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Neonatal Iron Status is Impaired by Maternal Obesity and Excessive Weight Gain during Pregnancy

Alyssa K. Phillips, BS[1,2], **Sheila C. Roy, MD**[1], **Rebecca Lundberg, BS**[1], **Theresa W. Guilbert, MD, MS**[1,2], **Anthony P. Auger, PhD**[3], **Sharon E. Blohowiak, MS**[1,2], **Christopher L. Coe, PhD**[3,4], and **Pamela J. Kling, MD**[1,2]

[1]School of Medicine and Public Health, University of Wisconsin, Madison WI

[2]Department of Pediatrics, University of Wisconsin, Madison WI

[3]Department of Psychology, University of Wisconsin, Madison WI

[4]Harlow Center for Biological Psychology, University of Wisconsin, Madison WI

Abstract

Objective—Maternal iron needs increase 6-fold during pregnancy, but obesity interferes with iron absorption. We hypothesized that maternal obesity impairs fetal iron status.

Study Design—316 newborns with risk factors for infantile iron deficiency anemia (IDA) were studied to examine obesity during pregnancy and neonatal iron status. Erythrocyte iron was assessed by cord blood hemoglobin (Hb), zinc protoporphyrin/heme (ZnPP/H) and reticulocyte-ZnPP/H and storage iron by serum ferritin.

Results—Women with body mass index ≥30 kg/m^2, as compared with non-obese women, delivered larger offspring with higher reticulocyte-ZnPP/H, and lower serum ferritin concentrations (p<0.05 for both). With increasing BMI, estimated body iron was relatively lower (mg/kg) and the ratio of total Hb-bound iron (mg)/total body iron (mg) increased. Maternal diabetes compromised infant iron status, but multivariate analysis demonstrated that obesity was an independent predictor.

Address Correspondence to: Pamela J. Kling, M.D., UW School of Medicine and Public Health, Dept. of Pediatrics and Meriter Hospital, 202 S. Park St., Madison, WI 53715, pkling@pediatrics.wisc.edu, Tel: (608) 417-6236, Fax: (608) 417-6377.

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Author Contributions: Alyssa K. Phillips carried out the initial analyses, coordinated patient recruitment, supervised data collection, drafted the initial manuscript, reviewed and edited the manuscript, approved the final manuscript as submitted. Sheila C. Roy recruited subjects into the study, summarize portions of the data, reviewed and edited the manuscript, approved the final manuscript as submitted. Rebecca Lundberg processed samples, performed iron assays, reviewed and edited the manuscript and approved the final manuscript as submitted. Theresa W. Guilbert, Anthony P. Auger, and Christopher L. Coe were collaborators on the larger IDA project, aided study design and data analyses, reviewed and edited the manuscript, and approved the final manuscript as submitted. Sharon E. Blohowiak coordinated and supervised laboratory processing and testing, supervised laboratory study personnel, managed project funding, submitted progress reports for the human subject committees, reviewed and revised the manuscript and approved the final manuscript as submitted. Pamela J. Kling is the PI of the larger IDA study, initiated and designed the study, developed the collaborations, supervised study recruitment, recruited subjects, supervised the laboratory analyses, reviewed and edited the manuscript, and approved the final manuscript as submitted.

Conclusions—Obesity during pregnancy and excessive weight gain are independent risk factors for iron deficiency in the newborn.

Keywords

body mass index; weight gain; zinc protoporphyrin/heme; ferritin; iron transport; placenta; pregnancy; inflammation; iron deficiency; newborn

Introduction

Obesity affects 30% of women at childbearing age in the United States and is a key risk factor for poor perinatal outcome.[1] Pregnancy normally stimulates a 6-fold increase in intestinal iron absorption to fulfill fetal iron needs.[2] However, in non-gravid adults, obesity-related inflammation, especially with rapid weight gain,[3] can cause iron deficiency by functionally impeding intestinal iron transport.[4, 5] Excessive gestational weight gain may also precipitate the development of maternal iron deficiency anemia (IDA) in later gestation.[6, 7] The same membrane-bound iron transporters that are found in the intestine are also present on placenta. However, little is known about how obesity during pregnancy affects placental iron transfer. A small study demonstrated that both BMI at conception and maternal hepcidin, the recently recognized major regulator of iron trafficking, were inversely related to measures of neonatal transport iron.[8] However, it is unknown whether the findings generalize to impaired iron delivery to fetus.

IDA is the most common micronutrient deficiency in the world.[9] IDA acquired during rapid infant growth can be addressed with supplementation, but sustained anemia during infancy may impair neural development, emotionality and cognitive performance.[10] Half of the iron needed to sustain growth during the first year of life must normally be acquired before birth[11] and an inadequate fetal endowment increases risk for infantile IDA.[12]

Assessing iron status in early development is challenging, but zinc protoporphyrin/heme (ZnPP/H) is a sensitive index in the neonate and young infant. Zinc replaces iron in the protoporphyrin ring when iron is unavailable, causing steady state ZnPP/H to rise. Cord ZnPP/H in a reticulocyte-enriched fraction assesses iron within recently-made, late gestation erythrocytes,[13] during higher fetal iron need,[2] similar to the automated reticulocyte hemoglobin (Hb) measurement.[14, 15] Transport iron can be assessed using transferrin saturation (TSAT)[8, 16] and although values are higher than in older children, serum ferritin provides a general reflection of storage iron.[17]

The study tests the hypothesis that maternal obesity impairs fetal iron status. Putative mechanisms affecting fetal iron acquisition and tissue partitioning include: a) pre-existing maternal IDA limiting supply[12] or late-gestation onset of IDA, b) fetal demand for greater iron utilization from overgrowth, c) weight-related maternal and placental proinflammatory responses, and/or d) comorbid maternal diabetes impairing placental iron transport while stimulating fetal growth.[5, 12, 18] In a prospective study using perinatal demographic data and multiple cord blood indices the study aim was to examine the effect of maternal obesity and gestational weight gain on newborn iron status.

Methods

IDA Study Design and Eligibility

The Iron Deficiency Anemia in Infancy (IDA) study was a prospective, observational study approved by the Institutional Review Boards from the University of Wisconsin-Madison and Meriter Hospital. Infants delivered at Meriter Hospital, a tertiary care teaching hospital with 3900 deliveries per year, were recruited between June 2008 and August 2010.

The study inclusion criteria included healthy newborns, born to English and Spanish-speaking women between 18-40 years of age, who were delivered ≥35weeks gestation at the Meriter Hospital Birthing Center. The newborns were without infection or other complications, but exhibited at least one risk factor for developing IDA in infancy, with many having multiple risks factors.[19] Electronic medical records were screened for known risk factors for infantile IDA. Medical risk criteria included maternal anemia diagnosed at initiation of prenatal care, pre-gestational or gestational diabetes mellitus (most were diet-treated), fetal undergrowth (small for gestational age - SGA) or overgrowth (large for gestational age - LGA).[20] Demographic risk criteria included women of low socioeconomic status (Medicaid) and from certain ethnic/racial background (African American, Latina, or Asian). After informed consent, mothers and newborns were enrolled, and maternal anthropometric data were used to determine body mass index (BMI) before pregnancy and at delivery.

Sample Collection and Laboratory Tests

Umbilical cord blood was collected at delivery, stored at 4 °C, and assayed within 8 days.[19] Hb was assayed using a pocH-100i hematology analyzer (Sysmex, Mundelein, IL). Erythrocyte iron was assessed by ZnPP/H with hematofluorometry (Aviv Biomedical Co., Lakewood, NJ) after washing.[13] RE-ZnPP/H was measured using the top, most immature fraction of erythrocytes to improve ZnPP/H sensitivity,[13] in a concept analogous to reticulocyte Hb. Iron status was assessed by serum non-heme iron,[21] serum transferrin (Immunology Consultants Laboratory (Newberg, OR), and serum ferritin (Bio-Quant, San Diego, CA). The inflammatory state was assessed by high sensitivity serum C-reactive protein (CRP) (Bio-Quant).

Data Analysis

Maternal morphometric measures were used to calculate pre-pregnancy and delivery BMIs (weight [kg]/length^2[m^2]). Nongravid definitions of obesity were utilized: BMI ≥30 kg/m^2 defined obesity. Excessive pregnancy weight gain was defined as ≥18 kg (40 lbs), based on ACOG recommendations[22] prior to the 2013 revised IOM statement.[23] Newborn birth weight and gestational age determined the *z*-score,[24] based on gender-specific, sea level intrauterine growth curves.[25] LGA defined a *z*-score of >2 and SGA <-2. ΔZnPP/H was the difference between washed and RE-ZnPP/H.[13] The 95th percentile for ZnPP/H was defined as 138 μmol/mol[19] and the 5th percentile for serum ferritin defined as 40 ng/mL.[17, 19] TSAT was calculated from serum transferrin and iron levels. Total storage iron (mg) was calculated as weight (kg) × [(21.99)(log serum ferritin) − 29.04].[17] Total Hb iron (mg) was calculated by weight (kg) × Hb (g/dL) × 2.74 and total body iron (mg) calculated by

summing total Hb iron + total storage iron + functional iron (7 mg × weight [kg]).[17, 26, 27] Fisher's exact or chi square testing was used for nominal data, unpaired *t* tests used for normally distributed continuous data and the Mann-Whitney U test used for nonparametric continuous data. Linear regression, stepwise regression, factorial and multiple ANOVA/ANCOVA analyses were employed. Values were expressed as mean ± standard error. A *p* value of <0.05 was considered significant.

Results

Enrollee Demographic & Morphometric Data

The IDA study enrolled 316 mothers and healthy, but at-risk newborns between June 2008 and August 2010, with numbers of non-obese and obese at both pre-pregnancy and delivery shown in Fig. 1. Pre-pregnancy maternal obesity was evident in 28.5% of women, while 27.5% gained ≥18 kg during pregnancy, and 56% were obese at delivery (Table 1). The enrolled neonates included 52.8% male, 25% born by cesarean section, and 32% large-for-gestational age (LGA). Women obese at delivery birthed larger babies than non-obese, with a higher percent designated as LGA, $p<0.02$ for both (Table 1). Diabetes was more common in those obese at delivery vs. non-obese, $p<0.05$. The percentage of women with IDA diagnosed at the early pregnancy screening did not differ between those classified as obese and non-obese at delivery (Table 1).

A direct relationship was evident between the mother's BMI at delivery and newborn birth weight and birth weight *z*-score, $p<0.0001$ for both (Fig. 2). The influence of obesity on birth weight was more robust when including only the cohort with diabetes, compared to the cohort without diabetes (Fig. 2).

Cord Erythrocyte, Transport and Storage Iron

Cord Hb levels were 5% higher in the newborns of women who were obese at delivery, $p<0.03$ (Table 2) and cord serum erythropoietin levels were higher levels in the obesity group (35.8 ±4.0 U/L) than the non-obese group (27.9 ± 4.0 U/L), $p<0.03$. Newborns birthed by mothers obese at delivery were twice as likely to exhibit cord ZnPP/H ratios above the 95th percentile for term newborns[19] than their non-obese counterparts (11.7% vs. 5.5%, respectively), $p<0.05$ (Table 2). A correlation between delivery BMI and cord ZnPP/H was found, $p<0.02$, but this relationship was more robust in the women with diabetes, $p<0.002$ (Fig. 3A). The sensitive measures, cord RE-ZnPP/H and ΔZnPP/H, were 13% higher and 50% higher respectively in newborns born to mothers obese at delivery vs. those not obese, $p<0.05$ for both (Table 2). Maternal BMI at delivery was also correlated with cord RE-ZnPP/H ($F_{1,291}=12.7$, $R=0.28$, $p<0.0004$), as well as ΔZnPP/H ($F_{1,291}=14.2$, $R=0.22$, $p<0.0002$). Cord TSAT did not differ in obese compared to non-obese at delivery and was unrelated to delivery BMI (Table 2).

Cord serum ferritin levels were 13% lower in those obese at delivery than for non-obese, $p<0.002$, and were 4.5 times more likely to be <5th percentile norms for term newborns[17] vs. non-obese (6.8% vs. 1.5%), respectively, $p<0.05$ (Table 2). A modest inverse correlation

was observed between BMI at delivery and cord serum ferritin, $p<0.005$, which was more evident in newborns after gestational diabetic pregnancy, $p<0.0005$ (Fig. 3B).

Maternal Iron Status and Fetal Growth

The percent of women with IDA in early pregnancy did not differ based on obesity status at delivery, Table 1. Cord ZnPP/H, RE-ZnPP/H, TSAT, and serum ferritin in those born to obese mothers with anemia did not differ from non-obese mothers with anemia. It was not possible to reassess maternal iron status in late gestation because current guidelines do not recommend repeat late gestation screening.[28]

Fetal overgrowth increases fetal iron needs. Because male newborns were 161 grams heavier and had higher birth weight *z* scores, $p<0.04$, gender influence on ZnPP/H was examined. ZnPP/H was 10% higher in males vs. females, $p<0.03$, but ferritin was similar. With both genders combined, offspring of obese mothers were heavier, with larger birth weight *z*-scores, $p<0.007$, and a higher percentage having met criteria for a LGA classification, $p<0.02$, Table 1. In obesity, estimated newborn relative storage iron concentration (mg/kg) was 12% lower, $p<0.002$, but absolute total body iron allotment (mg) did not differ because erythrocyte iron was higher. However, because they were heavier, the relative total body iron concentration (mg/kg) was lower in offspring of obese than in non-obese women, $p<0.002$. Relative newborn body storage iron concentration (mg/kg)[17] was inversely related to maternal BMI at delivery, $p<0.01$, a relationship that was more robust in newborns of women with diabetes, $p<0.007$ (Fig. 3C). Maternal BMI at delivery was directly related with the ratio of total Hb iron (mg)/total body iron (mg), $p<0.002$, an effect also found when evaluating only newborns from women with diabetes, $p<0.003$ (Fig. 3D). Stepwise regression was employed to study the relative ability of newborn weight and maternal BMI to predict iron parameters. Newborn weight, as part of the total iron calculations, was better predictive of either Hb iron (mg), $p<0.0001$, or the ratio of total Hb (mg)/total body iron (mg), $p<0.0001$, than was maternal BMI. In contrast, maternal BMI was more predictive than newborn weight of either ZnPP/H, $p<0.03$, or RE-ZnPP/H, $p<0.001$ (direct relationships) and of plasma ferritin, $p<0.04$ (indirect relationship).

Maternal Weight Gain and Inflammation

High sensitivity CRP was determined in cord blood, but proved not related to obesity status prior to pregnancy or at delivery. The exception was in the case of excessive gestational weight gain of ≥18 kg with cord CRP higher than in newborns of women with more typical weight gain (0.31 mg/L vs. 0.17 mg/L), $p<0.03$. High gestational weight gain ≥18 kg was associated with poorer newborn iron status: 40% higher ΔZnPP/H, and 15% lower serum ferritin, in addition to 20% lower reticulocyte counts, $p<0.05$ for all.

Maternal Obesity and Diabetes

Diabetes was present in 30% of women obese at delivery, compared to 20% of the non-obese cohort, $p<0.05$, Table 1. In the absence of diabetes, newborn iron indices did not differ between the newborns gestated during obese and non-obese mothers. The most distinctive newborns were those born to obese, diabetic women. They had 30% higher RE-ZnPP/H, $p<0.02$, and 33% lower serum ferritin, $p<0.005$, than the newborns from mothers

who were diabetic but not obese. The relative effect of gestational diabetes and maternal obesity on cord iron status was examined further in a bivariate analysis. This approach demonstrated a main effect for maternal obesity, which affected newborn ZnPP/H, RE-ZnPP/H and serum ferritin levels, $p<0.001$ for all, without an interaction between obesity and diabetes. A highly significant interaction term was found between diabetes and LGA in predicting iron status. Two independent sets of analyses were undertaken. First, obesity, and not LGA or newborn sex affected RE-ZnPP/H, ΔZnPP/H, and plasma ferritin, $p<0.05$ for all, without an interaction between the other factors. Second, obesity, and not diabetes or newborn sex affected RE-ZnPP/H, ΔZnPP/H, and plasma ferritin, $p<0.01$ for all, without an interaction between the other factors.

Discussion

This study is the first systematic investigation of obesity during pregnancy and newborn iron status. These findings are important because obesity is increasingly prevalent in women of childbearing age. Although a previous paper reported that pre-pregnancy obesity was inversely related to transport iron indicators at birth,[8] the current study provides biochemical evidence of an independent negative relationship between maternal obesity on iron incorporation into both fetal erythrocytes (higher ZnPP/H) and to tissues (lower ferritin) that was independent of newborn birth weight in multivariate analysis. Although absolute total body iron (mg) did not differ between the groups, heavier babies in the obese group translate into a relative shortfall in both storage iron and total body iron (mg/kg) after maternal obesity.

As maternal BMI increased, trafficking of iron between compartments became more affected, as relative tissue stores (mg/kg) fell at the expense of greater relative Hb iron (mg/kg). A concurrent diagnosis of maternal diabetes further exacerbated the relative body storage iron concentration (mg/kg), but multivariate analyses demonstrated that maternal obesity was an independent predictor of cord blood tests of iron status and of calculated relative fetal tissue iron stores. These findings are concerning because approximately 50% of the iron needed for postnatal infant growth should be acquired before birth and overcoming this relative per kg shortfall to “catch up” is especially challenging with larger infants born to obese mothers commonly growing more rapidly than their peers.

Incomplete Hb iron incorporation was found because ZnPP/H rose, consistent with previous reports in newborns after maternal insulin-treated diabetes[18, 29] and in LGA newborns in the absence of diabetes.[30] During accelerated fetal erythropoiesis, transport of available fetal iron is prioritized to the erythrocytes at the expense of other tissues.[31] A direct relationship between BMI and the ratio of Hb/total body iron supports this view. However, iron was still insufficient to fulfill Hb needs because ZnPP/H rose,[24, 32] especially in reticulocytes. Lower cord ferritin values supported poorer iron stores. It was unclear why TSAT was not inversely related to maternal BMI as previously reported.[8] However, Dao, et al. evaluated only pre-pregnancy obesity in routine obstetrical patients,[8] excluding women with diabetes and other medical complications. In contrast, we actively recruited women with these clinical risks for developing infantile IDA.

Maternal obesity alone did not affect pre-existing gestational IDA, although maternal IDA was one of our enrollment criteria. Women diagnosed with anemia were provided supplements, which have been shown to improve fetal iron status.[33] Maternal iron status is not routinely assessed in the third trimester,[28] but may be warranted in obesity, especially with additional risk factors such as diabetes or groups prone to IDA.[34]

Fetal iron needs are normally proportionate to fetal size,[11] and the study found fetal size directly related to maternal BMI. We previously found that cord blood iron status worsened with increasing fetal size.[30] Large newborns may also grow more rapidly after birth, with rapid growth increasing risk for developing infantile IDA.[35, 36] Although the occurrence of LGA newborns was more common after pregnancies with maternal obesity,[1] being LGA was not the sole basis for poorer cord iron status.[30] Rather, we confirmed that fetal overgrowth compounded the effect of maternal diabetes on newborn iron status, supporting previous work.[24, 30, 32, 37] The effect of obesity on ZnPP/H, RE-ZnPP/H and serum ferritin was not lost in multivariate analysis, supporting that maternal obesity was independent of diabetes or newborn size in determining biochemical iron status.

As maternal BMI rose, iron compartmentalization within the fetus was progressively compromised, with fetal prioritization of iron to Hb at the expense of other tissues, as has been previously reported during insulin-dependent diabetes.[31] This maldistribution of iron with decreased relative tissue iron concentration (mg/kg) may include detrimental ramifications for the development in the immature central nervous system.[38] Obesity can functionally interfere with placental iron transfer and tissue partitioning via a number pathways, including through proinflammatory mediators such as interleukin-6 on hepcidin impairing intestinal iron absorption.[1, 4] Sequestering of iron into maternal tissue may also be seen.[5] Precisely how maternal obesity decreases the relative transfer of iron to fetus is not completely understood,[4] but newborn TSAT values were previously shown to be indirectly related to maternal BMI or hepcidin levels.[8] Of note, the relatively higher fetal Hb concentrations in the obese group may also prevent the normal fall in fetal hepcidin levels needed to upregulate placental iron transfer.[39,40] However, finding higher cord CRP levels after rapid, excessive pregnancy weight gain, as previously reported in nongravid adults,[37] supports that maternal intestinal iron absorption may be insufficient for needs. Our findings support the revised 2009 Institute of Medicine (IOM) recommendations to encourage weight loss consultation prior to pregnancy in obese women and to limit acceptable weight gain to only 5-9 kg for overweight women, an amount much lower than for non-obese women.[23]

Finding poorer iron status in the newborns of obese diabetic women was consistent with our previous work showing poorer newborn iron status after pregnancies complicated by insulin-dependent diabetes.[24] Prior research found dysfunctional placentae, functionally altered iron transporters, and increased fetal iron demand in pregnancies complicated by insulin-dependent diabetes.[39, 41] However, in contrast to our previous study of mothers with insulin-treated diabetes,[24, 32] the current data indicated that the main effects could be mediated in part through obesity.

Our conclusions are limited by several experimental features of the current study, which include that the primary recruitment criterion was not restricted to obesity. Thus, the

population of women included many with historical risk factors for their newborn to develop IDA. Nevertheless, the strengths include that the study sample was representative of newborns serviced by our hospital, and that our approach enabled us to achieve a large sample size. We were also able to obtain a unique panel of iron-related measures on cord blood. That approach allowed us to employ sensitive indices of both erythrocyte iron and storage iron, to calculate the partitioning of iron, and to demonstrate that these measures were abnormally low in otherwise healthy newborns.

The findings are of importance for obstetrical and pediatric practice because decreased relative (mg/kg) iron endowment was found in offspring of obese mothers, a concern compounded by the high prevalence of obesity in women of childbearing age. An effective prenatal transfer of maternal iron is critical to prevent the emergence of infantile IDA, especially in breastfed babies.[40, 42] If undiagnosed and untreated, strong evidence demonstrates that IDA can have lingering effects on neurobehavioral development, affecting emotionality and cognitive ability at school age and even into adolescence.[10] Our findings directly point to the conclusion that iron was prioritized and compartmentalized to erythrocytes at the expense of tissues, most likely including brain.[31, 41, 43] Although not currently recognized,[40] maternal obesity and excessive gestational weight gain could now be considered as risk factors for poorer fetal iron status.

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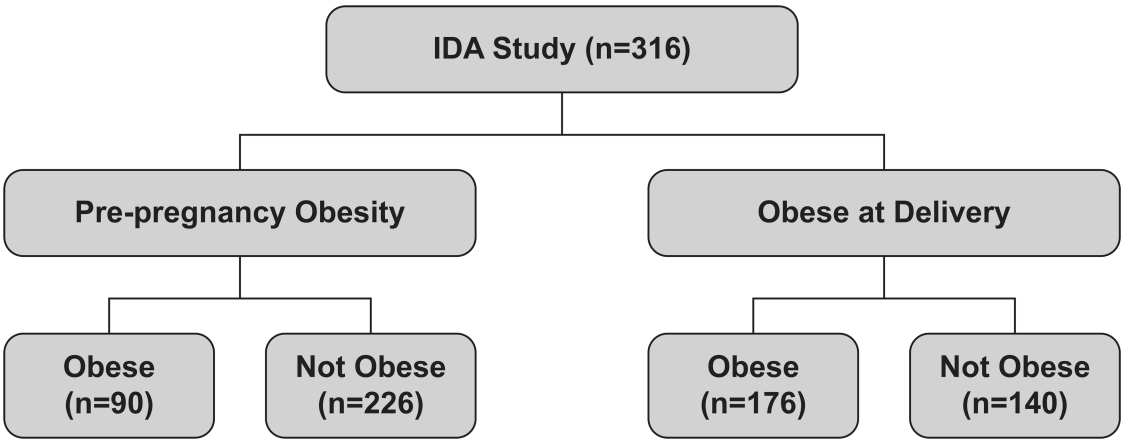

Figure 1.
Enrollees designated as obese or not obese either pre-pregnancy or at delivery.

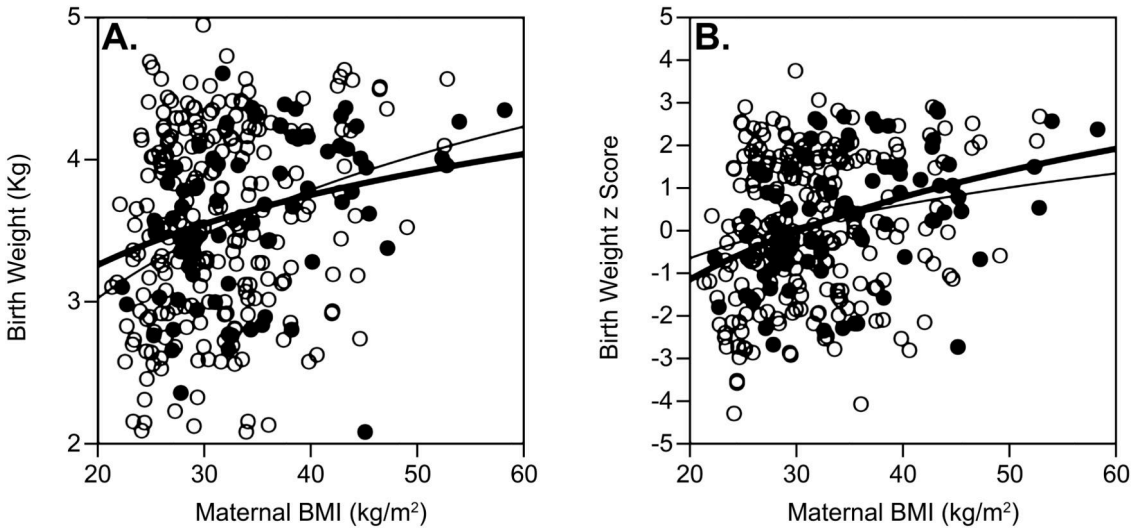

Figure 2.

Newborn size compared to BMI. Diabetes (closed circles, bold lines), non-diabetes (open circles, lighter regression lines). **A**. Birth weight all enrollees $F_{1,314}$=20.0, R=0.25, p<0.0001; diabetes $F_{1,79}$=17.2, R=0.42, p<0.0001; non-diabetes $F_{1,229}$=8.9, R=0.19, p<0.003. **B**. Birth weight z score all enrollees $F_{1,315}$=20.4, R=0.25, p<0.0001; diabetes, $F_{1,78}$=14.6, R=0.40, p<0.0005; non-diabetes, $F_{1,228}$=8.7, R=0.19, p<0.004.

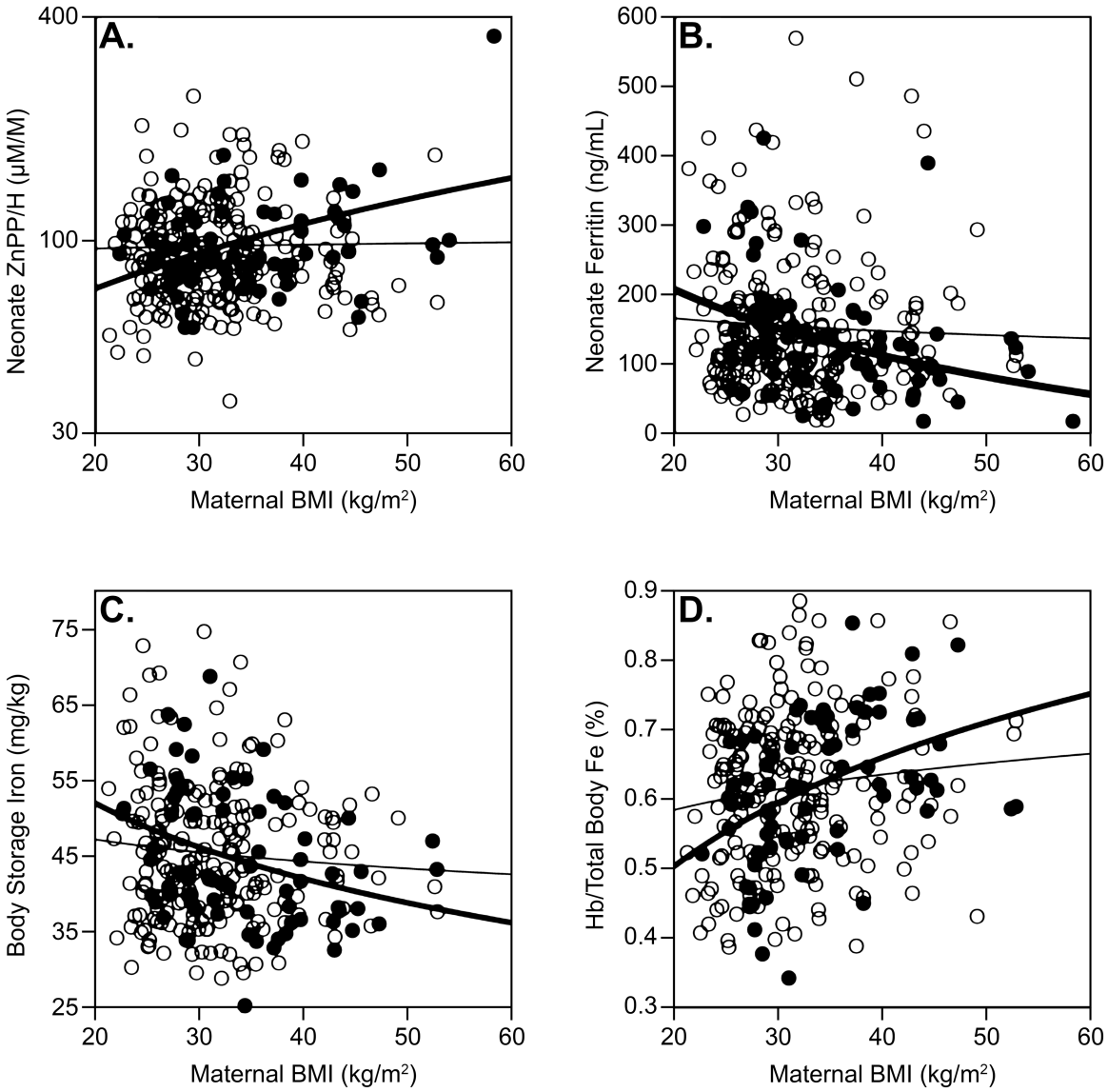

Figure 3.

Newborn iron indices compared to BMI. Diabetes (closed circles, bold line), non-diabetes (open circles, lighter regression line). **A**. ZnPP/H all $F_{1,291}$= 6.3, R=0.14, p<0.02; diabetes $F_{1,70}$=10.8, R=0.36, p<0.002; non-diabetes p=0.88. **B.** Plasma ferritin all $F_{1,291}$=8.2, R=0.17, p<0.005; diabetes $F_{1,72}$=12.2, R=0.39, p<0.0005; non-diabetes, p=0.35. **C**. Relative body stores of iron (mg/kg) all $F_{1,287}$=6.6, R=0.16, p=0.01; diabetes $F_{1,70}$=7.9, R=0.32, p<0.007; non-diabetes, p=0.20. **D.** Calculated total body Hb/body iron (mg/mg) all $F_{1,273}$=10.3, R=0.19, p<0.002; diabetes $F_{1,69}$=14.6, R=0.42, p<0.003; non-diabetes, p=0.10.

Table 1

Maternal & Neonatal Data

	All	Delivery BMI BMI<30 n=140	Delivery BMI BMI≥30 n=176	*p*-value By Delivery BMI
Race (C/AA/H/O) %	72/11.5/11.5/5	72/9/13/6	72/13.5/10.5/4	NS
Prenatal Vitamins %	86%	84%	87%	NS
Iron Supplements %	20%	23%	17%	NS
Maternal IDA %	37%	41%	34%	NS
Maternal Diabetes %	26%	21%	30%	$p<0.05$
Birth Weight (kg)	3.57±0.07	3.47±0.05	3.67±0.05	$p<0.007$
Birth Weight z score	0.18±0.09	(-)0.110±0.138	0.434±0.121	$p<0.004$
LGA Newborns %	32%	25%	38%	$p<0.02$
Male %	52.80%	48%	56%	$p=0.17$

Demographic variables of all enrollees at birth and then divided by BMI status. Data are mean ± SEM. Race includes C=Caucasian, AA=African American, H=Hispanic, O=Other. LGA=large for gestational age.

Table 2

Newborn Biochemistry Data

	All	Delivery BMI <30 mg/m2	Delivery BMI ≥30 mg/m2	*p*-value By Delivery BMI
Newborn Hb (g/L)	162±2	159±2	166±2	$p<0.03$
Newborn ZnPP/H (μmol/mol)	97.5±2	95.0±2.6	100.8±3.0	$p=0.08$
Newborn ZnPP/H >95%	9%	5.5%	11.4%	$P<0.05$
Newborn RE-ZnPP/H (μmol/mol)	124±4	115.7±3.7	130.5±5.7	$p<0.02$
Newborn serum Ferritin (pmol/L)	331±12	256±8	289±13	$p<0.002$
Newborn serum Ferritin (ng/mL)	149±5	168.2±9.5	137.7±7.5	$p<0.002$
Newborn serum Ferritin <5%	5%	1.5%	6.8%	$p<0.05$
TSAT (%)	50%	52±2%	50±2%	NS
CRP (mg/L)	0.213±0.44	0.215±0.048	0.212±0.052	NS

Newborn cord blood laboratory values of all enrollees at birth and then divided by BMI status. Data are mean ± SEM. Hb=hemoglobin, ZnPP/H=zinc protoporphyrin/heme, TSAT=transferrin saturation, CRP=C-reactive protein.

Table 3

Calculated Iron Pool Sizes

	All	Delivery BMI <30 mg/m2	Delivery BMI ≥30 mg/m2	*p*-value By Delivery BMI
Relative Hb Iron (mg/kg)	27.5±0.3	27.0±0.4	27.9±0.4	$p<0.05$
Absolute Hb Iron (mg)	98.95±1.44	93.4±2.2	102.9±3.9	$p<0.0001$
Relative Storage Iron (mg/kg)	17.2±0.4	18.3±0.5	16.2±2.0	$p<0.002$
Absolute Storage Iron (mg)	63.3±1.5	67.1±2.2	59.3±2.0	$p<0.001$
Relative Total Body Iron (mg/kg)	45.5±	46.1±0.9	44.5±0.7	$p<0.002$
Absolute Total Body Iron (mg)	160.7±2.0	158.9±3.1	162.0±2.6	$p=0.10$
Ratio of Total Hb (mg)/Total Body Iron (mg)	0.62±0.01	0.59±0.01	0.64±0.01	$p<0.001$