sectional study in Central Java, Indonesia, enrolled 84 full-term neonates whose mothers received iron supplementation. Subjects were stratified by maternal BMI into non-obese (<25 kg/m²) and obese (≥25 kg/m²) groups. Cord blood was analyzed for hematologic indices (hemoglobin, Mean Corpuscular Volume [MCV], Mentzer Index) and iron biomarkers (ferritin and hepcidin). **Results:** Neonates from obese pregnancies exhibited significantly higher MCV (103.4 ± 5.1 fL vs. 100.1 ± 4.9 fL, p=0.004) and Mentzer Index (21.8 ± 4.7 vs. 21.3 ± 2.9, p=0.040) compared to the non-obese group. These findings suggest macrocytic shifts and possible ineffective erythropoiesis. No significant differences were observed in hemoglobin, ferritin, or hepcidin levels between groups. **Conclusion**: Maternal obesity during late pregnancy is associated with altered neonatal hematologic profiles, despite standardized iron supplementation. These results underscore a potential inflammation-mediated disruption in placental iron transfer and erythropoiesis, reinforcing the need for tailored antenatal strategies that incorporate maternal metabolic screening and functional hematologic monitoring for at-risk infants.*

## 1. INTRODUCTION

Iron is an essential micronutrient critical to fetal hematopoiesis and neurodevelopment (Georgieff, 2008). During the perinatal period, fetal demand for iron dramatically increases to support hemoglobin synthesis, red blood cell proliferation, and neurological maturation. Consequently, universal iron supplementation during pregnancy is widely implemented to prevent maternal and neonatal anemia. However, growing evidence from cohorts of overweight and obese pregnant women suggests that supplementation alone may not guarantee optimal neonatal iron status, indicating that maternal factors like obesity-associated inflammation may interfere with placental iron transfer (Dao et al., 2012; McArdle et al., 2022). This indicates that maternal factors, particularly obesity-associated inflammation, may interfere with placental iron transfer.

One such factor is maternal obesity, increasingly recognized as a pro-inflammatory condition that disrupts iron homeostasis. Obesity-induced inflammation elevates hepcidin—a central iron-regulatory hormone—which suppresses ferroportin-mediated iron export. These disturbances may impair fetal iron uptake, even when maternal iron stores are sufficient. While experimental models have demonstrated reduced placental expression of iron transporters such as transferrin receptor 1 (TfR1), DMT1, and ferroportin (FPN1) in response to elevated hepcidin, evidence in human studies remains limited and inconsistent (Dao & Eckel, 2021; Garcia-Valdes et

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al., 2015; Zaugg et al., 2024). This highlights a critical research gap and a potential mechanistic pathway for functional iron deficiency at birth in the context of maternal obesity, which warrants further investigation.

This issue is particularly relevant in Indonesia, where women of reproductive age experience overweight or obesity in approximately 29–35% of cases, according to national health surveys (Kementerian Kesehatan RI, 2022; UNICEF, 2022). This is consistent with local data showing that 14.5% of newborns were anemic at Dr. Hasan Sadikin Hospital in Bandung (Asfarina et al., 2020), highlighting a significant public health issue. The coexistence of maternal overweight/obesity and anemia suggests a pressing public health concern: whether universal iron supplementation remains effective in metabolically compromised pregnancies. Current guidelines may overlook metabolic influences that potentially compromise iron absorption, placental iron transfer, and fetal outcomes in high-risk populations (Flynn et al., 2018; Stoffel et al., 2020; Hansen et al., 2023).

To address this gap, the present study investigates the association between maternal BMI in the third trimester and neonatal hematologic and iron parameters among full-term infants. We aim to evaluate whether antenatal iron supplementation meets fetal iron requirements in obese pregnancies and assess its implications for neonatal hematologic status. Findings from this study may inform more tailored strategies for iron supplementation in populations experiencing nutritional transition.

## 2. METHOD

This cross-sectional study was conducted in three hospitals (one public and two private) in Purbalingga Regency, Central Java, Indonesia, between September and November 2015. Given the cross-sectional design, the observed relationships should be interpreted as associations rather than causal infere. A total of 84 neonates were enrolled following informed consent. To minimize potential bias, strict inclusion and exclusion criteria were applied. Inclusion criteria comprised full-term singleton deliveries via spontaneous vaginal birth, Apgar score ≥ 7, and birth weight within normal range (2,500 to <4,000 g), and absence of major congenital anomalies. Exclusion criteria included infants with hematologic/oncologic conditions or maternal postpartum hemorrhage. Additionally, to control for detection bias, laboratory personnel who analyzed the samples were blinded to the maternal BMI status of the participants.

The sample size for this study was determined based on the comparison of means between two independent groups (pregnant women with normal and obese BMI). To ensure the study's power to detect a statistically significant difference, the calculation was performed with a significance level (α) of 5% (0.05) and a statistical power of 80%. Based on a study by Hedengran et al. (2015), the standard deviation (σ) for hepcidin levels in the third trimester of pregnancy was assumed to be 1.29 ng/mL. To detect a minimum mean difference of 0.8 ng/mL in hepcidin levels between the two groups, the calculation indicated a requirement of 41 participants per group, for a minimum total of 82 participants. With a total of 84 participants recruited for this study, the sample size is considered adequate to address the study's objectives.

Participants were grouped based on maternal BMI in the third trimester into non-obese (<25 kg/m$^2$) and obese (≥25 kg/m$^2$) groups, in accordance with the World Health Organization's classification for Asian populations (Kementerian Kesehatan Republik Indonesia, 2015; Tham et al., 2022). Maternal demographic and clinical data—including age, parity, education, family income, hemoglobin, ferritin, CRP status, smoking exposure, and iron supplementation—were collected through structured interviews and medical records.

Umbilical cord blood samples were obtained immediately postpartum from the clamped and cut umbilical cord by trained healthcare personnel. A blood sample of 5 mL was collected into EDTA tubes (for hematological parameter analysis) and plain tubes (for serum). Samples for serum analysis were immediately centrifuged to separate the serum, which was then stored at -80°C until the time of examination to maintain the stability of ferritin, sTfR, and hepcidin levels. Hematological parameters (Hb, Ht, erythrocyte count, MCV, RDW, and Mentzer Index) were analyzed using the standardized Sysmex XN-1000 analyzer. Hepcidin, sTfR, and ferritin levels

were measured using the ECLIA (Electrochemiluminescence Immunoassay) method with the Elecsys 2010 analyzer. Meanwhile, serum iron and TIBC levels were measured using spectrophotometry. All examinations were performed at accredited laboratory facilities using standardized clinical laboratory methods.

Statistical analysis was conducted using SPSS 2022 software. Normality was tested using Kolmogorov–Smirnov or Shapiro–Wilk tests. Independent t-tests were used for normally distributed continuous variables, and Mann–Whitney U tests for non-normal distributions. Categorical data were analyzed using Chi-square or Fisher's exact tests. A p-value < 0.05 was considered statistically significant. Ethical approval was granted by the Health and Medical Research Ethics Committee, Faculty of Medicine, Diponegoro University/Dr. Kariadi Hospital Semarang (Approval No. 48/EC/FK-RSDK/2015).

## 3. RESULT AND DISCUSSION

### Result

### Maternal Characteristics by BMI Category

Among the 84 participating mothers (Table 1), 32 were classified as non-obese (BMI < 25 kg/m$^2$) and 52 as obese (BMI ≥ 25 kg/m$^2$). No significant differences were observed in maternal age (mean ± SD: 26.47 ± 5.50 vs. 28.02 ± 5.26 years; p = 0.201), parity distribution (p = 0.825), educational attainment (p = 0.441), or family income (p = 0.374) across groups. Maternal hemoglobin and serum ferritin levels in the third trimester were comparable (Hb: 12.28 ± 1.35 vs. 12.67 ± 1.38 g/dL; p = 0.200; ferritin median: 21.12 vs. 25.10 ng/mL; p = 0.796). All participants received iron supplementation during pregnancy, and rates of passive smoking exposure and CRP positivity did not differ significantly.

### Neonatal Demographic and Clinical Outcomes

All neonates were full-term with Apgar scores ≥7, and birth weights ranged within the normal clinical range (Table 2). Sex distribution was balanced (male: 40.6% vs. 48.1%; p = 0.652), with no difference in gestational age, clinical status at birth, or umbilical cord clamping time (p > 0.05 for all variables). Average birth weight did not differ significantly between groups (3157.81 ± 334.84 vs. 3209.90 ± 290.95 g; p = 0.454).

Table 1. Maternal Characteristics by BMI Category (Third Trimester)

| Variable | BMI<25 kg/m$^2$ *(Non-Obese) (n = 32)* | BMI ≥ 25 kg/m$^2$ (Obese) (n = 52) | *p*-value |
|---|---|---|---|
| Maternal Age (years), mean ± SD | 26.47 ± 5.50 | 28.02 ± 5.26 | 0.201[a] |
| Parity, n (%) | | | |
| < 2 | 17 (53.1) | 15 (46.9) | |
| ≥ 2 | 29 (55.8) | 23 (44.2) | 0.825 [b] |
| Education Level, n (%) | | | 0.441 [b] |
| ≤ Senior High School | 26 (81.3) | 38 (73.1) | |
| University | 6 (18.8) | 14 (26.9) | |
| Family Income | | | 0.374 [b] |
| < UMR* | 12 (37.5) | 20 (48.1) | |
| ≥ UMR | 25 (62.5) | 27 (51.9) | |
| Maternal Hb (g/dL), mean ± SD | 12.28 ± 1.35 | 12.67 ± 1.38 | 0.200 [a] |
| Anemia status, n (%) | | | 1.000 [d] |
| Anemia (Hb < 11 g%) | 4 (12.5%) | 8 (15.4) | |
| Non-Anemia (Hb ≥ 11 g%) | 28 (87.5%) | 44 (84.6) | |
| Maternal Ferritin (ng/mL), median (min-max) | 21.12 (9.29 - 505.40 | 25.10 (5.69 - 130.20) | 0.796 [c] |
| Fe supplementation, n (%) | 32 (100) | 52 (100) | - |
| CRP positivity, n (%) | | | 0.175 [b] |

| | | | |
|---|---|---|---|
| Positive | 17 (53.1) | 25 (48.1) | |
| Negative | 15 (46.9) | 27 (51.9) | |
| Passive Smoking Exposure, n (%) | | | 0.821 [b] |
| Passive smokers Not passive smokers | 15 (46.9) | 22 (42.3) | |
| Not passive smokers | 17 (53.1) | 30 (57.7) | |

Notes: a, independent t-test; b: Pearson Chi-Square; c, Mann-Whitney test; d, Fisher's Exact Test; *UMR, *upah minimal regional* (regional minimum wage)

Table 2. Neonatal Characteristics and Clinical Outcomes

| **Characteristics** | **BMI<25 kg/m²** (Non-Obese) (n = 32) | **BMI ≥ 25 kg/m²** (Obese) (n = 52) | ***p*-value** |
|---|---|---|---|
| Sex Distribution (M/F), n (%) | | | 0.652 [b] |
| Male | 13 (40.6) | 25 (48.1) | |
| Female | 19 (59.4) | 27 (51.9) | |
| Apgar Score ≥7 (in the first minute), n (%) | 32 (100) | 52 (100) | - |
| Gestational Age (weeks), n (%) | 32 (100) | 52 (100) | - |
| Birth Weight (g), mean ± SD | 3157.81 ± 334.84 | 3209.90 ± 290.953 | 0.454 [a] |
| Cord Clamping Time, n (%) | | | 0.822 [b] |
| Early (20 seconds) | 17 (53.1) | 25 (48.1) | |
| Delayed (3 minutes) | 15 (46.9) | 27 (51.9) | |

Notes: ($\overline{x}$±SD), mean±standard deviation; a, independent t-test; b: Pearson Chi-Square

## Neonatal Hematologic and Iron Parameters across Maternal BMI Categories

Association analysis of maternal BMI and neonatal hematologic and iron parameters (Table 3) found that neonatal hemoglobin, hematocrit, and erythrocyte counts were statistically similar across BMI groups (Hb: 17.56 ± 1.63 vs. 17.36 ± 1.79 g/dL; Ht: 49.42 ± 4.63 vs. 49.49 ± 5.25%; p > 0.05). However, a significant difference was detected in Mean Corpuscular Volume (MCV), with higher values observed in neonates born to obese mothers (103.44 ± 5.07 vs. 100.13 ± 4.90 fL; p = 0.004), suggesting a tendency toward macrocytic erythrocytes.

Table 3. Association analysis of maternal BMI and neonatal hematologic and iron parameters

| **Parameter** | **BMI<25 kg/m²** (Non-Obese) (n = 32)* | **BMI ≥ 25 kg/m²** (Obese) (n = 52) * | ***p*-value** |
|---|---|---|---|
| Hemoglobin (g/dL), ($\overline{x}$±SD) | 17,56±1,63 | 17,36±1,79 | 0,615[a] |
| Hematocrit (%), ($\overline{x}$±SD) | 49,42±4,63 | 49,49±5,25 | 0,668[b] |
| (median (min-max)) | 49,15 (39,50-62,50) | 47,75 (40,90-59,90) | |
| Erythrocyte Count ($\times 10^6/\mu L$), ($\bar{x}$±SD) | 4,94±0,48 | 4,79±0,53 | 0,203[a] |
| MCV (fL), ($\overline{x}$±SD) | 100,13±4,90 | 103,44±5,07 | 0,004[a] |
| RDW (%), (median (min-max)) | 16,65 (15,20-19,90) | 17,35 (15,90-19,50) | 0,818[b] |
| RDW Index ($\overline{x}$±SD) | 359,73±52,05 | 381,56±93,40 | 0,053[b] |
| Mentzer Index, ($\overline{x}$±SD) | 21,32±2,87 | 21,82±4,69 | 0,040[a] |
| Serum Iron (µg/dL), ($\overline{x}$±SD) | 112,56±53,55 | 119,17±41,36 | 0,300[a] |
| TIBC, µg/dL (median (min-max)) | 210,00 (113,00-296,00) | 210,00 (136,00-443,00) | 0,174[b] |
| Transferrin Saturation, % ($\overline{x}$±SD) | 60,46 ± 33,95 | 49,37 ± 24,05 | 0,086[a] |
| Ferritin (ng/mL), ($\overline{x}$±SD) | 411,00 ± 210,35 | 413,52 ± 205,83 | 0,958[a] |
| sTfR (nmol/L), ($\overline{x}$±SD) | 35,81±8,60 | 39,93±10,81 | 0,511[a] |
| Hepcidin Umbilical Cord (median (min-max)) | 3,86 (1,58-6,90) | 3,88 (1,66-6,25) | 0,364[b] |

Notes: Statistical tests using 95% confidence intervals; ($\overline{x}$±SD), mean±standard deviation; a, Independent T-Test; b, Mann-Whitney Test; *, n for TIBC and transferrin saturation in the non-obese maternal group = 31 subjects and in the obese = 52 subjects; n for infant ferritin in the non-obese maternal group = 31 subjects and in the obese = 50 subjects.

Red Cell Distribution Width (RDW) median values were comparable between groups (16.65% vs. 17.35%; p = 0.818). The Mentzer Index, used to evaluate iron-deficiency anemia, was slightly elevated in neonates from the obese group (21.82 ± 4.69 vs. 21.32 ± 2.87; p = 0.040).

Serum iron concentrations, transferrin saturation, and ferritin levels showed no significant differences between groups (iron: 112.56 ± 53.55 vs. 119.17 ± 41.36 µg/dL; saturation: 60.46% vs. 49.37%; ferritin: 411.00 ± 210.35 vs. 413.52 ± 205.83 ng/mL; all p > 0.05). Although transferrin saturation tended to be lower in the obese group, this did not reach statistical significance (p = 0.086).

Serum soluble transferrin receptor (sTfR) and umbilical cord hepcidin concentrations were likewise comparable across BMI groups (sTfR: 35.81 ± 8.60 vs. 39.93 ± 10.81 nmol/L; hepcidin: 3.86 vs. 3.88 ng/mL; p = 0.364).

**Discussion**

This study provides key insights into the hematologic profile of full-term neonates born to obese mothers who received a standardized antenatal iron supplementation. Our findings suggest a potential disruption in fetal erythropoiesis and iron utilization in this cohort, evidenced by significantly higher Mean Corpuscular Volume (MCV) and Mentzer Index values. These alterations were not reflected in conventional biochemical markers, as serum ferritin and hepcidin levels were comparable across groups. This paradox highlights the complexity of iron homeostasis in maternal-fetal dyads and indicates that universal iron supplementation may not adequately address functional iron deficits in metabolically compromised pregnancies.

Maternal obesity is a chronic inflammatory condition, with elevated pro-inflammatory cytokines (such as IL-6 and TNF-α) stimulating hepatic hepcidin expression (Nemeth & Ganz, 2021; Sangkhae & Nemeth, 2019). Hepcidin reduces iron availability for transplacental transfer by downregulating ferroportin, leading to functional iron deficiency in the fetus despite normal maternal stores (Zaugg et al., 2024). Beyond this hepcidin-mediated transport, maternal inflammation may also impair fetal iron availability through complex pathways affecting erythropoiesis (Dao et al., 2013). The elevated erythropoietin (EPO) found in cord blood of infants born to obese mothers suggests a compensatory response to reduced fetal iron (Brunner et al., 2018). Our observed changes in neonatal hematologic parameters may therefore reflect an iron-restricted erythropoiesis state rather than a classic iron deficiency, which is crucial for clinical interpretation (Dao et al., 2013).

The lack of significant differences in ferritin and hepcidin between groups warrants discussion. Ferritin can be elevated as an acute-phase reactant, potentially masking subclinical iron deficiency in inflammatory states (Daru et al., 2017). Similarly, hepcidin levels in cord blood may reflect transient regulatory states influenced by labor physiology or sampling timing, rather than cumulative intrauterine exposure (Koenig et al., 2014). This is further complicated by the fact that fetal hepcidin appears to regulate fetal iron status independently of maternal hepcidin, with little maternal-fetal concordance (Rehu et al., 2010). These factors, along with analytical challenges in hepcidin quantification, may explain the unexpected stability of these biomarkers in our study.

Our findings present a complex picture when compared to previous studies. While some research indicates that maternal obesity does not impact placental iron trafficking (O'Brien et al., 2021), others have shown that obesogenic diets can disrupt placental iron handling and fetal growth trajectories (Zaugg et al., 2024). Our results, which show functional hematological changes without significant changes in conventional biochemical markers, echo the paradox observed in other literature and reflect the complexity of obesity-related iron metabolism. From a clinical perspective, these results challenge the adequacy of "one-size-fits-all" iron supplementation strategies. In populations with rising maternal obesity, iron dosing may need to be tailored to the maternal metabolic profile. Screening for inflammatory markers (e.g., CRP or IL-6) during antenatal care could identify high-risk women, allowing for targeted intervention. Furthermore, monitoring functional hematologic indices like MCV and Mentzer Index in neonates may offer greater sensitivity than relying solely on ferritin or hepcidin.

These findings have long-term implications, as neonatal functional iron dysregulation can lead to neurodevelopmental and immunological vulnerabilities (Georgieff, 2020). Iron is indispensable for neurocognitive maturation, and suboptimal iron utilization at birth can predispose infants to iron-deficiency anemia and delayed developmental milestones (Berglund et al., 2017; Lozoff et al., 2018). Our results underscore the urgent need to revisit antenatal care guidelines to account for maternal metabolic status, as conventional supplementation may not adequately address these functional iron deficits.

This study has several limitations. Its cross-sectional design restricts causal inference, and cord blood biomarkers only provide a snapshot of intrauterine iron status. The absence of longitudinal data prevents us from assessing postnatal anemia trajectories or developmental outcomes. Furthermore, potential confounders such as maternal dietary patterns, micronutrient interactions, and genetic influences were not evaluated. Future research should prioritize longitudinal cohort designs to track iron status and cognitive outcomes beyond birth. Molecular studies examining placental transporter expression and clinical trials evaluating personalized supplementation protocols based on inflammatory and metabolic screening are needed to develop more precise solutions.

## 4. CONCLUSION

Maternal obesity in the third trimester is associated with significant alterations in neonatal hematologic indices, specifically elevated Mean Corpuscular Volume (MCV) and Mentzer Index. These findings suggest that inflammation-driven disruption of placental iron transport and fetal erythropoiesis may contribute to functional iron dysregulation at birth. Importantly, this condition may not be effectively detected by conventional biomarkers like ferritin and hepcidin, highlighting a critical gap in current assessment practices.

Clinically, these results underscore the inadequacy of a one-size-fits-all approach to iron supplementation. We recommend a more tailored strategy that incorporates antenatal screening for maternal metabolic status and inflammatory markers. Furthermore, integrating the monitoring of functional hematologic markers in neonates—perhaps within existing health systems such as Indonesia's Buku KIA—could improve early detection and ultimately enhance neonatal outcomes in populations facing a dual burden of maternal obesity and anemia.

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