EFFECT OF SHORT-TERM NUTRITIONAL SUPPLEMENTATION VIA THE WIC PROGRAM ON SUBSEQUENT DEVELOPMENT OF PREECLAMPSIA

A THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

FOR THE DEGREE OF MASTER OF SCIENCE

IN THE GRADUATE SCHOOL OF THE

TEXAS WOMAN'S UNIVERSITY

COLLEGE OF NUTRITION, TEXTILES AND HUMAN DEVELOPMENT

BY

STEPHANIE LAYNE MARTCH BARRERA, B.S.

DENTON, TEXAS

MAY, 1983

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INTRODUCTION

Preeclampsia of pregnancy, formally referred to by the misnomer "toxemia", is a syndrome characterized by increased blood pressure, abnormal edema, and proteinuria. It affects 5-7% of the pregnant population as a whole (1,2), while 8-10% of mothers who are socioeconomically deprived develop the syndrome. Preeclampsia is thought to be a disease of the arterioles since vasoconstriction and vasospasm of the peripheral arterioles occur. Such alterations decrease blood flow to vital organs, especially the uterus, and lead to increased blood pressure. If blood pressure elevations cannot be controlled, eclamptic seizures usually ensue, which may be due to increased central nervous system irritability caused by hypoxia from a decreased perfusion of blood through the system (1). The symptoms of the disease are completely reversible upon delivery of the infant. Perinatal mortality approximates 10% in preeclampsia and 30% in eclampsia (3). For mothers, preeclampsia ranks only behind hemorrhage and sepsis as a cause of death. Still, it is felt that this mortality could be reduced to almost zero by proper prenatal care and careful management of the syndrome and any complications (3). Perhaps therein lies

the problem. Preeclampsia has been called by many "the disease of theories" because no one is certain as to its mode of development.

Many researchers suggest that diet plays an important role in prevention of the syndrome. The effects of varying levels of dietary protein, carbohydrate, fat, sodium, iron, and other micronutrients have been investigated (4,5,6,7,8,9). Still, a biochemical link between preeclampsia and dietary status has not been established.

The Woman, Infants, and Children (WIC) Supplemental Food Program provides foodstuffs for pregnant women deemed to be at nutritional risk. Such a program provides the short-term nutritional supplementation necessary to determine any effect of such an addition on development of preeclampsia.

STATEMENT OF THE PROBLEM

The WIC program provides supplemental eggs, milk, cereal, juice, and cheese to pregnant women deemed to be at nutritional risk during pregnancy. By comparing blood pressures, incidence of proteinuria, edema, and diagnosis of preeclampsia, this study will attempt to answer the question: Is there a significant difference in incidence of preeclampsia in women enrolled in the WIC program compared to those not participating in the WIC program during their pregnancies?

REVIEW OF THE LITERATURE

Preeclampsia is a syndrome classically characterized by edema, proteinuria, and hypertension; and can include complaints of dizziness, headaches, visual disturbances, upper abdominal pain, anorexia, nausea, or vomiting (3). It is one of the leading causes of death among obstetric patients, following hemorrhage and sepsis. Once preeclampsia develops, all treatments relate to preventing the development of further complications in the mother--especially cardiovascular accidents, eclampsia, and accidental hemorrhage--and the assessment of fetal maturity to determine time and mode of delivery. Once the disease process is established, no form of therapy is available to reverse the process except delivery of the infant (10). Perinatal mortality approximates 10% in preeclampsia and 30% in eclampsia. Preeclampsia normally occurs after the 20th week of gestation but may develop before this time in the presence of trophoblastic disease. It is predominantly a disorder of the primigravida. Even with scrupulous prenatal care, the incidence is rarely less than 2.0-2.5% of delivered patients. Among mothers receiving little or no prenatal care and the socioeconomically deprived, the syndrome occurs in about 8-10% of patients (1).

Care must be taken in diagnosis of preeclampsia. Failure to recognize the onset of preeclampsia or misdiagnosis of the syndrome can occur if the major symptoms--edema, hypertension, and proteinuria--are not assessed properly. Edema of the lower extremities is considered to be a normal physiological process of pregnancy and should not be confused with the facial and hand swelling seen in preeclamptics. Venous capillary hydrostatic pressure in the lower extremities normally increases due to pressure on the inferior vena cava from an enlarging uterus, thereby causing normal development of edema in pregnancy (11).

Hypertension of preeclampsia has been defined as systolic blood pressure greater than or equal to 140 mm Hg or diastolic blood pressure greater than or equal to 90 mm Hg or both. A National Health Survey reports that 75% of women 18 to 34 years of age have blood pressure less than 120/80 and young teenagers have blood pressures of approximately 90/60 (11). Therefore, an increase in blood pressure of 20-30 mm systolic pressure or 10-15 mm diastolic pressure would provide a more conclusive diagnosis. Readings should be obtained on at least two occasions, six or more hours apart for a definite diagnosis.

Proteinuria is defined as the presence of urinary protein in concentrations greater than 0.3 g/l in a 24 hour

urine collection, or greater than 1 g/l (1+ or 2+ by standard turbidometric methods) in a random urine collection on two or more occasions at least six hours apart (3). The specimens must be clean, voided midstream, or obtained by catheterization.

If symptoms worsen--proteinuria of 5 g or more in 24 hours, blood pressure of 160/100 mm Hg or more, cerebral or visual disturbances, oliguria less than 400 ml in 24 hours, or hyperreflexia--aggressive therapy is initiated to prevent the seizures of eclampsia (3). If blood pressure continues to increase and other symptoms persist, delivery is normally induced, especially if the BUN is elevated. Fetal growth is poor when maternal BUN exceeds 25 mg% (1).

Etiology

In spite of research performed to this date, the cause of preeclampsia remains unknown. Many theories have been suggested to explain the syndrome.

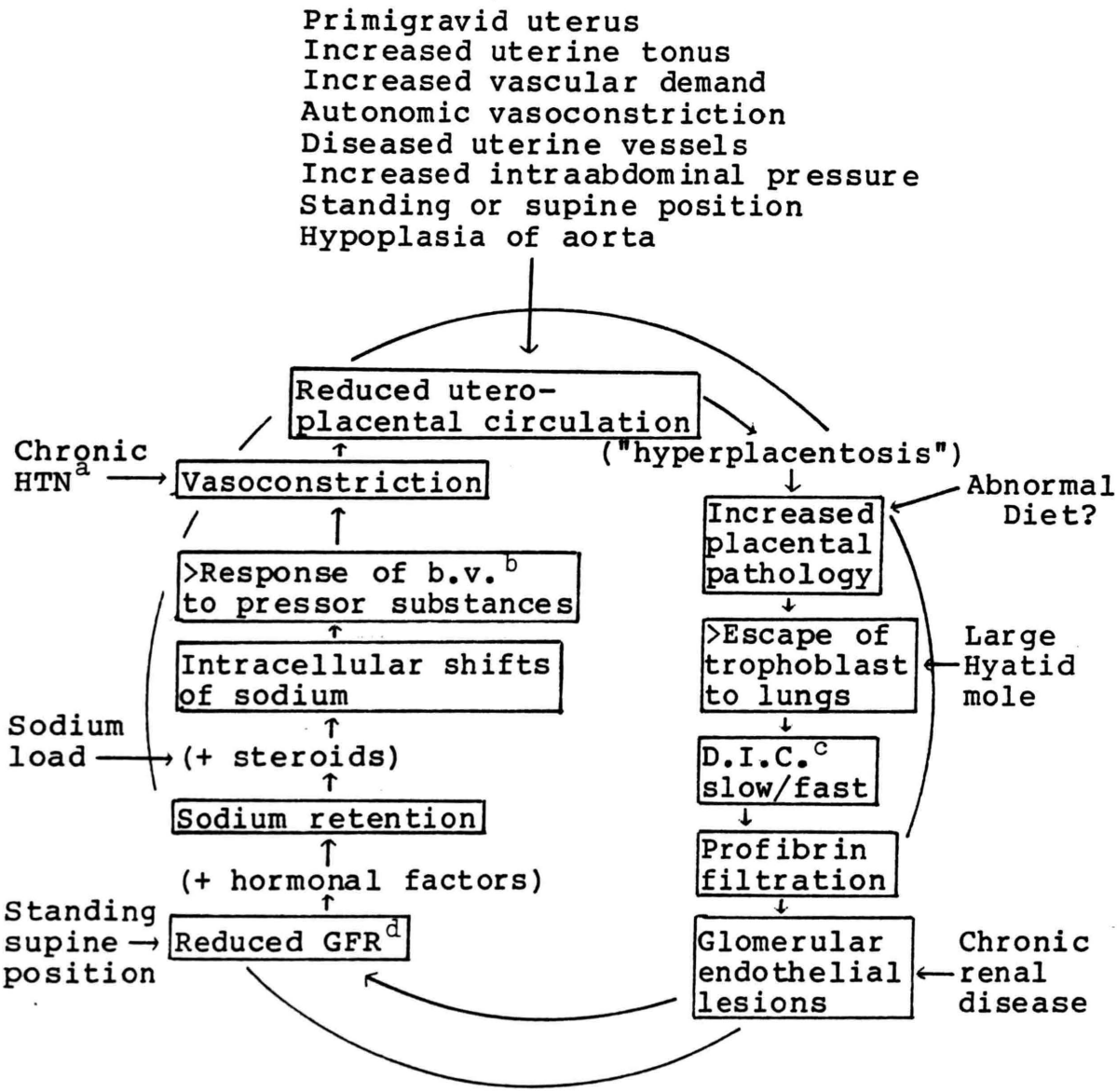
The uterine ischemia theory, first proposed in 1914, is probably the most widely accepted theory today. It states that preeclampsia is ultimately due to decreased blood supply through the myometrium resulting in stimulation of a vasoconstricting substance from the uterine contents (3). This substance is thought by some to be renin, which is produced by the uterus in the anephric human female (12,13).

Hodari offered experimental support of this premise by placing snug, but not constricting, Teflon bands around the uterine arteries of dogs before pregnancy (14). Once pregnant, the animals developed a relative uterine ischemia since the bands prevented normal pregnancies and provided for increased uterine artery size. Preeclampsia developed, characterized by hypertension, proteinuria, and hypernatremia.

Rogers speculates as to why this development occurs: The peripheral arteriolar vasoconstriction "leads to a decreased perfusion of blood and oxygen through vital organs and a concomitant elevation of blood pressure. The resultant hypoxia and edema of the major organ systems produce the classic syndrome of preeclampsia or eclampsia with: (1) cerebral edema and hypoxia which result in cerebral irritability and, ultimately, in convulsions; (2) decreased renal blood flow with arteriolar thickening and damage to the kidneys, producing albuminuria, sodium retention, and increased hypertension; (3) liver damage, secondary to tissue hypoxia, and possible vascular stasis; and (4) reduced placental perfusion. Hypertension and sodium retention may lead, ultimately, to pulmonary and generalized edema, cerebral hemorrhage, and cardiac, renal, or hepatic failure" (15).

Page further elaborates on the vicious circle of preeclampsia with its predisposing factors (Figure 1) (13). He defines the inner circle as "a chain of events which once initiated is self perpetuating, and persists until the circle is broken by the termination of the pregnancy". Page speculates that preeclampsia could be initiated at almost any point in the circle depending on the existing predisposing factors (outer circle).

Scott suggests a hypoimmune response as the causative agent in preeclampsia (16). He notes that after the first pregnancy or a blood transfusion, incidence of preeclampsia is reduced. A form of homograft (fetal) rejection is thought to predispose preeclampsia. Separate from the influence of blood transfusion, other antigenic stimuli such as exposure to sperm antigens through coitus may also have an effect (16). The very young primigravida, in which preeclampsia incidence is high, may have become pregnant from a sole act of intercourse. Marti found that primigravidas having little exposure to seminal fluid had significantly higher incidence of preeclampsia than those who had had many acts of intercourse without barrier contraception (17). Other studies, however, have failed to detect circulating immune (antigen-antibody) complexes related to the pathogenesis of preeclampsia (18).



[a] hypertensive disease
[b] blood vessels
[c] disseminated intravascular coagulation
[d] glomerular filtration rate

Figure 1. THEORETICAL VIEW OF ETIOLOGY OF PREECLAMPSIA, INCLUDING VICIOUS CIRCLE OF SYMPTOMS THAT FOLLOW (13)

There is considerable disagreement regarding whether or not diet plays a role in the etiology of preeclampsia. One can speculate that the more children a woman has, the less favorable her nutritional status would become. Often impoverished women are those who have pregnancies in rapid succession. If malnutrition was a predisposing factor in preeclampsia, one would expect to see an increasing incidence with each rise in parity (19). However, preeclampsia is primarily a disease of primigravidas, and is usually not seen in later pregnancies without predisposing factors such as multiple gestation, diabetes, or preexisting hypertension. Well-nourished women do develop preeclampsia, but they are more likely to receive early detection through prenatal care with subsequent hospital treatment (16).

Williams et al. could find no relationship between dietary protein levels and preeclampsia in 32 preeclamptics and 25 non-preeclamptics (4). Their study reported that women with preeclampsia tended to consume greater levels of protein and essential amino acids than controls, based on dietary recalls. Nevertheless, other researchers have documented a reduced incidence in the preeclampsia syndrome as nutritional status improves (20,21,22).

Physiologic alterations

Several physiological systems are involved in the pathology of preeclampsia, thereby complicating the etiology of the syndrome even further. Researchers cannot agree as to the physiological process occurring in preeclamptics, which further hinders a solution to preventing the syndrome.

Blood

Premature separation or breakdown of the placenta produces intravascular coagulation due to the escape from the uterus of trophoblastic fragments which travel to and are lysed by the lungs, their contents entering the greater maternal circulation. Trophoblast tissue has a higher thromboplastic activity per gram than any other body tissue (13). These molecules affect distant organs, which partly explains one of the major mechanisms involved in the necrosis of the brain, pituitary, kidney, lung, and placenta. If abruptio placenta occurs, the intravascular coagulation may be of such magnitude as to result in a hemorrhagic predisposition, which is potentially fatal (3).

The presence of profibrin in the plasma results from a slow, chronic disseminated intravascular coagulation (DIC) (13). Chatterjee et al. found plasma fibrinogen levels increased by 70% in preeclamptics and 145% in eclamptics,

while levels noted in patients with essential hypertension were roughly the same as normal pregnancy values (23). Whigham et al. found that platelets from patients with severe preeclampsia were less responsive than normal to a variety of aggregating agents, especially collagen and vasopressin (24). The authors felt that the platelets may have undergone aggregation followed by disaggregation within the circulation, leading ultimately to decreased clotting function. Crandon noted a decreased incidence of preeclampsia (4% compared to 12.3%) in women with a history of taking aspirin or aspirin-containing compounds compared to women with no such history (25). Aspirin inhibits platelet formation and is postulated to be responsible for the decreased incidence of preeclampsia.

Brain

Varying degrees of cerebral edema are found in postmortem studies of nonconvulsive preeclamptics, while edema and cerebral hemorrhages are common in patients dying with convulsions. The cause and effect relationship of hemorrhage to convulsions remains unclear--hemorrhaging may result from severe or prolonged cerebral hypoxia. Cerebral cortex cells are probably protected from seizure-inducing afferent stimuli by an enzymatic or metabolic block in the extracellular fluid compartment of the brain. In the

convulsions of eclampsia, metabolic changes and fluid and electrolyte imbalance may render this protective mechanism ineffective. The absence of permanent neurologic problems following uncomplicated convulsive eclampsia further supports this concept as a causitive agent in convulsions.

Kidney

Preeclampsia involves unique glomerular changes--capillaries swell due to enlargement of endothelial cells and deposits of amorphous material in their cytoplasm. When the capillary tuft becomes enlarged, the narrowing lumen of its vessels produce ischemia, interstitial cells between the capillaries proliferate, and the glomerulus becomes overcrowded with capillary tufts. Decreased glomerular blood flow and glomerular filtration rate then follow, with the renal tubules generally showing nonspecific abnormalities secondary to ischemia and proteinaceous material within the tubular lumen. In addition, interstitial edema is usually seen. Repair of the lesions normally occurs rapidly and completely after delivery, but a few patients may develop permanent vascular glomerular damage. This generally occurs only if preeclampsia is superimposed upon hypertensive vascular disease (3).

Liver

Characteristic changes of the liver include hemorrhage and necrosis beginning in the periportal areas and extending peripherally from these areas to involve the liver lobule. Fibrin thrombi associated with extensive thrombosis are found in the arterioles, capillaries, and smaller branches of the portal vein. These lesions may be transient. In rare cases, acute distention and rupture of the liver may occur. While the renal changes are quite variable, the liver exhibits regenerative capacity by seven days postpartum, which makes experimental studies of the changes in hepatic function in preeclampsia difficult.

Heart

Researchers have had problems correlating acute congestive heart failure of preeclampsia with the minimal histopathologic lesions in the myocardium. Primary cardiac failure is a less likely explanation for decompensation than the combination of hypovolemia, contracted intravascular compartment, hemoconcentration, decreased cardiac return, tachycardia, and the autotransfusion occurring with the evacuation of the uterine contents.

Placenta

Placental infarcts of varying size and distribution, larger and greater in number than those found in 60% of normal pregnant patients, are present in the preeclamptic. The red and more recent infarcts are thought to be due to vascular obstruction with subsequent necrosis, while white infarcts are associated with intervillous deposits of fibrin. When a large laminated clot forms beneath the endometrium, the clot may become extensive, which can lead to premature placental separation. Histologic changes in the placenta are suggestive of premature aging.

Retina

Temporary or permanent maternal blindness has been reported in 1-3% of eclamptics (26,27), but is rare in preeclamptics (28). In some eclamptics, there are no evident retinal alterations, while others demonstrate narrowing and focal constricton of the arterioles, resulting in a decrease in the arterio-venous ratio. The vasospastic phenomena and retinal edema may be so severe as to result in retinal detachment. Once the preeclamptic process has stopped, there is normally no demonstrable compromise of visual acuity.

Lung

Pulmonary edema and severe diffuse hemorrhagic bronchial pneumonia is extremely common in patients dying of eclampsia. In preeclamptic patients, intravascular coagulation, fibrin deposition, and hemorrhage into the aveolar spaces (resembling endotoxin shock) are found. Preeclampsia pulmonary edema may be induced by overzealous intravenous infusions of even small amounts of fluids.

Treatment

The basic treatment for preeclampsia is delivery. After 37 weeks of gestation, delivery is normally indicated regardless of severity of preeclampsia. If severe preeclampsia develops at any time, delivery is indicated regardless of fetal age, since it is futile and hazardous to postpone delivery in the hope of progression to fetal maturity. The hazards of prematurity to the infant are much less than those of preeclampsia and its sequelae.

Bedrest at home in the lateral recumbant position is usually sufficient for the management of insipient, but not developed, preeclampsia (characterized by edema and blood pressure increases of less than 30 mm Hg systolic or 15 mm Hg diastolic). Phenobarbitol administered three times a day is used to limit activity. Bedrest alone produces a

measurable increase in glomerular filtration and the resulting diuresis can mobilize considerable amounts of tissue fluid.

Salt restriction and diuretics were used for years as the cornerstone to therapy for insipient or mild preeclampsia. Evidence now demonstrates that not only are such treatments ineffective but they also may be harmful by disrupting optimal electrolyte patterns, causing purine metabolite accumulation, impairing production of placental estrogens, and by predisposing to postpartum vasomotor collapse (3). The normal salt intake (usually 4-6 g/day) can be continued without harm (3), but preeclampsia may be worsened by excessive salt intake; therefore, high sodium foods should be avoided.

With diuretic therapy, the kidneys excrete water and electrolytes. This effect produces reduction in circulating blood volume. Preeclamptics normally show hypovolemia in contrast to the gradually increasing blood volumes seen in normal gravidas (7,29). Maclean found six patients with severe preeclampsia to have hypovolemia despite the presence of edema (7). Treatment with intravenous saline and albumin to obtain normal central venous pressure resulted in improved renal function.

The role of hypovolemia in preeclampsia is uncertain, but its appearance may precede the development of

hypertension. With a decreased blood volume, cardiac output and diffusion through peripheral tissues can only be maintained by the increased blood pressure seen in the preeclamptic state. A further decrease in blood volume only serves to increase blood pressure further and reduce circulation of placenta, brain, kidney, and liver (7).

The preeclamptic patient potentially could benefit from a restoration of normal blood volume. The use of human albumin preparations, which expand plasma volume by osmotic effect, shows promise (7). The use of saline in patients with gross edema seems contradictory but does allow for rapid volume expansion. Excess sodium can normally be excreted.

Magnesium sulfate is a cerebral depressant which reduces neuromuscular excitability (3). It is used to prevent seizures in severe preeclampsia and eclampsia. It also acts peripherally to improve uterine blood supply and to stimulate vasodilation and a decrease in blood pressure (1). Since calcium and magnesium are mutually antagonistic, magnesium acts principally by antagonizing the action of calcium at myoneural junctions so that the nerve impulse cannot occur (30). After magnesium sulfate therapy, increased parathyroid hormone and decreased calcitonin in maternal serum indicate the body's attempts to conserve calcium (30). Magnesium toxicity can cause myocardial

infarction in the mother and poor ventilation for the newborn (1,31,32). However, the drug is effective in preventing seizures and the risks involved to the fetus can be minimized.

Sedatives are important for enforcing bedrest, and phenobarbitol is the standard agent (3). In eclampsia and certain cases of severe preeclampsia, a heavy sedation with morphine may be necessary. It should be used carefully if delivery is imminent within the next six hours, since the infant will be heavily sedated.

Antihypertensives have been studied extensively in treatment of high blood pressure prenatally and at labor and delivery. They were once thought to have great promise but it is realized now that they do not effect the underlying cause of the disease (3). They are still utilized, however, to prevent damage from hypertension. Regimens for the different stages and degrees of preeclampsia are presented in Appendix A.

Dietary relationships in preeclampsia

Malnutrition

De Alvarez notes that dietary deficiencies or severe malnutrition have been associated with a higher incidence of preeclampsia-eclampsia (2). He includes protein

deficiencies and possibly water soluble vitamin deficiencies, but notes that rigorous proof for specific deficiencies is poorly documented. In addition, a low socioeconomic status is highly correlated with incidence of the syndrome.

Zinc

Cherry et al. found significantly lower plasma zinc levels in pregnant women experiencing hypertension of preeclampsia (9). They suggested that the relationship of poor nutrition to preeclampsia could be caused by a zinc deficiency and speculated on the need for zinc supplementation during pregnancy.

Iron

Of all the nutritional deficiencies in pregnancy, anemia is the most common (8). Chaudhuri reports a significant increase in incidence of preeclampsia in anemic subjects (8). He states that anemia, especially that due to iron deficiency, "is one of the most important factors in the etiology of (preeclampsia), though not the sole cause of it". Chaudhuri speculates that anemia may cause edema by lowering the osmotic pressure of blood, producing tissue anoxemia, and by capillary damage. Proteinuria is produced by anoxemia damage to the glomerules. Hypertension is

affected through defective oxygenation of the kidneys and/or the placenta, which may lead to accumulation of high amounts of vasopressor substances either due to increased formation or defective inactivation.

Sodium

For the past three decades, sodium restriction has been part of the medical protocol for pregnant patients. It was felt that:

1. Pregnant women often exhibit symptoms of edema. Sodium is the key ion in water balance; therefore, during pregnancy the patient is not able to manage sodium properly.
2. Aggravation of preeclamptic symptoms are demonstrated upon saline injection (27).
3. Non-pregnant patients ingesting large amounts of salt eventually tend to develop essential hypertension more than those people on normal sodium intakes.
4. In treating hypertensives, blood pressure falls significantly following decreased dietary sodium.

Today researchers realize that normal pregnant women actually have problems retaining enough sodium for a normal pregnancy. Pregnancy calls for a 50% volume increase in maternal intercellular and intravascular fluid compartments.

The pregnant woman must retain approximately 19,500 mg of sodium to retain the extra fluid. To accomplish this during pregnancy, chorionic gonadotropin is secreted following fertilization which stimulates the corpus luteum to synthesize progesterone and estrogen (33). The progesterone increases sodium in the urine by a decrease of tubular reabsorption of sodium and a decrease in arteriolar tone triggered by baroreceptors in the kidney. With this increased sodium in the urine, hypovolemia and hyponatremia naturally follow. The potentially severe consequences of sodium loss do not occur due to a compensatory mechanism. Excess urinary sodium is detected by the juxtaglomerular apparatus in the kidney, and renin is secreted and converted to Angiotensin I, which converts to Angiotensin II (a potent vasoconstrictor normally, but not in pregnancy). Angiotensin II stimulates the adrenal gland to secrete aldosterone, which produces increased tubular reabsorption of sodium and the stimulation of antidiuretic hormone (ADH) secretion, which results in water retention.

With a sodium restriction, the normal renin-angiotensin-aldosterone mechanism works harder to ensure positive sodium balance. This system may be stressed excessively in preeclamptic patients on a sodium restriction.

Carbohydrate

Reports of carbohydrate metabolism in preeclampsia suggest that glucose homeostasis is impaired, but the relationship is uncertain (34,35,36,37). In normal pregnancies, fasting blood sugar levels are lower than in non-pregnant women (38). Patients with mild preeclampsia were found to have levels lower than normal pregnant patients, but the difference was not significant (5). In severe preeclamptics, fasting plasma glucose levels were found to be significantly decreased. An abnormality of pancreatic beta cell function may be attributable to the anoxia subsequent to vascular changes in preeclampsia. As many as one third of patients without preeclampsia, but with abnormal glucose tolerance tests, develop diabetes mellitus within fifteen years postpartum (39). A retrospective study of severe preeclamptics at 10-20 years postpartum revealed no significant difference in abnormal glucose indices compared with patients with no history of preeclampsia (40).

Fat

Little has been reported relating fat consumption to preeclampsia. Chung et al. suggested that if fat consumption and preeclampsia were interrelated, quantity rather than quality of fatty acid would be the important

variable (6). Nevertheless, he was unable to correlate either parameter with incidence of preeclampsia.

Protein

Characteristic clinical findings in preeclampsia and eclampsia include hypoalbuminemia and hypovolemia (41,42,43,44) with subsequent hemoconcentration often masking lowered total plasma albumin. Reports confirm that dietary protein deficiency leads to hypoalbuminemia (45), which lowers the colloid osmotic pressure of the intravascular compartment. In preeclampsia, significant decreases in serum albumin and globulin contents in close relationship to severity of the disease have been found in both maternal and neonatal blood (22). One could speculate that a decrease in dietary protein may somehow stimulate development of preeclampsia but such has not been determined. Human serum albumin infusion has shown promise in combating the hypovolemia and hemoconcentration of severe preeclampsia (45).

Other research has not borne out the relationship between protein intake and preeclampsia. Williams et al. found average intakes of protein and the essential amino acids to be significantly greater for preeclamptics compared to controls, when evaluating 24-hour recalls obtained bimonthly in the third trimester (4). Total protein intake

for both groups was greater than 100% of the 1980 Recommended Dietary Allowance (RDA). This study suggested that women could develop preeclampsia even if they consumed more than adequate quantities of essential amino acids and protein.

WIC program

Congress in 1972 passed legislation creating the WIC program for women, infants, and children who were potentially at nutritional risk for optimum growth. In Texas, until August, 1981, eligibility was not determined by income but rather by health status. Pregnant women were eligible to receive eggs, milk, cereal, juice, and cheese if they were underweight or overweight at conception, failed to gain weight properly, were anemic, had a poor obstetrical history, exhibited inadequate dietary patterns (see Appendix B), or were under 18 or over 35 years of age at conception. The women received cards monthly until six weeks postpartum. Program sites were located in low income public health clinics; therefore, the lower socioeconomic sector of the population was normally served. Women learned of the WIC program by medical referral while in the clinic or through literature or word of mouth.

Nineteen WIC projects in fourteen states participated in a study to evaluate nutritional benefit of the WIC

program (46). For pregnant women, researchers noted an increase in the consumption of protein, calcium, phosphorus, iron, vitamin A, thiamin, riboflavin, niacin, vitamin C, and folacin, but not of energy. A greater weight gain was seen in WIC participants compared to controls. WIC participants delivered babies whose mean birthweights had increased; however, WIC supplementation for a period less than three months had no effect on birthweight. Gestational duration was approximately five to six days longer for women who received food supplements for more than six months, compared to women receiving supplements less than three months. With longer gestational periods, infants had more time to gain weight by birth.

A second study assessed the effect of the WIC program on 907 pregnant women compared to 463 non-WIC participants. Part of the non-WIC group was from the same clinic as the WIC participants and part was from a different health facility (47). Birthweights of infants born to WIC mothers were significantly higher than those of infants born to non-WIC mothers. The incidence of low birthweight was 6.0% lower in the WIC group than the non-WIC group from the same clinic. The authors estimated that for every dollar spent on WIC, 3.1 dollars were saved in costs for medical care of low birthweight infants.

Although undetermined nutritional deficiencies may be related to onset of preeclampsia, theories as to the cause do not seem to implicate nutritional inadequacies. If a short-term improved nutritional status via the WIC program did reduce the incidence of preeclampsia, the need for further investigation into dietary relationships to the syndrome would be evident and the importance of the WIC program in prenatal care would be established.

HYPOTHESES

The null hypotheses are:

1. There will be no significant difference ($p<.05$) in blood pressure throughout pregnancy in supplemented versus non-supplemented mothers.
2. There will be no significant difference ($p<.05$) in incidence of edema in supplemented versus non-supplemented mothers.
3. There will be no significant difference ($p<.05$) in incidence of proteinuria in supplemented versus non-supplemented mothers.
4. There will be no significant difference ($p<.05$) in incidence of preeclampsia in supplemented versus non-supplemented mothers.

METHODOLOGY

All subjects of this study were maternity patients at Riverside Health Center, Houston, Texas between May, 1979 and February, 1980. Each subject was prescreened by a nurse at every clinic visit to determine problems the patient may have been experiencing with her pregnancy. If the patient complained of facial, pedal, or other swelling, positive incidence of edema was documented in the patient's medical chart. Blood pressure and weight were recorded and a urine sample collected to determine protein and glucose content, if any. At the first visit only unless problems developed, blood samples were obtained for venereal disease, hematocrit, and rubella tests. After the initial screening, the patient was seen by a doctor, who would refer patients with nutrition-related problems to the nutritionist.

Three hundred and thirteen WIC and 191 non-WIC pregnant subjects were selected for study during a time period when the author was employed at the clinic as nutritionist. Differences in patient evaluation for WIC enrollment, methods of nutritional counseling, and types of education were therefore minimized, since the author was responsible for all screenings before WIC enrollment.

Subjects who visited the nutritionist but were not enrolled in the WIC program were not included in this study. All control subjects had had no contact with the clinic nutritionist during their pregnancies and therefore were not screened for WIC program eligibility, did not receive individual nutrition counseling and did not attend WIC group education.

To establish eligibility for the WIC program, the patient's medical record was evaluated by the nutritionist. A 24-hour recall and diet history were obtained and documented by S.O.A.P. (subjective, objective, assessment, plan) criteria. WIC eligibility was then determined (see Appendix C). Reasons for the patient's WIC eligibility were discussed, and the patient signed a governmental form agreeing to rules and regulations of the WIC program. The woman was told that the foods were for her consumption only.

The patient received counseling regarding appropriate food choices based on six food categories: milk and milk products, meat and meat substitutes, vegetables, fruits, breads, cereals and starchy foods, and fats and oils. Appropriate counseling was given for nutrition-related problems such as rapid weight gain, weight loss, nausea, and anemia. Clerical staff then completed paperwork for WIC enrollment and issued cards to be redeemed for WIC foods.

Each month, including the first, the patient received food coupons for 4 1/2 gallons of milk (whole, low fat, or skim), two pounds of cheese (cheddar, swiss, longhorn, colby, American, or Monterrey Jack only), four 13-ounce cans evaporated milk, 276 fluid ounces of vitamin C-fortified unsweetened fruit juice (orange, grapefruit, orange-grapefruit, pineapple, orange-pineapple, grape or apple), 36 ounces of highly fortified, unsweetened cereal, and two dozen Grade A large eggs. Authorized grocery stores would redeem the coupons for the designated foods.

The certification period for the program continued until six weeks past the delivery date. Before receiving food cards each month, the patient was given the opportunity to receive nutritional education in the form of an informal class led by the nutritionist, a film, or a short discussion followed by a true-false quiz to determine retention. If the patient refused nutrition education, she signed a release form to fulfill government regulations. Even if a release form was signed, a true-false quiz was administered to ensure some form of nutrition education. Each month the subject for nutrition education was based on a nutrient of WIC foods (iron, vitamin C, protein, calcium) or a concept to promote good nutrition for pregnancy (basic four food groups, breastfeeding, infant feeding, food cost).

To collect data, all medical records of pregnant patients, whose first visit to the clinic fell between May, 1979 and February, 1980, were reviewed. To ensure confidentiality and avoid use of patient names, each subject was given a random number for identification purposes. Subjects were selected who

1. first visited the clinic before the 30th week of pregnancy
2. kept at least two clinic appointments
3. kept at least one clinic appointment after the 35th week of pregnancy and
4. were enrolled in WIC before the 30th week of pregnancy (if a WIC subject).

When a subject conformed to these criteria, a random number was assigned and the following were documented:

1. WIC status
2. if a WIC subject, date enrolled and weeks pregnant at enrollment
3. date enrolled in clinic
4. weeks pregnant when enrolled in clinic
5. prepregnancy weight
6. last weight recorded from 34-37 weeks
7. last weight recorded after 37 weeks
8. race
9. gravidity

10. delivery date
11. initial hematocrit
12. hematocrit at six weeks postpartum
13. edema, incidence and severity
14. proteinuria, incidence and severity
15. blood pressure readings for all clinic visits
16. infant birthweight
17. infant Apgar score.

If a patient had enrolled in the WIC program before the 30th week of her pregnancy, she was labelled a "WIC" patient. If the patient had never been enrolled in the program, she was labelled a "non-WIC" patient.

To analyze the data, edema, proteinuria, and blood pressures were separated by occurrence into first, second, and third trimesters. The number of patient complaints of edema were totalled and analyzed by trimester. The proteinuria results were coded as to severity (negative = 0, trace = 1, +1 = 2, +2 = 3, +3 = 4). The scores for each trimester were added together to determine the sum for that trimester.

One way analyses of variance were utilized to determine separate effects of WIC status, race, gravidity, hematocrit, and time of WIC and clinic enrollment on both infant outcome (birthweight and Apgar scores) and separate indicators of maternal preeclampsia (blood pressure, edema, proteinuria).

Preeclampsia was defined by the author as any incidence of systolic blood pressure greater than 140 mm Hg or any diastolic blood pressure greater than 90 mm Hg combined with any incidence of edema or any incidence of proteinuria. Each variable was analyzed to determine effects on incidence of preeclampsia as defined. In addition, effects of anemia on preeclampsia were examined as well as effects of preeclampsia on incidence of low birthweight infants.

RESULTS AND DISCUSSION

According to the selection criteria for preeclamptics above, 10.5% of the population studied exhibited symptomology of the syndrome. This would be the expected incidence, indicating that the selection criteria for preeclamptics in this study could be a useful clinical measure. Cross tabulations analysis showed that 9.9% of WIC and 11.5% of non-WIC patients had symptoms of preeclampsia by time of delivery (Table 1). This difference was not significant, although a trend emerged, indicating the need for further investigation. After determining that short-term nutritional supplementation did not have significant effect upon development of preeclampsia, analyses were made to determine what effects other recorded variables may have had on development of preeclampsia or infant outcome.

The purpose of creating optimal maternal conditions is to produce an infant of maximal health. Two of the most commonly used indicators of infant health are birthweight and Apgar score. Overall infant outcome was not significantly affected by WIC participation (Table 2),

TABLE 1

Incidence of Preeclampsia in WIC and Non-WIC Groups and in the Population as a Whole

	Preeclampsia	Without Preeclampsia
WIC, % (no.)	9.9% (31)	90.1% (282)
Non-WIC, % (no.)	11.5 (22)	88.5 (169)
Total, % (no.)	10.5 (53)	89.5 (451)
F-ratio	0.327	
Degrees of freedom	1. 502	
F-probability	0.5675	

TABLE 2

Effect of WIC Status on Infant Outcome

	Birthweight (g) (Mean ± SD)	Apgar Score (Mean ± SD)
WIC	3120.4 ± 537.4	17.3 ± 1.5
Non-WIC	3164.1 ± 585.5	17.8 ± 1.6
F. ratio	0.438	1.129
Degrees of Freedom	1, 310	1, 49
F. probability	0.5084	0.2932

although 8.8% of WIC infants were of low birthweight compared to 13.0% of non-WIC infants (Table 3). Maternal nutritional health is a function of long-standing eating habits, and results herein suggest that a "nutritional boost" after conception may be inadequate to produce significant effects on measurable infant health parameters.

TABLE 3

Incidence of Low Birthweight Infants in WIC and Non-WIC Groups

	Birthweight	
	<2500 g	>2500 g
WIC, % (no.)	8.8% (18)	91.2% (186)
Non-WIC, % (no.)	13.0% (14)	87.0% (94)
Total, % (no.)	10.3% (32)	89.7% (280)

Infants born to black mothers were of significantly lower weight at birth (Table 4). One might expect differences due to race to disappear when socioeconomic status was controlled for, as in this study, but such was not the case.

TABLE 4

Effect of Race on Infant Outcome in the Population as a Whole

	Birthweight (g) (Mean ± SD)	Apgar Score (Mean ± SD)
Black	3109.2 ± 559.2 (285 cases)	17.4 ± 1.6 (44 cases)
White	3460.1 ± 441.4 (15 cases)	18.5 ± 0.6 (4 cases)
Hispanic	3444.9 ± 384.2 (9 cases)	17.0 (1 case)
F. ratio	4.354	0.867
Degrees of freedom	2, 306	2, 46
F. probability	0.0137	0.4269

Results in Table 5 indicate that gravidity did not significantly affect Apgar scores or infant birthweight. As gravidity increases, one would suspect that maternal nutritional status would decline, especially in the lower socioeconomic groups. However, if maternal nutritional status did decline with increasing gravidity in this study, such a decline did not affect measured infant health indicators herein. Although gravidity did not significantly affect infant birthweight, there was a slight correlation between these variables, suggesting the need for further investigation. WIC and non-WIC groups did not differ in gravidity (Table 6) and race had no effect on number of pregnancies (Table 7).

Indicators of preeclampsia as defined in this study had no effect on infant outcome (Tables 8 and 9), although larger group size would be necessary to accurately evaluate correlations. Patients complained of edema an average of 2.6 times in the third trimester, while the mean score for proteinuria was 2.2 (based on the method described previously). Preeclampsia did not significantly affect infant birthweight ($p = 0.1507$) or Apgar score ($p = 0.0886$). Each symptom of preeclampsia was correlated separately with both parameters of infant outcome. Perhaps tighter correlations were not found because one symptom alone does not define preeclampsia, but must be coupled with another to

TABLE 5

Effect of Gravidity on Infant Outcome
in the Population as a Whole

	Birthweight (Mean ± SD)	Apgar Score (Mean ± SD)
Gravidity		
1	3087.8 ± 532.3 (172 cases)	17.5 ± 1.3 (25 cases)
2	3154.1 ± 565.3 (76 cases)	17.5 ± 1.9 (15 cases)
3	3270.2 ± 653.1 (35 cases)	18.2 ± 0.9 (4 cases)
4	3299.8 ± 460.5 (12 cases)	16.0 (1 case)
5	3058.1 ± 542.4 (11 cases)	17.0 ± 2.6 (3 cases)
6	3900.0 (1 case)	18.0 (1 case)
F-ratio	1.324	0.418
Degrees of freedom	5, 301	5, 43
F-probability	0.2539	0.8335

TABLE 6

Gravidity of WIC and Non-WIC Groups and of Population as a Whole

	WIC	Non-WIC	Total
Gravidity			
Mean ± SD	1.97 ± 1.4	2.0 ± 1.3	1.98 ± 1.4
Percentage reported	99.4	98.4	99.0
Total pregnancies			
1	52.6%	47.9%	50.2%
2	23.7	25.0	24.4
3	12.3	16.0	14.2
4	4.5	5.3	4.9
5	3.6	3.7	3.6
6	1.0	1.1	1.1
7	1.6	0.5	1.1
8	0.3	0.0	0.3
9	0.3	0.5	0.4

TABLE 7

Correlation of Race to Gravidity
in the Population as a Whole

	Gravidity Mean ± SD (no.)
Race	
Black	1.95 ± 1.3 (433)
White	1.78 ± 1.3 (27)
Hispanic	2.31 ± 1.7 (26)
F-ratio	1.113
Degrees of freedom	2, 483
F-probability	0.3294

TABLE 8

Effect of Third Trimester Edema on Infant Outcome in the Population as a Whole

	Birthweight Mean ± SD (no.)	Apgar Score Mean ± SD (no.)
Incidence of Edema		
1	3112.1 ± 376.8 (58)	16.9 ± 2.1 (13)
2	3094.4 ± 616.1 (39)	17.0 ± 1.9 (7)
3	3225.6 ± 654.1 (31)	17.3 ± 0.7 (7)
4	3237.4 ± 607.6 (19)	18.0 ± 1.4 (2)
5	3493.1 ± 643.2 (11)	
6	3073.6 ± 563.2 (8)	
7	3341.4 ± 438.4 (7)	18.0 ± 1.4 (2)
9	2733.0 (1)	
F-ratio	1.063	0.303
Degrees of freedom	7, 166	4, 26
F-prob.	0.3898	0.8731

TABLE 9

Effect of Third Trimester Proteinuria on Infant Outcome in the Population as a Whole

	Birthweight Mean ± SD (no.)	Apgar Score Mean ± SD (no.)
Incidence of Proteinuria		
1	3083.0 ± 451.5 (21)	18.0 ± 1.0 (3)
2	3332.6 ± 493.4 (21)	19.0 (1)
3	3177.6 ± 534.1 (8)	18.0 (1)
4	3116.0 ± 678.5 (6)	18.0 (1)
5	1762.5 ± 1043.0 (2)	
9	3050.0 ± 565.7 (2)	
F-ratio	3.445	0.278
Degree of freedom	5, 54	3, 2
F-prob.	0.0090*	0.8405

*Too few cases to establish significance

confirm diagnosis. Edema had no effect on proteinuria or systolic blood pressure, indicating that one symptom of preeclampsia does not cause the others (Table 10). As stated previously, two or more symptoms occur simultaneously in preeclampsia.

Participation in the WIC program had no effect on individual indicators of maternal preeclampsia (Table 11). Participants either became enrolled in WIC during the second trimester (85.9%) or third trimester (14.1%) of pregnancy. Symptoms of preeclampsia normally are seen after the 20th week of gestation. One could assume that the actual beginning of the disease occurs sometime earlier than that. Once preeclampsia occurs, development of the syndrome continues unless the infant is delivered. This study found that women enrolled in the WIC program in the second trimester exhibited no significant difference in incidence of preeclampsia over those enrolled in the third trimester. This could be attributed to the supposition that the predisposing factors of preeclampsia were already developing before WIC enrollment. In addition, one cannot assume that a nutritional inadequacy would be overcome immediately upon WIC supplementation. Had WIC foods been given for a period of several months prior to conception, the benefits of an improved nutritional status possibly would have been more measurable. If preeclampsia was nutrition-induced,

TABLE 10

Effect of Third Trimester Edema on Indicators of Maternal Preeclampsia in the Population as a Whole

	Systolic BP Mean ± SD	Diastolic BP Mean ± SD	Proteinuria Mean ± SD
Incidence of Edema			
1	109.7 ± 12.0	66.9 ± 8.6	2.1 ± 1.9
2	112.7 ± 9.8	69.8 ± 9.2	2.1 ± 1.1
3	111.9 ± 10.0	69.3 ± 7.4	2.5 ± 1.3
4	109.8 ± 11.3	69.0 ± 8.1	3.4 ± 3.4
5	109.9 ± 6.7	67.6 ± 5.3	2.2 ± 1.2
6	102.8 ± 3.5	63.9 ± 2.4	2.3 ± 0.6
7	110.6 ± 8.5	68.6 ± 7.2	2.0
8	103.1	67.4	
9	118.1 ± 9.6	69.5 ± 8.5	1.5 ± 0.7
Total	110.6 ± 9.8	68.3 ± 7.5	2.3 ± 1.7
F-ratio	0.914	0.383	0.419
Degrees of freedom	8, 67	8, 67	7, 46
F-prob.	0.5107	0.9259	0.8852

TABLE 11

Effect of WIC Status on Third Trimester Indicators of Maternal Preeclampsia

	Systolic BP Mean ± SD (no.)	Diastolic BP Mean ± SD (no.)	Edema Mean ± SD (no.)	Proteinuria Mean ± SD (no.)
WIC	109.8 ± 9.7 (75)	69.7 ± 21.5 (76)	2.5 ± 1.6 (171)	2.3 ± 1.4 (56)
Non-WIC	109.3 ± 9.1 (39)	66.7 ± 7.3 (39)	2.9 ± 2.0 (111)	2.0 ± 1.7 (35)
F-ratio	0.069	0.677	2.858	1.296
Degrees of freedom	1, 112	1, 113	1, 280	1, 89
F-prob.	0.7938	0.4124	0.0920	0.2580

induction probably occurred before WIC foods were implemented.

Worthington-Roberts et al. (11) speculates that development of preeclampsia is not dependent on race, and the results of this study support this premise (Table 12). Since the population studied herein was overwhelmingly black (Table 13), the results in Table 12 are not uniquely conclusive. A larger percentage of black patients were WIC subjects; however, all other races showed a greater percentage of subjects in the non-WIC group. Knowledge of the WIC program was largely due to patient word of mouth, and one could assume that the people of each racial group would more than likely associate with each other. Since such a large percentage of the population was black, this would tend to "dilute" the other races in clinic interactions; therefore causing fewer people of the other racial groups to learn of WIC.

Gravidity had no effect on isolated symptoms of preeclampsia (Table 14) or on incidence of the syndrome as defined in this paper. Preeclampsia is a disease of the primagravida; however, no trend could be discerned from this study to support such a premise.

TABLE 12

Effect of Race on Third Trimester Indicators of Maternal Preeclampsia in the Population as a Whole

	Systolic BP Mean ± SD (no.)	Edema Mean ± SD (no.)	Proteinuria Mean ± SD (no.)
Race			
Black	109.3 ± 9.7 (97)	2.6 ± 1.7 (231)	2.2 ± 1.4 (83)
White	113.2 ± 7.8 (8)	3.6 ± 2.4 (23)	1.5 ± 0.7 (2)
Hispanic	108.9 ± 8.1 (7)	3.2 ± 2.2 (14)	2.6 ± 1.1 (3)
F-ratio	0.643	4.129	0.392
Degrees of freedom	2, 109	2, 265	2, 85
F-prob.	0.5277	0.0171*	0.6772

*Non-parametric test failed to substantiate significance ($p = 0.071$)

TABLE 13

Racial Parameters of WIC and Non-WIC Subjects

	WIC	Non-WIC	Total
Black	91.2%	80.9%	86.1%
White	2.9	9.6	6.3
Hispanic	4.9	5.9	5.4
Asian	0.3	1.6	0.9
Indian	0.6	2.1	1.3

TABLE 14

Effect of Gravidity on Third Trimester Indicators of Maternal Preeclampsia in the Population as a Whole

	Systolic BP	Edema	Proteinuria
	Mean ± SD (no.)		
Number of Pregnancies			
1	109.8 ± 9.4 (68)	2.8 ± 1.9 (142)	2.1 ± 1.2 (55)
2	109.2 ± 10.7 (19)	2.4 ± 1.6 (66)	2.8 ± 2.1 (18)
3	109.6 ± 8.4 (14)	2.6 ± 1.5 (40)	1.8 ± 1.0 (10)
4	106.7 ± 8.2 (5)	3.0 ± 2.0 (14)	2.2 ± 1.0 (4)
5	110.2 ± 13.0 (6)	2.6 ± 2.0 (10)	2.0 (1)
6	106.0 (1)	2.0 ± 1.4 (4)	
7		1.0 ± 0.0 (2)	
Total	109.5 ± 9.5 (113)	2.7 ± 1.8 (278)	2.2 ± 1.4 (88)
F-ratio	0.135	0.958	1.212
Degrees of freedom	5, 107	6, 271	4, 83
F-prob.	0.9839	0.4544	0.3119

Table 15 shows that WIC and non-WIC groups were not significantly different in prepregnancy weight or height. At conception, the "average" mother was within normal weight range according to Metropolitan Life Insurance tables for weight.

TABLE 15

Initial Height and Weight Status of WIC and Non-WIC Subjects

	Height (inches)	Weight (pounds)
	Mean ± SD, Percentage Reported	
WIC	63.8 ± 2.7, 95.5%	135.0 ± 31.7, 94.9%
Non-WIC	64.1 ± 2.8, 91.6%	134.4 ± 27.5, 97.8%
Total	63.9 ± 2.7, 94.0%	134.8 ± 30.2, 94.8%

Results in Table 16 suggest that WIC mothers are more concerned about prenatal care, since as a whole, they enrolled in the clinic approximately 2.8 weeks earlier than non-WIC mothers. (This difference was significant). One

TABLE 16

Number of Weeks Pregnant at Initial Clinic Enrollment

	Number of Weeks Pregnant	
	Mean ± SD	Percentage Reported
WIC	15.6 ± 5.3	99.7%
Non-WIC	18.4 ± 5.7	100.0
Total	16.6 ± 5.6	99.8
F-ratio	7.726	
Degree of Freedom	1	
F-probability	0.0056	

should also consider the possibility that WIC mothers enrolled in the clinic earlier simply to receive WIC supplements, being more concerned about "free food" than

prenatal care; however, this study did not attempt to measure such intentions. Subjects having their first clinic visit in the second trimester (475 total) had 10.3% incidence (49 cases) of preeclampsia, whereas subjects first visiting the clinic in their third trimester (29 total) demonstrated 13.8% incidence (4 cases) of preeclampsia. Although this difference was not significant due to small group size, further research is needed before discounting any relationship.

Table 17 demonstrates that WIC and non-WIC groups had similar hematocrits at initial clinic visits and six weeks postpartum. When defining anemia as a hematocrit of less than 33%, more mothers were anemic at their initial clinic visit than at six weeks postpartum (Table 18), which was probably due to the iron supplements received during pregnancy. Development of preeclampsia was not related to initial incidence of anemia (F-probability = 0.1707).

Although this study did not attempt to measure nutritional benefits of WIC participation, past studies nevertheless indicate that improved nutritional status probably occurs with WIC supplementation. Edozien (46) found that diets of WIC participants contained greater amounts of protein, calcium, phosphorus, iron, vitamin A, thiamin, riboflavin, niacin, vitamin C, and folacin. Therefore, the assumption could be made that WIC mothers of

TABLE 17

Initial and Final Hematocrit Results
for WIC and Non-WIC Subjects

	Hematocrit		
	Mean ± SD	% Reported	No. Reported
Initial			
WIC	36.6 ± 3.0	96.8%	303
Non-WIC	36.1 ± 3.1	97.4	186
Total	36.4 ± 3.1	97.0	489
Six weeks postpartum			
WIC	39.1 ± 3.0	54.6	171
Non-WIC	39.5 ± 3.2	46.6	89
Total	39.2 ± 3.1	51.6	260

TABLE 18

Incidence of Anemia in WIC and Non-WIC Subjects at First Clinic Visit and at Six Weeks Postpartum

	Hematocrit >33%	Hematocrit <33%
Initial		
WIC, % (no.)	84.6% (252)	15.4% (46)
Non-WIC, % (no.)	82.0 (150)	18.0 (33)
Total, % (no.)	83.6 (402)	16.4 (79)
Six Weeks Postpartum		
WIC, % (no.)	98.8 (168)	1.2 (2)
Non-WIC, % (no.)	95.5 (85)	4.5 (4)
Total, % (no.)	97.7 (253)	2.3 (6)

this study were better nourished after WIC enrollment than non-WIC mothers. WIC participants received not only supplementary foods; individual nutritional counseling and opportunity for group nutritional education was also provided. If diet indeed played a role in the development of preeclampsia, one would expect incidence to decrease as diet improved with WIC supplementation. Still, supplementation may have been implemented too late to produce measurable improvements.

CONCLUSIONS

De Alvarez (2) associated the development of preeclampsia with severe malnutrition. Since subjects of this study attended a medical facility for prenatal care, one could assume that clinical symptoms of severe malnutrition would have been recognized by a trained staff, i.e., bleeding gums, sores around the mouth, hair loss, etc., if such symptoms were present. These symptoms of severe malnutrition are rarely seen in the United States and were not noted in the medical records of subjects in this study. Perhaps WIC supplementation would have been more important in the prevention of preeclampsia if nutritional status prior to clinic enrollment had been at the severely malnourished state. The diets of subjects studied may have already contained the minimum amounts of nutrients required, so that WIC foods only supplemented a diet that was already basically sound.

A simple increase in dietary protein as provided by WIC supplements likewise does not seem to be a preventative factor in the development of preeclampsia. Edozien (46) demonstrated that dietary protein increased with WIC supplementation; nevertheless, subjects of this study had

no less incidence of symptomology with the addition of WIC supplements.

From an opposite standpoint, theories which speculate as to the cause of preeclampsia give little support to a nutrition-related cause. If a poor nutritional state contributed to preeclampsia, one would expect a woman's chances of developing symptomology to increase as her number of pregnancies increased. Preeclampsia is, however, a disease of the primigravida, and subsequent pregnancies show a sharp drop in incidence of the disease.

Several factors in the design of this study could have contributed to a bias in the results obtained. A true diagnosis of preeclampsia would involve blood pressure greater than or equal to 140/90 and positive incidence of proteinuria or edema, especially in the hands and face. For patients at Riverside Health Center, once a positive diagnosis of symptomology was determined, the patient was immediately referred to a high risk clinic for confirmation and subsequent hospitalization if diagnosis was confirmed. Therefore, further follow-up of this patient was lost. The parameters of this study allowed for any blood pressure in the third trimester greater than or equal to 140/90 combined with any third trimester incidence of edema or proteinuria to give positive confirmation of diagnosis. This allowed for very liberal definition of the disease and possibly an

overestimation of the incidence. In addition, women experiencing problems at home related to preeclampsia probably did not wait for their next clinic visit, but went directly to the hospital for diagnosis. Therefore, fewer of these women would have been diagnosed at the clinic. In defining parameters for diagnosis of preeclampsia, two or more blood pressure readings and proteinuria findings with at least six hours in between are required for true diagnosis. Only one reading of each were utilized herein to verify diagnosis. As Worthington-Roberts et al. discussed (11), teenagers could experience an increase in systolic blood pressure of 30 mm Hg or diastolic blood pressure of 15 mm Hg and still never realize a blood pressure of 140/90. A positive symptom of the disease (for teens) would have been missed in this study if such occurred. Finally, the method of edema documentation at Riverside Health Center was by record of patient complaint--not actual testing for edematous pitting. Edema of pregnancy is a normal occurrence, unlike that found in preeclampsia. Perhaps the edema noted was normal edema of pregnancy and not abnormal edema of preeclampsia. Nevertheless, differentiation between normal and abnormal edema is difficult to make; therefore, in this study both were recorded as simply "edema", without specification as to duration or location of swelling.

Studies which have shown that tight control over diet resulted in minimal incidence of preeclampsia fail to point out that if such a regimented control of eating habits were dictated and attained, more than likely close attention was also paid to maternal health, infection control, and other factors of good prenatal care. Certainly the answer to control of preeclampsia involves a total package: good prenatal care, a healthy mother prior to conception to include, but not be limited to, a well-balanced diet.

Future studies on diet and preeclampsia should provide dietary supplements several months prior to conception to allow ample time for nutritional replenishing. For both experimental and control groups, blood and urine samples should be analyzed for nutritional adequacy and blood pressure should be documented prior to food supplementation, prior to conception, during pregnancy, and again at delivery, to better evaluate any changes resulting from improved diet. In addition, dietary recalls and diet histories should be obtained at intervals prior to and during pregnancy to document nutrients consumed. Such a study of this magnitude would indeed be costly and perhaps impractical, since one cannot control when and if a woman will become pregnant.

Null hypotheses for this study were accepted. No significant differences in blood pressure, proteinuria.

edema, or incidence of preeclampsia were noted in WIC and non-WIC groups.

IMPLICATIONS FOR FURTHER STUDY

Although results of this study failed to significantly correlate development of preeclampsia to WIC status, several trends merit further study. WIC patients tended to exhibit less incidence of preeclampsia, had fewer low birthweight infants, entered the clinic earlier, and tended to have less incidence of initial and postpartum anemia. Patients first entering the clinic in their second trimester tended to have less incidence of preeclampsia than those entering in the third trimester. All of these relationships deserve additional investigation with larger group sizes to better establish correlations.

Review of the literature failed to biochemically explain any relationship between nutritional status and development or prevention of preeclampsia. Future studies must obtain tighter correlations between diet and preeclampsia than those found herein before assigning further cause and effect relationships.

APPENDIX A

REGIMENS FOR TREATMENT OF PREECLAMPSIA AND ECLAMPSIA (13)

INCIPIENT PREECLAMPSIA. The patient with incipient, but not developed preeclampsia (excessive weight gain with edema of the ankles; blood pressure elevation, but below 140/90 or less than 30 mm Hg systolic and 15 mm Hg diastolic above prior levels; no proteinuria) may remain at home. The therapeutic regimen includes:

1. If before 32 weeks, a diet high in protein (1.5 g/kg); no salt restriction
2. Bedrest for 12 hours of the 24, especially in lateral incumbent position
3. Phenobarbital, 30-60 mg three times daily
4. Hospitalization if preeclampsia is diagnosed

MILD PREECLAMPSIA. All patients with preeclampsia should be hospitalized. For the patient with mild disease, the following therapeutic regimen is suggested:

1. Bedrest with bathroom privileges
2. Daily weighing
3. Evaluation of fetal heart tones every 4 hours
4. Phenobarbital, 30-60 mg every 6 hours while awake
5. Diet with 1.5 g protein per kilogram body weight

6. Evaluation of fetal maturity, favorability of cervix, and adequacy of maternal pelvis in anticipation of possible need for delivery
7. Delivery at 37 weeks gestation with or without improvement; prior to 37 weeks without improvement, need for delivery must be weighed against hazard of fetal prematurity

SEVERE PREECLAMPSIA. For the patient with severe preeclampsia, the regimen includes:

1. Close observation
2. Absolute bedrest
3. Magnesium sulfate, 2-10 g intramuscularly at intervals, or intravenously at a rate of 1 g/hour, to suppress hyperreflexia
4. Determination of water intake and output
5. Fetal monitoring continuously until delivery
6. Delivery after adequate sedation

ECLAMPSIA. For the patient with eclampsia, the regimen is as follows:

1. Constant observation
2. Absolute quiet, in a darkened room
3. Hourly determination of urinary output and monitoring of fetal heart tones

4. Presence of equipment to maintain airway and prevent trauma to tongue and lips during seizure
5. Magnesium sulfate, 1 g/hour, intravenously, with calcium gluconate available as an antidote for magnesium overdosage as evidenced by loss of deep reflexes
6. Morphine sulfate to maintain deep sedation (but respirations should be kept above 12/minute)
7. If seizures persist in spite of above, sodium diphenylhydantoin intravenously
8. Delivery, when seizures are controlled.

APPENDIX B

DEFICIENCY METHOD OF DIETARY ASSESSMENT

If the woman's 24 hour intake being compared to the Guide for Daily Intake (column 2 below) has three or more deficiencies, inadequate dietary pattern may be applied as a condition of nutritional need.

Food Group	Guide for Daily Intake	Counting Deficiencies
Milk	3 servings	Count each missing serving as one
Meat	3 servings	Count each missing serving as one
Fruits/ Vegetables	4 servings	Maximum of one deficiency to be counted if total servings are not consumed
(Vitamin C -rich)	1 serving	Count one when serving missing
(Vitamin A -rich)	1 serving	Count one when serving missing
Bread/Cereal	4 servings	Count one for every two missing or Count one for every two servings in excess of six

Other Dietary Considerations	
Non-nutritious foods--soft drinks, sugar-sweetened tea or coffee, candy, popsicles, chips, cookies	Count one for every two servings
Pica (the consumption of non-food items, such as clay, cooking or laundry starch, refrigerator ice, dirt, etc.	Count one if participant admits to consuming one or more times daily

APPENDIX C

CRITERIA FOR WIC ELIGIBILITY (1981)

A pregnant woman was deemed eligible for the WIC program if she:

1. was under 18 years of age or over 35 years of age at conception
2. was 15% underweight or 20% overweight at the time of conception (based on Metropolitan Life Insurance Actuarial Tables, 1969)
3. had insufficient or excessive weight gain (weight under or over the optimal curves on prenatal weight grid of U.S. Department of Health, Education, and Welfare)
4. had a hematocrit of 35% or less or hemoglobin of 11.9 g/100 ml or less in first trimester; or a hematocrit of 33% or less or hemoglobin of 10.9 g/100 ml or less during the second or third trimesters
5. was considered a high risk pregnancy by one of the following:
   a) Current pregnancy:
      (1) preeclampsia, eclampsia
      (2) chronic hypertension
      (3) renal disease

(4) cardiovascular disease

(5) diabetes mellitus (overt or gestational)

(6) tuberculosis

(7) multiple births expected

b) Maternal History:

(1) three or more pregnancies within the last two years

(2) five or more previous pregnancies

(3) history of stillbirth, miscarriage, infant born with congenital defect, premature infant (born at less than 36 weeks gestation), post term infant (born at more than 42 weeks gestation), low birthweight infant (born weighing less than 2500 g) or neonatal deaths.

6. was considered a high risk pregnancy by two of the following:

a) history of preeclampsia, eclampsia or hypertension

b) history of renal disease, cardiovascular disease, diabetes, tuberculosis

c) history of multiple births

d) chronic use of drugs, alcohol (ingesting more than 2 ounces/day--either currently or within three months prior to conception) or current

use of tobacco (smoking more than one package of cigarettes/day)

e) current hypoglycemia

7. had an inadequate dietary pattern of three or more nutritional deficiencies (see Appendix B).

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