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Relationship between maternal prenatal vitamin use and infant iron status

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**RELATIONSHIP BETWEEN MATERNAL PRENATAL VITAMIN
USE AND INFANT IRON STATUS**

Jennie P. Wilkins

A Thesis submitted to the
College of Agriculture, Forestry, and Consumer Sciences
at
West Virginia University
for partial fulfillment of the requirements
for the degree of

Master of Science
in
Human Nutrition and Foods

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ABSTRACT

Relationship Between Maternal Prenatal Vitamin Use and Infant Iron Status

Jennie P. Wilkins

Subjects were fifty-six women and their infants who were between the ages of six months and twenty-four months. All of the participants were clients of the Special Supplemental Nutrition Program for Women Infants and Children (WIC) clinics. The WIC clinics were in the West Virginia counties of Ritchie, Gilmer, Calhoun, Braxton, Mason, Jackson, Pleasants, and Greenbrier. These counties had anemia rates of greater than 10%.

Information concerning WIC participation, prenatal vitamin use, history of breast feeding, infant birth weight, and pre-term delivery were obtained through questionnaires. The infants' serum ferritin level was obtained through venous blood and analyzed by LabCorp.

Independent-samples *t*-test was used to analyze maternal prenatal vitamin use and infant's serum ferritin level ($p=.736$). Fisher's Exact Test was used to analyze WIC participation to vitamin use ($p=1.00$), ever breast fed ($p=.719$), and low birth weight ($p=1.00$). Pearson Chi-square Test was used to analyze WIC participation and birth weight ($p=.276$).

Fisher's Exact Test was also used to compare maternal vitamin usage to ever breast fed ($p=.20$), low birth weight ($p=.445$), pre-term delivery ($p=.615$). Pearson Chi-square was used to analyze maternal vitamin usage to infant's birth weight ($p=.327$)

This ex post facto study found no significant result in mothers who took prenatal vitamins or not and the serum ferritin levels of their infants. Also, no statistically significant relationships were found between prenatal vitamin use or non-use and participation or non-participation in WIC to the following: ever breast-fed, gestational age, birth weight, and incidences of low birth weight.

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CHAPTER 1

INTRODUCTION

Despite the apparent availability of high-quality diets, iron deficiency remains a major health risk in the United States (Baker, 2000). At least 7.8 million adolescent girls and pre-menopausal women are iron deficient (Baker, 2000). In West Virginia there are eleven counties where the anemia rate in low-income infants and children is greater than 10% according to the data collected from the Pediatric Nutrition Surveillance System (PedNSS).

Maternal anemia has been linked to death, low birth weight, pre-term delivery, low infant iron status and lower cognitive development of the infant. Death can result in young children and pregnant women who have severe anemia because of the limited capacity of the blood to carry oxygen to the body tissues (Yip, 1998). Maternal iron deficiency has also been linked to premature contraction and pre-term delivery as well as to low birth weight (Goepel, Ulmer, & Neth, 1988; Steer, 2000). Long-term iron deficiency anemia in a child affects his/her cognitive development. Such children tend to have lower scores on mental and motor function tests when they enter school (Lozoff, Jimenez, & Wolf, 1991).

Despite public health programs targeting the increased consumption of iron-fortified and iron-enriched products by lactating women and young infants, iron-deficiency anemia in low-income and pregnant women continues to be a problem (Beard, 2000). People who earn lower incomes may have problems purchasing animal products, which contain more bioavailable iron (Yip, 1998). An alternative is iron

supplementation. While iron supplementation is encouraged through public health programs such as the Special Supplemental Nutrition Food Program for Women, Infants, and Children (WIC) and Right From the Start (an educational program for promoting a healthy pregnancy and delivery of a healthy baby), it is not clear how many women take these supplements. The potential benefit of supplementation to newborns, infants and to their mothers is substantial (Baker, 2000).

Purpose:

Few studies have sampled iron levels beyond those obtained from cord blood. Is there a relationship between maternal iron supplementation during pregnancy and infant iron status after birth? If so, does this relationship continue into the first two years of life?

The objective of this research is to determine if there is a significant relationship between maternal prenatal vitamin use or non-use during pregnancy and serum ferritin levels in infants from six months to two years of age. A secondary objective is to determine if there is a relationship between the following variables: initiation of breast feeding, gestational age, birth weight, and incidence of low birth weight to prenatal vitamin use or non-use and participation or non-participation in WIC. In order to achieve the objectives of this study, the following hypotheses will be tested:

Hypothesis 1: There is a significant difference between the serum ferritin levels of infants born to women who took prenatal vitamins and serum ferritin levels of infants whose mothers did not take prenatal vitamins.

Hypothesis 2: There is a relationship between WIC participation or non-participation and any of the following: Maternal prenatal vitamin use or not, whether the infant was ever breast fed or not, infant birth weight, and incidence of low birth weight.

Hypothesis 3: There is a relationship between maternal prenatal vitamin use or not and any of the following: Whether the infant was ever breast-fed or not, pre-term delivery or not, infant birth weight, and incidence of low birth weight.

Problem Statement:

Mother and child are connected physically from conception through birth. There is a need to determine whether after birth if this connection, although no longer a physical connection, still affects the child. Iron is a critical element in our bodies, which affects virtually every aspect of our growth and development. In order to better understand whether maternal iron stores affect infant iron deficiency, the relationship between maternal intake of iron during pregnancy and infant iron status should be explored. The purpose of this study is to discover whether there is a correlation between maternal use or non-use of prenatal supplements and iron status of their infants between six and twenty-four months of age in rural West Virginia. Serum ferritin will be used as an indicator of infant iron status.

Definitions:

Serum Ferritin is an indicator of the body's iron stores.

Transferrin is the protein that transports iron from the intestine into the blood stream.

Erythrocyte is a mature Red Blood Cells (RBC) that lives for 120 days and contains hemoglobin.

Mean Corpuscular Volume (MCV) is a measure of volume of RBC.

Hemoglobin is the iron containing pigment of RBC, which contains four globin molecules.

Hematocrit is the measure of packed cell volume of RBC and is expressed as a percent of total blood volume.

Pre-term delivery is when a child is delivered at less than 37 weeks gestation.

Low birth weight is when the weight of an infant born is less than five and one half pounds.

CHAPTER 2

REVIEW OF LITERATURE

Overview:

Iron is critical in the formation of hemoglobin, capacity of the tissue to store oxygen as myoglobin, and for cellular aerobic metabolism in cytochromes (Baker, 2000; Yip 1998). Iron is primarily stored in tissue as ferritin or hemosiderin, an iron-rich pigment. Transportation of iron throughout the body is by the protein transferrin (Baker, 2000). Iron deficiency is the result of an imbalance between intake, loss, and tissue stores of iron.

The body of a pregnant woman tries to meet the extra iron demands of pregnancy by increasing the absorption rate of iron from the gut. This absorption increase is especially evident in the second half of the pregnancy (Haram, Nilsen, & Neth, 2000). When such an increase is large enough to meet the increased requirements of pregnancy, sufficient iron will be provided for the maternal and fetal erythrocytes (Harthoorn-Lastuizen, & Lindemans, Langenhuisen, 2001).

The absorption of iron from the epithelial cells in the small intestine is dependent on several factors including the composition of the ingested food, the chemical form of the iron in the diet, the type of iron - ferrous iron (Fe +2) or ferric iron (Fe +3), and the iron status of the individual (Haram et al, 2001; Sherwood, L., 2001). There are some dietary components that inhibit iron absorption such as tea and coffee. There are other dietary components that enhance iron absorption such as citrus juices and other vitamin C

containing foods (Haram et al, 2001). These foods increase absorption by reducing Fe^{+3} to Fe^{+2} (Sherwood, 2001). The composition of the diet, which includes the inhibitors and enhancers of absorption, establishes the baseline for daily iron absorption as does the gastrointestinal environment from which the iron compound will be absorbed (Beard, 2000). Phosphate and oxalate in the diet combine with iron to form insoluble iron salts that are not absorbable (Sherwood, 2001).

After iron is absorbed, it does one of two things. If iron is needed immediately for red blood cell (RBC) production, it is absorbed by the blood and transported by transferrin in the plasma to the bone marrow. This is where the iron is used for RBC production. If the iron is not needed right away, it is stored in the intestinal epithelial cells as ferritin. Ferritin is lost in the feces from the epithelial cells, which are sloughed off every three days, taking the stored iron with them (Sherwood, 2001).

Types of Anemia:

Iron deficient anemia results from not enough iron in the diet or if there is poor absorption from the digestive tract. When there is not enough iron for the synthesis of hemoglobin, the hemoglobin is decreased in size, which decreases the oxygen carrying capacity of the hemoglobin (Sherwood, 2001).

Folic acid is vital for the formation of DNA, cell division, and maturation of erythrocytes. A dietary deficiency of folic acid, a B vitamin, which is used to mature erythrocytes, causes the erythrocytes to remain immature, and is called megaloblastic anemia. A decrease in folic acid leads to a decrease in erythrocytes. Those that are formed are large and more fragile than normal erythrocytes. These large erythrocytes still

contain the normal amount of hemoglobin (Anderson, Anderson, & Glase, 1998; Sherwood, 2001).

Pernicious anemia is a vitamin B₁₂ deficiency that results from the lack of intrinsic factor. Intrinsic factor is essential for the maturation of RBC (Anderson et al, 1998, Sherwood, 2001).

Other types of anemia include aplastic anemia, renal anemia, hemorrhagic anemia, and hemolytic anemia (Sherwood, 2001). These categories of anemia will not be discussed.

Differences in Lab Values:

TABLE 1: The Spectrum of Iron Status in Relation to the Major Tests Available for Clinical Application

	Iron Overload	Normal	Depleted Iron Stores	Iron Deficiency Without Anemia	Iron Deficiency Anemia
Serum Ferritin	↑	N	↓	↓	↓↓
Transferrin Saturation	↑↑	N	N	↓	↓
Erythrocyte Protoporphyrin	N	N	N	↑	↑↑
MCV	N	N	N	N	↓
Hemoglobin	N	N	N	N	↓

Kleinman, ed., Committee on Nutrition American Academy of Pediatrics (1998)

Key: ↑, increased; ↑↑, markedly increased; ↓, decreased; ↓↓, markedly decreased; and N, normal

As evident from the above graph, serum ferritin is the most sensitive indicator to different types of iron changes. This is the reason serum ferritin was chosen as the measure of infant iron stores in this research.

Stages of Anemia:

There are three stages of progressive iron deficiency that are recognized. During the first stage, iron stores are depleted and serum ferritin is low; but hemoglobin synthesis is not affected. In the second stage, iron deficient erythropoiesis occurs but without overt anemia. During this stage there is an inadequate supply of iron to the erythropoietic cells. Percent transferrin saturation and serum iron are low, and total iron binding capacity is high. In the third stage, when the iron supply is insufficient to maintain normal hemoglobin, iron deficiency anemia results. The hemoglobin, hematocrit, MCV and mean corpuscular hemoglobin concentration are low with iron deficiency anemia (Harthoorn-Lastuizen et al, 2000: Chicago Dietetic Association, The South Suburban Dietetic Association, & Dietitians of Canada, 2000).

When an increase in iron needs occurs, iron is supplied from two different sources: First, iron is taken from available stores, and absorption is not altered. Second, as reserves become depleted, absorption is altered to allow for increased absorption from exogenous sources (Harthoorn-Lastuizen et al, 2000).

Complications of Anemia:

Fetal and maternal health are affected by severe iron deficiency anemia (hemoglobin concentration of 90-100 g/L) (Baker, 2000). Birth weight is decreased, complications occur (e.g., toxemia, labor and delivery complication, small-for-gestation age infants), and maternal functioning is impaired with anemia (Baker, 2000). During

pregnancy, a modest drop in hemoglobin appears to be a normal physiologic event.

Hemoglobin concentrations reach their lowest point in the middle of the second trimester and then rise again during the third trimester (Harthoorn-Lastuizen et al, 2000).

When iron intake is adequate during pregnancy, there is a rise in both hemoglobin and hematocrit to pre-pregnancy levels during the third trimester. Iron deficiency is indicated when hemoglobin levels become lower than pre-pregnancy levels. When iron levels increase above pre-pregnancy levels, this usually indicates poor maternal blood volume expansion. This type of situation is associated with hypertension, fetal growth retardation, death, premature delivery, and low birth weight (Baker, 2000).

Effects of Iron Deficiency:

Iron deficiency in early life is likely to have detrimental impact on normal neural development and functioning (Beard, 2000). In addition, adults with iron deficiency anemia experience a reduction of 10-15% in productivity. Oxygen transport to body tissues is compromised with severe anemia, which can cause death in young children and pregnant women (Yip, 1998). Premature contractions, pre-term delivery, and low birth weight have been associated with iron deficiency (Haram et al, 2001).

Neonatal iron stores are dependent on maternal iron stores. Iron deficiency during the first two trimesters of pregnancy is associated with double the risk of premature birth, triple the risk of low birth weight and with increased morbidity and mortality of both the mother and child (Baker, 2000, Gasper, Ortega, & Moreiras, 1993). Premature infants may also have lower iron stores because there has not been sufficient time to build iron stores before birth (Allen, 2000). Iron deficiency has been associated with higher rates of premature delivery, lower infant Apgar scores, delayed development, and disturbed

behavior (Allen, 2000; Baker, 2000), which affect infants' survival rate and development (Yip, 1998). Premature infants have much higher risks of neonatal complications, failure to thrive, and low stores of iron and other nutrients (Allen, 2000). Infants and children of iron-deficient mothers are at increased risk of developing iron-deficiency anemia, which was undetected at birth (Baker, 2000).

The most detrimental consequence of iron deficiency in young children is reduced mental development and cognitive function. These are aggravated by an increased tendency of the iron deficient child to absorb lead (Yip, 1998). Iron deficiency is not always evident at birth, but becomes evident during the first months of life (Haram et al, 2001). A study conducted by Blot, Tchermia, Chenayer, Hill, Hajeri, and Leluo (1980) in which mothers received either iron supplements or placebo during pregnancy, showed that serum iron and erythrocyte indices were greater at eight weeks and at six months in infants born to the supplemented group. There were no differences between the two groups at birth.

Iron Needs During Pregnancy:

The demand for iron during pregnancy is three to four times the quantity absorbed during the same amount of time by a non-pregnant person. Approximately 1245 mg of iron is needed in nine months, which is not equally distributed over time. As the pregnancy progresses, the need for iron increases (Blot, Diallo, & Tchermia, 1999).

During the first trimester, needs are small, 1 to 2 mg per day. In the second trimester, the need increases to 4 mg per day (Blot et al, 1999). Most of this iron is used to cover the increase in maternal erythrocyte mass. During the last trimester, the need for iron increases to 6 mg of iron per day in order for the fetus to establish its own iron

reserves (Beard, 2000). Of the 1245 mg needed for pregnancy (median requirement) and the growing fetus, 240 mg satisfies basal needs during pregnancy; 450 mg is used to augment maternal blood volume; 80 mg is deposited in the placenta; 225 mg satisfies fetal needs; and 250 mg is lost through maternal blood loss during delivery (Blot et al 1999). The median requirement for absorbed iron is 1018 mg for six months of lactation. This is an additional need above the needs for pregnancy. Iron-deficient erythropoiesis and anemia can result from a decrease in iron stores during pregnancy and lactation, which is caused, in turn, by the high demands for iron during pregnancy and lactation. The decline in iron stores makes it difficult to attain a positive or neutral iron balance (Beard, 2000).

The body needs more than 500 mg of storage iron to avoid iron deficiency during pregnancy. Only 20% of women have more than 500 mg of storage iron. Forty percent of women have stores of 100 to 500 mg, and 40% of women who have less than 100 mg of storage iron. Even with the increased iron absorption during pregnancy, at least 20% of women not taking supplements have iron-deficiency anemia (Baker, 2000). Women who have multiple births have a greater risk of iron deficiency if they do not replenish their iron reserves between pregnancies (Blot et al, 1999). Each additional pregnancy compounds the problem of the iron deficiency. In addition lactation pulls more iron from the maternal iron stores with a loss of 0.5 to 1.0 mg/day. After delivery a women needs up to two years of normal dietary intake to replenish the iron loss with each pregnancy (Baker, 2000). Oral iron supplements given to compliant pregnant woman are effective at reducing the incidences of anemia and will help provide enough exogenous iron to cover the increased physiological need of pregnancy (Haram et al, 2000).

The effectiveness of intervention with a iron supplement is dependent on several factors including the composition of the diet, the presence of a physiologic or pathologic condition that would alter iron absorption or loss, the composition of the supplement, the severity of the iron deficiency at baseline, and the duration of the intervention (Beard, 2000).

Factors That Can Affect Iron Levels:

Serum ferritin is the most specific blood test for iron deficiency. There is a correlation of about 75% between low serum ferritin and the absence of bone marrow stainable iron. The specificity of low serum ferritin for absent marrow iron is 98%. There are limitations when using serum ferritin, percentage of saturation and transferrin. Besides being influenced by pregnancy, a misleading increase in serum ferritin can be caused by premenstrual state, ingestion of iron supplements, progesterone-based oral contraceptives, iron dextran injection, hepatitis, and hemochromatosis. Diurnal variation, menstruation, acute or chronic inflammation, infection and malignancy can cause a decrease in serum ferritin. Progesterone-based oral contraceptives can increase total iron-binding capacity. Ingestion of iron supplements, progesterone-based oral contraceptives, iron dextran injection, and hemochromatosis can increase transferrin saturation. A decrease in transferrin saturation can be caused by acute or chronic infection, inflammation and malignancy (Baker, 2000). There is a lack of simple and reliable tests that can be used easily on a large group to assess iron status. This is a reason why there is across the board supplementation.

Maternal and Infant Iron Stores:

Numerous studies have researched the relationship between maternal iron status and infant iron status by analysis of cord blood. Some studies indicate no correlation between infant cord blood and maternal iron (MacPhail, Charlton, Bothwell, & Torrance, 1980; Wallenburg & van Eijk, 1984; Harthoorn-Lasthuizen et al, 2001), while others have found a correlation (Gasper et al, 1993; Singla, Tyagi, Shankar, Dash, & Kumar 1996; Kilbride, Baker, Parapia, Khoury, Shuqaidef, & Jerwood, 1999). These differences could be due to different techniques, history of maternal anemia in the population samples, and supplement use. None of these studies followed the infants of anemic and non-anemic mothers for any duration after birth. Infant iron stores would have most likely decreased during periods of rapid growth.

Puolakka, Janne, and Vihko (1980) studied forty-seven Finnish women during pregnancy and birth. Fifteen of the women took no iron supplementation during pregnancy. All the women had normal pregnancies and deliveries. The blood samples at delivery were obtained from the maternal antecubital vein and from the umbilical cord. Thirty-one of these women and their infants returned for the six-month follow-up. At that time venous blood was drawn from the mothers and capillary blood was drawn from the infants. None of the infants were treated with iron during the study. Mothers with low ferritin levels gave birth to babies with significantly lower mean serum ferritin values ($p < 0.05$) than babies born to mothers with normal ferritin levels. These results did not show a difference between the infants of mothers who received supplementation and the infants of mothers who did not receive supplementation during pregnancy when

comparing Apgar score, birth weight, weight at six months of age, and duration of breast feeding.

Puolakka et al (1980) also found that women who had taken iron supplements had higher serum ferritin levels than women who had not taken iron (328 and 224 $\mu\text{g/L}$, respectively) ($p>0.05$). At six months, the difference in iron stores between infants born to iron treated mothers and those born to non-treated mothers still persisted, as assessed by serum ferritin. The authors concluded, "...iron supplements given to the mother during pregnancy may be of value in preventing childhood iron deficiency anemia"

A double blind study out of Niger by Preziosi, Prual, Galan, Daouda, Boureima, and Hercberg (1997) included ninety-nine pregnant women who were given iron supplements and ninety-eight pregnant women in a placebo group. All the women were at 28 week \pm 21 day of pregnancy and were randomly assigned to either the iron or the placebo group. The women who were assigned to the iron group were given 100mg of iron per day for the remainder of their pregnancy. Maternal venous blood samples were obtained at the beginning of the study, during the first stage of labor, and at three and six months postpartum. Infant blood was collected from cord blood, and from heel sticks at three and six months. The cord blood revealed no differences between the two groups. Three months after delivery, the serum ferritin concentrations were significantly higher in infants whose mothers had received iron-supplementation, and these differences persisted at the six-month blood sample. Also, in the iron supplemented group, the mean lengths and Apgar scores were significantly higher than in the placebo group. The prevalence of iron deficiency was lower in the iron supplemented group (48.1%) than in the placebo group (72.1%), but the difference was not significant.

Colomer, Colomer, Guitierrez, Jubert, Nolasco, Donat, Fernandez-Delgado, Bonat, and Alvarez-Dardet (1990) followed 156 neonates for one year in a prospective cohort study. Their hypothesis was that a relationship exists between maternal iron deficiency during pregnancy and development of iron deficiency in the newborn. Confounding variables such as socio-economic variables, feeding practices, and other issues linked with the iron status of infants were used in a stratified analysis to control the effects of these factors variables. Blood was obtained from the mothers and infants at birth, and at three, six, nine and twelve months. Iron deficiency anemia was defined as a hemoglobin level below 11g/dl and either one of the following; serum ferritin <12ng/ml or erythrocyte protoporphyrin >35 mcg/100ml of whole blood. The odds ratio of 6.57 (95% confidence limits 1.81-25.97) indicated infants born to anemic mothers were more likely to become anemic themselves.

Morton, Nysenbaum, and Price (1988) studied ferritin and hemoglobin levels in 81 mothers and the cord blood of their infants. Ferritin and hemoglobin levels were measured at six months in fifty-five of the infants, and the levels were measured again at one year of age in fifty-one infants. No relationship was found between the mothers' iron status and their babies' iron status at birth. However, there was a significant relationship between infants' ferritin at six months and cord ferritin at birth ($r = 0.42$, $p < 0.05$). The infants' ferritin was still related to cord ferritin one year after birth ($r = 0.55$, $p < 0.01$).

“The worldwide anemia prevalence data suggest that normal dietary intakes of iron are insufficient to meet peak daily requirements for a significant proportion of pregnant women” (Beard, 2000). In West Virginia there are eleven counties where the anemia rate in low-income infants and children is greater than 10% according to the data

collected from WIC clinics. Understanding the mother's supplement use and the effects of the use or non-use of supplements on her infant can contribute to the knowledge of the effectiveness of iron supplementation.

CHAPTER 3

METHODOLOGY

The information for this ex post facto study was obtained from an ongoing research study conducted by Dr. Cindy Fitch titled *Factors Associated with Iron Status among WIC Infants in Rural West Virginia*. Dr. Fitch's study was reviewed and approved by the West Virginia University Institutional Review Board for the Protection of Human Subjects. Funding was provided by the West Virginia Agriculture and Forestry experiment station and a grant from The United States Department of Agriculture, Economic Research Service. Dr. Fitch's study was a cross-sectional study that determined and analyzed the iron status, health status, dietary patterns, and quantitative nutrient intake of a group of infants in the West Virginia counties of Ritchie, Gilmer, Calhoun, Braxton, Mason, Jackson, Pleasants, and Greenbrier. Prevalence of anemia is greater than the state average in these counties.

Research Design

This ex post facto study addressed two primary objectives. The first objective was to determine if there was a relationship between maternal prenatal vitamin usage or non-usage during pregnancy and infant iron status after birth. The two secondary objectives were to determine if there was a relationship between prenatal vitamin use or non-use and participation or non-participation in WIC to the following: ever breast fed, gestational age, birth weight, and incidences of low birth weight in rural West Virginia participants of WIC.

Hypothesis 1: the independent variable was maternal prenatal vitamin usage or non-usage and the dependent variable was the infant's serum ferritin level.

Hypothesis 2: the independent variable was WIC participation or non-participation and the dependent variables were maternal prenatal vitamin use or non-use, history of breast-feeding or non-breast feeding, infant birth weight and the incidence of low birth weight.

Hypothesis 3: the independent variable was maternal prenatal vitamin use or non-use and the dependent variables were history of breast feeding or non-breast feeding, pre-term delivery or not, infant birth weight and incidence of low birth weight.

Subjects

There were fifty-six participants in the study. All of the participants were from rural counties of West Virginia and were from low-income families. This information was known due to the fact that in order to be a WIC client, they have to meet certain low-income guidelines.

The counties mentioned above were identified by the PedNSS as having an anemia rate of >10% in the population served by WIC. Dr. Fitch and her research assistants met with the WIC nutritionists in the counties selected to ask for their help in recruiting participants. Infants who were known to have a chronic disease were excluded because of the effects of chronic disease on nutritional status and nutrient intake. All other infants between the ages of six and twenty-four months who were clients of the WIC program were considered eligible for the study and their parents were approached for informed consent. When the WIC personnel identified an appropriate family with infants in the age range, a parent was contacted and given an information sheet explaining

the study, and a postcard. If the family was interested in participating in the study they mailed the postcard to Dr. Fitch's office at West Virginia University.

Procedures

After a postcard was received, the research assistants contacted the family to set up an appointment at the participant's WIC clinic. At this meeting, informed consent was obtained, information on the DATA COLLECTION FORM (Appendix A) and the DIET HISTORY (Appendix B) sheets were completed. These information sheets were obtained from fifty-six participants.

The parent and infant were then sent to the local clinic for the infant's blood to be drawn. The clinic, using approved procedures from Laboratory Corporation of America (LabCorp), sent the blood sample to LabCorp to be analyzed. Parents, whose children participated in the study, spent approximately two hours with the investigators. They were offered compensation of \$40.00 for their time. Payment was mailed to them after completion of the session. Compensation of \$10.00 was provided to parents who complete part, but not all, of the session.

First hypothesis:

The information on serum ferritin levels was obtained from research concerning another aspect of Dr. Fitch's study. Abnormal serum ferritin levels were established from Laboratory Corporation of America's guidelines printout. The infant's serum ferritin levels were compared to whether the mother took prenatal vitamins or not. The information on prenatal vitamin use or non-use was obtained from the Diet History question fourteen. This information was analyzed using independent-samples *t*-test..

Second Hypothesis:

The information on WIC participation or WIC non-participation was obtained from Diet History question thirteen. Several different factors were used to determine if there was a relationship. The first relationship was maternal prenatal vitamin use or non-use, and this information was obtained from Diet History question fourteen. The second relationship was whether the infant was ever breast-fed or not: Diet History questions nine and Diet History question ten. These were analyzed using Fisher Exact Test because of the small sample size in several of these groups. The third relationship concerned infant birth weight. This information was obtained from the Data Collection Form and analyzed using independent-samples *t*-test. The fourth comparison was the incidence of low infant birth weight (infant weighing less than 5.5 pounds), which was obtained from the Data Collection Form. These were analyzed using Fisher's Exact Test because of the small sample size (Hermansen, 1990).

Third hypothesis:

An investigation of a relationship was also made among maternal prenatal vitamin use or non-use and several other factors. Prenatal vitamin use or non-use (Diet History question thirteen) was compared to whether the infant was ever breast-fed or not (Diet History questions nine and ten), pre-term delivery or not (Data Collection Form), and incidence of low infant birth weight (Data Collection Form). These were analyzed using Fisher's Exact because of the small sample size. Another factor for comparison prenatal vitamin use or not was infant birth weight (Data Collection Form), which was analyzed using independent-samples *t*-test.

Statistical Program for the Social Sciences 10.0.7 (SPSS) was used to enter and analyze data collected. A statistician provided statistical expertise.

CHAPTER 4

RESULTS

The first objective of this study was to determine if there was a significant relationship between maternal prenatal vitamin use or non-use during pregnancy and the serum ferritin level of their infants from six month to two years of age. The secondary objective was to determine if there was a relationship between the following variables: initiation of breast feeding, gestational age, birth weight, incidence of low birth weight and/or to prenatal vitamin use or non-use and participation or non-participation in WIC.

There were fifty-six participants in the study. All hypotheses were tested at the $\alpha = .05$ level of significance.

Hypothesis 1:

Infant's serum ferritin was obtained from thirty-seven participants. Of the nineteen participants that serum ferritin levels were not obtained: In three there wasn't enough blood drawn to perform the test and in sixteen there was no infant's blood drawn. Two attempts were made to draw the infant's blood. If the procedure was not successful after two attempts, no further attempts were made to obtain blood from the sixteen infants. This resulted in a 66% participation rate in infant's serum ferritin.

Equality of variances was supported by Levene's Test for Equality of Variances with results of $F = .211$ and $p = .649$. An independent-samples t -test revealed there was no significant difference between the serum ferritin levels of infants born to women who took prenatal vitamins and the infants of women who did not take prenatal vitamins. Infants born to mothers who did not use prenatal vitamins ($n = 6$) had a mean serum

ferritin level of 25.83 with a standard deviation of 27.40. The results for the independent-samples t – test are $t = -.339$, $df = 35$, and $p = .736$. Table 2 summarizes the results from the analysis of maternal prenatal vitamin use or non-use when compared to the infant’s serum ferritin level.

TABLE 2: Results of Maternal Prenatal Vitamin Use or Non-use Compared to Infant’s Serum Ferritin Level

	Prenatal Vitamin Use $n = 31$	Prenatal Vitamin Non-use $n = 6$	Significance Level
Ferritin	29.13 \pm 20.69	25.83 \pm 27.40	$p = .736$
Mean \pm SD			

The null hypothesis that there is no significant difference between the serum ferritin levels of infants born to women who took prenatal vitamins and serum ferritin levels of infants whose mothers did not take prenatal vitamins is accepted. The results of the analysis of the data indicated that there was no difference between the maternal prenatal vitamin use and non-use when compared to the infant’s iron status.

Hypothesis 2:

Information on WIC participation or non-participation, vitamin use or non-use, breast-fed or never breast-fed and low birth weight or not were obtained from fifty-six participants. Data was obtained from all of the participants regarding this information. This resulted in a 100% participation rate.

There were a total of 56 participants and the combinations were WIC participation and prenatal vitamin use ($n = 42$), WIC participation and prenatal vitamin non-use ($n = 5$), WIC non-participation and prenatal vitamin non-use ($n = 1$) and WIC non-participation and prenatal vitamin non-use ($n = 8$). There was no relationship between WIC participation or non-participation and prenatal vitamin use or non-use. Fisher's Exact Test was used for analysis of this relationship ($p = 1.00$).

Twenty-one mothers participated in the WIC program and breast-fed their infants for some period of time, twenty-six participated in the WIC program and never breast fed, four did not participate in WIC while they were pregnant and they never breast-fed and five did not participate in WIC while they were pregnant did breast-fed their infants. There was no statistically significant relationship between WIC participation or non-participation and ever breast-fed or never breast-fed. Fisher's Exact Test was used for analysis of this relationship ($p = .719$).

To determine the relationship between WIC participation and other variables, and the combinations were WIC participation and low birth weight ($n = 4$), WIC participation and birth weight within normal limits (WNL) ($n = 43$), WIC non-participation and low birth weight ($n = 5$) and WIC non-participation and birth weight WNL ($n = 4$). There was no statistically significant relationship between WIC participation or non-participation and incidences of low birth weight. Fisher's Exact Test was used for analysis of this relationship ($p = 1.00$).

WIC participation or non-participation and the relationship to the infant's birth weight was calculated using Pearson Chi-square Test. This analysis resulted in Pearson Chi-square Test = 52.293 ($df = 47$), and $p = .276$. Therefore, no relationship was found

between WIC participation or non-participation and incidences of low birth weight. The null hypothesis that there is no relationship between WIC participation or non-participation and incidences of low birth weight is accepted.

Table 3 summarizes the results from the analysis of WIC participation or non-participation and prenatal vitamin use or non-use, incidences of low birth weight and ever breast-fed or not. Table 4 summarizes the results of WIC participation or non-participation in relationship to infant’s birth weight.

TABLE 3: Maternal WIC Participation or Not Compared to Whether the Mother Took Prenatal Vitamins or Not, Whether the Infant Was Low Birth Weight (< 5.5 lb.) or WNL, or Whether the Infant Was Ever Breast Fed or Not

	Prenatal Vitamin Use	Prenatal Vitamin Non-Use	Low Birth Weight	Birth Weight WNL	Ever Breast fed Yes	Never Breast fed No
WIC Participation Yes	42	5	4	43	21	26
WIC Participation No	8	1	1	8	5	4
Significance Level *	$p = 1.00$		$p = 1.00$		$p = .719$	
* Using Fisher’s Exact						

Hypothesis 3:

Information on prenatal vitamin use or non-use, breast-fed or never breast-fed, incidences of low birth weight and infant's birth weight were obtained from all fifty-six participants. This resulted in a 100% participation rate.

Of the fifty-six women indicated in the study, the combinations were prenatal vitamin use and ever breast-fed ($n = 25$), prenatal vitamin use or never breast-fed ($n = 25$), prenatal vitamin non-use and ever breast-fed ($n = 1$) and prenatal vitamin non-use and never breast-fed ($n = 5$). There was no relationship between prenatal vitamin use or non-use and if the infant had ever been breast-fed or not. Fisher's Exact Test was used for analysis of this relationship ($p = .20$).

Prenatal vitamin use and low birth weight ($n = 4$), prenatal vitamin use and birth weight WNL ($n = 46$), prenatal vitamin non-use and low birth weight ($n = 1$) and prenatal vitamin non-use and birth weight WNL ($n = 5$) were analyzed and no relationship was found between prenatal vitamin use or non-use and incidences of low birth weight. Fisher's Exact Test was used for analysis of this relationship ($p = .445$).

The combinations used for prenatal vitamin use or non-use and pre-term delivery or full term delivery were prenatal vitamin use and pre-term delivery ($n = 11$), prenatal vitamin use and full term delivery ($n = 39$), prenatal vitamin non-use and pre-term delivery ($n = 2$) and prenatal vitamin non-use and full term delivery ($n = 4$). There was no relationship between prenatal vitamin use or non-use and incidences of pre-term delivery. Fisher's Exact Test was used for analysis of this relationship ($p = .615$).

Prenatal vitamin use or non-use and the relationship to infants' birth weight was calculated using Pearson Chi-square Test. This analysis resulted in Pearson Chi-square

Test = 50.773 ($df = 47$), and $p = .327$. Therefore, no relationship was found between vitamin use or non-use and incidences of pre-term delivery. The null hypothesis that there is no relationship between vitamin use or non-use and incidences of pre-term delivery is accepted.

Table 4 summarizes the result of prenatal vitamin use or non-use in relationship to the infants' birth weight. Table 5 summarizes the results from the analysis of prenatal vitamin use or non-use and ever breast-fed or not, pre-term delivery or full term and incidences of low birth weight.

Table 4: Infant Birth Weight in Relationship to WIC Participation or Non-participation and Prenatal Vitamin Use or Non-Use

	Pearson Chi-square	Degrees of Freedom	Significance Level
Prenatal Vitamin use or non-use/ Infant Birth Weight	50.773	47	$p = .327$
WIC Participation or non-participation/ Infant Birth Weight	52.293	47	$p = .276$

TABLE 5: Maternal Prenatal Vitamin Use or Non-use Compared to Whether the Infant Was Ever Breast Fed or Not, Pre-term Delivery or Not and Born Low Birth Weight (< 5.5 lb.) or Not

	Ever Breast fed Yes	Never Breast fed No	Pre-term Delivery	Full Term Delivery	Low Birth Weight	Birth Weight WNL
Prenatal Vitamin Use	25	25	11	39	4	46
Prenatal Vitamin Non-use	1	5	2	4	1	5
Significance Level *	$p = .200$		$p = .615$		$p = .445$	
* Using Fisher's Exact						

CHAPTER 5

DISCUSSION

Iron supplementation is effective in the prevention of iron deficiency anemia in pregnant women (Harthoorn-Lasthuizen et al, 2001). However, it is not clear whether that benefit is passed on to the fetus. The logic follows if prenatal iron supplements increase maternal iron and maternal iron increases infant iron status after birth then prenatal iron supplements would increase infant iron status. However, this study did not support this logic.

The Harthoorn-Lasthuizen et al (2001) study along with Wallenburg et al (1984) and MacPhail et al (1980) found no significant relationship between maternal prenatal vitamin use or non-use compared to the infants' iron status. The unexpected result of this research was the acceptance of the null hypothesis concerning prenatal vitamin use or non-use and the infants' serum ferritin level. The results of this research confirm the null hypothesis that there is no significant difference between the infants' ferritin level and the maternal prenatal vitamin use or non-use. This supports the findings of Harthoorn-Lasthuizen et al (2001), Wallenburg et al (1984) and MacPhail et al (1980) that there is no correlation between maternal iron deficiency and neonatal serum ferritin. The results of Harthoorn-Lasthuizen et al (2001) Wallenburg et al (1984) and MacPhail et al (1980) were based on cord blood. The researchers of these studies did no follow-up tests on the infants after birth to see the effects of being born to an iron deficient mother presented itself a few months after delivery.

The results of this study were contrary to Puolakka et al (1980) findings. Puolakka et al (1980) found a significant difference in infant iron status at six months between infants born to anemic mothers and those infants who were born to non-anemic mothers. Other studies that obtained similar results were Preziosi et al (1997), Colomer et al (1990) and Morton et al (1988).

All of the studies mentioned above were conducted in countries other than the United States and may not have the same food fortification programs available to their citizens. This could have affected the initial serum ferritin levels of the mothers. Also, many of the above studies contain subjects of different ethnic backgrounds. There are ethnic differences in the iron stores between people of African descent and Caucasians (Zacharski, Ornstein, Woloshin, & Schwartz, 2000). All of the WIC participants in this study were of Caucasian descent. Therefore, generalization across a diverse ethnic population would not be suggested.

Limitations of this present research could have contributed to the acceptance of the null hypothesis. Some limitations of this research could explain the acceptance of the null hypothesis one: small total sample size ($n = 56$), small sample size of the infant ferritin level ($n = 37$) and ex post facto study. Possibly, if the sample size was larger, there would have been a larger number of mothers who did not take prenatal vitamins and/or possibly a larger number of infants would have had their blood drawn to allow a broader subject base for the comparison of infants' serum ferritin levels to maternal prenatal vitamin use or non-use.

The iron content of the prenatal vitamins vary. There is the rare instance when a prenatal vitamin is prescribed that contains no iron. The amount of iron in the subjects'

prenatal vitamins could have been useful information because the assumption was made that all of the prenatal vitamins contained iron. The large *SD* could be attributed to the disproportional sample size of the prenatal vitamin use ($n = 31$) and non-use ($n = 6$).

The null hypothesis two and three were also accepted. Again the limitations of the study probably had a factor in the acceptance of these null hypotheses. The fact that there was a small participation base ($n = 56$), ex post facto study, and that the answers to the questions on the DATA COLLECTION FORM and the DIET HISTORY sheet are answered by the participants from memory. There is the possibility that questions were answered incorrectly, or that they were not answered truthfully. The results of this research were based on the answers the participants gave on these sheets. Retrospectively linking the information on the DATA COLLECTION FORM to the subjects WIC chart would have negated the possibility of incorrect answers to several of the questions.

Recommendations for future research:

- Link research information to a reliable source of information such as medical records.
- Establish a broader subject base.
- Obtain iron status of newborn infants.
- Divide the children in to different age groups. Possibly three to six months, six to nine months, and ten to eighteen months.
- Possibly follow the same children at birth, three months, and six months.

Further research is needed to establish through understanding of this complicated issue. The existing research to date is difficult to compare, due to the fact that there is no

uniform standardization (none were found by this researcher), such as the range of severe anemia, which blood values to use in the research, ages of subjects, and parity

The subjects for this research were from rural West Virginia WIC clinics; therefore, generalization of the results to other populations is not suggested. Additional research is suggested to better understand the effects of maternal iron status on the newborn.

CHAPTER 6

SUMMARY

Hypothesis 1: There is a significant difference between the serum ferritin levels of infants born to women who took prenatal vitamins and serum ferritin levels of infants whose mothers did not take prenatal vitamins.

Hypothesis 2: There is a relationship between WIC participation or non-participation and any of the following: maternal prenatal vitamin use or not, breast-feeding or not, infant birth weight, and incidence of low birth weight.

Hypothesis 3: There is a relationship between maternal prenatal vitamin use or not and any of the following: breast-feeding or not, pre-term delivery or not, infant birth weight, and incidence of low birth weight.

The first objective of this study was to determine if there was a significant relationship between maternal prenatal vitamin use or non-use during pregnancy and the serum ferritin level of their infants from six month to two years of age. The secondary objective was to determine if there was a relationship between the following variables: initiation of breast feeding, gestational age, birth weight, incidence of low birth weight and/or to prenatal vitamin use or non-use, and participation or non-participation in WIC.

The results of this research did not support the hypotheses. Infant iron status may be related to maternal iron supplementation, but it was not supported by this research. Further research is needed on this important topic to better understand the effects of maternal iron status on the newborn.

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Appendices

Appendix A

DATA COLLECTION FORM

Date _____

Patient Name _____

Subject Number _____

Date of Birth _____

Gender _____

	<u>% For Age</u>	<u>z-score</u>
Weight (lbs) _____ or _____ kg	_____	_____
Length (cm) _____ or _____ cm	_____	_____
Weight for Length _____	_____	_____

Birth/Delivery Status:

Pre-Term
< 37 weeks

Full-Term
40 weeks

Post-Term
> 42 weeks

Length of Gestation _____

Length at Birth (cm) _____ Weight at Birth (lbs) _____

MEDICAL HISTORY

History of chronic illness:

Asthma	yes	no
Disability	yes	no
Constipation/Vomiting	yes	no

DISEASES

Diabetes	yes	no
Kidney Disease	yes	no
Cancer	yes	no
Heart Disease	yes	no
Liver Disease	yes	no

How long ago did the child last have

a) a cough or cold _____ days _____ weeks _____ months

b) diarrhea _____ days _____ weeks _____ months

c) ear infection _____ days _____ weeks _____ months

d) bacterial infection requiring antibiotics _____ days _____ weeks _____ months

Appendix B

Vitae

Personal Data

Name: Jennie Pringle Wilkins
Date of Birth: April 28, 1960
Residence: Westover, West Virginia

Education

August 2000 – May 2002
West Virginia University
Morgantown, West Virginia
Masters of Science in Human Nutrition and Foods

August 2000 – May 2002
West Virginia University
Morgantown, WV
Accredited Masters Level Dietetic Internship

January 1998 – May 2000
West Virginia University
Morgantown, WV
Bachelor of Science in Human Nutrition and Foods

August 1978 – May 1981
West Virginia University
Morgantown, WV
Course of Study – Child Development

Experience

August 2001 – May 2002
West Virginia University
Dietetic Internship

August 2000 – May 2001
West Virginia University
Graduate Teaching Assistant

May 1996 – November 1999
Weight Watchers
Charleston, WV
Group Leader

August 1996 – February 1998
Junior Elementary School
Junior, WV
Library Aid

Scholarships, Awards and Honors

Phi Upsilon Omicron — National Honor Society in Family and
Consumer Science

Outstanding Senior Award in Human Nutrition and Foods 2000

Dean's Honor Roll

Scholarship – Foundation of Monongalia General Hospital