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MODIFIABLE DETERMINANTS OF VITAMIN A DEFICIENCY

AMONG PREGNANT WOMEN

PARTICIPATING IN THE MAMA SASHA COHORT STUDY

OF VITAMIN A IN WESTERN KENYA

By

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MODIFIABLE DETERMINANTS OF VITAMIN A DEFICIENCY AMONG PREGNANT WOMEN PARTICIPATING IN THE MAMA SASHA COHORT STUDY OF VITAMIN A IN WESTERN KENYA

By Victor Akelo

Master of Public Health

Global Health (Infectious Diseases)

Thesis Committee Chair: Amy Webb Girard, PhD

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Mothers' vitamin A (VA) status during pregnancy and lactation determine infants' VA levels. We estimated VA status during pregnancy and assessed its modifiable determinants using data on 505 pregnant women attending first antenatal care visit in Western Kenya. VA and iron status were assessed using plasma retinol binding protein (RBP), and ferritin and transferrin receptor, respectively, corrected for inflammation as measured by C-reactive protein (>5 mg/L) and α-1-acid glycoprotein (>1 g/L)]. Anemia was assessed with Hemocue hemoglobinometer. Mean RBP was 1.44 µmol/l (±0.02) and the prevalence of VA deficiency (VAD) was 21.8%. Prevalence of inflammation was 24%. Only 34% of women had heard of vitamin A, and 26% of them could not specify its importance. School was the most common source of vitamin A information (68%), followed by health facility (19%). Anemia, but not iron deficiency, was the only factor associated with VAD (OR (CI): 1.68 (1.05, 2.71) in the single predictor analysis. In multiple logistic regression analysis, maternal consumption of VA rich food was significantly associated with reduced odds of VAD (AdjOR (CI): 0.53, (0.33, 0.83) but only among women with mid upper arm circumference (MUAC) ≤ 24.5cm. There was no significant association between household (HH) consumption of VA-rich food and VAD (AdjOR 0.96, 95% CI: (0.59, 1.54). The prevalence of VAD is high among pregnant women in Western Kenya and could be associated with anemia but not iron deficiency. Promotion of consumption of Vitamin A rich foods in pregnancy may reduce odds of VAD in pregnancy, especially among women with lower nutritional status. Additional research is needed to understand the etiology of VAD in this population.

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1. Chapter 1: Introduction

1.1.Background

Vitamin A (VA) is one of the essential nutrients needed for normal functioning of the human body [1]. It helps to regulate and promote growth and cell differentiation, maintenance of epithelial integrity, red blood cell production, immunocompetence, and reproduction [1, 2]. Retinoic acid, a vitamin A metabolite, has been shown to be involved in induction of the blood brain barrier which protects and safeguards optimal brain neural activities [3, 4]. In pregnancy, vitamin A is important for normal fetal growth and could be essential in maintaining normal pregnancy in the later stages when there is extensive fetal organ cell development and proliferation [5]. Fetal and newborns' VA levels depend on maternal VA levels in pregnancy and early lactation respectively hence an adequate maternal vitamin A level is essential for normal fetal/neonate's VA levels [2, 6-10]. Breast fed infants VA levels are dependent on VA levels in breast milk of their lactating mothers [9]. With breast milk being highly prevalent in Sub-Saharan Africa, maternal vitamin A levels are important in determining infant VA levels in this context.

Despite these beneficial effects of vitamin A, vitamin A deficiency (VAD) remains one of the major global micronutrient disorders of public health significance, especially in developing countries[1, 2]. Pregnant and lactating women and children are considered at high risk of VAD due to their increased metabolic demands [1]. In addition, women who have marginal vitamin A levels are at increased risk of developing VAD when they become pregnant [1]. WHO approximates that VAD among preschool children is a public health concern in 45 countries (as measured by night blindness) and 122 countries (as measured by serum retinol concentration of <0.70 µmol/liter) [1]. About 19.1 million (15.3%) pregnant women are estimated to be at risk of VAD globally [11]. VAD of sufficient duration or severity can lead to night blindness, xerophthalmia, anemia, weakened host resistance to infection, and non-response to antiviral therapy [1, 12]. Maternal VAD is also associated with low birth weight, reduced fetal nephron endowment and growth, and increased risk of mother-to-child transmission of HIV [11, 13, 14].

In Kenya, Vitamin A deficiency is one of the leading micronutrient deficiencies in children [15]. According to WHO regression-based estimates, Kenya is one of the countries with severe and moderate biochemical vitamin A deficiencies of public health concern among pre-school children and pregnant women respectively [1]. Among adults, VAD is compounded by the high HIV prevalence levels (7.4% in age group 15-49 years) [16]. HIV increases the risk of diarrheal and respiratory diseases which in turn lower not only intake of vitamin A through depressed appetite, but also absorption and depletion through increased metabolism [1]. However, limited data exists documenting vitamin A status of women during pregnancy and breastfeeding. A pregnant woman who has VAD is not only at risk of developing night blindness, increased risk of infections, and giving birth to a baby with VAD with its accompanying complications, but she is also at risk of producing breast milk with inadequate levels of vitamin A which would further worsen her baby's VA status. This makes it important to ensure pregnant women have adequate levels of vitamin A intake.

1.2.Problem Statement

While vitamin A deficiency has been shown to be as high as 29% among children between one to three years in Western Kenya [17], there is little information on the actual burden and determinants of vitamin A deficiency among pregnant women in this region**.** There is need to estimate maternal vitamin A status and its modifiable determinants during pregnancy to inform monitoring of maternal VA trends and delivery of targeted and timely maternal nutritional counseling and dietary interventions.

1.3.Purpose Statement

An ongoing cohort study of the impact of an integrated agriculture, nutrition and health proof of concept project (Mama SASHA project) on the Vitamin A and health status of mothers and their infants from pregnancy through 9 months postpartum in Western Kenya affords the opportunity to assess vitamin A status and its modifiable determinants in pregnant women. The following research questions will frame these analyses;

1. What is the estimated prevalence of vitamin A among pregnant women participating in the Mama SASHA cohort study in Western Kenya?

2. Is there a relationship between maternal vitamin A status and modifiable factors such as household food insecurity, household food diversity, household consumption of VA rich food and maternal consumption of VA rich food?

2. Chapter 2: Review of Literature

A literature review was conducted in PubMed, Web of Science, EMBASE, Cochrane library, and Google Scholar databases using the following key words and phrases: micronutrients deficiency, micronutrient deficiency in pregnancy, vitamin A, vitamin A biochemistry and physiology, role of vitamin A, vitamin A deficiency (VAD), predictors of vitamin A, determinants of Vitamin A, maternal and household consumption of vitamin A, vitamin A in pregnancy, VAD in pregnancy, VAD in children, vitamin A teratogenicity, effects of vitamin VAD, vitamin A supplementation, vitamin A bio-fortification, and agricultural-nutritional vitamin A programs. An initial group of articles were identified and reviewed. These articles informed additional searches until no new article could be identified. Further, secondary data sources including WHO reports, standard nutritional text books, government reports and various professional bodies' reports and recommendations were used to yield more information on VAD in children and pregnancy.

A summary of key findings were organized in the following sub-headings: Biochemistry of vitamin A; role of vitamin A in various physiological systems; Food sources of vitamin A and beta carotene; VAD in Pregnancy and children; strategies and interventions for preventing VAD.

2.1.Biochemistry of vitamin A

Vitamin A is a fat soluble vitamin that occurs in nature in three forms; All-trans-retinol (the biologically active form), 3-dehydroretinol and beta-carotene [18, 19]. Both all-trans-retinol and 3-dehydroretinol are found in animal products. Bile salts is required during absorption to solubilize retinyl esters in mixed micelles and to activate the hydrolyzing enzymes [18, 19]. In the intestinal mucosa, the ester is incorporated into chylomicrons in the enterocytes for transport into the lymph and eventually the blood [18, 20]. Peak levels are usually reached 3-4 hours after an oral dose [18]. Food sources that naturally exist as dietary retinyl esters follow the same pathway. Retinol is stored mainly in the liver but can also be stored in the lungs, the gonads and the kidneys [9, 18]. Total body retinol varies between 300-900mg [18]. From the liver, retinol is secreted bound to retinol-binding protein (RBP) to various body parts e.g. the eye where it is needed [18, 20]. RBP is synthesized in the liver and its secretion is tightly regulated by the

availability of retinol [19]. During VAD, RBP secretion is inhibited; upon an increase in retinol levels, RBP is secreted into the blood [19]. Hepatic RBP synthesis depend on adequate protein therefore the blood levels of retinol can be affected by protein deficiency as well as by chronic vitamin A deficiency [9]. In addition to retinol being esterified for storage, it can also be oxidized into retinal by enzyme retinol dehydrogenase and then into retinoic acid by enzyme retinal dehydrogenase [9, 19]. Retinol can also be conjugated into retinyl glucuronide for excretion. Retinoic acid, retinol and retinal are degraded by the microsomal cytochrome P450 system to oxidized metabolites [19]. Vitamin A that is usually employed in parenteral nutrition and supplementation in children is usually in the form of an ester (available in liquid forms), an acetate, or palmitate.

Beta-carotene is the most abundant carotenoid present in green, yellow, and orange fruits and vegetables [20]. It is a provitamin-A compound found in plants. Following ingestion of beta-carotene, the body enzymatically cleaves some of it to retinol, which is esterified to a fatty acid (usually palmitate) in the intestinal cell to retinyl esters (VA is absorbed in the body as ester) [20]. Due to inefficient conversion, 12 µg of beta-carotene in food yields only about 1 µg retinol, and therefore a serving of food that contains 12 µg of beta-carotene is said to contain 1 µg beta retinol activity equivalent (RAE). Other carotenoids are even less efficiently converted to retinol (1 µg RAE equates to 24 µg of these other carotenoid species) [20]. Dietary carotenoids are less bioavailable than purified beta-carotene therefore greater amount is needed to meet vitamin A requirement.

2.2.Role of Vitamin A in various physiological systems

Vitamin A is an essential nutrient that plays a significant role in the human body. The American Academy of Pediatrics Committee on Nutrition cites VA as one of the most critical vitamins during pregnancy and lactation period especially in terms of lung function and maturation [10]. First, VA plays a key role in ocular retinoid metabolism and visual function [10]. The main vitamin A form that circulates in the blood, all-trans-retinol, is taken up into the eye by the retinal pigment epithelium where it is involved in the development or maintenance of the mucus membranes, cornea, and conjunctiva [19, 20]. In a process known as phototransduction, all-trans

retinal is linked to a protein to form rhodopsin in the rod cells, and iodopsin in the cone cells of the retina which are necessary for good vision [20].

Second, retinoic acid act as a hormone that affect gene expression thus affecting protein synthesis and many body processes [9, 20]. Some of these processes include morphogenesis in embryo development and epithelial cell function (including differentiation and production of keratin proteins). VA maintains normal fetal organ cell proliferation and development which are extensive during the later stages of pregnancy [5, 6]. Congenital nephron number has been shown to vary with fetal retinoic acid (RA) and infant with low RA may develop hypertension later in life due to reduced number of nephrons [21]. Third, β-carotene acts as antioxidants thereby counteracting the harmful and damaging effects of oxidation in the body [20]. Fourth, VA is also involved in maintenance of normal integrity and growth of skin and tissue cells, including the mucus membranes of the mouth, intestinal, respiratory, genitals, and urinary tracts. Vitamin A is also essential for normal reproduction, bone development and function, and immune system function, although its actions in these roles are currently not well understood [9, 22]. Whereas VA is an important immuno-modulator nutrient [22], its levels are affected by infections that cause inflammation, indicated by rise in acute phase proteins [C-reactive protein (>5 mg/L) and α-1-acid glycoprotein (>1 g/L)]. Therefore, assessment of retinol and RBP levels require adjustment for the influence of inflammation especially in settings where infections are common. This adjustment is done by correcting the VA & RBP levels using the inflammatory markers C-reactive protein [CRP] and α-1-acid glycoprotein[AGP] [23].

2.3.Food sources of vitamin A and beta carotene

Pre-formed vitamin A exist only in food of animal origin in storage areas such as liver, milk and eggs [9]. Very high concentrations are found in liver and liver oils of marine animals [9, 10]. Provitamin A carotenoids are found in red palm oil, green leafy vegetables, yellow-orange root vegetables and fruits [9, 18]. Some examples of provitamin A rich foods include carrots, greens, spinach, mangoes, pumpkin fruit, ripe papaya and sweet potatoes (especially orange flesh sweet potatoes) [9]. However, high doses of provitamin A are needed to substitute pre-formed retinol [10]. In many of these foods, vitamin A bioavailability is limited by binding of carotenoids to proteins which can be overcome by cooking, which disrupts the protein association and frees the

carotenoids [9]. Other artificial sources are VA fortified food such as margarine and cereals with added VA. Adult dietary reference intakes (DRIs) of VA are based on adequate blood levels and liver stores adjusted for the differences in body sizes [9]. The recommended Daily Allowance (RDA), the amount that should be taken to prevent VAD, is about 0.8mg/day [10]. A large heterogeneity, however exists with recommended intakes varying from 0.77mg/day in UK to 1.1mg/day in D-A-CH countries (European countries with German-speaking majorities) [10].

2.4.VAD in Pregnant women and Children

Primary VA deficiencies result from inadequate intake of VA and its precursors whereas secondary VA deficiencies occur from malabsorption, liver disease, nephrotic syndrome and alcohol abuse [18, 20]. Malabsorption can result from insufficient dietary fat, biliary or pancreatic insufficiency, impaired transport from abetalipoproteinemia, protein-energy malnutrition or zinc deficiency [9]. Clinically, VAD results in night blindness due to decreased rod function, dryness of the conjunctiva (xerophthalmia), hyperkeratosis of the skin, and impairment of both humoral and cell-mediated immune systems [18, 20]; this latter effect is known to increase susceptibility to infection due to reduction of glycoprotein synthesis [18]. Loss of mucous membrane integrity as a result of VAD increases host susceptibility to bacterial, viral, or parasitic infections [1, 9]. The xerosis of the eye is manifested as a shiny gray foamy triangular areas called Bitot's spots and may cause corneal ulceration and blindness [18]. Biochemically, VAD is shown by low serum levels of retinol <0.70 µmol/L [1, 19]. Women RBP levels of <1.17µmol/L correspond to <0.70µmol/L retinol levels (sensitivity and specificity for the cut-offs 81.8 and 93% respectively) [24].

There is a high prevalence of vitamin A deficiency among pregnant and lactating women, and children under 6 years globally [1, 5, 25]. VAD is associated with increased maternal morbidity and mortality, and increased infant mortality rates during the first year of life [26]. VAD during pregnancy is associated with preterm birth, low birth weight, and low neonatal liver stores [2, 10]. This has further been corroborated by a number of cross-sectional retrospective studies that have shown an association between low vitamin A concentrations in umbilical cord blood and prematurity, and fetal growth retardation (low birth weight and reduced length) [5]. Furthermore, newborns of mothers with VAD have been shown to have lower mean cord retinol

concentrations [6]. VAD is the most significant cause of blindness, especially among children in the developing world [1, 15]. An estimated 250 million children are at risk and between 250,000-500,000 cases of blindness from VAD occur annually [9]. According Kathleen et al (2008), research shows that 4.4 million preschool children, most from South Asia, had clinical disease (xerophthalmia) caused by VAD [9]. A cross sectional study of 3571 children in Uttar Pradesh, India showed a high prevalence of xerophthalmia of 9.1% which was of public health significant [27]. In a randomized clinical trial of vitamin A supplementation in children aged 6 months to 2 years in rural Guinea-Bissau, baseline VAD prevalence was 65.7% (as measured by RBP) and was associated with rainy season but not sex, age or arm circumference of the child in a multivariate analysis [28]. VAD has also been associated with anemia, even though the mechanism of how this is achieved remains unclear [22].

2.5.Strategies and Interventions for Preventing VAD

Vitamin A supplementation has been shown to effectively reduce the risk of VAD in children [25]. In India, a decline in prevalence of xerophthalmic blindness, keratomalacia and Bitot's spots has been reported among pre-school children due to vitamin A supplementation programme [29, 30]. There is however mixed evidence of whether vitamin A supplementation is also beneficial in pregnant women. Consumption of too much vitamin A, especially retinoic acid during the first trimester of pregnancy can cause birth defects to the developing embryo [20, 31]. 13-cis-retinoic acid (Accutane) used in treatment of severe cystic acne, has been shown to cause craniofacial, central nervous system, cardiovascular, and thymic malformations in the fetus, and is currently contra-indicated for pregnant women [1, 9]. Fetal malformations have also been linked to daily exposures of 600 to 7500 RAEs of vitamin A supplements among pregnant women [9]. Teratology Society of the United States (TS) recommends that the daily VA dose for women should never exceed 3,000 µg retinol equivalents [19]. Further, TS recommends that it is reasonable to replace part of vitamin A supplement for pregnant women with β-carotene [19]. Excessive intake of beta-carotene has not been shown to be teratogenic because only limited amounts are converted to VA [19, 20] . However, people who consume large amount of beta-carotene may develop a yellow tinge to skin (carotenosis) which is not harmful [20].

In a cross-sectional study of 225 mother-infant pairs in Senegal, maternal VA supplementation during lactation period was associated with an increase in breast milk VA and infant VA levels [8], indicating the potential benefit of maternal VA supplementation. Other studies have also shown that maternal vitamin A levels correlates with infant VA levels and the amount of VA intake for infants is based on the amount of retinol in breast milk [9]. This has been supported by a systematic review by Hovdenak, N., and Haram, K. (2012) [32] that concluded that vitamin A supplementation in pregnancy enhances birth weight and infant growth born to HIV positive women. Vitamin A supplementation has also been shown to help treat anemia in pregnancy. Combined vitamin A and iron supplementation among anemic pregnant women in Indonesia (Hb<11g/dl) was shown to correct the hematological deficit in 97% of the women compared to 68% use of iron supplementation only [33]. The World Food Programme provides vitamin A fortified skimmed milk in countries with populations at risk of VAD to mitigate potential negative effects of VAD [33].

In contrast, a large-scale, randomized study performed in Ghana with more than 200,000 women of reproductive age found no improvement in perinatal or infant survival with VA supplementation [2]. This has raised the question whether VA supplementation may only be beneficial in some VAD sub-groups (e.g. severe VAD) [2]. VA supplementation as a policy for pregnant women is not recommended in some settings as a result of these mixed results. Other alternative recommendations include use of alternative low dosing strategies; use of water soluble precursors- beta carotene instead of retinol and dietary diversification of VA rich foods. In literature, food dietary diversification has also been emphasized as one of the preventive strategies against micronutrient deficiencies like VAD [34].

Other strategies involve fortification of flour with VA levels, or home gardening with improved micronutrient rich fruits, vegetables or tubers such as OFSP which has high content of provitamin A carotenoids [35-37]. Other food based strategies include supporting small scale livestock production such as poultry rearing or fish farming alone or in combination with home gardening [37]. Whereas supplementation and fortification have been shown to improve maternal VA intake, the approaches may not be sustainable, accessible or acceptable in rural areas where the burden of VAD is greatest [37]. Agricultural-nutritional interventions not only offer a safer

alternative of improving maternal VA levels, but also increase food available for consumption by raising purchasing power of food and increasing quantity and quality of food available for consumption [37]. An integrated OFSP approach for example, has been shown to be feasible and potentially increase VA intake of young children [35, 36, 38].

The Mama SASHA initiative is a proof of concept project in Western Kenya that links delivery of maternal and child health services with distribution of orange flesh sweet potato vines for the purposes of improving vitamin A status and health of women during pregnancy and breastfeeding and their infants. The evaluation strategy for this initiative includes a nested cohort study of women from control and intervention communities who are enrolled in pregnancy and followed to 9 months postpartum. This thesis utilizes data collected at enrollment into this cohort study to assess prevalence of VAD and identify modifiable factors associated with VAD status in this population of pregnant Kenyan women for the purposes of prioritizing and informing future work on nutrition programming in Western Kenya.

3. Chapter 3: Manuscript

3.1. Title

Modifiable Determinants of Vitamin A Deficiency Among pregnant women participating in the MAMA SASHA Cohort Study of Vitamin A in Western Kenya.

3.2. Contribution of the Student

The student led the formulation of thesis research questions, data analyses and writing of the thesis with guidance from thesis faculty committee members.

3.3. Abstract

Mothers' vitamin A (VA) status during pregnancy and lactation determine infants' VA levels. We estimated VA status during pregnancy and assessed its modifiable determinants using data on 505 pregnant women attending first antenatal care visit in Western Kenya. VA and iron status were assessed using plasma retinol binding protein (RBP), and ferritin and transferrin receptor, respectively, corrected for inflammation as measured by C-reactive protein (>5 mg/L) and α-1-acid glycoprotein (>1 g/L)]. Anemia was assessed with Hemocue hemoglobinometer. Mean RBP was 1.44 µmol/l (±0.02) and the prevalence of VA deficiency (VAD) was 21.8%. Prevalence of inflammation was 24%. Only 34% of women had heard of vitamin A, and 26% of them could not specify its importance. School was the most common source of vitamin A information (68%), followed by health facility (19%). Anemia, but not iron deficiency, was the only factor associated with VAD (OR (CI): 1.68 (1.05, 2.71) in the single predictor analysis. In multiple logistic regression analysis, maternal consumption of VA rich food was significantly associated with reduced odds of VAD (AdjOR (CI): 0.53, (0.33, 0.83) but only among women with mid upper arm circumference (MUAC) ≤ 24.5cm. There was no significant association between household (HH) consumption of VA-rich food and VAD (AdjOR 0.96, 95% CI: (0.59, 1.54). The prevalence of VAD is high among pregnant women in Western Kenya and could be associated with anemia but not iron deficiency. Promotion of consumption of Vitamin A rich foods in pregnancy may reduce odds of VAD in pregnancy, especially among women with lower nutritional status. Additional research is needed to understand the etiology of VAD in this population.

Key Words: Vitamin A deficiency, Pregnancy, Household Consumption, Maternal Consumption.

3.4. Background

Vitamin A plays an important role in regulating and promoting growth and cell differentiation, maintenance of epithelial integrity, red blood cell production, immunocompetence, and reproduction [1, 2]. Besides, the dietary precursor of vitamin A, Carotenoids, has anti-oxidant properties [2]. Retinoic acid has also been shown to be involved in the induction of the blood brain barrier which is important in safeguarding optimal neural activities [3, 4]. Despite these important roles, vitamin A deficiency (VAD) remains one of the three micronutrient disorders of public health significance globally, especially in lower income countries [1]. WHO approximates that VAD among preschool children is a public health concern in 45 countries (as measured by night blindness) and 122 countries (as measured by serum retinol concentration of <0.70 µmol/liter) [1]. VAD of sufficient duration or severity can lead to xerophthalmia, anemia, weakened host resistance to infection, and non-response to antiviral therapy [1, 12].

Pregnant women are considered a high risk group for developing vitamin A deficiency, with about 19.1 million (15.3%) pregnant women estimated to be at risk of VAD globally [1, 11]. There is increased demand of vitamin A during pregnancy [39]. Consequently, women who have marginal vitamin A levels are at increased risk of developing night blindness, anemia and intercurrent infections when they become pregnant [1]. Infants born from mothers who have vitamin A deficiency are at increased risk of experiencing night blindness, dryness of the eye, low birth weight, death and if the mother is HIV positive, increased risk of mother to Child transmission of HIV [11, 13, 14]. Critically, vitamin A levels of mothers during pregnancy and lactation define the level of this vitamin in newborns [7]. Maternal vitamin A also affects kidney organogenesis of their neonates [21]. It's postulated that lower maternal vitamin A levels could results in fetal reduced nephron endowment and glomerular hypertrophy which may later predispose to hypertensive disease later in life [21]. For young children especially under 6 months, breast milk is the main source of vitamin A. Thus a pregnant woman who has VAD is not only at risk of developing night blindness, increased risk of infections, and giving birth to a

baby with VAD with its accompanying complications, but she is also at risk of producing breast milk with inadequate levels of vitamin A which would further worsen her baby's VA status. This makes it important to ensure pregnant women have adequate levels of vitamin A intake.

In Kenya, Vitamin A deficiency is one of the leading micronutrient deficiencies in children [15]. According to WHO regression-based estimates, Kenya is one of the countries with severe and moderate biochemical vitamin A deficiencies of public health concern among pre-school children and pregnant women respectively [1]. VAD is compounded by the high HIV prevalence levels (7.4% in age group 15-49 years) [16]. HIV increases the risk of diarrheal and respiratory diseases which in turn lower not only intake of vitamin A through depressed appetite, but also absorption and depletion through increased metabolism [1]. While vitamin A deficiency has been shown to be as high as 29% among children between one to three years in Western Kenya [17] , there is little information on the actual burden and determinants of vitamin A deficiency among pregnant women in this region.

We used data from an ongoing study of the impact of an integrated agriculture, nutrition and health intervention on the vitamin A and health status of mothers and their infants from pregnancy through 9 months postpartum in Western Kenya to assess vitamin A status and identify modifiable factors associated with VA status in pregnant women. We anticipate such analyses will inform monitoring of maternal VA trends and delivery of targeted and timely maternal nutritional counselling and dietary interventions, including for example orange sweet potatoes [40].

3.5.Methods

3.5.1. Overview

This is an analysis of baseline data collected from pregnant women involved in the Mama SASHA Proof of Concept Project (PoCP) cohort study of vitamin A (COVA). Mama SASHA is an agriculture-health linkage PoCP which is part of Sweet potato Action for Security and Health in Africa (SASHA); a five-year multi-partner project led by the International Potato Center (CIP) designed to improve the food security and livelihoods of poor families in sub-Saharan Africa by

exploiting the untapped potential of Orange flesh sweet potatoes (OFSP). The overall goal of Mama SASHA project is to improve the health status of pregnant women and the nutritional status of children up to two years through an integrated OFSP and health service delivery strategy through an existing health program in selected districts of Western Kenya. Mama SASHA is implemented through partnerships with 8 facilities in Western Kenya purposively selected according to size-related variables (number of service providers, antenatal clinics (ANC) attendance numbers, and population served), and coverage with community health workers (CHW) linked to the USAID/Kenya AIDS, Population and Health Integrated Assistance Program (APHIA plus) location criteria). COVA is a 28 month, community-based cohort study nested within the larger Mama SASHA PoCP. While COVA is designed to assess the impact of Mama SASHA project on maternal and infant VA status, this analysis only examines the modifiable determinants of VAD using COVA enrolment data.

3.5.2. Study Population

Between November 2012 and March 2013, 505 pregnant women were enrolled after screening for eligibility and referral to the study by the health care workers from the 8 facilities of the Mama SASHA PoCP. The enrolment criteria included: first antenatal clinic; 17-40 years of age; 10-24 weeks gestations determined by the last menstrual period or if absent, by fundal height; intending to breast feed; and live in the catchment area until child is 10 months of age. The exclusion criteria included the following: previous involvement with Mama SASHA during earlier pregnancy; mother does not live in Mama SASHA intervention village for intervention facilities; and mother resides in a Mama SASHA intervention village for control villages.

3.5.3. Data Collection and Procedures

3.5.3.1. Questionnaire Data

Data were collected using written study instruments with structured and semi-structured questions. Questionnaires were written in English and translated into Kiswahili and Kibukusu which are the predominant languages of the area. Pilot testing and revision of questionnaires were completed prior to initiation of the study. The enrolment questionnaire used for purposes of

this study consisted of four components: household characteristics; household food security and dietary diversity; maternal diet and uptake of health services. The questionnaire was verbally administered by research assistants who had been previously trained on the study protocol, data collection tools and study-specific interviewing methods.

Household food security score and scale, and household dietary diversity score (HDDS) were assessed using USAID Food and Nutrition Technical Assistance Project (FANTA) Household Food Insecurity Assessment Scale (HFIAS) and the FANTA dietary diversity scale respectively [41]. Maternal dietary consumption of VA rich food and household consumption of VA rich food were assessed using FAO Guidelines for Measuring Household and Individual Dietary Diversity [42].

3.5.3.2. Collection of Biochemical and Anthropometric Data

Biochemical and anthropometric data collected include: Maternal retinol binding protein (RBP), hemoglobin, transferrin receptor levels, ferritin levels, C-reactive protein (CRP), α1-acid-glycoprotein (AGP) and Mid Upper Arm Circumference (MUAC).

Retinol binding protein, α1-acid-glycoprotein, C-reactive protein, serum ferritin, transferrin receptor levels were measured from finger prick capillary blood sample (about 500µL) and hemoglobin measured using Hemocue machine (Hb201+). Hemocue devices were calibrated each morning and evening using standardized techniques. The capillary blood sample was collected in a microtainer with a clot activator and gel for serum separation. The microtainer was capped and inverted 10 times gently to prevent clotting. The blood was then centrifuged at 1500 × g for 5 min at 27^{o}C and serum transferred to suitable PCR tubes and cryovials. They were labeled and serum samples stored at the health facility at -20^{0}C using solar powered freezers until collected and transported on ice for longer term storage at Kenya Medical Research Institute/CDC Malaria Laboratory in Kisumu, Kenya. Samples were shipped to the laboratory of Juergen Erhardt in Germany where analyses took place. Analyses utilized a 5-protein ELISA method and simultaneously quantified RBP, ferritin, transferrin receptor, CRP and AGP. Abnormal biochemical indicators' values were as follows: ferritin, <12 mg/L; TfR, >8.3 mg/L; CRP, >5 mg/L; and AGP, >1.0 g/L [23]. Elevated TfR/ ferritin index defined as >500, was

calculated by dividing TfR (mg/L) by ferritin (mg/L) [23]. Correction factors (ratios of geometric means of the nutrients in the low and high groups [43]), were applied to correct for inflammation for RBP, ferritin, transferrin receptor levels using the acute phase proteins (CRP and AGP) [23]. Vitamin A and iron statuses were assessed using corrected RBP, ferritin and transferrin receptor levels. Cut-offs for VAD was set at <1.17µmol RBP/L for the women which correspond to <0.70µmol/L retinol levels (sensitivity and specificity for the cut-offs 81.8 and 93% respectively) [24]. WHO Serum retinol threshold levels of <0.70µmol/liter is the cut-off for vitamin A deficiency [1]. Mid-Upper Arm Circumference (MUAC) was measured using a stretchable, non-flexible tape at the mid-point of the non-dominant upper arm, between the acromion process and the tip of the elecranon. MUAC is a recommended WHO standard for assessing maternal nutritional status. Under-nutrition is defined as MUAC <18.5cm.

3.5.4. Ethics

The research protocol was reviewed and approved by the Ethical Review Committee of the Kenyan Medical Research Institute (KEMRI) and the Institutional Review Board of the Emory University, Atlanta, GA, USA. All participants provided written informed consent prior to data collection.

3.5.5. Statistical Analysis

An exploratory analysis was done by conducting frequency distributions on categorical variables and a univariate analyses on continuous variables. We assessed normality by probability plots, and Kolmogorov-Smimov test. RBP, CRP, and AGP were normally distributed. Continuous variables were reported as mean with standard deviations. Categorical variables were described as frequencies and percent. Anemia was calculated as hemoglobin < 11.0g/dl cutoff while iron deficiency anemia was based on ferritin and transferrin receptor levels after correcting for inflammation. Hemoglobin levels (Hb) <11.0g/dl were set as the anemia cut-offs for a pregnant mother; this was used to create dichotomous anemia variable. Corrected maternal RBP (from section 3.5.3 above) was used to determine vitamin A deficiency. Subclinical inflammation was defined as CRP >5 mg/L and/or AGP >1 g/L) [23].

The association of five main independent variables (HDDS, HFIA scale & score, household, and maternal consumption of VA rich food) and VAD were each assessed with bivariate and multiple logistic regression analyses. For each of the variables with more than two levels (i.e. HFIAS; household consumption of VA rich food; maternal & HoH occupation; and maternal marital status), when no significance difference was found at an alpha of 0.05 between its levels and VAD, it was dichotomized for further testing in a multiple logistic regression analyses. With no difference in the risks of VAD among the various categorical levels of these variables, the further dichotomization served to stabilize the estimates especially at the tail of the distribution. Multicollinearity testing was done using variance of inflation (VIF) for pairwise comparison and further using condition indices (CI) and variance decomposition proportion (VDP). VIF > 2, CI >30 with its corresponding VDP of >0.5 indicated presence of multicollinearity.

Tests of mediation were conducted to estimate empirically and test hypotheses of direct and indirect pathways through which exposure variables (the main modifiable determinants in our analyses) carry their effects on VAD. Mediation analysis helps establish if there is an intervening variable (mediator) located causally between an exposure variable being tested and VAD. A significant mediation analysis result means that the exposure variable influences VAD through the mediator. The result is used to answer the question as to how the exposure variable transmits its effect on VAD. In our mediation analyses, sobel test was used to determine whether there was any mediator between each of the main independent variables (HDDS, HFIA scale & score, household, and maternal consumption of VA rich food) and VAD. This was compared with the results generated using SAS Process Macro [44]. The main independent variables were also tested if they mediated each other. All the mediation tests were insignificant at an alpha of 0.05.

Interaction assessment was then performed using hierarchical backward elimination approach in logistic regression. Finally, confounding was assessed using 10% change around the crude estimate in logistic regression. The following socio-demographic variables of interest were coded as categorical and assessed for confounding: participant's age, education, work; and marital status; who served as Head of Household (HoH) and occupation and education of HoH head. Other maternal variables of interest included gestational age, birth interval, previous place of delivery, previous pregnancy outcome, awareness of vitamin A, source of vitamin A information and HIV status.

3.6. Results

Socio-demographics, nutritional, reproductive and uptake of health services factors of 505 pregnant mothers at baseline are shown in (table 1). Overall, maternal median age was 24 years old (range 17-44 years). The majority of women (61.39%) had completed primary education while 37.62% had completed Secondary education and about 1% had completed college or university education. Most of the household (86%) were headed by husbands or partners of the women. These husbands or partners had higher secondary education at 51% compared to the women's 38%. On the other hand, most women (39.8%) did not have any work (formal or informal) compared to their husband or partners (14%). Seventy nine percent of the women were in monogamous marriage with a further 7% in polygamous marriage. The median gestational age at enrollment was 20 weeks (range 8-31 weeks). About 70% of the women reported previous pregnancies prior to enrolment. Of these pregnancies, 5% resulted in miscarriages, 1% still births and 94% in live births. Of the most recent births, 55% were delivered at home, with only 43% delivered in a health facility. Further, for 33% of participants the birth interval between the current and the preceding birth was less than 2 years.

The prevalence of maternal VAD in this population of pregnant women was 21.8% adjusted for inflammation. The prevalence of subclinical inflammation was 23.6% and 4.6% for CRP >5mg/l and AGP > 1.0g/l respectively. The general prevalence of anemia was 22%, similar to the prevalence of iron deficiency at 22.6%. Of those who were classified as anemic using Hb measurement, 43.4% were iron deficient. However, 16.6% of women with iron deficiency were not anemic by Hb measurements. Of these women, about 5% had both VAD and iron deficiency while 17% had VAD only. Mean MUAC was 26.03cm and only 5 women (1%) had MUAC < 21cm. In terms of knowledge of VA, only 34% of the women had ever heard of vitamin A. Of those who had heard of vitamin A, 26% could not name one important function of vitamin A (figure 1). School was the most commonly cited source of vitamin A information at 68%, followed by health facility at 19% and the others at 13% (figure 2).

In single predictor analysis (Table 1), women who were anemic had a 1.7 times greater odds of being vitamin A deficient compared to women who were not anemic (crude odds ratio [COR] 1.68, 95% confidence interval [CI]: 1.05 2.71). Food Security Status, HFIAS Score, HDDS, days of Maternal VA consumption and HH Consumption of VA rich food were not associated with

VAD in single preditcor analyses. Although statistically significant, only 0.78% of variability in number of days of maternal VA intake could be explained by HH consumption of VA rich food in a univariate analysis (R-square=0.0078, p=0.03), figure 4. From the mediation results, food security status, HIFAS score, HDDS, days of maternal consumption of VA rich foods, and HH consumption of VA rich foods did not indirectly affect VAD through the effect of each other.

Overall, maternal consumption of VA rich food was not significantly associated with VAD (AdjOR 0.80, 95% CI: 0.63, 1.03; table 2). However, when stratified by MUAC tertiles, maternal consumption of VA rich food was significantly associated with 47% reduction in the odds of VAD among women with MUAC ≤24.5cm, (AdjOR 0.53, 95% CI: 0.33, 0.83; Figure 3). No significant effect was observed between maternal VA consumption and VAD for women with MUAC > 24.6cm. HDDS, HH food insecurity or HH consumption of VA rich food were not significantly associated with VAD (table 3, 4 & 5).

3.7. Discussion

Vitamin A deficiency is a significant public health concern among this cohort of pregnant women in Western Kenya. Similarly high prevalence of VAD among pregnant women has been described in other studies [1, 10, 45-47]. In Congo, the prevalence of VAD among pregnant women in Brazzaville was 26% [45]. VAD prevalence ranged from 23.5% among Ethiopian poor mothers in one study to 37.9% among women in Southern Ethiopia [26, 46]. However, the high VAD prevalence in Southern Ethiopian study was not adjusted for inflammation. In India, prevalence of VAD among adolescent mothers assessed by the presence of night blindness was 16% [47]. The true prevalence of VAD may be higher however given that night blindness underestimates biochemical prevalence. In addition, the high prevalence in our finding is similar to prevalence of VAD that has been reported among pregnant women in Brazil of 18% [48, 49].

Maternal consumption of vitamin A rich food appears to be significantly associated with reduction in VAD but only among women of poorer nutritional status. This may highlight the need to target women with reduced nutritional status with strategies that promote maternal consumption of VA rich foods policies for maximum impact. This is further supported by a study that had suggested the need for improved targeting to enhance sensitivity of nutrition programs

[50]. The protective effect of maternal consumption on VAD among women who had MUAC of ≤24.5cm raises the need to target women with higher MUAC than the WHO recommended cut-off of <21cm [51]. Other studies have shown that the WHO cut off of <21cm might be too low for screening pregnant women at risk of developing other adverse fetal outcome such as low birth weight [51].

Whereas maintenance of high nutritional status is important during pregnancy, women with reduced nutritional status as measured by low MUAC are more likely to have VAD compared to women with higher nutritional status [52]. Adequate dietary intake of VA rich food is thus more important especially among women with reduced nutritional status to avert possible adverse effects of VAD in pregnancy [18, 22]. This differential effect of maternal consumption of VA rich food by MUAC could also explain mixed results of VA consumption and supplementation on VAD among pregnant women [2, 8]. While some studies showed beneficial effects [8, 9], others have shown no benefit in VA supplementation [2]. These studies did not look at the effect of VAD supplementation stratified by the women's nutritional status. Maternal consumption of VA rich food in our study was only significantly associated with reduced VAD when stratified by MUAC level. This is however under the assumption that consumption of VA rich food and supplementation of VA both have the potential of raising VA levels thereby reducing the risk of VAD, which may not be the case in all circumstances.

The significant reduction of VAD among women who consume Vitamin A rich food, especially in agricultural areas such as Western Kenya, calls for designing of policies that encourage women to grow VA rich foods for consumption to reduce the high prevalence of VAD. Agricultural-nutritional interventions are feasible, cost effective with potential to enhance women empowerment and increase access to diverse diets [35, 50]. In most rural parts of Kenya, breast milk is the main source of VA for children under 6 months; and with a substantial proportion of mothers either having marginal or deficient VA levels, exclusively breastfed infants may not receive adequate VA [53]. Thus strategies that promote production and consumption of VA rich foods by pregnant and lactating women have the potential to be efficient, effective and offer synergistic effects on childhood nutrition and developments [50]. Furthermore, the food based interventions are likely to be safer alternatives than VA supplements in first trimester which would require monitoring for potential teratogenic effects [54]. In

addition, food based interventions may be readily acceptable, sustainable and accessible in rural areas where the prevalence of VAD is greatest [37].

We did not observe any significant association between household consumption of VA rich food, household dietary diversity, household food insecurity status and VAD in this group. Household consumption of VA rich food was insignificantly associated with a small reduction in VAD. The large difference in protective effect between household and maternal consumption could be due to the proximal influence of maternal consumption on VA levels compared to household consumption. The two (maternal consumption of VA rich food and HH consumption of VA food) may be acting through two different pathways to influence maternal VA levels. In our analysis, they did not mediate the effect of each other and they were not strongly correlated. Furthermore, the estimation of household consumption is limited by the difficulty in assessing intra-household allocation of food that would otherwise allow quantification of food intake of individuals household members [55]. Women may not be automatically eating the household foods due to differences in intra-household food allocation. A qualitative study conducted among a sample of women involved in MAMA SASHA project found that mothers were the last to eat when food is limited in the household [56]. Other studies have shown that women are less likely to meet their nutrients requirements for beta-carotene compared to men of the same age due to late position in the serving order, giving special and higher portions of foods to males due to their 'greater contribution' to the family [57-59]. This is despite the fact that women need increased nutrients during pregnancy and lactation. The household level proxies of women dietary intake can be affected by cultural beliefs and dietary practices such as a woman not consuming particular foods even though they are present in the household [10] . Improving household diets therefore may not necessarily translate into improved maternal diets [37]. These contextualized factors may explain the difference between our finding and a study conducted in Southern Ethiopia which had reported a significant negative association between food diversity score and VAD among pregnant women [46]. Further research is needed to better understand how to improve the use of household level factors as tools to gauge individual nutritional intake.

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3.7.1. Limitations

Our study has several limitations. First, the data was limited to baseline; thus, we are not able to assess changes over the course of pregnancy. Second, we did not collect data on malaria, a prevalent inflammatory condition in western Kenya (70% at risk of infection) and we had limited data on HIV status (most women were missing HIV status from their antenatal cards) in order to control and/or assess for their association with VAD [60]. This is especially important on the background of high subclinical inflammation of 24% in our study. Infectious diseases, malaria and HIV included, can potentially cause VAD [22]. We attempted to reduce this potential bias by adjusting for inflammation in VA levels. Third, recall bias and social desirability bias are also likely to have played a role since the enrolment questions were based on recall of past events, and the women might have reported to the interviewer what they perceived to be most acceptable.

Fourth, even though the women were mobilized by community health workers, the eight health facilities from which they were eventually recruited from were conveniently sampled and were all primary public hospitals (dispensaries and health centers) hence the sample may not be representative of all women in the catchment areas. According to Kenya Demographic and health survey (KDHS 2008-09), about 56% of women in Western province received antenatal care for their most recent births in primary health facilities (dispensaries and health facilities); 23.1% in secondary and tertiary hospitals; and 18.7% in private hospitals [60]. Whereas the health centers mostly serve the poor, it is possible that the ultra-poor women who are most vulnerable may lack access to antenatal care from these facilities. Such scenarios would result in possible under-estimation of the protective effect of maternal consumption of VA rich food on VAD. We think it may be possible to see an even higher protective effect of consumption of VA rich food on VAD among this group of ultra-poor women since they are more likely to have lower MUAC than the women in our study. There is therefore need for a future community based survey to make these findings generalizable.

Sixth, the women were enrolled into the study in their first trimester. In Western Kenya, only 14% of women obtain antenatal care in their first trimester [61]. Majority of the women who received their antenatal care after first trimester could have been missed in our study. With the available evidence showing an increase in prevalence of maternal VAD with increasing

gestational age, our analysis could be under-estimating the burden of VAD among pregnant women in this population [46]. However, the choice of first trimester was important in COVA study for early intervention with OFSP in order to accrue maximum health benefits. Finally, data were collected from study participants attending antenatal clinic in health facilities in Western Kenya and the findings may not be generalizable to other settings within and outside of Western Kenya.

3.7.2. Conclusion

VAD among pregnant women in Western Kenya is of public health significant. Women awareness and knowledge of VA should be improved and incorporated in nutritional counseling of pregnant women. Whereas health facilities would be the natural source of VA information, other sources such as schools could be used to reinforce the importance of VA since they are already offering such information. To increase maternal consumption of vitamin A rich food, innovative, feasible and effective targeted approaches are needed in resource limited setting. Such approaches could include integration of nutritional and agricultural interventions aimed at improving maternal VA levels, while targeting women with reduced nutritional status for maximum impact.

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3.9. Tables and Figures

Table 1: Enrolment Characteristics of 505 Pregnant Women Participating the Mama SASHA Cohort Study of Vitamin A (COVA) Study in Western Kenya and associations with Retinol Binding Protein and vitamin A status					
Variable	**Overall(Frequency (%) or Mean (± sd)**	**RBP (n=504) β (95% CI)**	**VAD n(% or Mean)**	**VAS n(% or Mean)**	**OR (95% CI)**
Food Security Status (n=481)					
Secure	163 (33.89%)	Referent	35 (21.47%)	128 (78.53%)	Referent
Mild	113 (23.49%)	-0.03 (-0.12, 0.05)	31 (27.68%)	81 (72.32%)	1.40 (0.80, 2.45)
Moderate	102 (21.21%)	0.04 (-0.05, 0.13)	18 (17.65%)	84 (82.35%)	0.78 (0.42, 1.47)
Severe	103 (21.41%)	0.02 (-0.06, 0.11)	22 (21.36%)	81 (78.64%)	0.99 (0.54, 1.81)
HFIAS Score (n=481)	5.44 (±6.05)	0.002 (0.003, 0.01)	106 (5.46)	374 (5.54)	0.99 (0.95, 1.03)
Household Dietary Diversity score (n=505)	5.50 (±1.43)	-0.01 (-0.03, 0.01)	110 (5.67)	394 (5.45)	1.12 (0.96, 1.29)

Table 1: Enrolment Characteristics of 505 Pregnant Women Participating the Mama SASHA Cohort Study of Vitamin A (COVA) Study in Western Kenya and associations with Retinol Binding Protein and vitamin A status

Variable	Overall(Frequency (%) or Mean (± sd)	RBP (n=504) β (95% CI)	VAD n(% or Mean)	VAS n(% or Mean)	OR (95% CI)
Days of Maternal VA consumption (n=505)	0.84 (±0.91)	-0.01 (-0.05, 0.02)	110 (0.82)	394 (0.87)	0.80 (0.63, 1.03)
Consumption of VA HH (n=505)					
None	302 (59.80%)	Referent	64 (21.19%)	238 (78.81%)	Referent
Natural food	171 (33.86%)	-0.07 (-0.13, -0.002)	42 (24.56%)	129 (75.44%)	1.21 (0.78, 1.89)
Fortified food	32 (6.34%)	-0.02 (-0.15, 0.11)	4 (12.90%)	27 (87.10%)	0.55 (0.19, 1.63)
Facility site (n=505)					
Intervention	250 (49.50%)	-0.04 (-0.10, 0.03)	54 (21.69%)	195 (78.31%)	0.98 (0.65, 1.50)
Control	255 (50.50%)	Referent	56 (21.96%)	199 (78.04%)	Referent
Anemia (n=505)					
Yes	113 (22.38%)	-0.08 (-0.16, -0.01)	33 (29.20%)	80 (70.80%)	1.68 (1.05 2.71)
No	392 (77.62%)	Referent	77 (19.69%)	314 (80.31%)	Referent

Table 1: Enrolment Characteristics of 505 Pregnant Women Participating the Mama SASHA Cohort Study of Vitamin A (COVA) Study in Western Kenya and associations with Retinol Binding Protein and vitamin A status

Variable	Overall(Frequency (%) or Mean (± sd)	RBP (n=504) β (95% CI)	VAD n(% or Mean)	VAS n(% or Mean)	OR (95% CI)
Iron deficiency (n=504)					
Yes	114 (22.62%)	-0.06 (-0.13, 0.01)	26 (22.81%)	88 (77.19%)	1.08 (0.65, 1.77)
No	390 (77.38%)	Referent	84 (21.54%)	306 (78.46%)	Referent
Aware of VA (n=505)					
Yes	171 (33.86%)	-0.03 (-0.10, 0.03)	41 (23.98%)	130 (76.02%)	1.21 (0.78, 1.87)
No	334 (66.14%)	Referent	69 (20.72%)	264 (79.28%)	Referent
Demographics					
Maternal education (n=505)					
Primary	310 (61.39%)	Referent	66 (21.36%)	243 (78.64%)	Referent
Post Primary	195 (38.61%)	-0.03 (-0.10, 0.03)	44 (22.56%)	151 (77.44%)	1.07 (0.70, 1.65)
Education HoH (462)					
Primary	216 (46.75%)	Referent	45 (20.83%)	171 (79.17%)	Referent
Post Primary	246 (53.25%)	-0.03 (-0.10, 0.03)	56 (22.86%)	189 (77.14%)	1.13

Table 1: Enrolment Characteristics of 505 Pregnant Women Participating the Mama SASHA Cohort Study of Vitamin A (COVA) Study in Western Kenya and associations with Retinol Binding Protein and vitamin A status

Variable	Overall(Frequency (%) or Mean (± sd)	RBP (n=504) β (95% CI)	VAD n(% or Mean)	VAS n(% or Mean)	OR (95% CI)
					(0.72, 1.75)
Maternal age (yrs) (=505)	24.59 (±5.53)	0.01 (0.001, 0.012)	110 (24.32)	394 (24.67)	0.99 (0.95, 1.03)
Marital Status (n=503)					
Single	69 (13.72%)	Referent	14 (20.29%)	55 (79.71%)	Referent
Married/Partnered Monogamous	399 (79.32%)	0.03 (-0.06, 0.12)	88 (22.11%)	310 (77.89%)	1.12 (0.59 2.10)
Polygamous	35 (6.96%)	0.12 (-0.03, 0.26)	7 (20.00%)	28 (80.00%)	0.98 (0.36, 2.71)
Maternal Occupation (n=503)					
Does not work	200 (39.76%)	Referent	45 (22.61%)	154 (77.39%)	Referent
Agriculture	168 (33.40%)	0.05 (-0.02, 0.12)	33 (19.64%)	135 (80.36%)	0.84 (0.51, 1.39)
Salaried employment	25 (4.97%)	-0.06 (-0.21, 0.09)	7 (28.00%)	18 (72.00%)	1.33 (0.52, 3.39)
Informal business	54 (10.74%)	0.08 (-0.03, 0.19)	10 (18.52%)	44 (81.48%)	0.78 (0.36, 1.67)

Table 1: Enrolment Characteristics of 505 Pregnant Women Participating the Mama SASHA Cohort Study of Vitamin A (COVA) Study in Western Kenya and associations with Retinol Binding Protein and vitamin A status

Variable	**Overall(Frequency (%) or Mean (± sd)**	**RBP (n=504) β (95% CI)**	**VAD n(% or Mean)**	**VAS n(% or Mean)**	**OR (95% CI)**
Others	56 (11.13%)	0.025 (-0.08, 0.13)	14 (25.00%)	42 (75.00%)	1.14 (0.57, 2.27)
Occupation HoH (n=503)					
Does not work	68 (13.52%)	Referent	11 (16.18%)	57 (83.82%)	Referent
Agriculture	94 (18.69%)	0.08 (-0.03, 0.19)	17 (18.09%)	77 (81.91%)	1.14 (0.50, 2.63)
Salaried employment	81 (16.10%)	-0.01 (-0.13, 0.10)	22 (27.16%)	59 (72.84%)	1.93 (0.86, 4.34)
Informal business	71 (14.12%)	0.02 (-0.10, 0.13)	20 (28.57%)	50 (71.43%)	2.07 (0.91, 4.74)
Others	189 (37.57%)	0.02 (-0.08, 0.11)	40 (21.16%)	149 (78.84%)	1.39 (0.67, 2.90)
Primagravida (n=505)					
Yes	153 (30.30%)	-0.07 (-0.13, 0.001)	35 (22.88%)	118 (77.12%)	1.09 (0.69, 1.72)
No	352 (69.70%)	Referent	75 (21.37%)	276 (78.63%)	Referent
Gestational age in weeks	19.4 (±4.60)	-0.003	110 (20.13)	394 (19.20)	1.05 (1.00, 1.10)

Table 1: Enrolment Characteristics of 505 Pregnant Women Participating the Mama SASHA Cohort Study of Vitamin A (COVA) Study in Western Kenya and associations with Retinol Binding Protein and vitamin A status

Variable	Overall(Frequency (%) or Mean (± sd)	RBP (n=504) β (95% CI)	VAD n(% or Mean)	VAS n(% or Mean)	OR (95% CI)
(n=505)		(-0.01, 0.004)			
MUAC (n=505)	26.03 (±3.02)	0.01 (-0.003, 0.017)	110 (25.64)	294 (26.12)	0.94, (0.87, 1.02)
Previous pregnancy outcome (n=352*)					
Live births	330 (93.75%)	Referent	68 (20.67%)	261 (79.33%)	Referent
Still births/ Miscarriages	22 (6.25 %)	-0.05 (-0.21, 0.11)	7 (31.82%)	15 (68.18%)	1.79 (0.70, 4.57)
Birth Interval (yrs) (n=352*)					
<2	117 (33.24%)	Referent	26 (22.41%)	90 (77.59%)	Referent
≥2	235 (66.76%)	-0.03 (-0.11, 0.05)	49 (20.85%)	186 (79.15%)	0.91 (0.53, 1.56)
Place of Delivery (n=334*)					
Home or Way to H/facility	190 (56.89%)	Referent	32 (16.93%)	157 (83.07%)	Referent
Health Facility	144 (43.11%)	-0.02 (-0.10, 0.06)	36 (25%)	108 (75%)	1.64 (0.96, 2.79)

*Only prior maternal pregnancies

**Number less than total due to missing data

Table 2: Association of Maternal consumption of VA rich food and VAD among 505 Pregnant Women Involved in COVA Cohort Study in Western Kenya *(Single Predictor and multiple predictor adjusted Models)*

Variables	**Maternal consumption of VA rich food**		
	Beta Estimate (% change)	**OR**	**95% CI**
Crude	-0.22	0.80	(0.63, 1.03)
Adjusting For;			
Household Food Insecurity Status (HFIA)	-0.23 (6.13)[A]	0.79	(0.61, 1.03)
Household Dietary Diversity score (HDDS)	-0.23 (4.27)[A]	0.79	(0.61, 1.02)
Maternal age	-0.24 (7.58)[A]	0.79	(0.61, 1.02)
Maternal Occupation	-0.21 (3.91) [†]	0.81	(0.63, 1.04)
Maternal education	-0.22 (0.77) [†]	0.80	(0.63, 1.03)
Maternal MUAC	-0.21 (3.13) [†]	0.81	(0.63, 1.04)
Gestational age at Enrolment	-0.23 (4.45) [A]	0.80	(0.62, 1.02)
Maternal anemia	-0.21 (4.00) [†]	0.81	(0.63, 1.04)
Adjusted for All*	**-0.23 (3.04)[A]**	**0.80**	**(0.61, 1.04)**

[A] Change away from the null [†] Change towards the null

*Adjusted for all covariates

Table 3: Association of Household Food Consumption of VA rich food and VAD among 505 Pregnant Women Involved in COVA Cohort Study in Western Kenya *(Multivariate Analysis): Single Predictor and multiple predictor adjusted Model*

Variables	**Household consumption of VA Rich food**		
	Beta Estimate (% change)	**OR**	**95% CI**
Crude	0.09	1.10	(0.71, 1.69)
Adjusting For;			
Maternal consumption of VA rich food	0.13 (43.17) [A]	1.14	(0.74, 1.76)
Household Food Insecurity Status (HFIA)	-0.04 (142.62) [C]	0.96	(0.62, 1.50)
Maternal Occupation	0.11 (16.70) [A]	1.11	(0.72, 1.71)
HoH Occupation	0.07 (22.13) [†]	1.07	(0.70, 1.65)
Marital status	0.11 (19.63) [A]	1.12	(0.73, 1.72)
HoH Education	0.03 (63.67) [†]	1.03	(0.66, 1.62)
Maternal MUAC	0.106 (7.16) [A]	1.10	(0.72, 1.70)
Gestational age at Enrolment	0.13 (37.74) [A]	1.14	(0.74, 1.75)
Maternal anemia	0.07 (22.67) [A]	1.07	(0.70, 1.65)
Adjusted for All*	**-0.05 (155.55)[C]**	**0.96**	**(0.59, 1.54)**

[A] Change away from the null [†] Change towards the null [C]Switch-over

*Adjusted for all covariates

Table 4: Association of Household food insecurity status and VAD among 505 Pregnant Women Involved in COVA Cohort Study in Western Kenya *(Multivariate Analysis): Single Predictor and multiple predictor adjusted Models*			
Variables	**Household Food Insecurity Status (HFIA)**		
	Beta Estimate (% change)	**OR**	**95% CI**
Crude	0.05	1.06	(0.67, 1.67)
Adjusting For;			
Household Dietary Diversity score (HDDS)	0.09 (59.07) [A]	1.09	(0.69, 1.73)
Maternal consumption of VA rich food	0.04 (25.56) [†]	1.04	(0.66, 1.65)
Household consumption of VA Rich food	0.04 (27.96) [†]	1.04	(0.66, 1.65)
Maternal age	0.06 (13.15) [A]	1.06	(0.67, 1.68)
Maternal Occupation	0.03 (41.30) [†]	1.03	(0.65, 1.63)
Marital status	0.02 (55.56) [†]	1.02	(0.65, 1.62)
Maternal education	0.06 (15.74) [A]	1.06	(0.67, 1.69)
Gestational age at Enrolment	0.08 (52.22) [A]	1.09	(0.69, 1.72)
Maternal anemia	0.09 (70.56) [A]	1.10	(0.69, 1.74)
Adjusted for All*	**0.10 (100.00) [A]**	**1.11**	**(0.68, 1.79)**

[A] Change away from the null [†] Change towards the null

*Adjusted for all covariates

Table 5: Association of Household dietary diversity and VAD among 505 Pregnant Women Involved in COVA Cohort Study in Western Kenya *(Multivariate Analysis): Single Predictor and multiple predictor adjusted Models*			
Variables	**Household Dietary Diversity score (HDDS)**		
	Beta Estimate (% change)	**OR**	**95% CI**
Crude	0.11	1.12	(0.96, 1.29)
Adjusting For;			
Household Food insecurity Status (HFIA)	0.09 (18.94) †	1.09	(0.94, 1.27)
Maternal consumption of VA rich food	0.13 (14.73)A	1.13	(0.98, 1.31)
Household consumption of VA Rich food	0.12 (13.08) A	1.13	(0.95, 1.35)
Maternal age	0.11 (1.10)A	1.12	(0.96, 1.29)
Maternal Occupation	0.12 (10.98)A	1.13	(0.97, 1.31)
Marital status	0.13 (15.46)A	1.14	(0.98, 1.32)
Maternal education	0.11 (0.91) †	1.11	(0.96, 1.29)
HoH Education	0.09 (15.46) †	1.10	(0.94, 1.28)
Maternal MUAC	0.12 (7.04) A	1.12	(0.97, 1.30)
Gestational age at Enrolment	0.12 (12.08) A	1.13	(0.97, 1.31)
Maternal anemia	0.11 (0.82) A	1.12	(0.96, 1.29)
Adjusted for All*	**0.13 (18.12)A**	**1.14**	**(0.96, 1.34)**

A Change away from the null † Change towards the null

*Adjusted for all covariates

Figure 1: Maternal Knowledge of Vitamin A importance among 171 Pregnant Women who were Aware of Vitamin A- nutritional knowledge table

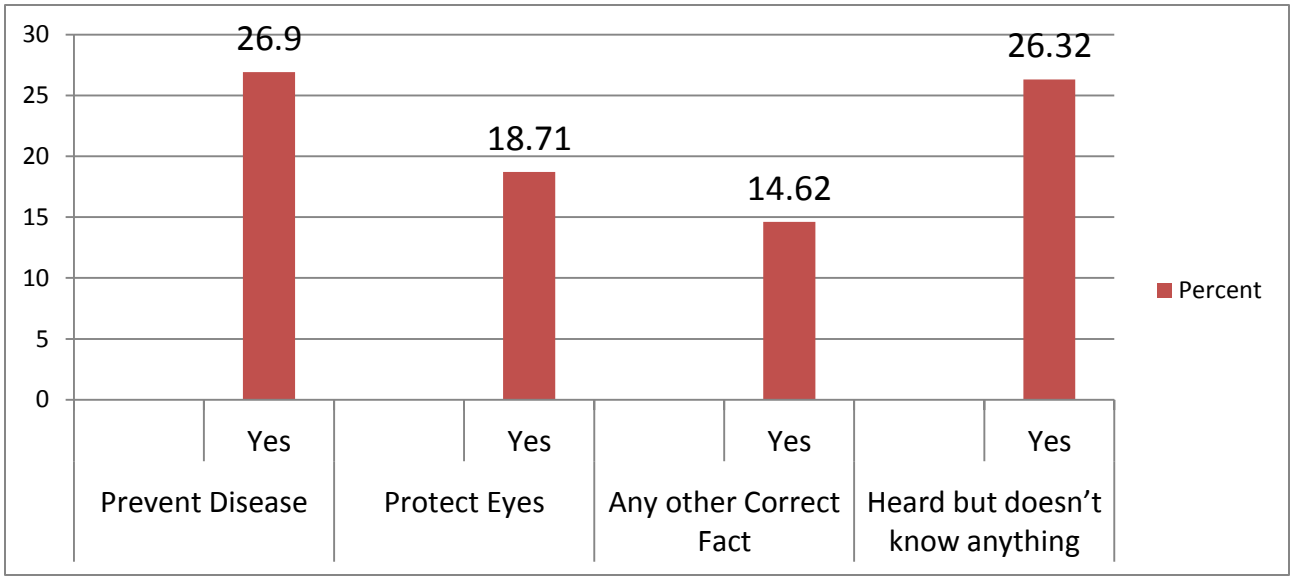


Figure 2: Sources of information on Vitamin A Importance among 171 Pregnant Women who were Aware of Vitamin A

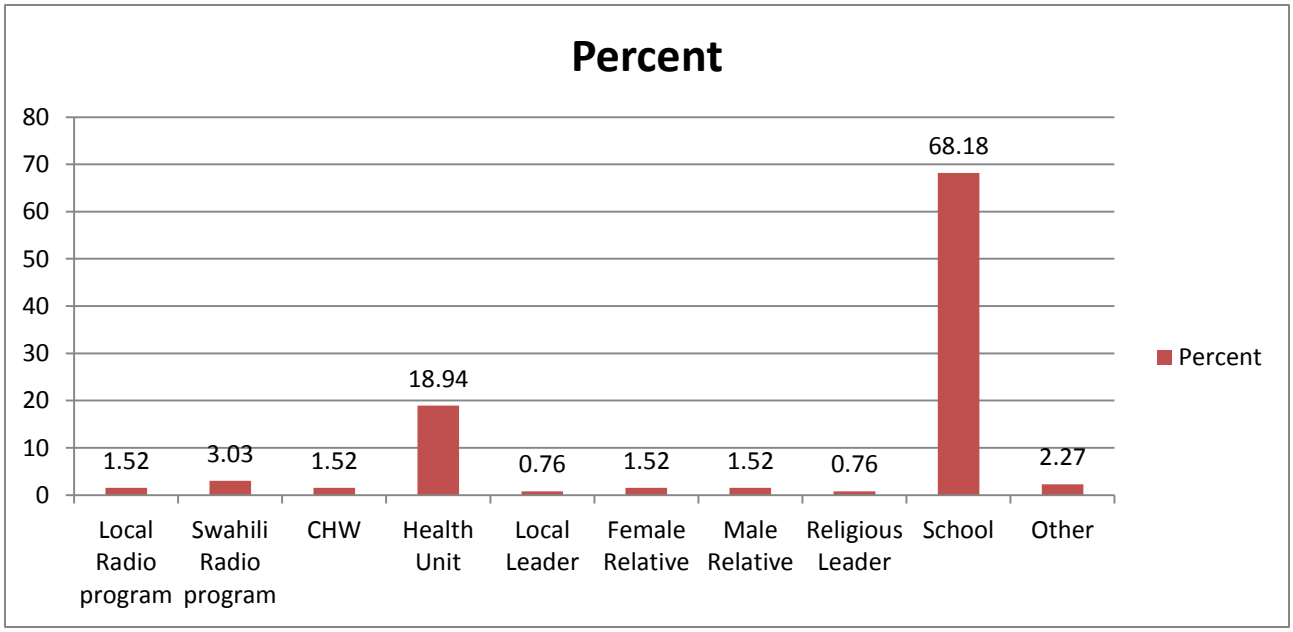


Figure 3: Interaction of Maternal consumption of VA rich food and Maternal MUAC in the Adjusted Model

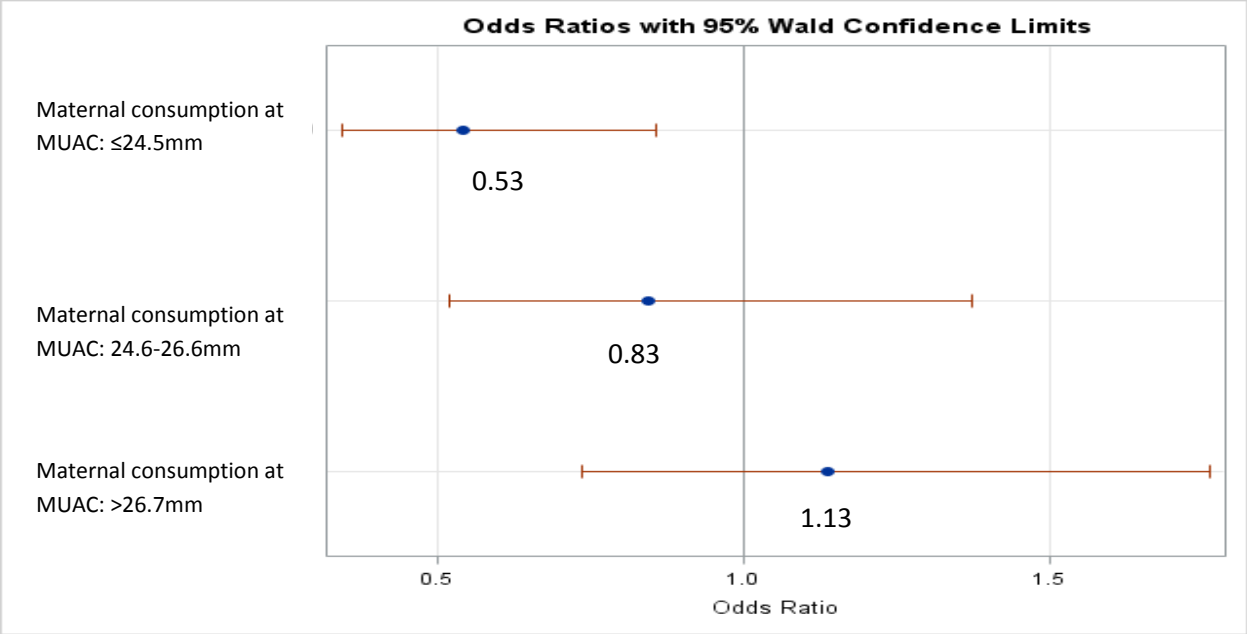
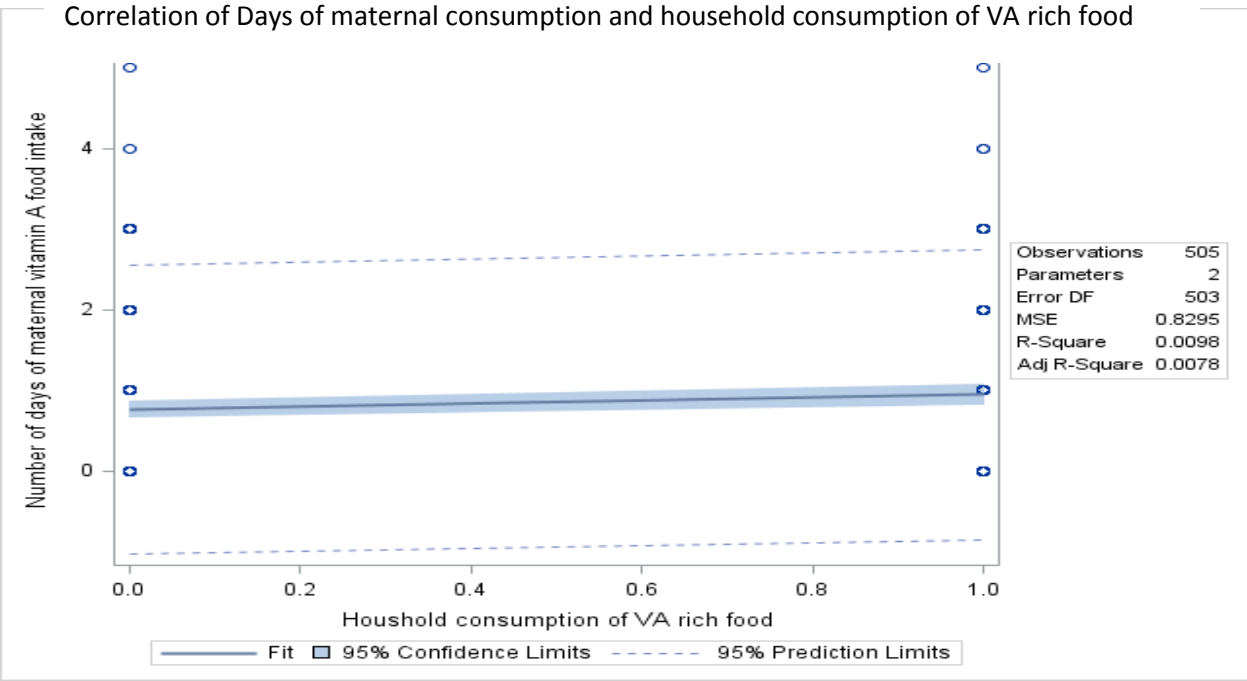


Figure 4: Correlation of number of days of maternal vitamin A food lntake and HH vitamin consumption of VA rich foods



4. Chapter 4: Recommendations and Conclusions

4.1. Summary of the Findings

The prevalence of VAD among pregnant women in this study is of significant public health concern. Besides, majority of the women had no knowledge of VA or its importance. Among the few who could mention any importance of VA, their main source of information was school rather than health facility, pointing to the need to promote VA awareness and knowledge beyond the traditional health settings. Pregnant women in poor nutritional status had greater odds of VAD. Maternal consumption of VA rich can potentially reduce this risk with the greatest impact being among women with the lowest MUAC. Targeting women with lower MUAC with interventions that aims to increase maternal consumption could thus be more cost-effective. Such interventions include agricultural-nutritional programs that promote production and consumption of VA rich foods by pregnant and lactating women. However, program implementers need to employ caution when promoting consumption at household levels with the targeted endpoint being improved VA levels of pregnant women in the households. This is because household consumption is affected more by intra-household allocation of food biases, lower social status of women, cultural beliefs and dietary practices. In the presence of such factors, household consumption would not be a good measure of maternal dietary intake.

4.2. Recommendations

4.2.1. Future Research

First, additional evidence is needed to show whether targeted promotion & support of maternal consumption of VA rich foods reduces VAD in a longitudinal study. Since our study demonstrated an association between maternal consumption and reduced VAD despite having women with relatively good nutrition, there is a possibility of even higher effect among women of poorer nutritional status. Second, further research is needed to better understand how to improve the use of household level factors to measure more accurately individual nutritional intake. This should include determining factors that affect the relationship between household consumption and maternal consumption. Third, in settings where infectious diseases are prevalent, future studies should explore how such morbidities influence maternal VA levels. Fourth, future studies should involve the use of rigorous sampling techniques (unlike

convenience sampling) to improve on representativeness, precision while reducing potential biases of the results. Fifth, additional evidence is needed to show whether maternal consumption of VA rich food reduces VAD beyond first trimester especially in settings such as Western Kenya where most women attend their first antenatal clinic after the first trimester. Nevertheless, intervention in first trimester, or better still in the preconception stages, should be encouraged as this is when VAD has most adverse effects on developing fetus. Lastly, population based rather than health facilities' based studies should be conducted to confirm our findings at the population level.

4.2.2. Programming

To improve VA levels among pregnant women, programs should target women of low nutritional status to achieve maximum impact. This could be done by promoting production and consumption of home gardened foods rich in VA, small poultry rearing and fish farming techniques. For programs that offer interventions at the household level, they should address barriers that could be hindering actual maternal consumption of VA rich foods. In addition, VA interventions programs should promote awareness and importance of VA among pregnant women in Western Kenya. This should include going beyond traditional sources of information such as health facilities and target other sources such as institutions of learning.

4.2.3. Policy

In agricultural areas such as Western Kenya, policies should aim at encouraging women to grow vitamin A rich foods for consumption to reduce the high prevalence of VAD. For example, inter-sectorial collaborations between the Ministry of health and Ministry of agriculture in Kenya can develop policies that ensure seeds of VA rich foods e.g. OFSP vines are accessible and affordable to women. The women should also be trained on nutrition and best agronomical practices to increase crop yields and ensure that nutritious crops are consumed or, if sold, that nutritious foods are purchased with earned income. Home gardens and animal production are some of the agricultural strategies that have been associated with increased dietary diversities and intake among women [37]. In resource limited setting, the policies can target women of poor nutritional status for cost-effectiveness. In addition, policies that aim to increase maternal consumption of VA rich food through household food production strategies approaches should address factors such as intra-household allocation of foods that could divert nutritious foods

produced by the home away from those that need it most, namely pregnant and lactating women and young children. The main decision makers of food allocation in the household should be counselled on the importance of adequate maternal VA intake.

4.3.Conclusion

Promotion of maternal consumption of Vitamin A rich food could hold the key to significant reduction in VAD in pregnancy, especially among women with lower nutritional status in agricultural areas. Agricultural and nutritional integrated interventions which are feasible and affordable should be promoted to improve nutritional status of pregnant mothers. These programs could have synergistic economic and nutritional benefits to pregnant women and their children. By cultivating, consuming, and selling for example OFSP, the women can use the cash proceeds to buy other varieties of foods thereby diversifying her diet.